

CONCISE CLINICAL GUIDANCE

Outpatient Management of Isolated Left-to-Right Shunt Lesions in Pediatric Patients: 2026 ACC Concise Clinical Guidance



A Report of the American College of Cardiology Solution Set Oversight Committee

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PREFACE

The American College of Cardiology (ACC) has a long history of developing documents (eg, decision pathways, appropriate use criteria) to provide clinicians with guidance on both clinical and nonclinical topics relevant to cardiovascular care. In most circumstances, these documents have been created to complement clinical practice guidelines and to inform clinicians about areas where evidence is new and evolving or where sufficient data is

more limited. Despite this, numerous gaps persist, highlighting the need for more streamlined and efficient processes to implement best practices in patient care.

Central to the ACC's strategic plan is the generation of actionable knowledge—a concept that places emphasis on making clinical information easier to consume, share, integrate, and update. To this end, the ACC has shifted from developing isolated documents to creating integrated “solution sets.” These are groups of closely related activities, policy, mobile applications, decision-support tools, and other resources necessary to transform care, improve heart health, or both. Solution sets address key questions facing care teams and offer practical guidance to be applied at the point of care. They use both established and emerging methods to disseminate information about cardiovascular conditions and their related management. The success of the solution sets rests firmly on their ability to have a measurable impact on the delivery of care. Because solution sets reflect current evidence and ongoing gaps in care, the associated tools will be refined over time to match changing evidence and member needs.

Concise Clinical Guidance (CCG) documents are a key component of solution sets. Highly focused and limited in scope, CCGs provide recommendations where none currently exist and/or outline actions required for evidence to be implemented in practice for specific patient populations. CCGs aim to illustrate clinical decision-making processes using tools (ie, figures, tables, and checklists) and are limited in scope, focusing on patient populations that share certain characteristics, such as conditions, subtypes, or lines of therapy. In some cases, covered topics will be addressed in subsequent expert consensus decision pathways, appropriate use criteria, clinical practice guidelines, and other related ACC clinical policy as the evidence base evolves. In other cases, these will serve as stand-alone policy and represent best standards.

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Chair, ACC Solution Set Oversight Committee*

1. INTRODUCTION

Among the various types of congenital heart diseases (CHD), the left-to-right shunt lesions, including atrial septal defect (ASD), ventricular septal defect (VSD), patent ductus arteriosus (PDA), and atrioventricular septal defect (AVSD), are the most prevalent. Significant advances in surgical- and catheter-based interventions have greatly improved outcomes in children with these defects. Despite these advances and improved survival, currently no evidence-based clinical practice guidelines exist for management of these conditions in pediatric patients. Significant variability remains in outpatient management of these lesions, which impact patient care as well as resource utilization. The purpose of this CCG is

to fill this gap by providing guidance for outpatient management of both unrepaired and repaired left-to-right shunt lesions. This document may provide a framework for future implementation studies that may ultimately lead to the development of evidence-based clinical practice guidelines for managing these patients.

In accordance with ACC's Relationships With Industry and Other Entities policy, relevant disclosures for the writing committee and comprehensive disclosures for external peer reviewers can be found in [Appendixes 1 and 2](#). To ensure complete transparency, a comprehensive table of the writing committee's relationships with industry, including those not pertinent to this document, is provided in the online [Supplemental Appendix](#).

1.1. Acknowledgments

The writing committee acknowledges the Adult Congenital and Pediatric Cardiology Section of the ACC for proposing left-to-right shunt lesions as a CCG topic. We also extend our sincere gratitude to the primary reviewers of this CCG—Drs. Aimee K. Armstrong, Jonathan Johnson, Leo Lopez, and Shubhika Srivastava—for their thoughtful and constructive feedback. Their critical review and helpful suggestions improved the clarity, rigor, and overall quality of this work.

2. ASSUMPTIONS, DEFINITIONS, AND ABBREVIATIONS

2.1. Assumptions and Scope

1. This document addresses only the outpatient follow-up of patients with established diagnosis of ASD, VSD, PDA, or AVSD using appropriate testing modalities. Less common lesions with left-to-right shunts (eg, aortopulmonary window, partial anomalous pulmonary venous return, extracardiac arteriovenous malformations) have not been included.
2. It is assumed that a complete anatomic cardiac diagnosis has been established by a qualified clinician using appropriate diagnostic tests.
3. The goal of this document is to provide guidance for isolated left-to-right shunt lesions in the absence of other major cardiac lesions or noncardiac comorbidities. Genetic syndromes and chromosomal anomalies are excluded from this document except for trisomy 21. It is assumed that the clinicians will use their best judgment while applying the algorithms in this document and will consider the inclusion and exclusion criteria for each algorithm before use. It is important to rule out multi-level shunts or co-existing lesions that could worsen the clinical condition (eg, coarctation in a patient with a VSD).
4. To determine whether the patient is symptomatic, it is assumed that a comprehensive history and physical

examination have been conducted by a qualified clinician and that the signs and symptoms accurately reflect the patient's clinical status.

5. It is assumed that the tests are ordered by clinicians with expertise in CHD and are performed and interpreted by qualified personnel in facilities that adhere to national standards for the relevant testing modality. The risks and benefits of each testing modality will be considered with the goal of providing the highest value of care, with value defined as quality divided by cost.
6. Some scenarios include a broad time range for surveillance to account for a wider spectrum of anatomic variations, pathophysiology, natural history, and a range of symptomatology in patients. It is assumed that clinicians will use their best judgment to determine the timing of follow-up visits within this range for an individual patient.
7. It is assumed that in a few scenarios, the type and timing of intervention, as well as periprocedural management, may be influenced by institutional guidelines.
8. It is beyond the scope of this document to discuss the specifics of candidacy for catheter-based procedures versus surgical intervention. It is assumed that the clinician will use the currently available literature, appropriate consultation, institutional preference and experience, and a shared decision-making model with discussion of pros and cons of each approach with the families.
9. It is beyond the scope of this document to define the grading of size or severity for each cardiac lesion in a granular manner, and it is assumed that the clinician is using the currently available literature to determine these while interpreting algorithms. Similarly, the document does not address the controversies in nomenclature of the cardiac defects included here and uses the terminology that is most often used in the currently available literature.
10. This document does not include managing transition to adulthood for adolescents with CHD or care of CHD patients >18 years of age due to preexisting clinical practice guidelines.^{1,2}
11. This document does not cover all diagnostic modalities that may be used in surveillance of left-to-right shunt lesions; rather, the focus is on echocardiography and electrocardiography because they are most frequently used in the outpatient setting. Clinical scenarios that warrant additional diagnostic testing, such as cardiac catheterization, are alluded to, but the specific indications and timing are not discussed.
12. Detailed management strategies for left-to-right shunts in patients residing at high altitudes are beyond the scope of this document; however, these individuals should be monitored more closely for earlier development of pulmonary vascular disease,

with consideration of a lower threshold for intervention to reduce the risk of irreversible pulmonary hypertension.

13. It is assumed that clinicians are familiar with current recommendations for exercise participation, including guidance for competitive sports in patients with cardiovascular disease.^{3,4} Regardless of repair status, exercise eligibility should be based on the hemodynamic significance of the shunt and associated clinical findings. Patients with small shunts and no markers of increased risk, such as pulmonary vascular disease, complex arrhythmias, or significant ventricular dilation or dysfunction, may participate in competitive sports without restriction. In contrast, for those with moderate or large shunts and markers of increased risk, activity recommendations should be individualized through shared decision-making with the patient and family.

2.2. Definitions

To maintain consistency, the following definitions have been used in accordance with a previous ACC document on CHD.⁵

- **Asymptomatic:** Lacking characteristic signs, symptoms, or both for a given condition.
- **ASD:** A congenital cardiac malformation in which a hole is observed between the atrial chambers.⁶
 - **Small ASD:** Hemodynamically insignificant.
 - **≥ Moderate ASD:** Hemodynamically significant.
 - **Secundum ASD:** A congenital cardiac malformation in which an interatrial communication is confined to the region of the oval fossa (fossa ovalis), most commonly attributable to a deficiency of the primary atrial septum (septum primum), but deficiency of the septum secundum (superior interatrial fold) may also contribute.⁶
- **AVSD:** This cardiac defect is subcategorized into complete, intermediate, transitional, and partial AVSD.⁶
 - **Complete AVSD:** A congenital cardiac malformation in which both atria connect to a common atrioventricular valve, which characteristically has 4 or 5 leaflets, including superior and inferior bridging leaflets, and a single annulus. An interatrial communication is just above the atrioventricular valve and interventricular communication just below it.
 - **Intermediate AVSD:** A variant of complete AVSD that has distinct right and left atrioventricular valve orifices despite having only 1 common annulus. An interatrial communication that is just above the atrioventricular valve and interventricular communication just below it.
 - **Transitional AVSD:** A variant of complete AVSD with an interatrial communication is immediately above the atrioventricular valve and a restrictive interventricular communication immediately below the atrioventricular valve.
- **Partial AVSD:** A congenital cardiac malformation comprised of an interatrial communication just above the atrioventricular valve, no interventricular communication just below the atrioventricular valve, separate right and left atrioventricular valvular orifices, and varying degrees of malformation of the left atrioventricular valve.
- **Cardiovascular computed tomography (CCT):** Computed tomography imaging of the heart and blood vessels.⁷
- **Cardiovascular magnetic resonance (CMR):** Magnetic resonance imaging of the heart and blood vessels.
- **Double-chambered right ventricle (RV):** A congenital cardiac malformation in which the RV is divided into 2 chambers—1 inferior that includes the inlet and trabecular portions of the RV and 1 superior that includes the trabecular portion and infundibulum.⁶
- **Eisenmenger syndrome:** Pulmonary arterial hypertension in the setting of CHD with elevation of pulmonary vascular resistance, resulting in bidirectional or right-to-left shunting through a systemic-to-pulmonary artery connection or septal defect and cyanosis.⁸
- **Hemodynamically significant defect:** A defect that can result in symptoms and/or cardiac chamber dilation.
- **Infant:** A child from 28 days of age to 1 year of age.
- **Neonate:** A child from birth until 28 days of age.
- **PDA:** A congenital cardiovascular finding in which the arterial duct (ductus arteriosus) is open beyond the normal age of spontaneous closure.⁶
 - **Trivial and silent PDA:** No left atrial or left ventricular dilation; murmur not heard on auscultation.
 - **Small and audible PDA:** Audible on examination; may or may not be associated with left atrial or ventricular dilation.
 - **≥ Moderate PDA:** Hemodynamically significant.
- **Pulmonary hypertension:** A resting mean pulmonary artery pressure ≥20 mm Hg.^{9,10}
- **Symptomatic:** Demonstrating characteristic signs, symptoms, or both of a given disease.
- **Transthoracic echocardiography (TTE):** Noninvasive ultrasound imaging to assess cardiac structures and valvular and ventricular function. It may include 2-dimensional, 3-dimensional, M-mode, and strain imaging; color, spectral, and tissue Doppler imaging; and use of contrast agents for opacification.
- **VSD:** A congenital cardiac malformation in which a hole or pathway is between the ventricular chambers.⁶
 - **Small VSD:** Hemodynamically insignificant.
 - **≥ Moderate VSD:** Hemodynamically significant.

2.3. Abbreviations

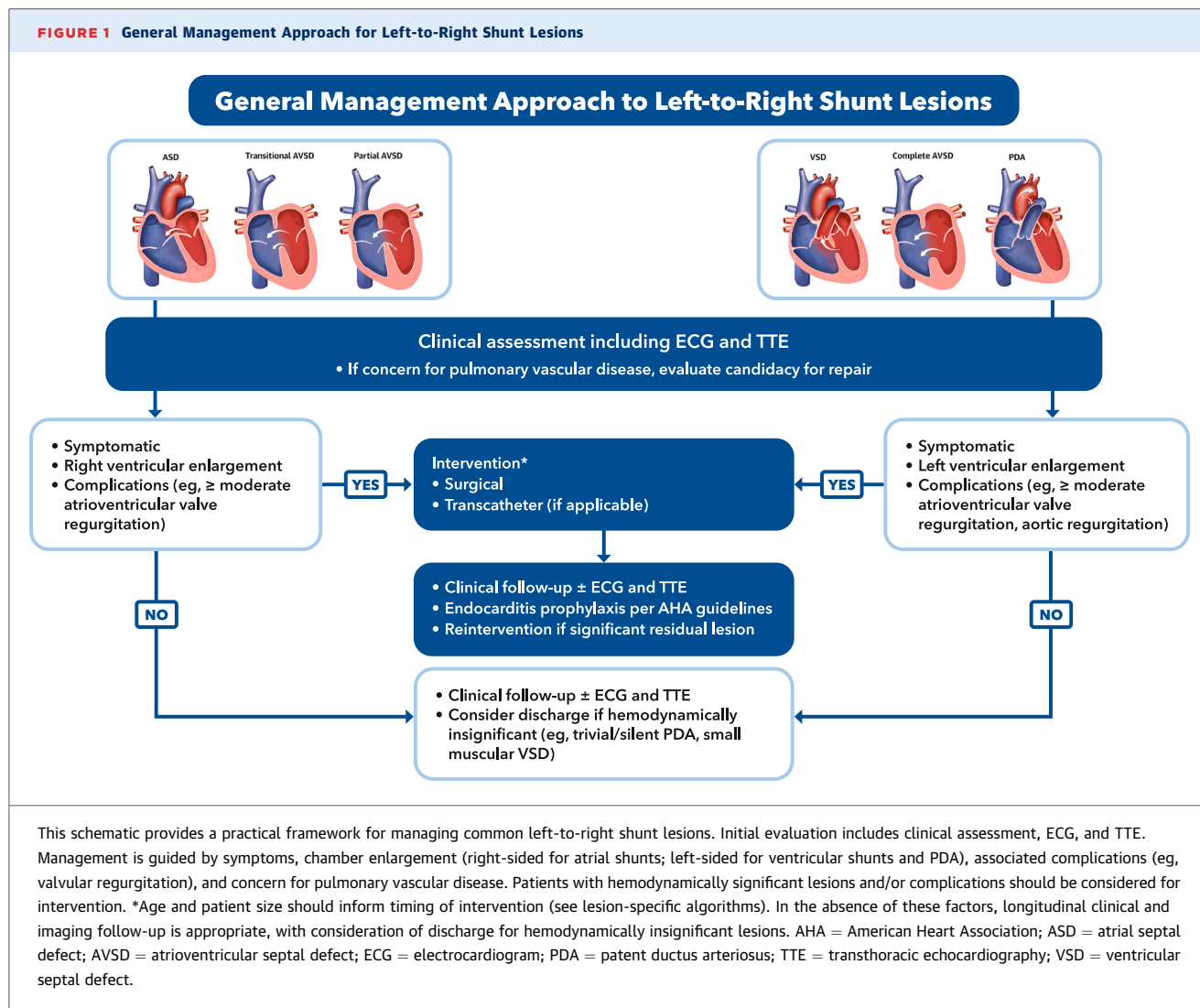
ASD = atrial septal defect

AVSD = atrioventricular septal defect

CCT = cardiovascular computed tomography
CHD = congenital heart disease
CMR = cardiovascular magnetic resonance
LA = left atrium
LPA = left pulmonary artery
LV = left ventricle

LVOTO = left ventricular outflow tract obstruction
PDA = patent ductus arteriosus
Qp:Qs = ratio of pulmonary to systemic blood flow
RV = right ventricle
TTE = transthoracic echocardiography
VSD = ventricular septal defect

3. SUMMARY GRAPHIC



4. DESCRIPTION, RATIONALE, AND IMPLICATIONS

4.1. Rationale

The goal of this CCG is to provide cardiologists and other clinicians who care for patients with CHD with a decision support tool for the outpatient management of left-to-right shunt lesions (Figure 1). Although common in clinical practice, management varies significantly within and across institutions, particularly in serial outpatient follow-up and testing.^{11,12}

In drafting this document, the writing committee performed a comprehensive review of the available literature, including natural history studies, single and multicenter studies, and practice survey reports. Existing clinical practice guidelines were used whenever applicable.⁵ Although outpatient management includes pharmacologic and nutritional therapy, this document focuses primarily on resource utilization, including the frequency of outpatient visits as well as serial noninvasive imaging and other testing used for surveillance of these lesions before and after intervention. To create an easy-to-use

decision support tool, the approach to management of specific shunt lesions is presented as clinical algorithms with distinct branching points.

Considerations for individual lesions are specified under each section. Broadly, this approach was used to subdivide the clinical pathway algorithms. The algorithms were based on age when appropriate. For example, secundum ASDs without associated comorbidities are unlikely to result in symptoms in children <5 years of age, and therefore require infrequent outpatient surveillance, while children who present at or above this age trigger the decision point of whether intervention (surgical or transcatheter) is required based on clinical findings. Age was also a factor in determining the frequency of outpatient follow-up. Transitional changes in newborn physiology and the decrease in pulmonary vascular resistance during the first few weeks of life can cause patients with $\geq$ moderate-sized VSDs or PDAs to become symptomatic and thus require closer follow-up. Conversely, some defects such as perimembranous VSDs or PDAs can become restrictive, and the follow-up duration can be decreased accordingly. For shunt lesions such as VSDs and PDAs, the size and characteristics of the shunt as well as symptomatology determine the branch points in the decision tree, with $\geq$ moderate shunts requiring more frequent follow-up and ultimately intervention. To make the algorithms more concise and easier to follow, lesions were separated based on natural history and potential for additional complications rather than a purely morphological approach. For this reason, although various terminologies exist for the nomenclature of VSDs, they are divided into “muscular” and “nonmuscular” for the purpose of this document. Muscular VSDs have a high incidence of spontaneous closure and are unlikely to have complications when hemodynamically insignificant. However, perimembranous and outlet defects, even when small, require surveillance for aortic valve prolapse, development of a double-chambered right ventricle or subaortic membrane, or both. Similarly, the predominant physiology (ie, ASD or VSD physiology) dictates the branching points of the decision tree in the AVSD algorithm.

Although comprehensive, these algorithms are not designed to be all-encompassing; rather, they provide guidance for the most frequently encountered clinical scenarios. Pulmonary artery hypertension with resultant Eisenmenger physiology is rarely encountered in the pediatric age group in the United States due to early detection of CHD in most cases. Therefore, although references are made to alert clinicians to the possibility, the algorithms do not address outpatient follow-up and testing frequency in this high-risk population. Similarly, the testing frequency is suggested

based on applicability to the most common presentation or natural history and is intentionally broad, and not intended to be overly prescriptive. Given the inherent heterogeneity in CHD, this approach allows clinicians the flexibility to tailor their approach to each patient based on patient factors and institutional standards. Similarly, the presence of genetic syndromes and other comorbidities may require more frequent follow-up; thus, they are excluded except for trisomy 21, given its frequent association with left-to-right shunt lesions.

Considerations for postintervention follow-up are specific to the shunt lesion and the method of intervention, whether surgical or catheter-based. With transcatheter devices, outpatient follow-up and imaging may vary by manufacturer recommendations; therefore, a broader range is provided. For relatively newer devices, for which long-term data are lacking, a conservative approach to continued follow-up has been suggested. The presence and severity of residual shunting or procedure-related complications dictate the frequency of surveillance and referral for reintervention. Finally, these clinical practice algorithms are not intended to replace the independent professional judgment of health care clinicians, which may vary on an individual case-by-case basis.

4.2. Atrial Septal Defect

4.2.1. Anatomic and Physiologic Considerations

Isolated ASDs are among the most common types of CHD. Atrial-level shunting can arise from various anatomic defects, and their management is guided by their size, location, and the presence of associated sequela. The various types of ASDs are defined by their anatomic features and location in the atrial septum. This document focuses on surveillance and intervention for the most common type—secundum ASDs—which account for 75% of ASDs and excludes more complex anatomy, including sinus venosus ASDs, which are frequently associated with pulmonary venous anomalies. Notably, despite their high prevalence, significant variation exists in the clinical management of secundum ASDs.^{11,13}

Patent foramen ovale is a remnant of fetal circulation that allows a very small amount of shunting at the atrial level and may be present in as many as one-fourth of the healthy adult population.¹⁴ Given that these are generally considered benign in childhood and rarely intervened on, they are not addressed in this document.

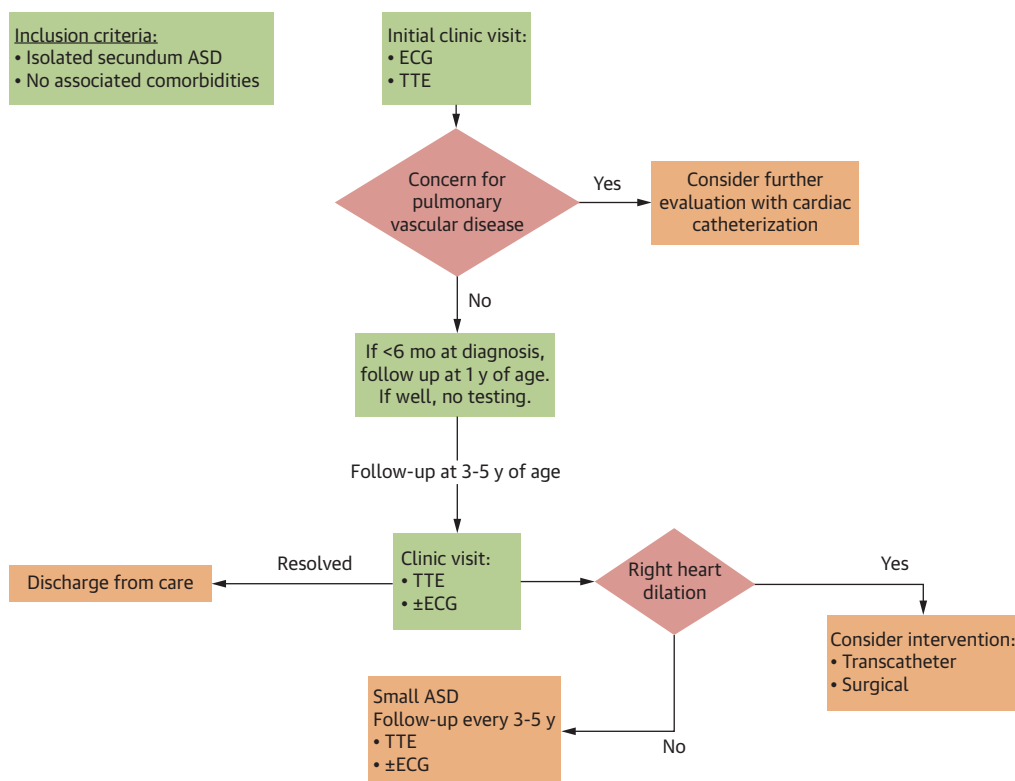
4.2.2. Management

Surveillance

Small secundum ASDs

In small secundum ASDs, the atrial-level shunting is not hemodynamically significant. Studies have demonstrated high rates of spontaneous closure for small secundum

FIGURE 2 Outpatient Management of Isolated Secundum ASD in Patients <5 Years of Age



ASD = atrial septal defect; ECG = electrocardiogram; TTE = transthoracic echocardiography.

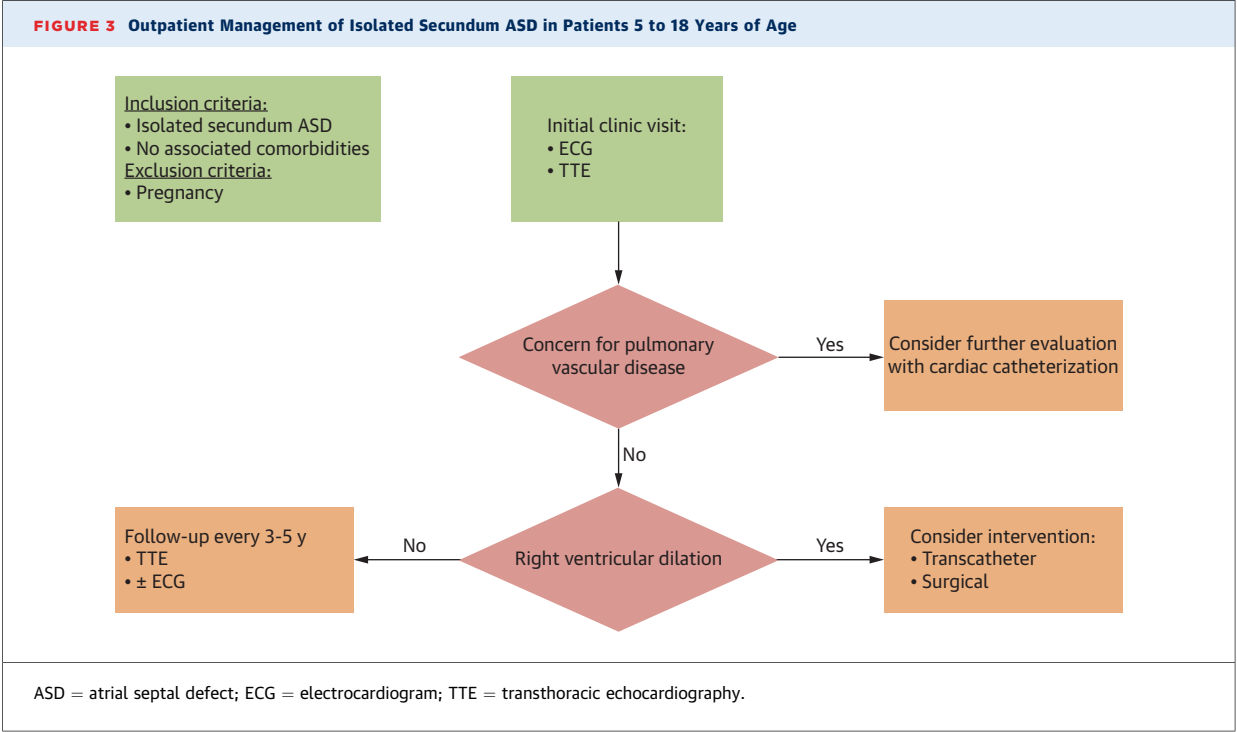
ASDs that are diagnosed in infancy.^{15,16} Appropriate counseling on the low but real risk of thromboembolic events, including stroke and the increased risk of decompression sickness with deep sea diving, should be provided. Small secundum ASDs may be followed every 3 to 5 years to reassess for right ventricular dilation by TTE (Figure 2).⁵

Moderate or large secundum ASDs

Moderate or large secundum ASDs result in volume overload and subsequent dilation of the RV. Although it is generally accepted that hemodynamically significant ASDs diagnosed early in life should be closed prior to the child being school-age (ie, typically at 3-5 years of age), wide variation is seen in the surveillance of these patients in the period prior to considering intervention. Even large ASDs are typically asymptomatic in infants and young children in the absence of other comorbidities such as chronic lung disease. Thus, frequent follow-up prior to considering intervention is not necessary, and an evaluation at 3 to 5 years of age for intervention planning is recommended (Figure 2).

Intervention

ASD closure is indicated when there is a hemodynamically significant shunt with a Qp:Qs $\geq 1.5:1$, right ventricular dilation, or both. Because most patients with isolated ASDs do not warrant diagnostic catheterization, indicators of a significant shunt include right ventricular dilation on TTE, cardiomegaly or increased pulmonary vascular markings on chest x-ray, and right ventricular hypertrophy/enlargement on ECG (Figure 3). As most patients are asymptomatic in childhood, closure of hemodynamically significant shunts is performed to prevent late complications, including pulmonary hypertension, atrial arrhythmias, right ventricular dysfunction, paradoxical embolism, and hemodynamically significant tricuspid regurgitation.¹⁷ For secundum ASDs that meet the criteria for closure, intervention can be performed via transcatheter device placement or surgery after discussion of the pros and cons of each approach with the family.¹⁸ Candidacy for catheter-based intervention is governed largely by the anatomic features of the defect, including the size, associated rims, and patient size. The technology for transcatheter device closure has continued



to evolve over time, and because of the less invasive nature and excellent long-term outcomes, it is generally preferred in the current era, provided the anatomy is favorable and the shared decision-making with the patient, family, or both supports this approach.¹⁸

Postintervention

After ASD closure, periodic long-term surveillance is indicated (Figure 4). If the closure is performed in a preschool-aged child, and the child is asymptomatic without residual sequelae, follow-up can be scheduled at 2 to 5 years after the first year following closure.⁵ Some ASD devices have the risk of late erosion, warranting periodic surveillance. If there is a residual atrial-level shunt, more frequent follow-up is warranted—every 1 to 2 years to determine the significance of the shunt and intervene as needed. Although late complications are rare, an increased risk is possible in those who undergo closure much later in life. These complications include atrial arrhythmias, progression of pulmonary vascular disease, continued right ventricular dilation and dysfunction, left ventricular dysfunction, and functional mitral regurgitation. In these scenarios, more frequent follow-up (every 3-12 months) with appropriate management will be needed, including additional testing such as Holter monitor, exercise testing, and CMR.^{1,5} Bacterial endocarditis is a rare complication that can occur after both surgical and device closure, and antibiotic prophylaxis is recommended for the first 6 months

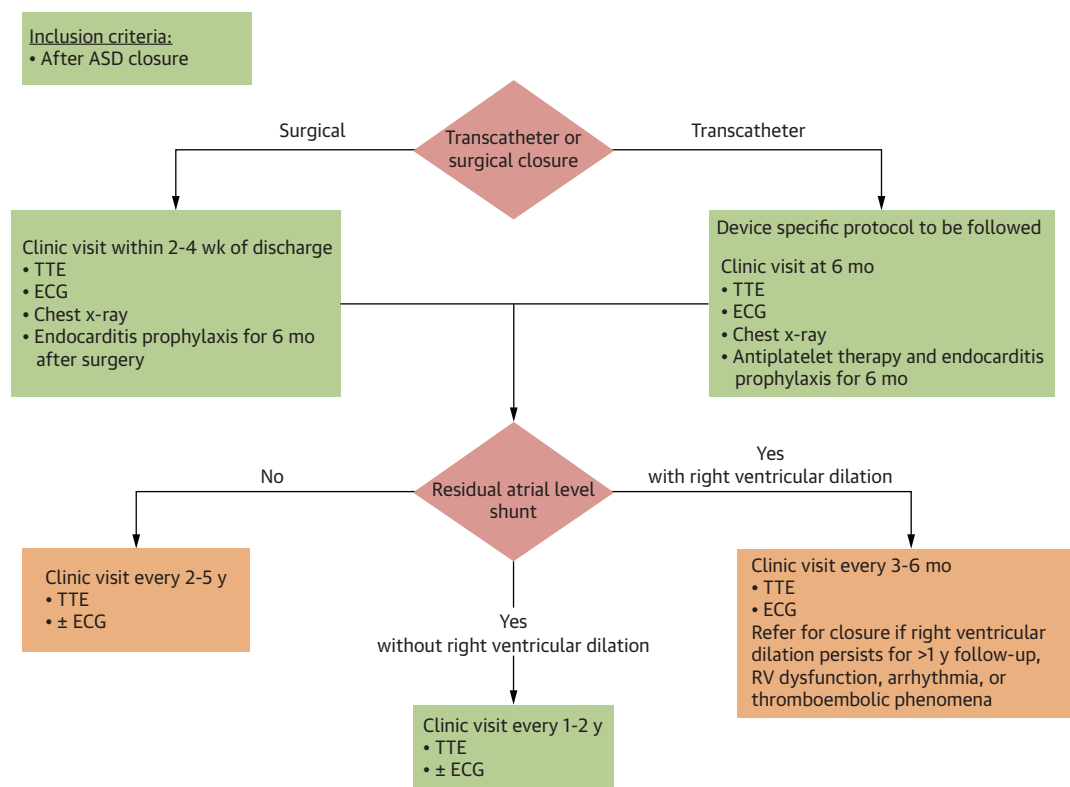
after intervention, until the device or prosthetic material is endothelialized.^{19,20}

4.3. Ventricular Septal Defect

4.3.1. Anatomic and Physiologic Considerations

VSDs are the most commonly diagnosed CHD in childhood. Several nomenclatures for VSDs are used; however, from a clinical management standpoint, they can be classified as muscular or nonmuscular VSDs based on their distinct natural histories and potential for additional complications. VSD location and size, patient age, symptomatology, and shunt direction are helpful in determining ongoing cardiology follow-up. Muscular VSDs are most likely to decrease in size or close spontaneously due to progressive growth of surrounding cardiac tissue. Perimembranous VSDs may also become partially or completely occluded by aneurysmal tissue formed by the septal leaflet of the tricuspid valve. Aortic valve prolapse into a perimembranous VSD may, in uncommon cases, reduce shunt flow. Small defects are typically asymptomatic, whereas moderate or large defects present with signs and symptoms of pulmonary overcirculation, including tachypnea, poor feeding, and failure to thrive. A hemodynamically significant VSD refers to a defect that can result in these symptoms and/or left heart dilation. With compensated moderate shunts, symptoms may be minimal, but progressive left heart dilation can be noted. Untreated and long-standing hemodynamically significant VSD shunts can result in Eisenmenger syndrome with irreversible pulmonary vascular changes.

FIGURE 4 Outpatient Management of Secundum ASD Postintervention



ASD = atrial septal defect; ECG = electrocardiogram; RV = right ventricle; TTE = transthoracic echocardiography.

4.3.2. Management

Wide variation exists in the management of VSDs,^{11,12} including frequency of follow-up and imaging, despite evidence that many of these defects are not hemodynamically significant and can close spontaneously²¹ without the need for medical or surgical intervention.¹² VSDs other than small muscular VSDs, whether repaired or unrepaired, will require surveillance because they can cause symptoms or have associated complications.^{22,23}

Surveillance

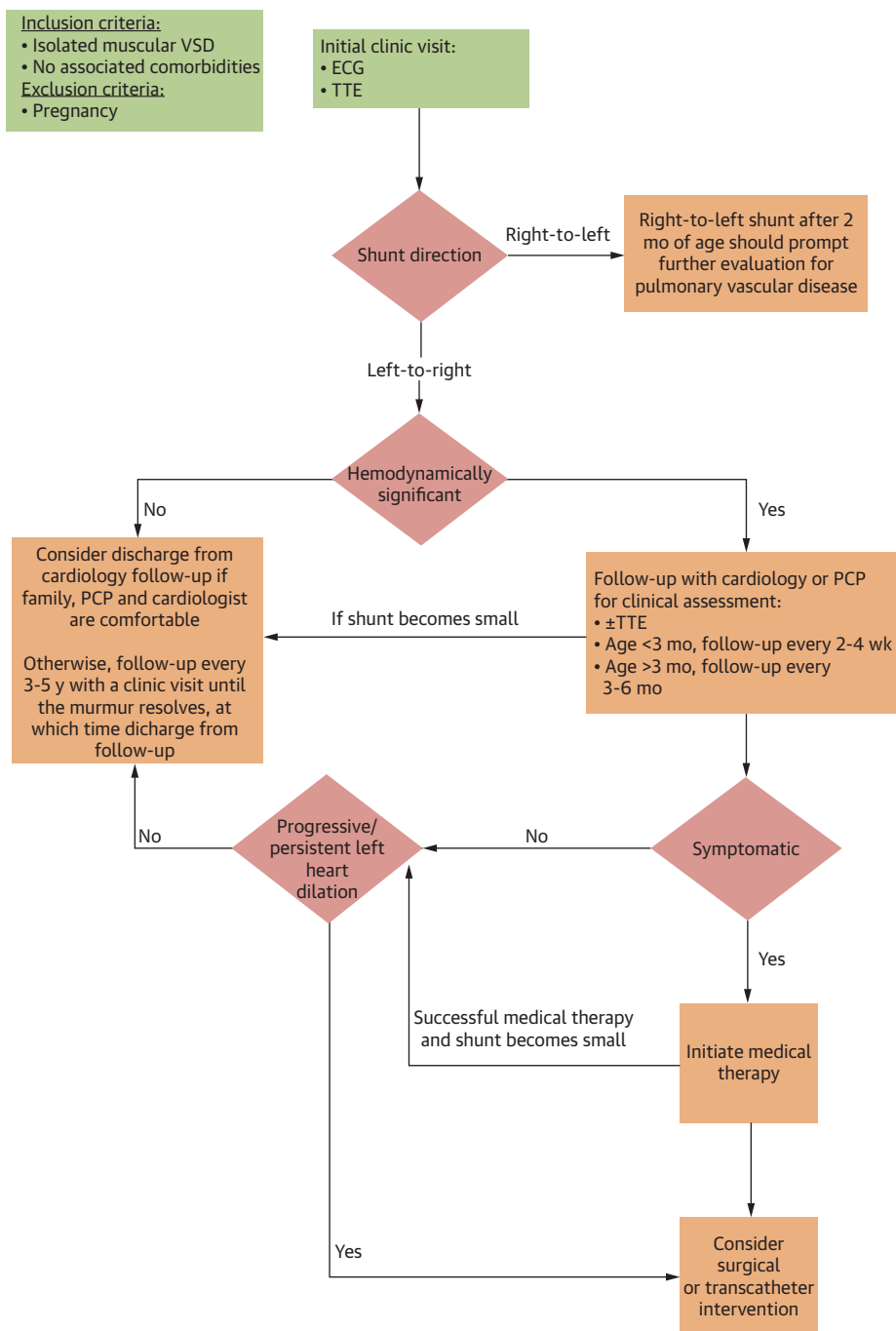
Unrepaired Muscular VSDs

Patients with small muscular VSDs can be discharged from the clinic using a shared decision-making model if the patient, their family, and the primary care provider are comfortable with the recommendation (Figure 5).

Otherwise, a cardiology follow-up every 3 to 5 years can be continued until the associated heart murmur resolves, at which time the patient should be discharged from cardiology. Given the natural history of small muscular VSDs, repeated TTE does not add further value to care unless a change in clinical status is observed. The family should be educated about an extremely rare but possible risk of endocarditis with small muscular VSDs.

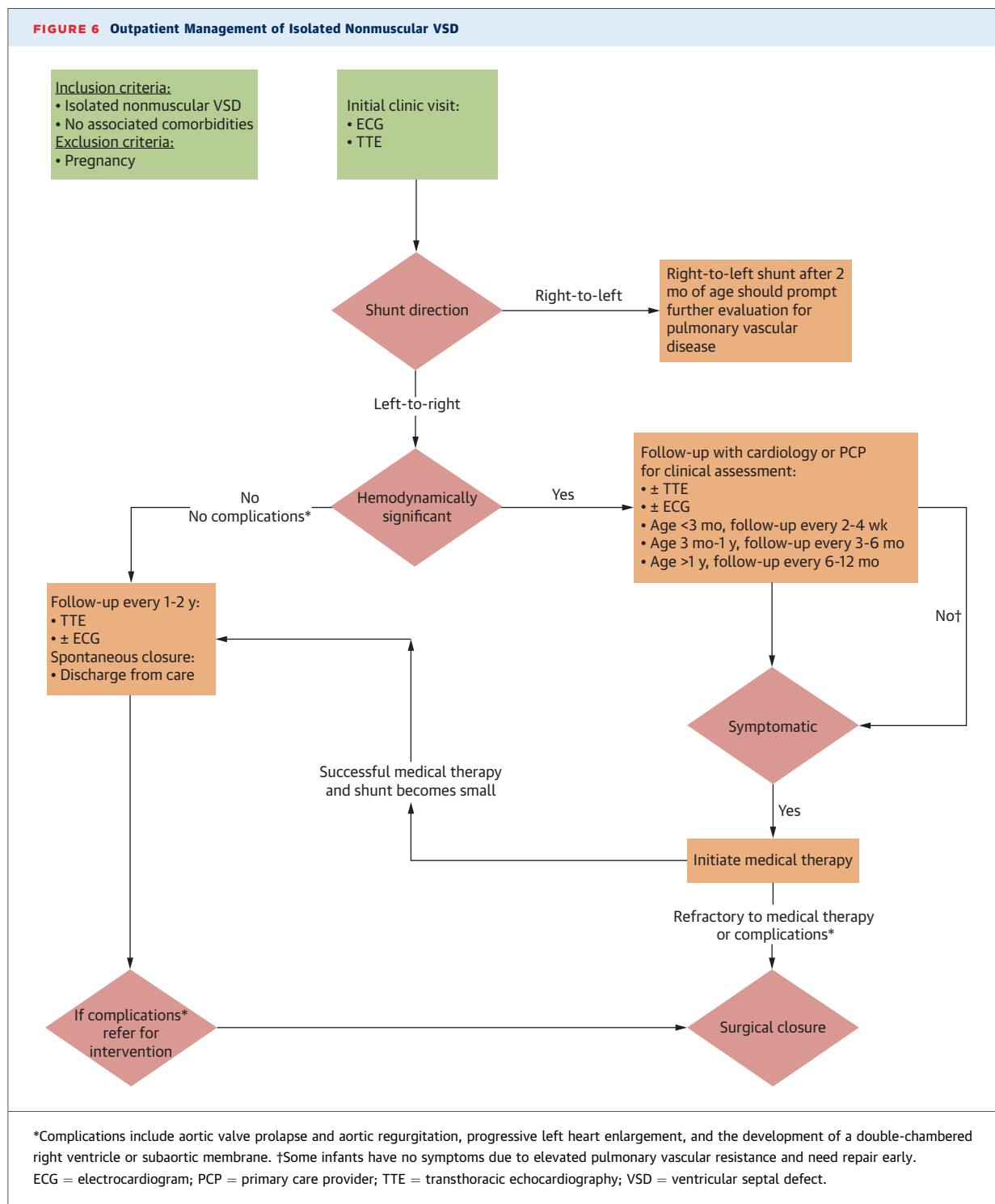
Moderate or large muscular VSDs should be followed more closely in infants. Frequent clinical assessment to evaluate respiratory status and weight gain is necessary. Medical therapy should be initiated if clinical signs and symptoms of pulmonary over-circulation are observed. Infants <3 months should be followed every 2 to 4 weeks because of the changes in pulmonary vascular resistance that can lead to the development of symptoms, although patients >3 months of age can be followed every 3 to 6 months depending on their clinical

FIGURE 5 Outpatient Management of Isolated Muscular VSD



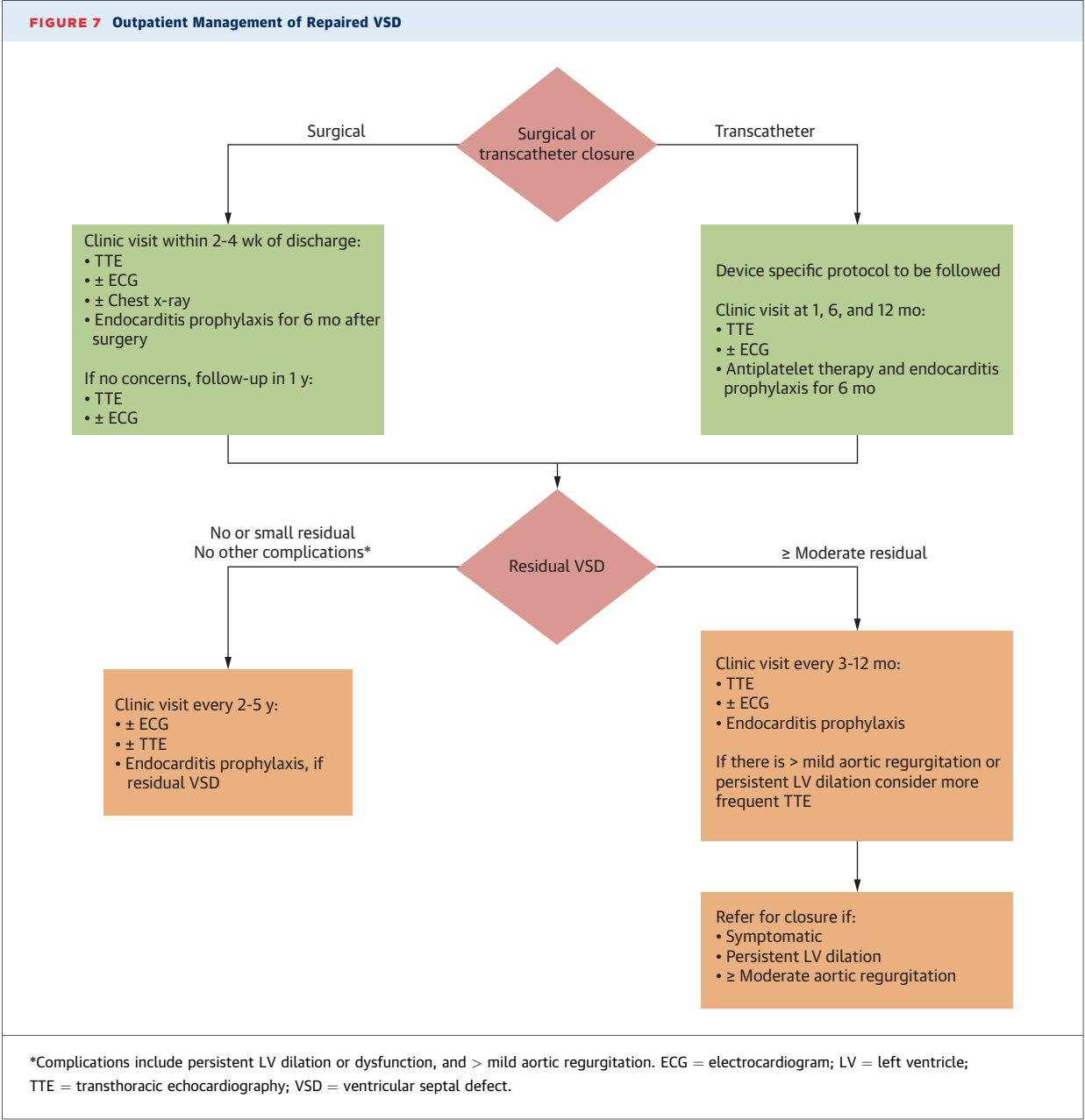
ECG = electrocardiogram; PCP = primary care provider; TTE = transthoracic echocardiography; VSD = ventricular septal defect.

FIGURE 6 Outpatient Management of Isolated Nonmuscular VSD



status. Children >6 months of age with persistent or progressive left ventricular dilation or symptoms should be considered for surgical or device closure of the VSD. The timing of intervention will depend on the number, location, and size of the VSD, as well as clinical symptoms. In some cases, where a complete repair is not

feasible in a young infant, a pulmonary artery band may be considered for initial palliation. Although TTE does not need to be obtained at every visit, it should be obtained periodically to assess the magnitude and direction of shunt flow and the development of pulmonary vascular disease.⁵



Unrepaired Nonmuscular VSDs

Nonmuscular VSDs have additional potential complications beyond left heart enlargement and the development of pulmonary vascular disease. A risk of the development of double-chambered right ventricle, aortic valve prolapse, and subaortic membrane have been observed.²⁴ Similar to muscular VSDs, the initial surveillance in infants and children centers around assessment of symptoms and left heart enlargement. Unlike muscular VSDs, all nonmuscular VSDs should continue to be followed by a cardiologist unless

spontaneous closure occurs (Figure 6).⁵ For hemodynamically significant nonmuscular VSDs, clinical assessment should be more frequent during infancy. Although TTE is not needed at every visit during infancy, it should be considered periodically to assess shunt volume and development of any associated complications. If the shunt is small and no left heart dilation is observed, cardiology clinic follow-up every 1 to 2 years, including a TTE, should be obtained, unless spontaneous closure occurs. Outlet VSDs that have a deficiency of the muscular or fibrinous support

underneath the aortic valve are predisposed to aortic valve prolapse and regurgitation. Elective surgical repair of these defects in the absence of aortic valve prolapse or regurgitation remains controversial. However, surgery should be considered in the presence of aortic valve prolapse, progressive aortic regurgitation, or both.

Intervention

For both muscular and nonmuscular VSDs, initiation of medical therapy is recommended if the patient is symptomatic. Signs and symptoms can include tachycardia, tachypnea, hepatomegaly, sweating, poor weight gain, and feeding intolerance. Medical management can include diuretics, afterload-reducing drugs, and other strategies such as nasogastric feeding and calorie-dense feeds.^{25,26} Referral for closure, either surgical or catheter-based, is indicated if medical therapy does not improve symptoms or left heart dilation, or if additional complications manifest.²⁴ Closure of large defects should be considered during infancy, regardless of symptoms, because of the risk of irreversible pulmonary vascular changes. Depending on the age of presentation and clinical manifestations, cardiac catheterization may be considered in some patients to assess the pulmonary vascular resistance prior to intervention.

Postintervention

Patients with VSDs, even after repair, have higher morbidity and death compared with the general population, thus requiring lifelong follow-up.^{22,23} Patients should be monitored for arrhythmias, conduction abnormalities, ventricular dysfunction, and any residual ventricular shunt. Much of the initial follow-up is currently based on institutional or device manufacturer protocols when available. Initial TTE may show a reduced left ventricular systolic function after repair of large shunts, but this typically improves within a few months.²⁶ For patients who are more than 1 year post-intervention, with no or small residual shunt and no other sequelae, follow-up frequency can be reduced to every 2 to 5 years, as can cardiac testing (Figure 7). ECG and TTE do not need to be performed at every visit in asymptomatic patients but should be performed intermittently to assess complications related to the VSD repair and if any new clinical symptoms or findings.⁵ Those with moderate or more residual shunts can be followed more closely and may ultimately need to be

considered for reintervention. Endocarditis prophylaxis is recommended for 6 months after surgical or device closure and should be continued in those with a residual shunt at the site or adjacent to the prosthetic material or device.^{19,20}

4.4. Patent Ductus Arteriosus

4.4.1. Physiologic Considerations

Isolated PDA in term infants is a relatively common defect, accounting for 5% to 10% of CHD.²⁷ Given the rise in use of TTE, incidental detection of asymptomatic “silent” PDA is increasingly common. The size of the PDA correlates with the physiologic consequences. Patients with trivial or small PDAs are usually asymptomatic, while moderate and large PDAs can cause tachypnea, tachycardia, poor weight gain, left heart dilation, and eventually, elevated pulmonary vascular resistance. Symptomatic patients and those with left heart dilation related to PDA (ie, patients with a hemodynamically significant shunt) are referred for closure.²⁷ A wide variation remains in the timing of follow-up and testing of PDA, including that of asymptomatic newborns with small shunts.¹¹

4.4.2. Management

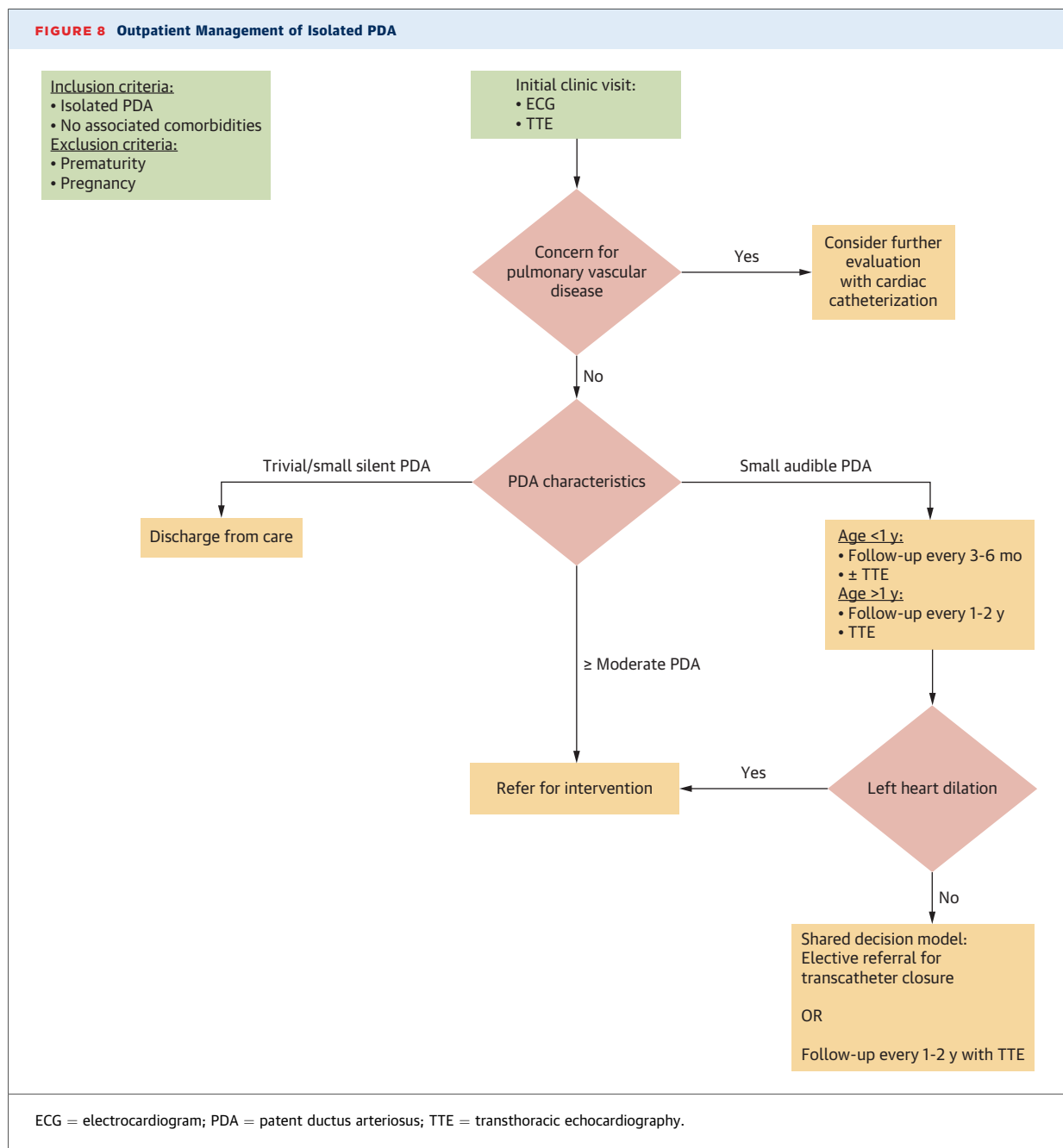
Surveillance

Trivial “Silent” and Small PDAs

Patients with trivial or small “silent” PDA can be discharged from care (Figure 8).^{5,28} A small, audible PDA should be followed during infancy every 3 to 6 months. Intervention is indicated if evidence of left heart dilation is observed. Beyond infancy, in those without left heart dilation, a shared decision-making model should be used to either refer for elective transcatheter closure or continue conservative follow-up every 1 to 2 years.^{5,27}

Moderate or Larger PDAs

Moderate or larger PDAs should be followed more frequently, particularly in infants, and prompt referral for intervention should be made.²⁸ Frequent clinical assessments for tachypnea, tachycardia, poor weight gain, and left heart dilation are recommended until intervention can be performed. In a large, nonrestricted PDA, pulmonary hypertension will always be present but not necessarily elevated pulmonary vascular resistance. If the shunt is all left-to-right in an infant, even when the



PDA is nonrestrictive, then the patient can be referred for closure of the PDA without the need for further evaluation by cardiac catheterization to determine pulmonary vascular resistance. Although medical therapy such as diuretics may be used in the short term while awaiting intervention, it is not the standard management strategy.

Intervention

Referral for PDA closure is indicated when patients are symptomatic, have left heart dilation, or both. Elective

closure may also be performed when a small and audible PDA is detected.²⁸ Most PDAs can be closed percutaneously in the cardiac catheterization laboratory, although surgical closure may be indicated in cases when the anatomy is unfavorable or in premature infants.²⁷ However, due to the rise in transcatheter-based closure strategies for premature infants, surgical closure is becoming less common at centers with the expertise to do so. The closure of the PDA in premature infants is a relatively recent advancement, and the literature in this area is still

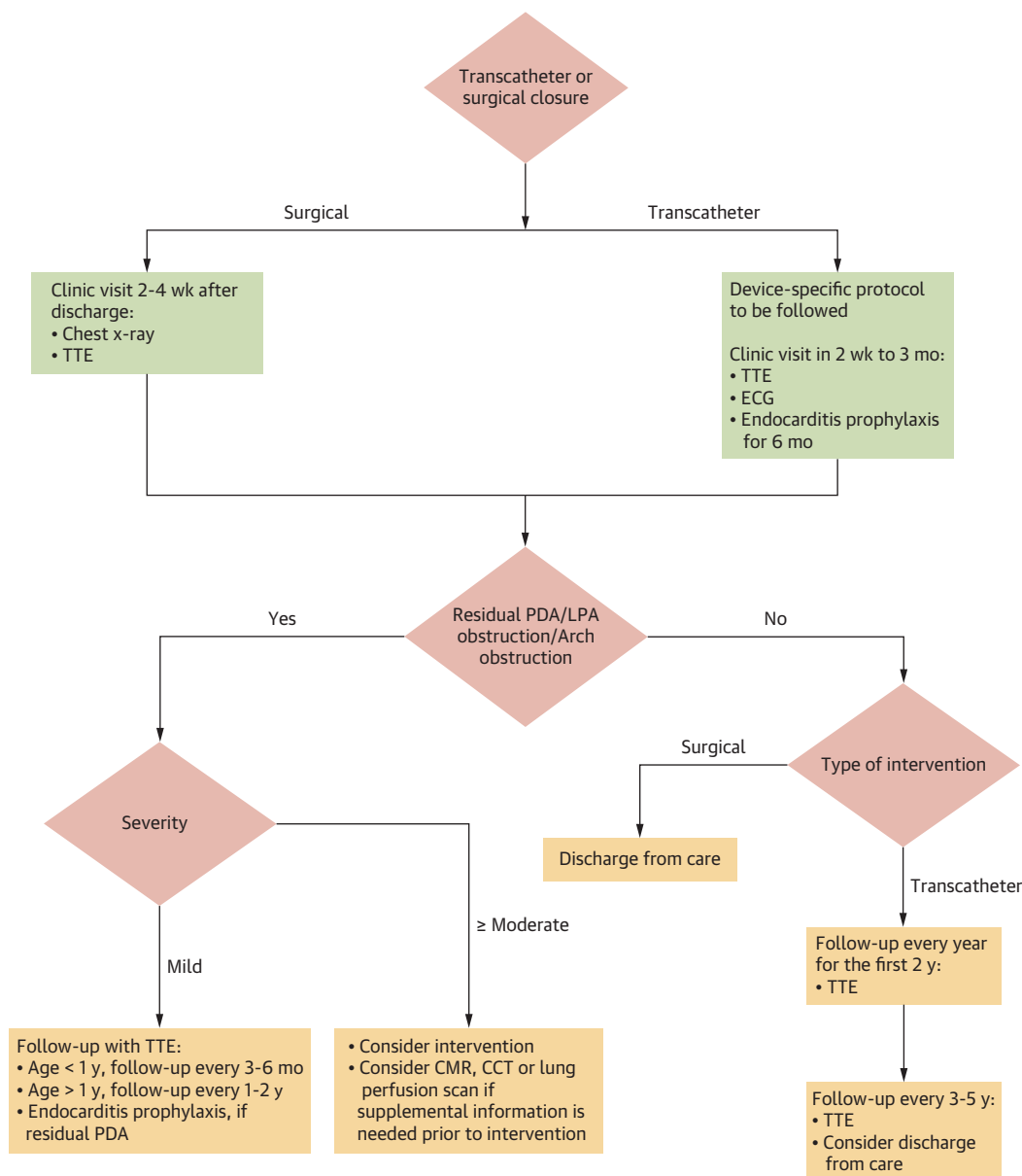
evolving. As such, we have excluded this from our current algorithm and recommend that clinicians use the most up-to-date literature when considering this approach.

Postintervention

After surgical or transcatheter PDA closure, clinic follow-up with a TTE is recommended within 2 weeks to 3

months (or based on device protocol) (Figure 9). Patients without residual PDA, left pulmonary artery (LPA), or aortic arch obstruction, following surgical PDA closure may be discharged from care. In cases of transcatheter PDA closure without these residual lesions, annual follow-up for the first 2 years following closure is recommended. Subsequently, the follow-up could either be spaced out every 3 to 5 years with a TTE,⁵ or discharge

FIGURE 9 Outpatient Management of PDA Postintervention



CCT = cardiovascular computed tomography; CMR = cardiovascular magnetic resonance; CXR = chest X-ray; LPA = left pulmonary artery; PDA = patent ductus arteriosus; TTE = transthoracic echocardiography.

from care may be considered in uncomplicated patients.²⁹ Regardless of a surgical or transcatheter closure approach, patients with residual PDA, LPA, aortic arch obstruction, or cardiac dysfunction require ongoing and more frequent monitoring. In the absence of cardiac dysfunction, follow-up frequency depends on the size of the residual PDA, the severity of the LPA aortic arch obstruction, or both.³⁰⁻³² In mild cases, follow-up with TTE is recommended every 1 to 2 years with consideration for supplemental information from CMR, CCT, or a lung perfusion scan.⁵ Patients with moderate or severe findings require reintervention.³⁰⁻³² Given the limitations of TTE in visualizing structures such as the branch pulmonary arteries and aortic arch, additional imaging with CCT or CMR may be required for assessment of severity and intervention planning. Endocarditis prophylaxis is indicated for the first 6 months in all patients and indefinitely in those with a residual shunt following device closure.^{19,20}

4.5. Atrioventricular Septal Defect

4.5.1. Anatomic and Physiologic Considerations

AVSD (also referred to as atrioventricular canal defect) represents a spectrum of CHD that results from abnormal embryological development of the endocardial cushions in the primitive atrioventricular canal. Primum ASD, inlet VSD, and atrioventricular valve abnormalities are among the possible pathologic features of AVSD. AVSD can be subclassified based on the atrioventricular valve anatomy and the presence and degree of ventricular shunting. A complete AVSD consists of a primum ASD, an inlet ventricular septal defect, and an atrioventricular valve with a common annulus. Intermediate AVSD is a variant of complete AVSD that has distinct right and left atrioventricular valve orifices despite having 1 common annulus, and a large inlet VSD. Transitional AVSD refers to a subtype where the VSD is small and restricted by dense chordal attachments to the ventricular septum. A partial AVSD describes the presence of a primum ASD, a separate right and left atrioventricular valve annulus, and a cleft in the left atrioventricular valve.³³ Broadly, physiology can be predominantly ventricular-level or atrial-level shunt physiology, thereby determining the surveillance and management approach. The presence and degree of atrioventricular valve regurgitation, particularly of the left atrioventricular valve, also has a significant impact on the clinical course and physiology. Subsets of partial AVSD may have small left-sided structures, or a common atrium. These rare cases have been excluded from this document because their physiology, symptoms, and timing of intervention may differ.

4.5.2. Management

Surveillance

Small or No Ventricular-Level Shunt

Patients without a hemodynamically significant VSD are at low risk of developing symptoms in the absence of significant left atrioventricular valve regurgitation. However, infants should be followed every 3 to 6 months to monitor growth and assess symptomatology (Figure 10). Periodic TTE should be performed to assess cardiac chamber dilation and degree of left atrioventricular valve regurgitation.⁵ Infants with $\geq$ moderate atrioventricular valve regurgitation may be symptomatic and follow-up every 2 to 3 months is prudent. Beyond infancy, children with mild or less atrioventricular valve regurgitation require less frequent surveillance, provided that they are asymptomatic, and can be evaluated every 1 to 2 years with TTE until the time of repair.⁵ Children with $>$ mild atrioventricular valve regurgitation should continue to be followed at 6- to 12-month intervals.

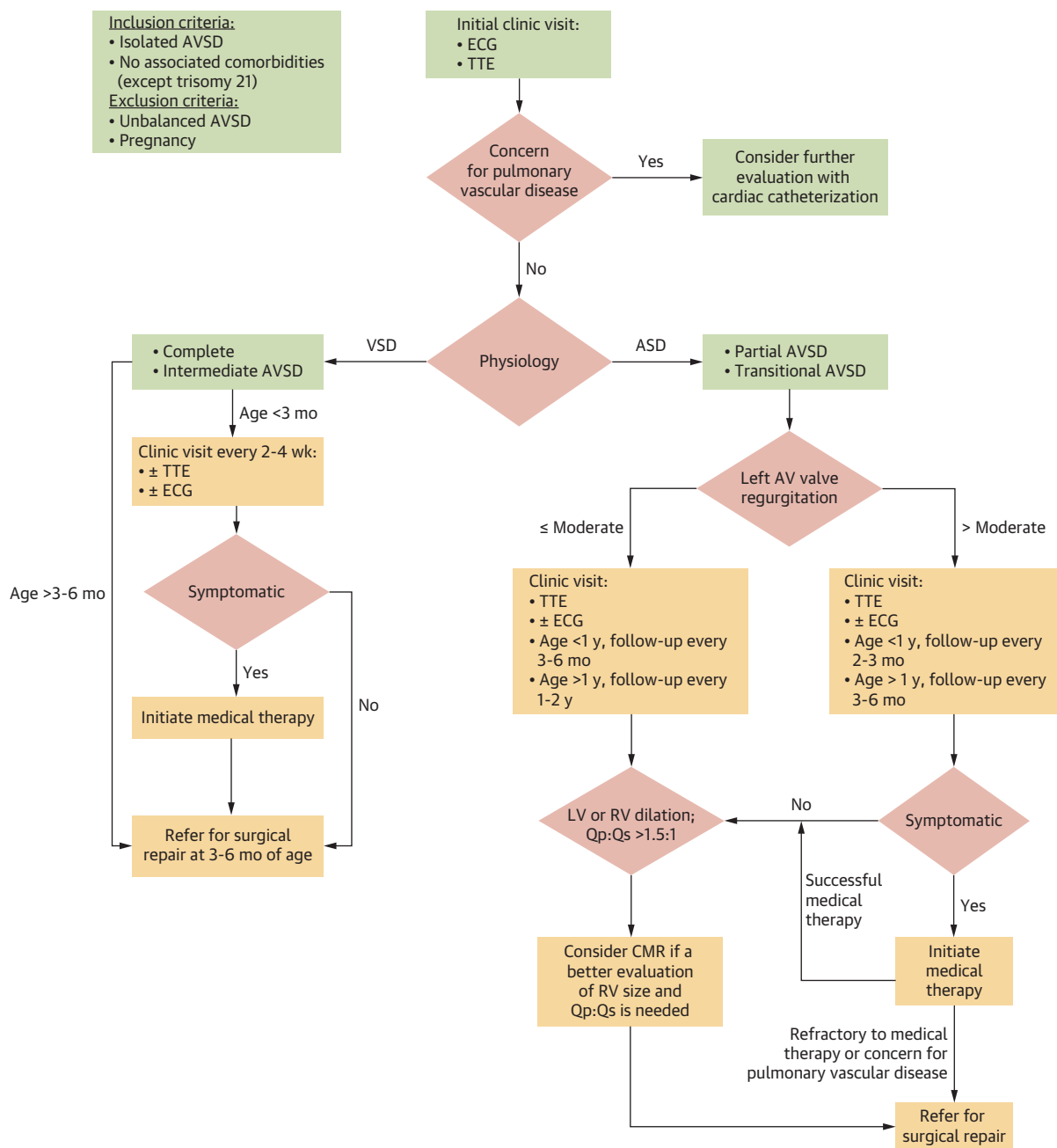
Moderate or Large Ventricular-Level Shunt

Patients with a $>$ small VSD are at risk for symptoms related to pulmonary overcirculation and thus require close follow-up in infancy for evaluation of symptoms and growth. Infants should be followed at 2- to 4-week intervals for clinical assessment. However, TTE is not necessary at every follow-up visit but should be performed periodically to assess chamber size, the degree of atrioventricular valve regurgitation, and the presence of pulmonary vascular disease.⁵

Intervention

Diuretics and afterload reduction may provide symptomatic relief related to pulmonary overcirculation. Additionally, feed fortification, enteral tube feeding, or both may be required to augment oral intake or compensate for increased metabolic demands. Surgical repair of all patients with complete AVSD should be considered between 3 and 6 months of age; earlier repair has been associated with worse outcomes.³⁴ Repair within this window is of particular importance in individuals with trisomy 21 or other risk factors for pulmonary hypertension to avoid progression to irreversible pulmonary vaso-occlusive disease. In partial and transitional AVSD, surgical repair is typically performed later because of the physiology of a primary atrial-level shunt and provided that no significant left atrioventricular valve regurgitation is observed. Repair of partial or transitional AVSD during infancy has been associated

FIGURE 10 Outpatient Management of Unrepaired Isolated AVSD



AV = atrioventricular; AVSD = atrioventricular septal defect; CMR = cardiovascular magnetic resonance; ECG = electrocardiogram; LV = left ventricle; Qp:Qs = ratio of pulmonary blood flow to system blood flow; RV = right ventricle; TTE = transthoracic echocardiography.

with a higher risk of reoperation, whereas repair in toddlers and older children has been associated with better outcomes.³⁵ Because of the increased risk of atrioventricular valve regurgitation with repair beyond 4 years of age in partial and transitional AVSD, the data support the

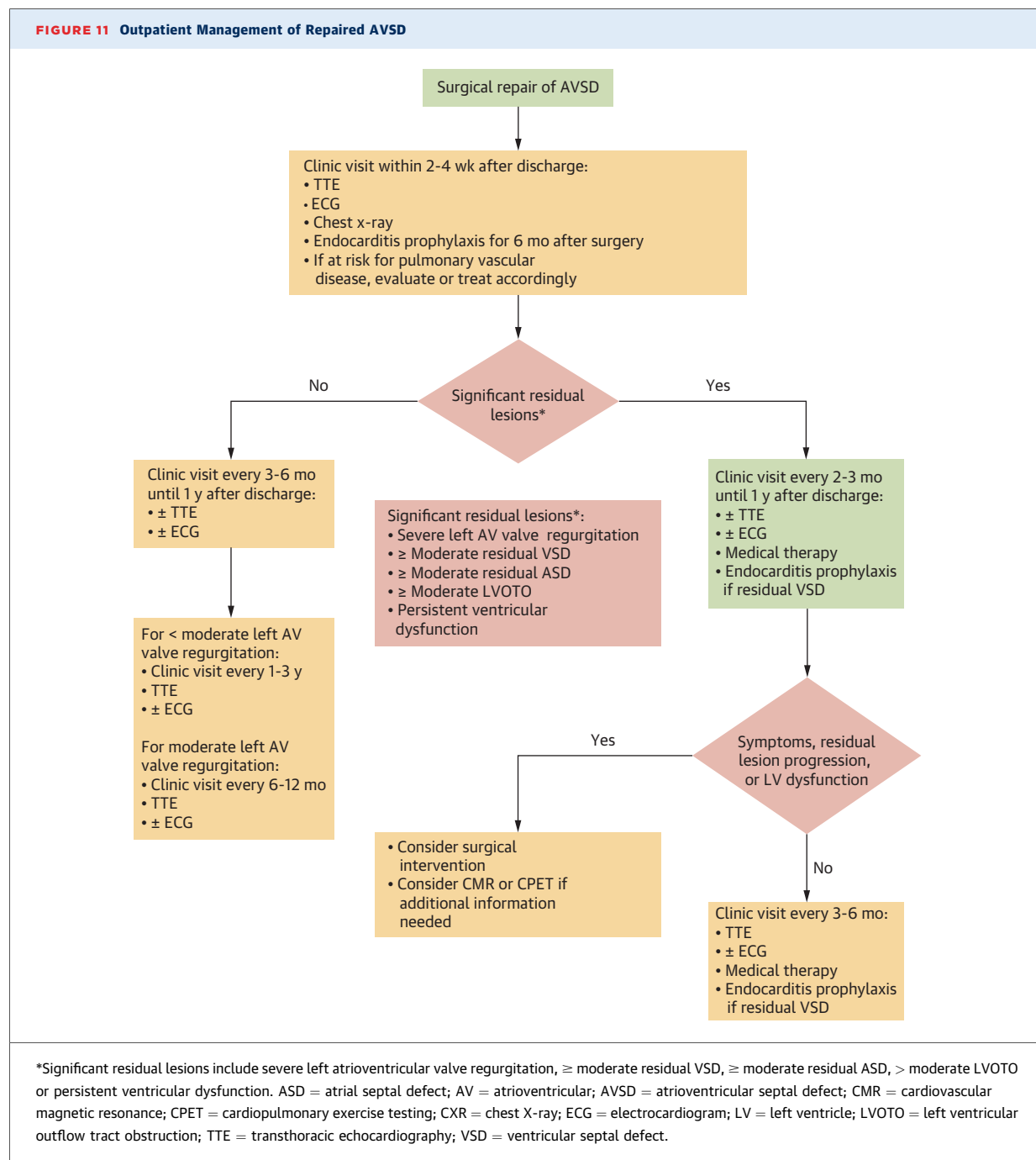
toddler years as the optimal window for surgical intervention.³⁶ Despite minimal perioperative mortality, the long-term burden of reoperation for left atrioventricular valve disease and left ventricular outflow tract obstruction (LVOTO) is not negligible.^{35,37}

Postintervention

After surgical repair of complete and partial AVSD, all patients will require lifelong surveillance to monitor any residual lesions, ventricular dysfunction, arrhythmias, or development of complications such as LVOTO and atrioventricular valve dysfunction. Approximately 15% of patients with both partial and complete AVSD can require surgical reintervention.^{35,37} The most common indications for reintervention are residual left atrioventricular valve regurgitation and LVOTO. In patients

without significant residual lesions, follow-up can occur every 1 to 3 years after the first postoperative year (Figure 11).⁵ For those with significant residual lesions, follow-up should occur every 3 to 12 months to assess residual intracardiac shunts, hemodynamically significant atrioventricular valve regurgitation, and LVOTO.^{1,5} Diagnostic testing includes primarily TTE, but also transesophageal echocardiography, CMR, or both in select cases. Electrocardiography is not indicated with every visit but may be performed if clinical concerns arise

FIGURE 11 Outpatient Management of Repaired AVSD



and for periodic surveillance of arrhythmia, particularly in patients with a history of postoperative arrhythmia. Endocarditis prophylaxis is recommended for 6 months after surgery and should be continued in those with a residual ventricular shunt at the site or adjacent to the prosthetic material.^{19,20}

4.6. Implications and Summary

This CCG offers cardiologists and other clinicians a practical decision-support tool for the outpatient management of common CHD lesions with left-to-right shunts. The overarching goal is to provide broad guidance for frequently encountered clinical scenarios, while allowing flexibility for clinicians to tailor care to individual patients. Although simplified for practicality and ease of use, the algorithms may not apply to every patient or encompass all possible clinical presentations. In the absence of evidence-based clinical practice guidelines for left-to-right shunt lesions in the pediatric age group, this document aims to bridge the current knowledge gap while acknowledging the limitations.

Ultimately, implementation of these clinical practice algorithms may help reduce practice variation and

promote appropriate utilization of diagnostic testing and directed therapies.³⁸ Single and multicenter implementation studies (eg, incorporation in the electronic medical record ordering and decision trees for outpatient imaging) based on this algorithm may help provide the foundation for future evidence-based management guidelines. Finally, clinician groups may use these algorithms for practice-based learning activities to support maintenance of certification.

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KEY WORDS ACC concise clinical guidance, atrial septal defect, atrioventricular septal defect, congenital heart disease, echocardiography, patent ductus arteriosus, pediatrics, ventricular septal defect

APPENDIX 1. AUTHOR RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES (RELEVANT)— OUTPATIENT MANAGEMENT OF ISOLATED LEFT-TO-RIGHT SHUNT LESIONS IN PEDIATRIC PATIENTS: 2026 ACC CONCISE CLINICAL GUIDANCE

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