ASD and VSD Flow Dynamics and Anesthetic Management

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Atrial septal defects and ventricular septal defects are often encountered in patients presenting for treatment under anesthesia. The flow mechanisms for both defects are predominantly left to right shunting prior to long-term maladaptive changes that may occur. Close examination of the shunt dynamics demonstrates a minor right to left shunt that occurs as well. The article discusses these dynamics and the impact on an anesthetic plan.

Key Words: Atrial septal defect; Ventricular septal defect; Shunt; Flow dynamics.

Atrial septal defects (ASDs) and ventricular septal defects (VSDs) are common congenital heart defects (CHDs) likely to be encountered by the anesthesia provider. The flow dynamics of such defects are discussed in this article with an emphasis on how they may impact anesthetic management. Epidemiological data and long-term prognosis of these defects are also discussed to assist in perioperative decision making.

ASD INTRODUCTION

Solitary ASDs constitute approximately 6–10% of all CHDs, making them the third most common CHD. They affect females with a frequency of 2–3 times more than males. CHDs can be detected in up to 75/1000 live births of the general population.¹ There are 4 major types of ASDs: ostium primum, ostium secundum, sinus venosus, and coronary sinus types. The ostium secundum type is by far the most common, making up over 75% of all ASDs. Ostium primum, which is a form of atrioventricular septal defect involving the mitral and tricuspid valves (canal defect), is next and the other 2 types are very rare.

Of particular concern to the anesthesia provider is the number of undetected or asymptomatic ASDs, which can be quite frequent before the age of 2 because of the lack of significant signs and symptoms. Detecting an ASD by physical examination alone can be difficult. Regular screening with electrocardiogram and echocardiogram analysis is not indicated as a routine part of primary care pediatrics. Though many ASDs will spontaneously close, especially those smaller than 3 mm, it is rare for defects to close after the second year of life.

Regardless of the number of undetected ASDs in the general population, it can be said without doubt that the anesthesia provider is likely to encounter patients with an ASD during his or her career. The flow dynamics of this defect are interestingly complex and a thorough understanding of these processes is an invaluable asset to the library of knowledge we carry for managing our patients.

In the early stages of an ASD, the developing heart has not undergone significant muscular or anatomic compensation in response to the defect. Very large ASDs may lead to earlier changes, but we will focus on the medium to small ASDs—those that are more likely to be encountered either as an intentionally unrepaired defect or a previously undetected one.

SHUNT DYNAMICS

The direction of any shunt (a passage that allows movement of fluid from one part of the body to another)

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requires that a gradient be formed between the 2 sides of the defect. Either a pushing force or a pulling force can generate this gradient. In other terms, a gradient can be established by squeezing on a given chamber, X, into chamber Y. Conversely, if chamber Y acts as a vacuum, the direction of the gradient towards Y can be generated.

Rudimentary understanding of the differences between the left and right sides of the heart would suggest that an ASD should result in a left to right shunt. In a left to right cardiac shunt, oxygenated blood flows from the left side of the heart to the right side, where it will reenter the pulmonary circulation. Indeed, this is the predominant feature that we observe. However, the ASD shunting actually occurs in 2 phases, each in a particular period in the cardiac cycle. The first stage of left to right shunting occurs in the late stages of ventricular contraction. As the force of the left ventricle rises, the majority of left to right shunting takes place shortly after the peak of left ventricular contraction. This coincides with our existing knowledge that the left side of the heart has a larger force-generating capability because of the increased pressure needed to open the aortic valve versus the pulmonic valve. Chronologically, the next phase of left to right shunting occurs during diastole at the time of atrial contraction.

The unexpected feature of an ASD is that most patients will also demonstrate a small but appreciable right to left shunt in the normal cardiac cycle. This shunt also occurs in 2 phases, a “double-bump” characteristic that reflects subtle changes in muscular and valvular activity.

The first bump is during rapid ventricular filling during early diastole, not dissimilar to a drawing or siphoning away from the left atrium, which favors a right to left shunt. The second bump is during initial ventricular contraction. At the time the mitral valve closes prior to ventricular contraction, the atrial pressures tend to equilibrate, thus allowing the minor right to left shunt to occur. However, very quickly the ventricular contraction will begin to build global pressure in the atria and form a left to right gradient.

In summary, from a chronological standpoint we first observe a minor right to left shunt during diastole and ventricular filling, followed by a secondary increase in right to left shunt as the mitral valve closes and ventricular contraction begins. Then, as left ventricular pressure rises towards peak pressure, the shunt shifts to its predominant direction of left to right. A secondary increase in left to right shunting occurs in early diastole as the atria contract. The cycle then restarts. See the Figure regarding flow directions.

Both ASDs and VSDs lead to volume overload of the heart. This overload is initially compensated by adjustment of the Frank-Starling curve and increasing total blood volume as the body attempts to maintain cardiac

output and forward flow. Over time, this volume overload leads to right ventricular dilation and increasing pulmonary vasculature pressures. VSDs have a tendency to lead to pulmonary hypertension more easily and earlier than ASDs. The compliance of the right heart allows for significant compensation before symptoms and irreversible consequences are observed.

ANESTHETIC MANAGEMENT

By applying these physiologic data to clinical scenarios, it may become clear why certain precautions are taken while treating these patients. Ensuring that the intravenous line is free of air bubbles, however minute, is especially important in patients with ASDs because of the potential for a bubble to cross from the right to the left heart and into systemic circulation. Once on the left side of the heart, these bubbles may travel up towards the brain via the brachiocephalic and left common carotid arteries, thus causing paradoxical embolisms. The evidence demonstrates very low rates of permanent brain injury, most recently as low as 1/40,000 cases.² However, there is no way of knowing how these small embolisms may affect the developing brain, and thus the anesthesia provider should maintain vigilance in preventing this complication. Looking at non-anesthesia-related evidence, up to 40% of strokes are considered cryptogenic, meaning no identifiable cause can be found.³ Of those cryptogenic strokes, 42% have a patent foramen ovale (functionally similar to ASD in shunt dynamics), up to 50% in young patients.⁴ In terms of relative likelihood, this potential of right to left movement is not simply an academic matter, but a potential that occurs during every single beat of the heart. Additionally, the potentially high number of undetected ASDs in children magnifies the importance of bubble avoidance in the entirety of the pediatric population.

Anesthetic management of an ASD involves reducing the magnitude of the shunt and yet also preventing the establishment of a hypoxic shunt phenomenon (right to left shunt). Changes in systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR) are 2 parameters likely to be impacted by sedation and anesthesia, and they are also the main determinants of how the shunt behaves. Heart rate, contractility, and other features do not play as important a role as they do not impact the flow dynamics the way that SVR and PVR do.

Preventing the establishment of a hypoxic shunt phenomenon involves avoiding a state in which there is low SVR relative to PVR, which may occur, for example, in the setting of bronchospasm, laryngospasm with vigorous ventilatory effort, and deep anesthesia combined with hypovolemia. The need to avoid these states is heightened in the setting of an ASD because it favors a

The figure depicts the direction and relative magnitude of shunt flow. Increasing values in the y axis indicate flow from left to right, whereas negative values indicate flow from right to left. Notice the primary left to right shunt occurs during late ventricular systole with a secondary increase during atrial contraction. The right to left shunting occurs in a double-bump fashion during rapid ventricular filling and the period just after the mitral valve closes before ventricular contraction begins.

right to left shunt that results in a hypoxic scenario that will be much more difficult to manage than a typical hypoventilation or low FiO_2 (fraction of inspired oxygen) scenario. Minor decreases in SVR will decrease the magnitude of the left to right shunt by promoting systemic blood flow but will not result in a hypoxic right to left shunt without a concurrent increase in right-sided heart pressures.

Alternatively, medications that lead to prolonged increases in SVR should be avoided because they will increase the magnitude of left to right shunting. In a similar manner, use of high FiO_2 for long periods may be problematic as well as it decreases PVR, again favoring an increase in left to right shunting. It is preferable to have a minor decrease in SVR, as it will decrease the magnitude of the left to right shunt, as noted above.

In summary, there are 3 distinct outcomes that can occur with changes in SVR and PVR. First, high PVR relative to low SVR is the main state that should be avoided as it causes a hypoxic shunt. Second, if PVR can be maintained at normal low pressures, increasing SVR will increase the magnitude of the left to right shunt, which is not ideal either. Third, if PVR remains normal, decreasing SVR will result in the most favorable circumstance, as it will decrease the magnitude of the left to right shunt while avoiding a hypoxic shunt flow.

Medications such as benzodiazepines and opioids are appropriate for the anesthetic management of patients with either ASDs or VSDs as they are typically hemodynamically stable, especially at sedative dose ranges. Minor decreases in SVR and heart rate seen with both types of medications decrease the magnitude of the left to right shunt without encountering enough of a gradient change to promote right to left shunting. General anesthetic agents such as volatile anesthetics, propofol, barbiturates, and possibly etomidate all lead to decreases in SVR, so care must be taken to minimize these responses and maintain SVR as much as possible. Adequate volume resuscitation, careful titration of general anesthetic agents, and avoiding relative hypoxia are all important management strategies.

The use of nitrous oxide reportedly increases PVR,⁵ particularly in higher doses in intubated patients, so it may not be the most ideal sedative adjunct as it would favor a right to left shunt. The concomitant increase in FiO_2 with the use of nitrous oxide may lead to an offsetting decrease in PVR, but ultimately, the impact of nitrous oxide would likely be significant only in cases in which the VSD or ASD already demonstrates profound cardiovascular instability.

A medication to generally avoid would be ketamine, as the increases in sympathetic stimulation can lead to sustained increases in SVR. Although there may be

Table 1. Anesthetic Management*

Anesthetic Management of ASD and VSD
 Avoid cyanotic shunt (right to left) by preventing profound decreases in SVR and acute increases in PVR
 Avoid increasing the magnitude of the left to right shunt by avoiding significant, prolonged increases in SVR
 Maintenance of SVR and PVR is ideal
 Minor decreases in SVR are preferable to increases in SVR
 Avoid introducing bubbles to the circulation
 Minimize the use of nitrous oxide
 Avoid ketamine in most circumstances

* ASD indicates atrial septal defect; VSD, ventricular septal defect; SVR, systemic vascular resistance; and PVR, pulmonary vascular resistance.

increases in PVR as well, it is uncertain which effect will predominate and it would remain prudent to avoid a medication that has a higher potential to impact the major physiologic determinants of shunt flows. The judicious use of ketamine to counteract the adverse hemodynamic effects of propofol could be considered, for instance, but close monitoring is essential. See Table 1 for a summary of anesthetic management for septal defects.

PROGNOSIS AND TREATMENT

ASDs may go undetected and untreated for several years because they initially fail to produce significant symptoms. However, once the ratio of pulmonary to systemic flow exceeds 1.5, closure of the defect is indicated. This can be accomplished via open heart surgery or a transcatheter approach. There are several transcatheter devices; however, only 2 are U.S. Food and Drug Administration–approved: the Amplatzer septal occluder and the Gore helix septal occluder. Surgical closure techniques include direct stitching of the defect or the use of a graft material such as a Dacron patch, bovine pericardium, or an autograft. In most cases, antibiotic prophylaxis against bacterial endocarditis is unnecessary unless one of the following criteria is met (taken from American Heart Association guidelines⁶ on prevention of infective endocarditis):

- Completely repaired CHD with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure.
- Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibits endothelialization).

Consultation with the cardiologist can be useful in the

Table 2. Atrial Septal Defect Summary

Major direction of shunting is left to right
 Minor right to left shunting can be observed
 Shunt directions do not remain static; volume overload eventually leads to right ventricular dilation, increasing pulmonary vasculature pressures, and Eisenmenger's syndrome

decision to provide antibiotic prophylaxis, especially regarding the second criterion.

See Table 2 for a summary of the flow dynamics and long-term sequelae for ASDs.

VENTRICULAR SEPTAL DEFECTS

VSDs are similar to ASDs in many ways, but they differ in a few significant areas, including disease progression and prognosis. VSDs are by far the most common CHD, found in 50% of all CHDs, 20% as an isolated defect. They show an incidence of 1.56 to 53.2 per 1000 live births and a prevalence of 0.3 per 1000 in the general population. Seventy-five percent of VSDs are of the perimembranous type, but there are also muscular, infundibular, and inlet types, with most involving more than one area. A large number of small muscular VSDs will spontaneously close.⁷

SHUNT DYNAMICS

Similar to ASDs, VSDs show a bidirectional flow pattern. There is minor right to left shunting during isovolumetric diastole, then the major component of the shunting occurs during isovolumetric systole from left to right.⁸ The flow dynamics are more straightforward regarding the shunt, as there are fewer chambers of the heart that are involved during the time that shunt is active. However, the subsequent result of this shunt can be more significant and carry greater morbidity.

Given equal shunt volumes, VSDs generate greater pressures in the pulmonary artery compared to ASDs. Because the shunt volume in an ASD can be compensated for by left and right atrial dilation, the pressure transmission is usually tempered. In a VSD, the shunt is flowing during ventricular systole and directly to the pulmonary artery. Consequently, the pulmonary vasculature is taxed more quickly and to a greater extent, which ultimately can lead to pulmonary hypertension.⁹ As in the case with either ASD or VSD, pulmonary hypertension that exceeds the aortic outflow pressure will result in a shift to a predominantly right to left shunt. Though possible in both ASDs and VSDs, it is more likely to be observed in VSDs, sometimes as early as 2

Table 3. Ventricular Septal Defect Summary*

Major direction of shunting is left to right
Minor right to left shunting can be observed
Faster developing and more pronounced effects on pulmonary vasculature compared to ASD
Shunt reversal and Eisenmenger's syndrome can occur by age 2

* ASD indicates atrial septal defect.

years of age. This reversal of shunt direction—known as Eisenmenger's syndrome—leads to desaturation or hypoxia, cyanosis, and compensatory erythrocytosis.⁷

Anesthetic management of a VSD resembles that of an ASD. The same directional forces that dictate ASD shunt dynamics will also affect how a VSD shunt behaves. Maintenance of normovolemia (avoidance of hypovolemia) and normal SVR and preventing acute increases in PVR, such as might occur during relative hypoxia, are the hallmarks of management. These strategies are in place to prevent a hypoxic shunt from occurring.

PROGNOSIS AND TREATMENT

Treatment for VSDs also includes transcatheter and surgical approaches. The Food and Drug Administration–approved transcatheter devices are the Cardio-SEAL and both the Perimembranous and Muscular Amplatzer VSD Occluders. Surgical repair options include direct stitching or the use of grafting material, similar to that of the ASD.

Up to 5% of patients with repaired VSDs can experience some form of conduction blockade, such as complete AV blockade requiring pacemaker implantation.¹⁰ Practitioners should be aware of the possible impacts this may have on anesthetic management. Late closures should also raise suspicion of ventricular function impairment and other longer-term sequelae that persist past invasive repair.

See Table 3 for the summary of flow dynamics and long-term sequelae of VSDs.

Understanding the details of dynamic flow patterns in ASDs and VSDs allows the practitioner to appropriately address the perioperative management of patients with known defects. In the setting of unknown defects,

knowledge of these flow patterns can help broaden the differential diagnosis and assist in management of perioperative crises. Delayed awakening from air thrombus strokes and persistent hypoxia in the setting of adequate ventilation are 2 rare events that can be attributed to shunt dynamics in ASDs and VSDs.

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CONTINUING EDUCATION QUESTIONS

This continuing education (CE) program is designed for dentists who desire to advance their understanding of pain and anxiety control in clinical practice. After reading the designated article, the participant should be able to evaluate and utilize the information appropriately in providing patient care.

The American Dental Society of Anesthesiology (ADSA) is accredited by the American Dental Association and Academy of General Dentistry to sponsor CE for dentists and will award 1 CE credit for each article completed. You must answer 3 of the 4 questions correctly to receive credit. Articles are eligible for CE credit for one year following the date of publication.

Submit your answers online at www.adsahome.org. Click on “On Demand CE.”

1. Which type of atrial septal defect (ASD) is the most common?
 - A. coronary sinus
 - B. ostium primum
 - C. ostium secundum
 - D. sinus venosus
2. Which of the following scenarios is the most ideal in anesthetic management for ASD?
 - A. increased pulmonary vascular resistance (PVR) and decreased systemic vascular resistance (SVR)
 - B. increased PVR and increased SVR
 - C. normal PVR and decreased SVR
 - D. normal PVR and increased SVR
3. Which of the following is true regarding the differences between ASDs and ventricular septal defects (VSDs)?
 - A. ASDs typically demonstrate minor right to left shunting, VSDs do not
 - B. ASDs can be repaired nonsurgically using transcatheter devices, VSDs cannot
 - C. ASDs are less common than VSDs
 - D. ASDs are more likely than VSDs to progress to Eisenmenger's syndrome, especially when left untreated
4. Which of the following drugs should be used with caution in patients with ASDs and VSDs?
 - A. diazepam
 - B. fentanyl
 - C. ketamine
 - D. ondansetron