# **Ventricular Septal Defects**

Markus K. Heinemann

# 14.1 Classification and Localizations – 464

- 14.1.1 Defect of the Inlet Septum (Inlet VSD) 465
- 14.1.2 Muscular VSD 465
- 14.1.3 Conoventricular VSD 465
- 14.1.4 Conus VSD 465
- 14.2 Natural Course and Indications for Operation 465

# 14.3 Cardiac Access – 466

- 14.3.1 Transatrial Access 467
- 14.3.2 Transventricular Access 467
- 14.3.3 Transvalvular Access 467

# 14.4 Closure Techniques – 467

- 14.4.1 Conoventricular VSD 468
- 14.4.2 Muscular VSD 470
- 14.4.3 Inlet VSD 470
- 14.4.4 Conus VSD 470

# 14.5 Complications – 471

# 14.6 Special Forms of Operative Treatment – 471

- 14.6.1 Pulmonary Artery Banding 471
- 14.6.2 VSD with Secondary Aortic Valve Insufficiency 472
- 14.6.3 VSD and Coarctation of the Aorta 473
- 14.6.4 «Swiss-Cheese-Type» VSD 475
- 14.6.5 VSD and Aneurysm of a Valsalva Sinus 475
- 14.6.6 Catheter Interventional Techniques 475
- 14.6.7 Pushing the Limits 477
- 14.7 Perioperative Aspects 477

# References – 478

G. Ziemer, A. Haverich (eds.), Cardiac Surgery, DOI 10.1007/978-3-662-52672-9\_14

# 14.1 Classification and Localizations

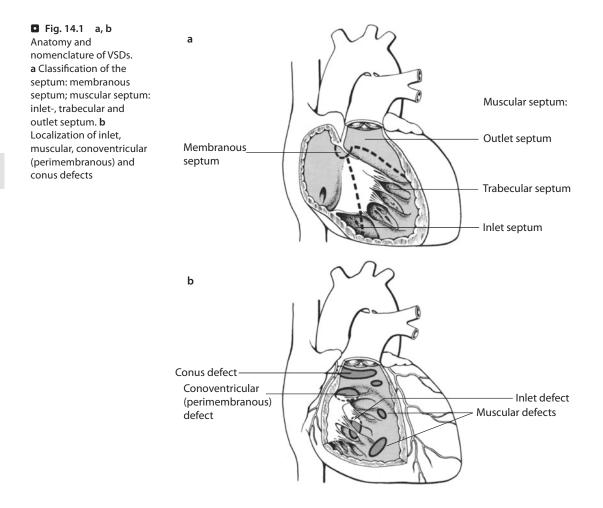
A ventricular septal defect (VSD) is defined as a communication between the left and the right ventricular chamber in the area of the interventricular septum. It may be isolated or can occur in association with other cardiac or vascular malformations. This chapter deals primarily with the treatment of isolated VSD which accounts for 20–25 % of all congenital heart disease.

As is so often encountered in terminology, several classifications for VSD exist, each with its own particular emphasis reflected in the literature. In this chapter, the recommendation by van Praagh (van Praagh et al. 1989) is followed, but the classifications according to Kirklin (Kirklin et al. 1957) and Anderson and Becker (Becker and Anderson 1982) are also common usage. All classifications share the description of the localization of the defect orientating themselves at features of the right ventricle, as surgical access is usually from the right side ( $\blacksquare$  Fig. 14.1).

During the complex embryonic cardiogenesis (Goor and Lillehei 1975), normally a complete separation of both ventricular chambers is formed by the development of a partition consisting mainly of muscle (muscular septum) and a small membranous part (membranous septum). The latter is positioned toward the outflow tract with its cranial portion connecting to the septum of the former truncus arteriosus which is separating the two great arteries (conus septum or conotruncal septum).

The right ventricle is subdivided into an inlet, a trabeculated, and an outlet part. Following the blood flow one would encounter the different defects in the following order and frequency:

- Inlet VSD (6%)
- Muscular VSD (10%)
- Conoventricular VSD (80%)
- Conus VSD (4%)



## 14.1.1 Defect of the Inlet Septum (Inlet VSD)

This defect lies directly behind the septal leaflet of the tricuspid valve and corresponds to the ventricular part of an atrioventricular septal defect or AV canal. Accordingly, it is often found in association with trisomy 21. The fact that some also call it an «AV canal-type VSD» is the cause of an ongoing morphological debate between the various schools of cardiac pathology (Spicer et al. 2013). Upstream its border is the tricuspid valve anulus. The conduction system usually lies rather superficial within the caudad muscular border. Chordae and papillary muscles of the sometimes atypically configured right-sided atrioventricular valve may obscure the overview from the right atrium. This can necessitate the detachment of a leaflet for secure closure (see below, ► Sect. 14.3.1).

# 14.1.2 Muscular VSD

Such a VSD is circumferentially bordered by muscle. It can occur in any area of the trabeculated part of the right ventricle. The view from the right ventricle with its crossing trabeculations can simulate multiple defects. Hence, the true arrangement can be judged much better from the smooth left ventricular side. Defects located anteriorly toward the apex are also known as «apical» VSDs. They are usually multiple and reflect an incomplete differentiation of the muscular septum. Spontaneous closure within the first months of life is therefore seen frequently.

The primarily netlike structure of the interventricular partition with numerous communications explains the commonly encountered multitude of defects. Whereas this is often only an illusion in the midmuscular part, apical VSDs are multiple as a rule. A prominent disruption of the myocardial differentiation of the septum is colloquially called a «Swiss-cheese-type.» Apart from a conoventricular VSD a separate muscular defect may coexist, the relevance of which often only becomes evident if it is overlooked or deliberately not closed at surgery for the leading defect.

# 14.1.3 Conoventricular VSD

This defect is situated between the true ventricular and the conus septum. The conus septum may deviate anteriorly (RVOT narrowing, embryogenesis of tetralogy of Fallot) or rarely posteriorly (LVOT narrowing in interrupted aortic arch). An isolated conoventricular VSD is the result of an insufficient development of the membranous septum and is therefore often also called a «membranous» VSD. Because of the usual extension into the muscular part, «perimembranous VSD» may be the better term. This localization is the most frequent one by far (>80%).

There is a close relation to the tricuspid valve in the area of the commissure between the septal and the anterosuperior leaflet. The conduction system lies within the posteroinferior border. If the membranous septum is missing completely, the superior border of the VSD reaches the base of the right/ noncoronary cusps of the aortic valve. Various extensions posteroinferiorly (muscular septum, inlet portion) or anterosuperiorly (conus septum, Fallot type) can occur and may impede orientation.

If there is only a small perforation of the membranous septum, the defect will be hemodynamically insignificant. The development of a sometimes quite impressive bulge of the membranous part may occasionally lead to subpulmonary obstruction. The impression of such a so-called aneurysm VSD is, however, more often created by an associated dysplasia of the tricuspid valve leaflet next to the defect which may be attached to its crest with several chordae. Septal and anteroseptal leaflet tissue can form a tunnel and direct blood from the left ventricle directly into the right atrium (Gerbode defect; Gerbode et al. 1958).

#### 14.1.4 Conus VSD

This VSD is characterized by an incomplete closure of the conus septum between the two great arteries and is sometimes also called «subpulmonary» or «outlet» VSD. With the upper rim being outlined by the pulmonary valve, its caudad border is muscular. The conduction system and the tricuspid valve are far away. For embryological reasons, this defect is frequently seen with a common arterial trunk, the isolated form being very rare.

## 14.2 Natural Course and Indications for Operation

The natural course depends on the size and localization of the VSD. According to the shunted volume and the pulmonary vascular congestion resulting therefrom, restrictive and nonrestrictive defects are differentiated. In the latter, right ventricular systolic pressure equals the left ventricular one. The degree of pulmonary vascular congestion is dependent on the variable of pulmonary vascular resistance (Rp). As this is physiologically increased early postnatally, the true hemodynamic significance often becomes apparent only with its decrease over several weeks of life. If the relation between pulmonary blood flow and systemic blood flow (Qp:Qs) is less than 1.5:1, a VSD is considered small and restrictive. Nonrestrictive VSDs often show a Qp:Qs ratio of more than 3:1. A left-to-right shunt of such dimensions soon causes cardiac failure, which in the infant manifests itself with tachypnea, sweating and feeding difficulties, leading to failure to thrive. This development determines the timing of the operation. Closure of a VSD should be performed before manifestation of cardiac failure, independent of age.

Chronic pulmonary arterial hypertension in untreated VSD stimulates remodeling of the pulmonary vasculature, which eventually becomes irreversible and causes a massive increase in pulmonary vascular resistance. When the relation of the resistances Rp:Rs (Rs = systemic vascular resistance) becomes greater than 1.0, the shunt flow is reversed to right to left, causing cyanosis. This phenomenon is known as «Eisenmenger's syndrome.» Because of the destruction of the pulmonary vasculature, the only «therapeutic» option may be combined heart-lung transplantation (HLTx). It should be mentioned that one of the first two successful HLTx in the world, performed by Bruce Reitz in 1981, was done for that very reason. The natural course of smaller defects is variable. Apical muscular defects show a tendency to close during ongoing myocardial differentiation within the first year of life. In the membranous septum such a spontaneous closure is often pretended by an increasing prolapse of accessory connective tissue («aneurysm VSD»). The development of a subaortic ridge at the left ventricular surface of the lower rim of an untreated conoventricular/ perimembranous VSD is seen in about 6% of cases. Upon closing the VSD, this must be resected to avoid leaving behind a subaortic stenosis which may become more prominent over time (Eroglu et al. 2003; Kidd et al. 1993; Kleinman et al. 2007).

Leaving a VSD with a load of the right ventricle untreated for years may cause the development of a trabecular hypertrophy up to an infundibular outflow tract stenosis («double-chambered right ventricle,» DCRV). During closure of the VSD this must be additionally corrected. A DCRV without VSD is extremely rare.

A direct relation of a high-positioned VSD with the aortic valve may cause prolapse of the right coronary, sometimes the non-coronary cusp into the VSD. The creation of a low-pressure zone at the border of an accelerated fluid is known as the «Venturi effect.» The acceleration of blood flow at the rim of a small VSD causes such traction on the affected valve cusps that an aortic valve insufficiency can result. The dynamics of this phenomenon have been repeatedly studied. The latest point in time to close such a high VSD is the appearance of valve regurgitation (see below, ► Sects. 14.6.2; Jian-Jun et al. 2006; Kostolny et al. 2006; Saleeb et al. 2007; Tatsuno et al. 1973; Tomita et al. 2004). Interestingly, such defects are more frequently encountered in patients of East Asian origin, which may be regarded as a hint to genetic disposition of the localization of VSDs.

Even a very small VSD must be feared to increase the risk of endocarditis because of the turbulence it creates on the endothelial surface. This is a weighty argument in favor of closure of defects which are barely or not hemodynamically significant (Backer et al. 1993).

It should be mentioned that a VSD may constitute a cardiac malformation in the context of a more complex genetic syndrome such as chromosome 22q11 deletion. Genetic investigations of these causes may help to further explain the mechanisms of cardiogenesis.

#### 14.3 Cardiac Access

Median sternotomy is considered standard access for VSD closure. With the constant development of so-called minimally invasive techniques reports about alternative incisions such as partial inferior sternotomy, right anterolateral thoracotomy, or axillary thoracotomy are increasing (Kadner et al. 2006; Mavroudis et al. 2005). These do compromise visualization, however, and their use should be limited to groups with a particular experience and to older patients. They are favored for hybrid approaches (see below, ► Sect. 14.6.6; Mo et al. 2011; Schreiber et al. 2012; Xing et al. 2011). For open repair the mainly cosmetically advantageous considerations must by no means compromise established security standards, e.g., direct suture instead of patch closure, or induced ventricular fibrillation instead of aortic cross clamping and cardioplegic arrest. Cannulation of a child's femoral or iliacal vessels remains highly controversial because of its long-term side effects.

When using a standard median sternotomy, arterial cannulation is via the distal ascending aorta, the venous one bicaval with snaring of both veins. This is followed by the induction of cardioplegic arrest under aortic cross clamping.

Depending upon its localization, the VSD is then approached through a cardiac incision.

#### 14.3.1 Transatrial Access

For transatrial access the right atrium is incised longitudinally, starting from the base of the right auricle. This can be extended if necessitated by poor exposure. A left atrial vent is placed through the foramen ovale or a small stab incision in the fossa ovalis. The anterosuperior leaflet of the tricuspid valve is then retracted with a blunt retractor. It may be helpful to additionally elevate the septal leaflet, too. This can be achieved with stay sutures. Through the tricuspid valve orifice one can then see the right ventricular aspect of the interventricular septum and all conoventricular (perimembranous) defects. The superior rim of the VSD may be difficult to visualize nevertheless. Sometimes it is then appropriate to partially detach the anterosuperior tricuspid leaflet. Confusing anatomy of chordae or papillary muscles sometimes need detaching of the septal leaflet. The leaflet in question is incised close and parallel to its fibrous anulus and reattached after VSD closure with a fine monofilament suture. A limited radial incision has also been described to provide very good exposure (Russell et al. 2011).

#### 14.3.2 Transventricular Access

A routine approach through the right ventricle must be regarded as historical. In case of a localization in or extension into the conus septum, a limited infundibulotomy may facilitate exposure. Care must be taken to spare the coronary artery branches, which may be quite prominent in the conal area. Apart from that, a right ventriculotomy is very rarely used for closure of anterior muscular VSDs. A conus VSD, however, can be approached through an oblique infundibulotomy.

In apical VSDs in the trabecular portion («Swiss-cheese-type»; see below,  $\blacktriangleright$  Sect. 14.6.4) a left ventriculotomy offers the theoretical advantage of unobstructed overview due to the lack of trabeculations. If done at all, the incision should be made at the very apex and to the right of the left anterior descending coronary artery. Closure of the ventricle usually requires fortification with buttressed sutures (e.g., with Teflon felt strips). Severe left ventricular dysfunction and arrhythmias were observed during long-term follow-up, making this approach almost obsolete (Hanna et al. 1991). If it comes into consideration at all, an alternative option should be discussed (see below,  $\blacktriangleright$  Sect. 14.6.6).

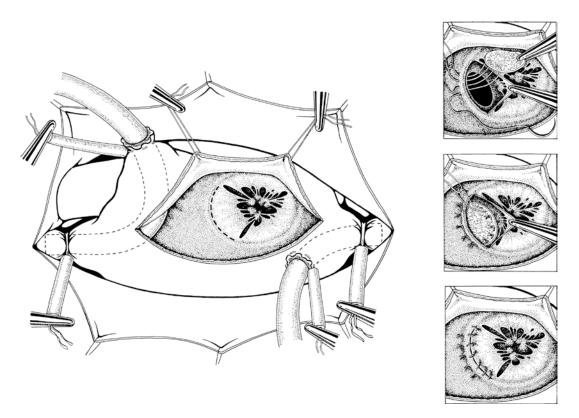
Access to defects close to the apex is often possible through an apical right ventricular incision. Obstructing trabeculations, however, must then be severed. Firm anchoring of the patch at the cranial rim may prove difficult (Myhre et al. 2004).

# 14.3.3 Transvalvular Access

Conotruncal defects immediately adjacent to the pulmonary or aortic valve can be reached through the respective valve orifice. Visualization of the interventricular septum remains limited. This approach is therefore reserved for smaller or recurrent defects in this area. In associated aortic valve insufficiency (see below, ► Sect. 14.6.2) or an aneurysm of the sinus of Valsalva (see below, ► Sect. 14.6.5), an aortotomy is obligatory and enables the precise exposure of the superior rim of any defect bordering the aortic valve.

## 14.4 Closure Techniques

The techniques for VSD closure described in the following paragraphs exclusively imply the use of patch material (Dacron, polytetrafluorethylene [PTFE], autologous or xeno-pericardium, tissueengineered matrix). The suture techniques used are urgently recommended. Individual expertise, however, allows for the routine use of alternative techniques: e.g., «always» running sutures, or «always» interrupted sutures with or without but-tressing material (Teflon felt, pericardium).

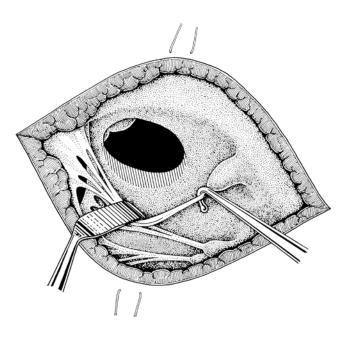


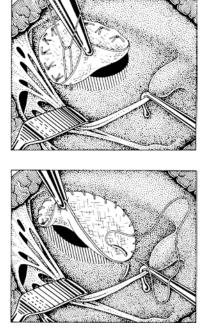
**Fig. 14.2** Transatrial closure of a VSD. Exposure of the VSD through the right atrium and the tricuspid valve. Shown is the partial detachment of the septal tricuspid valve leaflet at the anulus to expose the superior rim of the defect. VSD sutures in U-stitch technique

## 14.4.1 Conoventricular VSD

Almost all of these defects can be closed through a transatrial/transtricuspid approach. Initially, the upper rim is often obscured. Before starting any closure, however, the complete anatomy must be visualized unequivocally. It is also to be determined if the rim of the defect reaches the anulus of the tricuspid valve directly, or if a muscular ridge is present. If borderline structures remain obscured by valve tissue, detachment of the respective tricuspid valve leaflet directly above its anulus may be required.

For anchoring of a patch, the use of separate, double-armed, braided U stitches, buttressed with small Teflon pledgets, is generally recommended (**©** Fig. 14.2). It is advantageous to fix the first sutures on the anteroinferior aspect of the muscular septum. Armed with small rubber-shod clamps and draped on a hole towel for clear view, they facilitate stepwise exposure if put under gentle traction—similar to the implantation technique for a mitral valve prosthesis. Advancement is made in a clockwise direction first, caudally taking great care that the pledgets rest directly on the septum and that no chordae or papillary muscles are caught by the sutures. At the inferoposterior aspect the conduction system is approached ( Fig. 14.3). Here the sutures are to be applied stringently on the right ventricular aspect, somewhat detached from the true rim of the VSD. Adjacent to the tricuspid valve, especially if there is only a small ridge of tissue, one may need to pass some sutures from the atrium and through the tricuspid valve anulus to the ventricular side. Thus, the pledgets will be placed atrially. As soon as the most posterior point is reached, it is advised to return to the anterior aspect. Slight traction on the sutures already in place will expose the superior rim. Advancement, now counterclockwise, is in identical fashion. In the area of the outflow tract, generous portions of muscle can be used for secure anchoring. Reaching the aortic valve the available ridge can be distressingly narrow, probably the reason for the frequency of patch dehiscences in this area. This can be overcome by Chapter 14 · Ventricular Septal Defects





**Fig. 14.3** Right ventricular aspect of VSD closure. Opened right ventricle. The hatched area marks the part of the septum bearing the conduction system. Here, sutures must be placed stringently on the right ventricular aspect and away from the rim. Illustrated suture techniques are U-stitches and continuous suture. A combination is possible

placing several, usually only one to three, unbuttressed stitches in a longitudinal fashion along the rim. Under very delicate circumstances and in direct neighborhood of valve tissue, the use of fine monofilament material is expedient, enabling better guidance. It is helpful to fill the aortic root intermittently with cardioplegia. This problematic region is then overcome and one reaches the posterior aspect close to the tricuspid valve again, which is dealt with as described above. Usually, for closure of a nonrestrictive infant VSD, 10–15 sutures are needed to guarantee secure anchoring of the patch, at the same time avoiding compromising neighboring structures.

Woven Dacron is a well-proven patch material, being both tear-proof and flexible. PTFE is preferred by some. Autologous pericardium (usually impregnated with 0.6% glutaraldehyde for 10 min) and xenopericardium are controversial because of lower tear resistance. One should bear in mind that after closure an enormous pressure load is inflicted on the patch and that the muscular part of the circumference is contracting with every systole, putting the whole suture line in this area at risk. More recently, a tissue-engineered matrix of porcine intestine (CorMatrix<sup>®</sup>) came into use also. All stitches are now placed through the patch in their original order. The patch is then carefully lowered onto the septal defect where it comes to rest on its right ventricular aspect like a lid. It should therefore be tailored a bit larger than the actual defect size. When lowering the patch, great care must be taken again that it does not obstruct any chordae or parts of the tricuspid valve. Retracting these delicate structures with fine nerve hooks can be helpful. The sutures should only be tied after secure placement of the whole circumference of the patch.

If a tricuspid valve leaflet was detached for exposure's sake, it is then reattached, ideally with a fine monofilament running suture. Tricuspid valve competence is tested in any case by instilling fluid into the right ventricle. If necessary, reconstructive measures to alleviate valve incompetence are needed (adaptation of commissures, anuloplasty, but also replacement of awkward VSD patch sutures).

The left atrial vent catheter is now removed and the interatrial septum closed. We use absorbable monofilament material for closure of the right atriotomy in children. Before removing the aortic cross clamp, the left heart is meticulously de-aired through the cardioplegia site. For immediate quality control of the operative result and especially in the presence of any difficult anatomical circumstances, an intraoperative transesophageal echocardiography is strongly recommended. Moreover, pulmonary arterial oxygen saturations can be measured and compared to SVC saturations. A step-up of more than 5% in absolute saturation is suspicious for a residual VSD.

Direct closure of a VSD with separate buttressed U stitches is vehemently advised against because of the high risk of dehiscence and distortion of neighboring structures. This is also and particularly true for small defects. Very rare indications for direct closure (concomitant very small VSD with fibrous, scarred borders) may exist.

#### 14.4.2 Muscular VSD

Isolated midmuscular, inferior, or posterior defects can commonly also be reached through a right atriotomy. The more they are located in the trabecular part of the right ventricle, the more overview is obscured by these trabeculae. This may evoke the impression of multiple defects. Partial separation of muscle bundles may facilitate secure anchoring of a patch. The whole border of such a VSD is muscular, including that toward the tricuspid valve. Direct closure is therefore inappropriate.

If a muscular VSD is in close relation to a separate conoventricular or inlet defect, the conduction bundle often runs through the separating muscular bridge. Closure should be achieved by placing one large common patch covering both communications and avoiding sutures in the bridging area.

VSDs located in the very anterior region or apical ones may only be approachable through a ventriculotomy, which in case of an anterior VSD may be limited to an oblique infundibulotomy. Even if a left ventriculotomy enables good visualization, this access is rarely ever used due to its negative longterm sequelae. This has become possible because of the development of new and alternative closure technologies (see below, ► Sect. 14.6.6). The apical incision of the right ventricle has come somewhat back into fashion, especially where these new methods are not available (Myhre et al. 2004).

In very rare large anterior defects, which can be reached through an infundibulotomy, utmost care must be taken not to injure the left anterior descending coronary artery when placing the anterior stitches.

# 14.4.3 Inlet VSD

These defects can be easily reached through the tricuspid valve orifice because of their immediate proximity to it. By definition, they reach the valve anulus without an additional ridge of tissue. All sutures in this area must therefore be placed from the right atrial aspect. In the presence of multiple atypical chordae, it may be advantageous to use a single running stitch for the circumference bordering the valve. This can be woven in between the chordae and the usually short and plump papillary muscles like a mattress suture. The risk of dehiscence is low because the border of the defect in this area consists of firm connective tissue.

If the conduction system lies to the inferoposterior border, several separate U stitches are put some millimeters away from the edge and strictly on the right ventricular aspect. Thus, usually a combined running and single stitch closure technique is the result. The debate where the conduction system is most likely located in VSDs of the inlet septum is ongoing, depending on the pathological classification (Spicer et al. 2013).

#### 14.4.4 Conus VSD

This type of defect in a subpulmonary position is usually associated with more complex congenital heart disease such as common arterial trunk. If isolated, it can be reached through a limited infundibulotomy or through the pulmonary valve itself.

A limited longitudinal ventriculotomy sparing right coronary conal branches offers good exposure of the conus septum that forms the backwall of the funnel just opened. The superior aspect of these defects commonly reaches directly onto a narrow fibrous ridge between the pulmonary valve anteriorly and the aortic valve posteriorly. If this tissue appears too fragile for secure anchoring of the patch, as is often the case, the sutures must be placed from the respective sinus of the pulmonary valve. Again, separate buttressed U stitches are used. For better orientation and precise placement of the sutures, intermittent filling of the aortic root with small doses of cardioplegia is advised. At the caudad end, a continuous running suture is usually appropriate. As only the conus septum is involved, the conduction system is far away and generous portions of muscle can be used.

In smaller, semilunar defects or insufficient tissue between aorta and pulmonary artery, the latter is incised transversely above the commissures. Then the sutures for the superior aspect are placed from the sinus. The lower circumference can be sutured continuously through the valve orifice. Should the defect extend deep into the ventricle, exposure is compromised.

While oblique infundibulotomies can be closed directly, longitudinal infundibulotomies should be closed with a patch to avoid RVOT obstruction.

# 14.5 Complications

The anatomy of the *conoventricular/perimembranous VSD* bears three danger zones, the injury of which leads to a respective specific complication:

- The upper border of the defect reaches the aortic valve in many instances. The valve must be visualized through the VSD during closure. Any distortion in this area may lead to postoperative valve insufficiency (Chiu et al. 2007). For visual inspection of valve competence, cardioplegia can be repeatedly administered into the aortic root during surgery.
- The lower border of the defect in the posterior direction toward the tricuspid valve hides the conduction system. If hurt by stitches, a block of the atrioventricular conduction will result, which then is often permanent and complete (third degree heart block). If complete heart block is seen during reperfusion and depending upon the anatomy, one may consider replacing several stitches during a second time of ischemia. However, this will not guarantee complete remission (Andersen et al. 2006). If complete heart block persists early postoperatively, we wait for 10-14 days before we implant a permanent pacemaker system, as recovery of stable sinus rhythm is possible within this time frame. For additional safety throughout this time, we always suture a second set of temporary pacemaker wires on the right ventricle when a higher degree of heart block is present at the time of chest closure.
- If several stitches were placed from the right atrium through the tricuspid valve anulus or if a valve leaflet had to be detached for better exposure, significant tricuspid valve

insufficiency may result. Before closing the right atrium, the right ventricle must be filled with fluid for visual inspection, cross clamping the pulmonary artery if necessary.

Closure of *apically located muscular VSDs* via a left ventriculotomy will lead to an impairment of left ventricular function, especially in the long term. Late occurrence of life-threatening ventricular arrhythmias has also been observed (Hanna et al. 1991). For these reasons, usage of this approach should be made only very restrictively.

In *«Swiss-cheese-type» VSD*, smaller communications may remain or be overlooked, the hemodynamic relevance of which is hard to judge early postoperatively.

Defects located in the *inlet part of the septum*, directly adjacent to the tricuspid valve, often offer little space for safe anchoring of the sutures in the area of the conduction system. An increased risk of complete heart block has to be expected.

Closure of *conotruncal defects* through the pulmonary or aortic valve may lead to an insufficiency of the respective semilunar valve. Inappropriate exposure of the lower margin in larger defects leads to an increased incidence of residual defects in this area.

If a residual defect is known, regular follow-up is needed to judge its hemodynamic relevance (Dodge-Khatami et al. 2007). Intraoperative transesophageal echocardiography is able to detect most valve insufficiencies and residual defects and may thus decisively influence the further course of action, e.g., immediate revision. Significant septal defects can also be detected or excluded by measuring oxygen saturation in the pulmonary artery. A defect needing closure usually leads to saturations above 80% under ventilation with room air. As said above, when in doubt, a comparison with the SVC oxygen saturation is advisable.

#### 14.6 Special Forms of Operative Treatment

#### 14.6.1 **Pulmonary Artery Banding**

The first operative closure of a VSD was performed by Lillehei under «cross circulation» on March 26, 1954 (Lillehei et al. 1955). In the preceding years, there had been several attempts of palliation by lowering pulmonary blood flow, narrowing the pulmonary artery with the aid of a circumferential textile band (Muller and Dammann 1952). This had been based on the observation that moderate pulmonary stenosis would positively influence the prognosis of patients with single ventricle. The banding procedure enabled postponing correction into an age beyond infancy and had become quite common practice. In the current era of surgical treatment of VSD, pulmonary artery banding has almost completely lost its former role, being only considered in the presence of absolute contraindications for extracorporeal circulation or in pronounced forms of a «Swiss-cheese-type» septum (see below, ► Sect. 14.6.4). The technique still has its merits in the stepwise palliation of univentricular circulations with pulmonary vascular congestion in the newborn and is therefore described here.

Contrary to the historical technique which was done through a left posterolateral thoracotomy, access today is via a median sternotomy. Besides offering the advantage of equal ventilation and perfusion of both lungs, this route offers far better exposure, the possibility to establish cardiopulmonary bypass in an emergency, as well as the cosmetic aspect of just one scar for all subsequent operations. In addition, in cyanotic univentricular circulations, the development of post-thoracotomy chest wall-to-pulmonary collaterals complicating later Fontan type surgery is avoided.

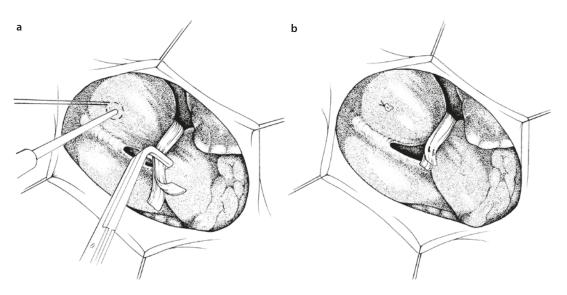
After displacement or partial resection of the thymus, the pericardium is incised over the great vessels. The connective tissue between the aorta and the often rather short main pulmonary artery is sparingly dissected over the width of the intended band. Instead of the conventional braided textile bands which may migrate through the vessel wall over time and lead to considerable adhesions in any case, the use of smooth, inert silicone, ideally reinforced with a Dacron mesh, or a PTFE band is recommended. Starting from the historical Trusler's formula (Trusler and Mustard 1972), the initial length of the circumference to be marked on the band should amount to 20 mm + 1 mm/kg body weight (e.g., 24 mm in a 4 kg baby). After marking, the band is pulled around the pulmonary artery and fixed according to the marked length. Depending upon the hemodynamic situation, it can then be loosened or tightened further. According to Poiseuille's law, the radius of a tube influences the flow through it in the fourth power. Thus, minimal changes can lead to sometimes dramatic effects. Before the band is eventually permanently fixed, the circulation must be given some time to adapt to the new resistances (■ Fig. 14.4). Usually we see a minimum increase of 10–15 mmHg systolic arterial pressure and a reduction of the heart rate by 10%. Pulmonary artery pressure does not need to be measured routinely. In a univentricular circulation, an arterial saturation between 75 and 80% (under ventilation with room air and with an open chest!) and a decrease of pulmonary arterial pressure to about one third systemic is considered the benchmark. When palliating a biventricular system, the arterial saturation should not drop below 85%, but rather be 90%.

To prevent distal migration of the band with the danger of compromising the bifurcation and inducing asymmetrical pulmonary blood flow, fixation sutures should be applied to the pulmonary root. In biventricular circulations the pulmonary valve must not be jeopardized in any manner. If the pulmonary trunk is very short, the band can be excised concavely at the underside of the right-angled take-off of the right pulmonary artery to prevent stenosis to the right side.

As the principle of palliation means transfer from one pathological state to another, which is presumably better tolerated, the time span with a banded pulmonary artery should be kept to a minimum. This is especially true for eventually curable congenital heart disease such as VSD or atrioventricular septal defect. The persistence of right-sided systemic pressures causes hypertrophy of the right ventricular muscle-up to the level of a hemodynamically relevant infundibular stenosis persisting after debanding, causing additional problems. With the growth of the child, the banding becomes constantly more effective and may induce critical spells of arterial desaturation. This should be prevented by early de-banding not later than 6 months post banding.

## 14.6.2 VSD with Secondary Aortic Valve Insufficiency

The increasing prolapse of an aortic valve cusp adjacent to a VSD can lead to distortion of the valve with consecutive regurgitation. Small conoventricular defects high in the septum or juxtaarterial conus defects are at particular risk. Usually it is the right coronary cusp which is closely



**Fig. 14.4** a, b Pulmonary artery banding. a Provisional adjustment of the band at the pulmonary artery by clamping. Direct distal pressure measurement. b Permanent fixation of the band with single sutures

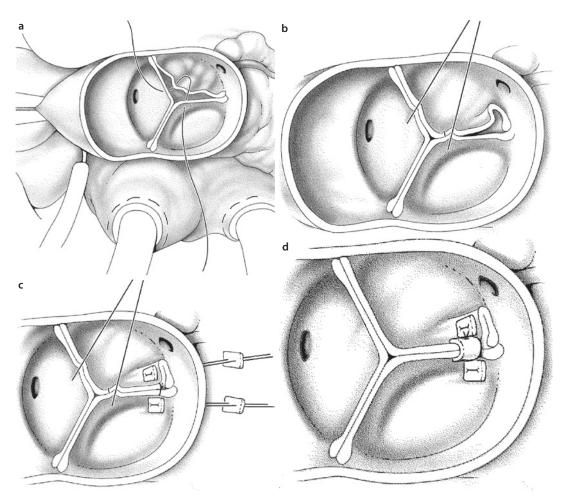
related to the VSD, sometimes the non-coronary one or both (Tomita et al. 2005). A lack of support of the afflicted area as well as the Venturi effect are considered mechanisms contributing to progressive valvular insufficiency (Tatsuno et al. 1973; Tweddell et al. 2006; see above  $\blacktriangleright$  Sect. 14.2). Closure of the defect before the development of valvular insufficiency or as long as it is only minor has the best prognosis (Jian-Jun et al. 2006; Kostolny et al. 2006; Saleeb et al. 2007; Tomita et al. 2004). In these cases closure of the VSD is often sufficient with the patch providing the necessary support. If the valve has already undergone morphological alterations, extension of the operation becomes necessary.

Reconstruction of the aortic valve according to the technique described by Trusler has very good long-term results (Trusler et al. 1973, 1992). After a transverse aortotomy above the sinotubular junction, the aortic valve is inspected. One usually finds an elongation of the cusp prolapsing into the defect. To correctly judge the extent of the asymmetry, it is recommended to pass a fine monofilament suture through the noduli Arantii to elevate the valve («Frater stitch»; Frater 1967). The afflicted cusp will crimp along its pathologically elongated part close to the commissure. A buttressed suture then fixes the resulting fold transmurally to the neighboring aortic wall, thus reestablishing the symmetry of the free cusp edges (**Fig. 14.5**). The central marking suture is then removed. Instillation of cardioplegia into the aortic root gives a first impression of valve patency. An intraoperative transesophageal echocardiography will exactly define the success of the reconstruction. In the presence of additional congenital valve asymmetries, e.g., a bicuspid valve or advanced changes, prognosis is obviously limited.

# 14.6.3 VSD and Coarctation of the Aorta

A VSD can be a defined, fixed part of a complex malformation, e.g. in tetralogy of Fallot or common arterial trunk. Often it is only loosely associated with a second malformation without this constituting a direct syndromal relationship. The most common malformation encountered in the presence of a VSD is coarctation of the aorta (see ▶ chapter «Congenital Anomalies of the Great Vessels», Sect. 21.2.1.1).

Whereas concomitant correction during a single procedure is well accepted for complex heart disease (Heinemann et al. 1990), the strategy for optimal treatment of VSD with aortic coarctation is still subject of debate (Gaynor 2003). A two-staged approach is the historically established one, in which the coarctation is corrected first through a left lateral thoracotomy. Then the VSD is closed transsternally in a second operation. In the presence of large defects, this



**Fig. 14.5 a**-**d** Trusler's plasty in aortic valve insufficiency. Transverse incision of the aortic root. **a** «Frater stitch» through the noduli Arantii. The right coronary cusp is elongated. **b** Tension on the «Frater stitch» creates crimping of the elongated cusp into a fold close to the aortic wall. **c** Right and non-coronary cusp are attached to each other. Then the fold is fixed transmurally with pledgeted sutures to gather the elongation. **d** Safety suture between the two cusps, creating a cover

strategy can be complemented with pulmonary artery banding during coarctectomy to gain some time until the second procedure. Disadvantages are the necessity of two different incisions, two anesthesias, and a longer or repeated hospital stay in combination with a state of palliation.

Technical advances have led to the increased endeavor of concomitant correction of an isolated VSD and aortic coarctation through a sternotomy in analogy to the strategy in more complex heart disease. The aortic isthmus is then approached during a short phase of deep hypothermia and, when necessary, circulatory arrest. During the cooling phase the aortic arch with its braches as well as the descending aorta can be dissected free extensively. A disadvantage is the use of hypothermia which would not be necessary for VSD closure alone and is therefore a considerable extension of the procedure with all associated risks. An increased rate of recoarctations, presumably because of worse exposure, has also been reported (Brouwer et al. 1996; Gaynor et al. 2000; Isomatsu et al. 2001). The successful combination of both strategies is also possible: First, a standard coarctation repair is done through a lateral thoracotomy, to be followed after repositioning but during the same anesthesia—by VSD closure through a sternotomy, avoiding deep hypothermia (Kanter et al. 2007).

Our experience leads us to recommend the following approach: in a small VSD and aortic coarctation, the aorta is repaired first and the VSD closely followed and closed at a later date as necessary. If size and extent of a VSD necessitate early closure, this is regarded an indication for single stage repair. If the aortic narrowing also extends into the aortic arch as a tubular hypoplasia, which is often observed in these cases, sternotomy and the use of hypothermic techniques for extended aortic reconstruction are appropriate anyway. If a VSD/coarctation patient presents in poor condition, even after compensation, we rather proceed with coarctation repair only, regardless of VSD size.

In adult patients an extra-anatomic bypass to circumvent the coarctation can be a very good alternative option (Myers et al. 2011; Reents et al. 2012).

#### 14.6.4 «Swiss-Cheese-Type» VSD

The malformation somewhat colloquially called a «Swiss-cheese-type» VSD represents a profound disturbance of the development of the muscular ventricular septum, primarily in its trabeculated part. The multiple connections between right and left chamber existing during embryonic and fetal life do not close, resulting in several defects in the muscular septum. In the case of anteroapically located small communications, spontaneous closure within the first year of life is highly likely.

A combination of larger VSDs extending into the midmuscular portion with a considerable combined passage area can become hemodynamically very significant at the time of the physiological postnatal drop in pulmonary vascular resistance. Number and location of these communications make their surgical exposure very difficult. A decision must be made between pulmonary artery banding and a right ventriculotomy (Seddio et al. 1999), if pulmonary artery banding is not the institutional approach in these cases anyway. A left ventriculotomy should be avoided if at all possible.

#### 14.6.5 VSD and Aneurysm of a Valsalva Sinus

Congenital aneurysm of a sinus of Valsalva represents a separation of the media of the afflicted sinus from that of the hinge point of the respective aortic cusp (Edwards and Burchell 1957). The structural weakness between the aortic root and the heart in combination with the aortic blood pressure leads to bulging of the sinus into the cardiac structures below, up to the point of perforation. Usually only the right coronary sinus is afflicted, sometimes in combination with the adjacent half of the noncoronary one. Perforation ensues into the right atrium or ventricle. Coexistence of a VSD bordering the aortic valve can also be part of the malformation, leading to valvular insufficiency. An increased incidence of this combination has been observed in Asia, giving an indirect hint to genetic predisposition (Brizard 2006; Sakakibara and Konno 1962, 1968; Lee et al. 2013).

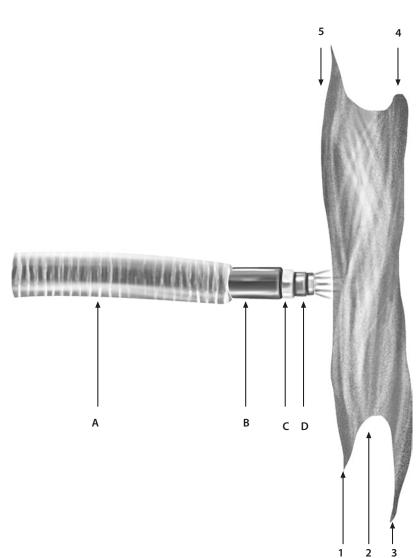
The operative strategy consists of resection of the «aneurysm,» VSD closure, and aortic valve reconstruction. After inspection of the anatomy through an aortotomy, an exposition from the ventricular side, e.g., through an infundibulotomy, is adequate in most cases. This allows for a definitive discrimination between a conoventricular and a pure conus defect and therefore the supposed course of the conduction system. The aneurysmatic tissue is resected, resulting in a larger defect in the right coronary sinus. The Dacron patch used for VSD closure is also utilized for reconstruction of the aortic root. First, complete VSD closure is performed with the anchoring of the middle part of the patch directly into the insertion zone of the right coronary cusp in the usual fashion. The excess patch length cranially can then be tailored into the aortic root defect with a continuous suture. In a classical conus defect, as is often seen in Asian patients, the whole procedure can be done transaortically. Asymmetry of a valve cusp may call for additional valve reconstruction or even replacement.

## 14.6.6 Catheter Interventional Techniques

The endeavor to avoid a ventriculotomy for closure of hemodynamically highly significant muscular defects led to the development of interventional techniques by catheter (Lock et al. 1988; Okubo et al. 2001; Waight et al. 2002; Bacha et al. 2005). Because of the limitations of vascular access in small children, these are often combined with open exposure of the heart through a thoracotomy as so-called hybrid procedures and have become well-established in several centers. Purely percutaneous closure of a muscular VSD can be regarded safe under certain conditions too. Further refinement of the devices has meanwhile led to their use for closure of conoventricular/perimembranous defects as an alternative to surgery. This was evaluated in several studies (Michel-Behnke et al. 2005; Holzer et al. 2006; Butera et al. 2007a, b; Hirsch et al. 2007; Xunmin et al. 2007; Mo et al. 2011). Because secure placement of these devices in a defect needs appropriate protrusions at the borders of the right and left ventricular aspect, several modifications of the established models for atrial septal defects became necessary. The «Amplatzer membranous septal occluder» shows recesses of the Nitinol meshwork for the aortic valve (■ Fig. 14.6).

This proved to be successful in older children and adults with accordingly relatively small defects. Direct contact of the device with the neighboring structures of the VSD did lead to some insufficiencies of the aortic and tricuspid valves, which was to be expected. Pressure upon the inferior border of the VSD also induced higher degree heart block up to the need of temporary transvenous stimulation over several weeks (Butera et al. 2006; Walsh et al. 2006; Zhou et al. 2008). Contrary to surgical therapy the possibilities to manage complications are limited, and even the theoretical benefit of a shorter hospital stay with low morbidity may then become obsolete. At present, elective interventional closure of a conoventricular VSD should be limited to a few centers and closely followed by rigid study protocols (Butera et al. 2007a, b; Carminati et al. 2007).

Fig. 14.6 «Amplatzer membranous septal occluder.» Lateral aspect. Device mounted on the catheter and expanded; left ventricular disk between 3 and 4; aortic excess length 4 with 0.5 mm being smaller than the septal one 3 with 5.5 mm. Right ventricular disk between 1 and 5, equal excess length of 2 mm. Waist fitting into the VSD (2) with a depth of 1.5 mm. A «delivery sheath»; B «pusher catheter»; C connector at catheter; D screw coupling at disk. The dacron-polyester patch material can be seen through the Nitinol mesh (Reproduced from Hijazi et al. (2002); with permission



The limited financial means of emerging countries have stimulated research to further modify occlusive devices for use in the setting of a hybrid approach through a (limited) thoracotomy but without cardiopulmonary bypass. Given the population density of China and the frequency of VSDs as isolated defects, impressive series were published over the recent years (Xing et al. 2010, 2011; Zheng et al. 2009; Chen et al. 2013). The remarkable early outcomes and the active participation in their development have now brought these new techniques to Europe (Schreiber et al. 2012). However, long-term results remain to be evaluated.

#### 14.6.7 Pushing the Limits

Advancements in microtechnology have led to amazing developments like that of a telemetrically adjustable banding device (Bonnet et al. 2004). This plays no role for standard treatment of a VSD. Transcutaneous tunneling of fixation sutures of a pulmonary band also serves the same purpose of avoiding operative readjustments (Choudhary et al. 2006).

Such notions are primarily generated in emerging countries which in theory do offer modern cardiac surgery, but where many patients are presenting only late or in dismal condition due to infrastructural shortcomings. If primary closure of a VSD is attempted in the presence of markedly elevated but still responsive pulmonary vascular resistance, patches with a valve mechanism may be considered (Novick et al. 2005; Zhang et al. 2007). In the event of a critical increase in pulmonary artery pressure, these allow for an overflow of desaturated blood from the right into the left ventricle and thus maintenance of systemic blood pressure at the expense of arterial oxygen saturation. We still use a simple fenestration of the Dacron patch with a standardized punch. Such an opening, usually with a diameter of 3 mm, can be closed consecutively with a catheter intervention if necessary. We feel this to be the safer approach-no risk of «valve» failure—as the limited fenestration should reliably prevent a suprasystemic right ventricular pressure rather than prevent rightsided volume overload which rarely will occur in these patients during long-term follow-up. The typical scenario is spontaneous closure once the pulmonary resistance drops.

#### 14.7 Perioperative Aspects

For getting informed consent, besides the general risks of open heart surgery, the typical risks of VSD closure must be mentioned: complete heart block with pacemaker dependency, residual defect, and valvular insufficiencies (aortic, tricuspid). On a positive note, it should be emphasized that quality and expectancy of life as well as exercise capacity can be considered normal after a successful transatrial operation (Roos-Hesselink et al. 2004).

Recommendations for the prevention of infective endocarditis have evolved over the years. Antibiotic prophylaxis should be administered in unrepaired VSD as well as any residual defects. Depending on the material, endothelialization of a patch can be considered complete after 6 months, making aggressive antibiotic prophylaxis unnecessary thereafter (Backer et al. 1993; Dajani et al. 1997; Wilson et al. 2007).

During postoperative intensive care therapy, the principles for preventing so-called pulmonary hypertensive crises must be followed, especially in young infants with a markedly raised preoperative Qp:Qs ratio (relaxation, NO application, etc.; see ► chapter «Critical Care in Pediatric Cardiac Surgery», Sect. 10.7 and Sect. 10.11). An early complete heart block may be caused by swelling of the tissue, which will usually resolve within 10–14 days. During this time period, the indication for permanent pacemaker implantation should be handled very restrictively. It is a recommended safety measure to implant two sets of temporary ventricular stimulation leads in any patient who has a higher degree heart block at the end of the operation.

Even if closure of a VSD historically belongs to the first successful cardiac procedures ever, it is by no means a bagatelle or a beginner's operation. Nowadays, it is a requirement to limit this operation only to centers with the necessary expertise in the treatment of congenital heart disease, which is particularly important because the majority of VSD closures must be performed during the newborn period or in early infancy. This is the only way to achieve the necessary quality regarding morbidity and mortality and long-term outcome.

# References

- Andersen HO, deLeval MR, Tsang VT et al. (2006) Is complete heart block after surgical closure of ventricular septal defects still an issue? Ann Thorac Surg 82:948–956
- Bacha EA, Cao QL, Galantowicz ME et al. (2005) Multicenter experience with perventricular device closure of muscular ventricular septal defects. Pediatr Cardiol 26:169–175
- Backer CL, Winters RC, Zales VR et al. (1993) Restrictive ventricular septal defect: how small is too small to close? Ann Thorac Surg 56:1014–1019
- Becker AE, Anderson RH (1982) Cardiac pathology. Churchill Livingstone, Edinburgh
- Bonnet D, Corno AF, Sidi D et al. (2004) Early clinical results of the telemetric adjustable pulmonary artery banding Flo-Watch PAB. Circulation 110(II Suppl 1):II158–II163
- Brizard C (2006) Surgical repair of infundibular ventricular septal defect and aortic regurgitation. Semin Thorac Cardiovasc Surg Pediatr Card Surg Ann:153–160
- Brouwer RM, Cromme-Dijkhuis AH, Erasmus ME et al. (1996) Decision making for the surgical management of aortic coarctation associated with ventricular septal defect. J Thorac Cardiovasc Surg 111:168–175
- Butera G, Chessa M, Carminati M (2006) Late complete atrioventricular block after percutaneous closure of a perimembranous ventricular septal defect. Catheter Cardiovasc Interv 67:938–941
- Butera G, Chessa M, Carminati M (2007a) Percutaneous closure of ventricular septal defects. State of the art. J Cardiovasc Med 8:39–45
- Butera G, Carminati M, Chessa M et al. (2007b) Transcatheter closure of perimembranous ventricular septal defects. J Am Coll Cardiol 50:1189–1195
- Carminati M, Butera G, Chessa M et al. (2007) Transcatheter closure of congenital ventricular septal defects: results of the European Registry. Eur Heart J 28:2361–2368
- Chen Q, Cao H, Zhang GC et al. (2013) Closure of perimembranous ventricular septal defects with intraoperative device technique: another safe alternative to surgical repair. Thorac Cardiovasc Surg 61:293–299
- Chiu SN, Wang JK, Lin MT et al. (2007) Progression of aortic regurgitation after surgical repair of outlet-type ventricular septal defects. Am Heart J 153:336–342
- Choudhary SK, Talwar S, Airan B et al. (2006) A new technique of percutaneously adjustable pulmonary artery banding. J Thorac Cardiovasc Surg 131:621–624
- Dajani AS, Taubert KA, Wilson W et al. (1997) Prevention of bacterial endocarditis. Recommendation by the American Heart Association. JAMA 277:1794–1801
- Dodge-Khatami A, Knirsch W, Tomaske M et al. (2007) Spontaneous closure of small residual ventricular septal defects after surgical repair. Ann Thorac Surg 83:902–905
- Edwards JE, Burchell HB (1957) The pathological anatomy of deficiencies between the aortic root and the heart, including aortic sinus aneurysms. Thorax 12:125–139
- Eroglu AG, Oztunc F, Saltik L et al. (2003) Evolution of ventricular septal defect with special reference to spontaneous closure rate, subaortic ridge and aortic valve prolapse. Pediatr Cardiol 24:31–35

- Frater RW (1967) The prolapsing aortic cusp: experimental and clinical observations. Ann Thorac Surg 3:63–67
- Gaynor JW (2003) Management strategies for infants with coarctation and an associated ventricular septal defect. J Thorac Cardiovasc Surg 125:887–889
- Gaynor JW, Wernovsky G, Rychik J et al. (2000) Outcome following single-stage repair of coarctation with ventricular septal defect. Eur J Cardiothorac Surg 18:62–67
- Gerbode F, Hultgren H, Melrose D, Osborn J (1958) Syndrome of left ventricular-right atrial shunt: successful surgical repair of defect in five cases, with observation of bradycardia on closure. Ann Surg 148:433–446
- Goor DA, Lillehei CW (1975) Congenital malformations of the heart. Grune & Stratton, New York
- Hanna B, Colan SD, Bridges ND, Mayer JE, Castaneda AR (1991) Clinical and myocardial status after left ventriculotomy for ventricular septal defect closure. J Am Coll Cardiol 17(Suppl):110A
- Heinemann M, Ziemer G, Luhmer I et al. (1990) Coarctation of the aorta in complex congenital heart disease: simultaneous repair via sternotomy. Eur J Cardiothorac Surg 4:482–486
- Hijazi ZM, Hakim F, Haweleh AA et al. (2002) Catheter closure of perimembranous ventricular septal defects using the new Amplatzer membranous VSD occluder: initial clinical experience. Cathet Cardiovasc Interv 56:508–515
- Hirsch R, Lorber A, Shapira Y et al. (2007) Initial experience with the Amplatzer membranous septal occluder in adults. Acute Card Care 9:54–59
- Holzer R, de Giovanni J, Walsh KP et al. (2006) Transcatheter closure of perimembranous ventricular septal defects using the Amplatzer membranous VSD occluder: immediate and mid-term results of an international registry. Catheter Cardiovasc Interv 68:620–628
- Isomatsu Y, Imai Y, Shin'oka T et al. (2001) Coarctation of the aorta and ventricular septal defect: should we perform a single-stage repair? J Thorac Cardiovasc Surg 122:524–528
- Jian-Jun G, Xue-Gong S, Ru-Yuan Z et al. (2006) Ventricular septal defect closure in right coronary cusp prolapse and aortic regurgitation complicating VSD in the outlet septum: which treatment is most appropriate? Heart Lung Circ 15:168–171
- Kadner A, Dodge-Khatami A, Dave H et al. (2006) Closure of restrictive ventricular septal defects through a right axillary thoracotomy. Heart Surg Forum 9:E836– E839
- Kanter KR, Mahle WT, Kogon BE, Kirshbom PM (2007) What is the optimal management of infants with coarctation and ventricular septal defect? Ann Thorac Surg 84:612–618
- Kidd L, Driscoll DJ, Gersony WM et al. (1993) Second natural history study of congenital heart defects. Results of treatments of patients with ventricular septal defects. Circulation 87(2 Suppl):138–151
- Kirklin JW, Harshbarger HG, Donald DE, Edwards JE (1957) Surgical correction of ventricular septal defect: anatomic and technical considerations. J Thorac Cardiovasc Surg 33:45–59
- Kleinman CS, Tabibian M, Starc TC et al. (2007) Spontaneous regression of left ventricular dilation in children with restrictive ventricular septal defect. J Pediatr 150: 583–586

- Kostolny M, Schreiber C, von Arnim V et al. (2006) Timing of repair in ventricular septal defect with aortic insufficiency. Thorac Cardiovasc Surg 54:512–515
- Lee SH, Kim JB, Park NH et al. (2013) Asymptomatic ruptured sinus of Valsalva aneurysm combined with perimembranous ventricular septal defect, and bicuspid aortic valve in adult patient. Thorac Cardiovasc Surg 61:327–329
- Lillehei CW, Cohen M, Warden HE et al. (1955) The results of direct vision closure of ventricular septal defects in eight patients by means of controlled cross circulation. Surg Gynecol Obstet 101:446–466
- Lock JE, Block PC, McKay RG et al. (1988) Transcatheter closure of ventricular septal defects. Circulation 78: 361–368
- Mavroudis C, Backer CL, Stewart RD, Heraty P (2005) The case against minimally invasive cardiac surgery. Semin Thorac Cardiovasc Surg Pediatr Card Surg Ann 8:193–197
- Michel-Behnke I, Le TP, Waldecker B et al. (2005) Percutaneous closure of congenital and acquired ventricular septal defects—considerations on selection of the occlusion rate. J Interv Cardiol 18:89–99
- Mo X, Zuo W, Ma Z et al. (2011) Hybrid procedure with cardiopulmonary bypass for muscular ventricular septal defects in children. Eur J Cardiothorac Surg 40:1203–1206
- Muller WH Jr, Dammann JF Jr (1952) The treatment of certain congenital malformations of the heart by creation of pulmonic stenosis to reduce pulmonary hypertension and excessive pulmonary blood flow: a preliminary report. Surg Gynecol Obstet 95:213–219
- Myers PO, Tissot C, Cikirikcioglu M et al. (2011) Complex aortic coarctation, regurgitant bicuspid aortic valve with VSD and non-compaction: a challenging combination. Thorac Cardiovasc Surg 59:313–314
- Myhre U, Duncan BW, Mee RB et al. (2004) Apical right ventriculotomy for closure of apical ventricular septal defects. Ann Thorac Surg 78:204–208
- Novick WM, Sandoval N, Lazorhysynets VV et al. (2005) Flap valve closure of ventricular septal defects in children with increased pulmonary vascular resistance. Ann Thorac Surg 79:21–28
- Okubo M, Benson LN, Nykanen D et al. (2001) Outcomes of intraoperative device closure of muscular ventricular septal defects. Ann Thorac Surg 72:416–423
- Reents W, Froehner S, Diegeler A et al. (2012) Ascendingto-descending bypass for simultaneous surgery of aortic coarctation with other cardiac pathologies. Thorac Cardiovasc Surg 60:210–214
- Roos-Hesselink JW, Meijboom FJ, Spitaels SE et al. (2004) Outcome of patients after surgical closure of ventricular septal defect at young age: longitudinal follow-up of 22–34 years. Eur Heart J 25:1057–1062
- Russell HM, Forsberg K, Backer CL et al. (2011) Outcomes of radial incision of the tricuspid valve for ventricular septal defect closure. Ann Thorac Surg 92: 685–690
- Sakakibara S, Konno S (1962) Congenital aneurysm of the sinus of Valsalva: anatomy and classification. Am Heart J 63:405–424
- Sakakibara S, Konno S (1968) Congenital aneurysm of the sinus of Valsalva associated with ventricular septal defect: anatomical aspects. Am Heart J 75:595–603

- Saleeb SF, Solowiejczyk DE, Glickstein JS et al. (2007) Frequency of development of aortic cuspal prolapse and aortic regurgitation in patients with subaortic ventricular septal defect diagnosed at <1 year of age. Am J Cardiol 99:1588–1592
- Schreiber C, Vogt M, Kühn A et al. (2012) Periventricular closure of a perimembranous VSD: treatment option in selected patients. Thorac Cardiovasc Surg 60:78–80
- Seddio F, Reddy VM, McElhinney DB et al. (1999) Multiple ventricular septal defects: how and when should they be repaired? J Thorac Cardiovasc Surg 117: 134–140
- Spicer DE, Anderson RH, Backer CL (2013) Clarifying the surgical morphology of inlet ventricular septal defect. Ann Thorac Surg 95:236–241
- Tatsuno K, Konno S, Ando M, Sakakibara S (1973) Pathogenetic mechanisms of prolapsing aortic valve and aortic regurgitation associated with ventricular septal defect. Circulation 48:1028–1037
- Tomita H, Arakari Y, Ono Y et al. (2004) Severity indices of right coronary cusp prolapse and aortic regurgitation complicating ventricular septal defect in the outlet septum: which defects should be closed? Circ J 68:139–143
- Tomita H, Arakaki Y, Ono Y et al. (2005) Impact of noncoronary cusp prolapse in addition to right coronary cusp prolapse in patients with a perimembranous ventricular septal defect. Int J Cardiol 101:279–283
- Trusler GA, Mustard WT (1972) A method of banding the pulmonary artery for large isolated ventricular septal defect with and without transposition of the great arteries. Ann Thorac Surg 13:351–355
- Trusler GA, Moes CAF, Kidd BSL (1973) Repair of ventricular septal defect with aortic insufficiency. J Thorac Cardiovasc Surg 66:394–403
- Trusler GA, Williams WG, Smallhorn JF, Freedom RM (1992) Late results after repair of aortic insufficiency associated with ventricular septal defect. J Thorac Cardiovasc Surg 103:276–281
- Tweddell JS, Pelech AN, Frommelt PC (2006) Ventricular septal defect and aortic valve regurgitation: pathophysiology and indications for surgery. Semin Thorac Cardiovasc Surg Pediatr Card Surg Ann 9:147–152
- van Praagh R, Geva T, Kreutzer J (1989) Ventricular septal defects: how shall we describe, name and classify them? J Am Coll Cardiol 14:1298–1299
- Waight DJ, Bacha EA, Kahana M et al. (2002) Catheter therapy of Swiss cheese ventricular septal defects using the Amplatzer muscular VSD occluder. Cathet Cardiovasc Interv 55:355–361
- Walsh MA, Bialkowski J, Szkutnik M (2006) Atrioventricular block after transcatheter closure of perimembranous ventricular septal defects. Heart 92:1295–1297
- Xing Q, Pan S, An Q et al. (2010) Minimally invasive perventricular closure of perimembranous ventricular septal defect without cardiopulmonay bypass: multicenter experience and mid-term follow-up. J Thorac Cardiovasc Surg 139:1409–1415
- Xing Q, Wu Q, Pan S et al. (2011) Transthoracic device closure of ventricular septal defects without cardiopulmonary bypass: experience in infants weighing less than 8 kg. Eur J Cardiothorac Surg 40:591–597

- Xunmin C, Shisen J, Jianbin G et al. (2007) Comparison of results and complications of surgical and Amplatzer device closure of perimembranous ventricular septal defects. Int J Cardiol 120:28–31
- Zhang B, Wu S, Liang J et al. (2007) Unidirectional monovalve homologous aortic patch for repair of ventricular septal defect with pulmonary hypertension. Ann Thorac Surg 83:2176–2181
- Zheng Q, Zhao Z, Zuo J (2009) A comparative study: early results and complications of percutaneous and surgical closure of ventricular septal defect. Cardiology 114:238–243
- Zhou T, Shen XQ, Zhou SH (2008) Atrioventricular block: a serious complication in and after transcatheter closure of perimembranous ventricular septal defects. Clin Cardiol 31:368–371