Assessment of Ventricular septal function after VSD transcatheter device closure Using Speckle Tracking Technique

Thesis
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Submitted by

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Introduction

Ventricular septal defect is the most common congenital heart disease accounting for 40% of all congenital heart diseases. An isolated VSD accounts for more than 20% of all congenital heart diseases ***(Penny and Vick, 2011)***.

It is classified according to its relation to septum as inlet, trabecular, outlet & membranous septum (***Praagh et al., 1989***). Another classification is based on VSD location on the right surface of the inter ventricular septum as; single or multiple, infundibular, perimembranous, inlet, muscular & Gerbode defect (***Cameron et al., 2006).***

Children with volume-overloaded left atrium and ventricle due to a VSD require surgical repair to prevent pulmonary artery hypertension, ventricular dilation, arrhythmias, aortic regurgitation, development of double chambered right ventricle and the risk of endocarditis (***Kidd et al., 1993***).

Until recently the only way of doing this was open-heart surgery. This is a major procedure that necessitates a thoracotomy, heart lung bypass, blood transfusion in some cases, permanent scar and potential risks of complete heart block, residual shunt, early and late arrhythmias, post pericardiotomy syndrome and even death ***(Backer et al., 1993).***

Surgery at present can be accomplished with minimal risk (less than 1%), but because of the obvious discomfort

and relatively long recovery period, alternative procedures have been developed.

The development of a transcatheter occlusion technique that can safely and effectively close these defects would be welcomed by cardiologists, patients, and their families ***(Sideris et al., 1999)***.

Multiple devices have been used to close VSDs including the Rashkind double umbrella device, CardioSEAL device, STARFlex device coils, Amplatzer Muscular and Membranous VSD device, Amplatzer Septal Occluder and the Amplatzer Duct Occluder and the Duct Occluder II as well as Chinese symmetrical and asymmetrical occluders which are variations of the Amplatzer devices. The most extensive experience in North America and Europe is with the Amplatzer family of devices. The use of the Chinese devices is most commonly reported from China itself ***(Fu YC et al., 2006).***

Two dimensional (2D) speckle tracking echocardiography (STE) is a promising new imaging modality. STE uses a completely different algorithm to calculate deformation: by computing deformation from standard 2D grey scale images, it is possible to overcome many of the limitations of TDI.

The clinical relevance of deformation parameters paired with an easy mode of assessment has sparked enormous interest within the echocardiographic community. This is also reflected by the increasing number of publications which focus on all aspects of STE and which

test the potential clinical utility of this new modality. Some have already heralded STE as 'the next revolution in echocardiography'. While both strain and LV ejection fraction (LVEF) measure LV function, there is a fundamental difference between the two: strain calculates the contractility of the myocardium, while LVEF is a surrogate parameter that describes myocardial pump function. Even if contractility is reduced, compensatory mechanisms (i.e, ventricular dilatation, geometry changes) can still assure that stroke volume remains normal (at least at rest) ***(Edvardsen et al., 2006)***.

Aim of The Study

To assess the global left ventricular systolic function and regional ventricular septal functions by 2D speckle tracking (before & after the procedure) in children who underwent closure of isolated VSD_s by transcatheter device in comparison to normal controls.

Chapter 1
Ventricular Septal Defect

Overview:

A ventricular septal defect (VSD) is a hole or a defect in the septum that divides the two lower chambers of the heart, resulting in communication between the ventricular cavities. A VSD may occur as a primary anomaly, with or without additional major associated cardiac defects. It may also occur as a single component of a wide variety of intracardiac anomalies, including tetralogy of Fallot (TOF), complete atrioventricular (AV) canal defects, transposition of great arteries (TGA), and corrected transpositions.

An isolated VSD occurs in approximately 2-6 of every 1000 live births and accounts for more than 20% of all congenital heart diseases (CHD). After bicuspid aortic valves, VSDs are the most commonly encountered CHD ***(Roger, 1879).***

Acquired VSD can occur as a result of a septal myocardial infarct but such a finding is much rarer than congenital lesions and prognosis tends to be poor ***(Alter et al., 2004)***.

Types of VSDs (Fig. 1):

The ventricular septum is divided into a small membranous portion and a large muscular portion. The muscular septum has three components: the inlet septum, the trabecular septum, and the outlet (or infundibular) septum.

Defects in the membranous septum often extend into different parts of the muscular septum and are labelled as perimembranous defects. These include inlet, trabecular and infundibular perimembranous defects. Perimembranous defects are the most common (about 70%).

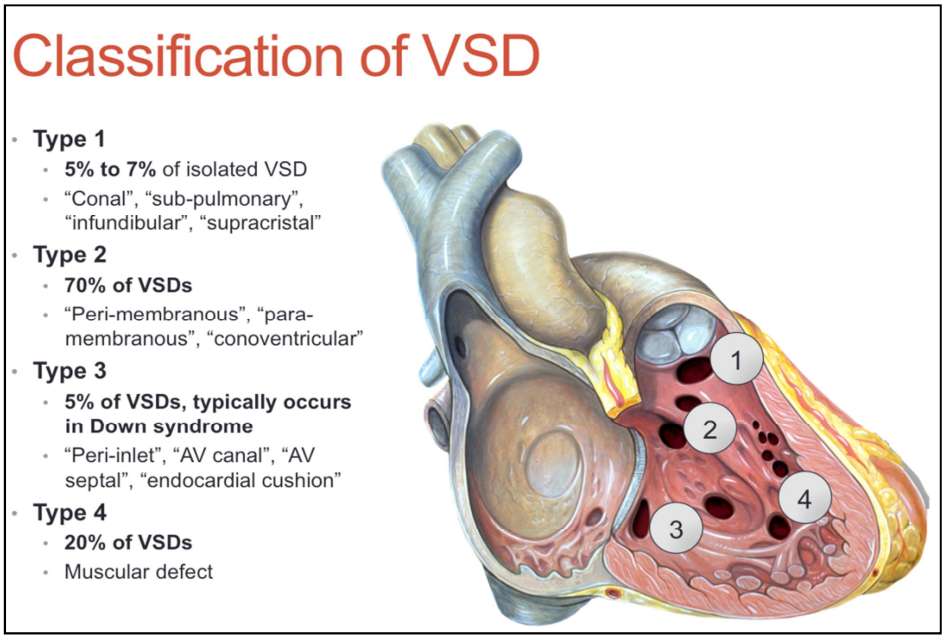

Figure 1: Types of VSD

Muscular defects are those confined to the muscular septum. They are further described according to their location in the muscular septum as they can be found in or between the inlet septum, trabecular septum or infundibular septum.

Defects in the area of the septum adjacent to the arterial valves are termed as subarterial infundibular defects. These defects are also called supracristal defects and because of the complete deficiency of the infundibular septum, allow prolapse of the aortic valve cusps into the right ventricular

outflow tract. This can result in development of aortic regurgitation. These defects account for 5% to 7% of all defects in the western world but about 30% in far eastern countries.

Inlet or AV canal VSDs lie beneath the septal leaflet of the tricuspid valve. They are not associated with defects in the AV valves and account for 5% to 8% of all VSDs.

Diagnosis:

• Pathophysiology:

How haemodynamically significant a VSD depends on its size, pressure in the individual ventricles and pulmonary vascular resistance (PVR). The presence of a VSD may not be obvious at birth because of nearly equal pressures in both ventricles with little or no shunting of blood. As the PVR drops, the pressure difference between the two ventricles increases and the shunt becomes significant allowing the defect to become clinically apparent ***(Minette and Sahn, 2006).***

• Clinical Picture:

The clinical presentation varies with the severity of the lesion. With a small VSD (A restrictive VSD is a defect that produces a significant pressure gradient between LV & RV so pulmonary-to-aortic systolic pressure ratio < 0.3), the infant or child is asymptomatic with normal feeding and weight gain and the lesion may be detected when a murmur is heard at a routine examination.

On the other hand, when a VSD is moderate-to-large (nonrestrictive VSD is accompanied by a large shunt (Qp/Qs

> 2.2) and a pulmonary-to-aortic systolic pressure ratio greater than 0.66.), although the babies are well at birth, symptoms generally appear by 5 - 6 weeks of age. The main symptom is exercise intolerance and since the only exercise babies do is feeding, the first impact is on feeding. Feeding tends to slow down and is often associated with tachypnea and increased respiratory effort. Babies are able to feed less, and weight gain and growth are soon affected. Poor weight gain is a good indicator of heart failure in a baby. Recurrent respiratory infections may also occur.

When there are very large VSDs, the features are similar but more severe. If appropriate management is not carried out promptly in infants with large VSDs excessive pulmonary blood flow may lead to increase in pulmonary vascular resistance and pulmonary hypertension. These babies may develop a right to left shunt with cyanosis or Eisenmenger's syndrome (VSD has a systolic pressure ratio of 1 and Qp/Qs less than 1:1 or a net right-to-left shunt).

Physical examination of a patient with a VSD usually reveals a heart murmur. The loudness of the murmur is related to the size of the defect and amount of blood crossing the defect.

• Diagnostic studies:

1- Chest X-ray (CXR):

Patients with small VSDs have normal CXR, whereas, with larger defects cardiomegally of varying degrees is present involving the left atrium (LA), the left ventricle (LV) and sometimes the right ventricle (RV). Pulmonary vascular markings are increased.

In those who have developed pulmonary hypertension the main pulmonary artery and hilar pulmonary arteries are enlarged significantly but the peripheral lung fields are oligaemic and heart size is usually normal.

2- Electrocardiography (ECG):

The ECG is usually normal in patients with small VSDs, whereas, with a moderate VSD, left ventricular hypertrophy (LVH) and occasionally left atrial hypertrophy (LAH) may be seen. Biventricular hypertrophy (BVH) with or without LAH can be seen with large VSD.

In those with pulmonary hypertension, right axis deviation, right ventricular hypertrophy (RVH) and right atrial hypertrophy (RAH) may be seen.

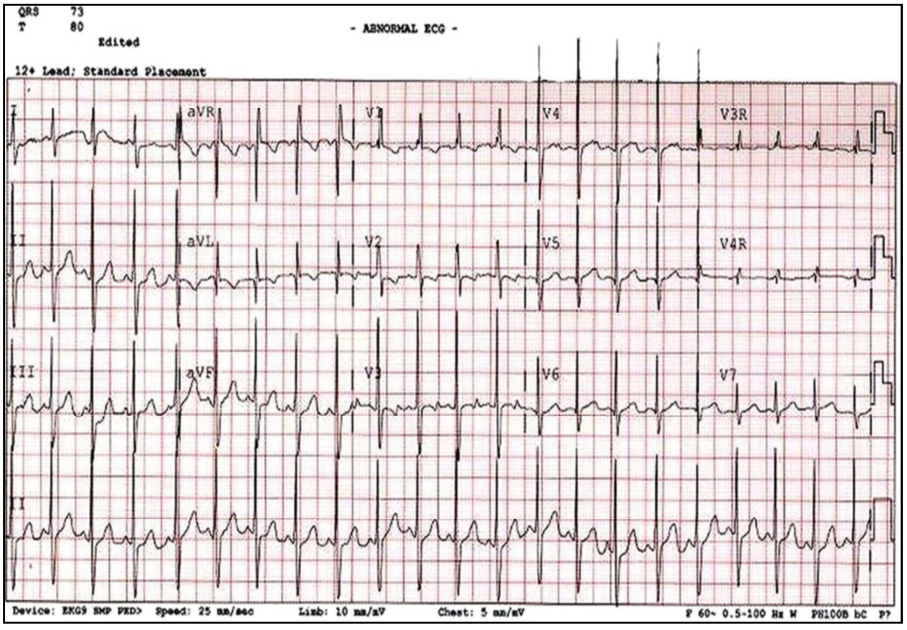

Figure 2: The electrocardiogram shows features of biventricular hypertrophy.

3- Echocardiography:

Transthoracic two-dimensional and Doppler echocardiography can identify the number, size and exact location of the defect. Any obstruction of the right ventricular outflow tract (RVOT) or insufficiency of the aortic valve can be identified. It can also provide a hemodynamic evaluation of the defect and estimate pulmonary artery pressure using the modified Bernoulli equation. In those with problems with image quality further information may be obtained using transoesophageal echocardiography.

Echocardiography can provide much information that previously required cardiac catheterization. Two dimensional echocardiography, along with Doppler echocardiography and color flow imaging, can assess the size and location of virtually all VSDs. Doppler echocardiography also provides physiological information including right ventricular pressure, pulmonary artery pressure and the difference in pressure between the ventricles.

Measurement of LA and LV diameter provides semi-quantitative information about shunt volume. Defect size is often given in terms of the size of the aortic root. Defects that are about the size of the aortic root are classified as large; those one third to two thirds of the diameter of the aorta are moderate, and those less than one third of the aortic root diameter, are small.

Three-dimensional echocardiography may be used to estimate the size of VSDs more accurately as part of

planning for surgical or catheter closure ***(Charakida et al 2013).***

4- Cardiac catheterization:

With advances in echocardiography, diagnostic cardiac catheterization is used far less frequently than in the past. Apart from information about the location and number of defects it can provide accurate measurement of pulmonary vascular resistance, pulmonary reactivity and volume of shunting.

• Management Strategies:

1- Medical management:

Management in the infant and child depends on symptoms, with small asymptomatic defects needing no medical management, and unlikely to need any intervention. First-line treatment for moderate or large defects affecting feeding and growth is with diuretics for heart failure and high-energy feeds to improve calorie intake. Angiotensin-converting enzyme inhibitors are used to reduce afterload which promotes direct systemic flow from the left ventricle, thus reducing the shunt. Digoxin can also be given for its inotropic effect. Any patient needing significant medical management should be referred for surgical assessment ***(Williams et al., 2004)***

2- Surgical management:

Surgical closure by direct suture or with a patch has been used for more than 50 years with low perioperative

mortality, even in adults, and a high closure rate. Patch leaks are not uncommon but seldom need reoperation. Late sinus node disease may occur ***(Roos-Hesselink et al., 2004).***

Overall results of surgery are excellent in the current era, but there is an increased risk of residual defects after multiple ventricular septal defects (mVSDs) surgery and the need for repeat operation when compared with perimembranous defects ***(Serraf et al., 1992*** *and* ***Kitagawa et al., 1998).***

3- Device Closure

Transcatheter occlusion of ventricular septal defects (VSD) was first reported in 1987 ***(Lock et al., 1988)***; however, the devices employed for transcatheter occlusion have not been particularly easy to use. The Rashkind or Clamshell umbrellas require a large sheath or front loading catheter (which limits retrievability) for introduction ***(Rigby and Redington, 1994)***, while the Sideris adjustable buttoned device has a two part introduction method and slow release mechanism ***(Sideris et al., 1997)***.

During the past few years a variety of devices have been used to close congenital or post-infraction mVSDs, but none has gained wide acceptance ***(Rigby and Redington, 1994*** *and* ***Janorkar et al., 1999).***

Large delivery sheaths, inability to recapture and reposition, structural failure, dislodgement and embolisation of the device, interference with the aortic valve resulting in aortic insufficiency and a very high rate of residual shunting are some of the limitations of previously used devices &

considered the major limitations of the previously described techniques.

Thanopoulos et al. (1999) and ***Hijazi et al. (2000***) reported on their preliminary experience in children with a new device, the Amplatzer ventricular septal defect occlude (AVSDO), to close MVSDs with very good results.

Figure 3: A close up view of the Amplatzer VSD occlusion device shows the two concave retention discs projecting4 mm beyond the 9 mm wide ´ 7 mm long central stent.

The retention discs contain polyester patches, and the central stent is filled with polyester fibres. The right ventricular disk has a microscrew attached. ***(Tofeig et al., 2000).***

Successful transcatheter device closure of trabecular (muscular) and perimembranous VSDs has been reported. Trabecular VSDs have proven more amenable to this

technique because of their relatively straightforward anatomy and muscular rim to which the device attaches well and, as such, result in excellent closure rates with a low procedural mortality ***(Thanopoulos and Rigby, 2005*** *and* ***Carminati et al., 2005).***

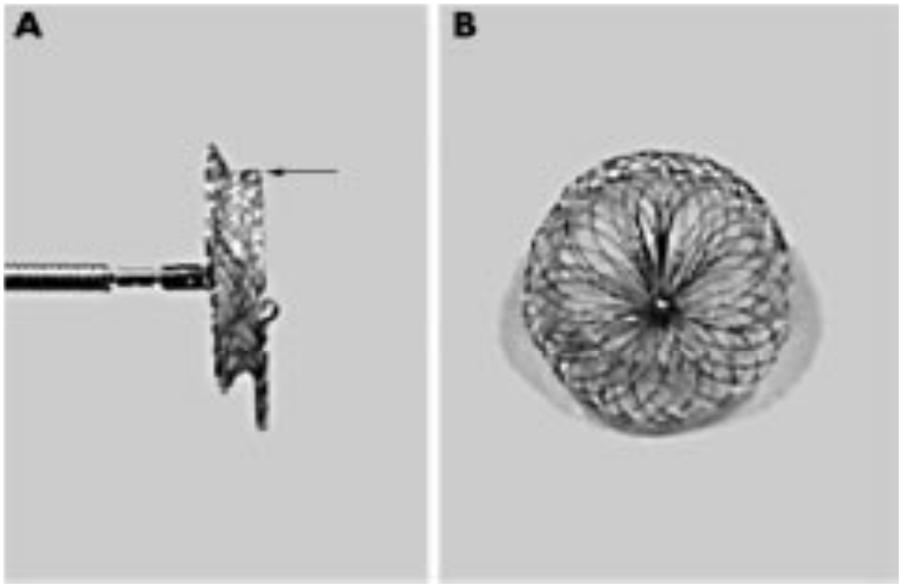

Figure 4: (A) Side and **(B)** frontal views of the Amplatzer asymmetric ventricular septal defect occluder (AAVSDO) made of woven Nitinol wires into two flat disks with a 1.5 mm connecting waist. Arrow indicates the 0.5 mm superior margin of the asymmetric left disk. ***(Thanopoulos et al., 2003).***

Management of moderate- to large-sized muscular VSD remains a challenge to both cardiologists and surgeons. The mortality and morbidity of surgical closure of mVSDs remain high ***(Serraf et al., 1992*** *and* ***Kitagawa et al., 1998).*** Therefore, cardiologists pursued the nonsurgical closure of such defects. Unfortunately, the previous devices (clamshell/buttoned or Rashkind) have not been originally designed for closure of the mVSDs; therefore, the success rate utilizing such devices was not high ***(Rigby and***