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PROJECT REPORT

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DECLARATION

I, Dr. Sanjeev H Naganur, hereby declare that the project in this book was undertaken by me under the supervision of the faculty, Department of Cardiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram

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Thesis Title

Intermediate and Long Term outcome after Transcatheter Closure of Atrial Septal Defects
Dedicated to My Parents
ACKNOWLEDGEMENTS

First and foremost I thank god for being there for me always.

There are so many people to thank, to express my gratitude and appreciation, that I find this space limited and inadequate. I shall however make a humble attempt.

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INTRODUCTION
Atrial septal defect (ASD) is the second most common congenital heart disease in adults, accounting for approximately 10% of all congenital heart lesions\(^1\). The incidence of ASD is approximately 1 per 1500 live births and 70% of ASDs are of the ostium secundum variety\(^2\). Need to treat such lesions has been studied and discussed extensively over the years. Small sized ASDs may not need to be addressed if found in an asymptomatic patient as an isolated defect. However, moderate to large size ASD causing right heart dilatation which may lead to increased pulmonary artery pressure should be closed. Interestingly, some ASDs can enlarge enough over time to require closure.

The presence and severity of functional limitation among patients with ASDs seem to increase with age. Congestive heart failure is rarely found in the first decade of life, but is well known to manifest in 2\(^{nd}\) decade as effort tolerance. However, symptoms are quite common once the patient is older than 40 years of age\(^3\). The onset of atrial fibrillation or, less commonly, atrial flutter can be a hallmark in the course of patients with ASDs. The incidence of atrial arrhythmias increases with advancing age\(^3\) to as high as 13% in patients older than 40 years of age\(^4\) and 52% in those older than 60 years of age\(^5\). Considering all these factors and possibility of development of pulmonary vascular disease the atrial septal defect when diagnosed and found to shunt significantly should be addressed at the earliest.

The surgical approach was once the standard of care for secundum ASD, proving to be both safe and effective. Surgical correction still is the standard of care for other types of ASD such as ostium primum and sinus venosus ASD. However, during the past 15 years, closure of secundum atrial septal defects (ASD) has moved from a surgical approach in the operating room to a percutaneous transcatheter approach in the cardiac catheterization laboratory.\(^6\)

Reports of transcatheter secundum ASD closure studies have shown improvement in cardiac dimensions and function, as well as right ventricular systolic pressure and pulmonary arterial hypertension.\(^7,9\) In contrast to surgical closure, the transcatheter approach avoids cardiopulmonary bypass, results in shorter hospitalization, reduces need for blood products, lessens patient discomfort, produces similar outcomes and decreases the overall cost.\(^10-13\)
Introduction

Following percutaneous closure of ASD, patients of all ages experience improved symptoms, reduction in pulmonary artery (PA) pressure and reverse remodelling of the right ventricle (RV) The transcatheater approach has evolved to become the new standard of care for children as well as adults with ASD. ASD closure should be considered in all hemodynamically significant secundum ASDs regardless of the patient's age or symptoms.

Short and intermediate-term studies have shown the device to be safe and effective.14-16 Among the available literature, studies to assess the long term clinical outcome, ECG changes, right ventricle (RV) size, pulmonary artery (PA) pressure, complications after device closure are sparse. Given that few long-term studies of the device exist17 and complications after device placement continue to be reported although rare.18-20

Hence the need of such a study to evaluate the long term outcome after transcatheter closure of OS ASD is in both adult as well as paediatric population.
REVIEW OF LITERATURE
ASD is one of the most common congenital heart defects requiring procedural intervention. Atrial septal defect (ASD) is the second most common congenital heart disease in adults, accounting for approximately 10% of all congenital heart lesions\(^1\). 70% of ASDs are of the ostium secundum variety. Secundum ASDs represent 6% to 10% of all cardiac anomalies, occurring with frequency of 1 per 1,500 live births and is more frequent in females than males by about 2:1\(^2\). Most ASDs are sporadic; few genetic defects are also been described. In 1960, Holt and Oram\(^21\) found out the association between ASDs and anomalies of the upper extremities; later proved in 1997, by Li et al\(^22\) to be caused by mutations in TBX5, a member of the Brachyury (T) gene family. NKX2.5 and GATA4 genes have been identified to cause familial ASD\(^23\)\(^24\). Recently, a missense mutation in myosin heavy chain 6 (on chromosome 14q12) has been found to cause familial ASD\(^25\).

**Anatomy, Embryology and Pathology of Atrial Septal Defects**

Atrial septal defects are classified according to their location in relation to the fossa ovalis and their proposed embryogenesis. Interatrial communications in the region of the fossa ovalis may represent either a true secundum ASD or a valvular incompetent patent foramen ovale. Defects anterior to the fossa ovalis (primum defects) often are associated with a cleft in the anterior leaflet of the mitral valve. Those posterior and superior to the fossa ovalis, the sinus venosus defects. Finally, interatrial communications at the expected site of the coronary sinus ostium are often associated with an unroofed coronary sinus and left atrial connection of a persistent left superior vena cava.

The sequence of atrial septation was well described long back by Van Mierop\(^26\). The septum primum- first septum to develop is an incomplete thin-walled partition in which the anteroinferior free edge is above the atrioventricular canal and becomes lined by tissue derived from the superior and inferior endocardial cushions. Before the resultant interatrial opening (ostium primum) becomes sealed by endocardial cushion tissue, programmed cell death in an area near the anterosuperior aspect of the septum primum creates small cribriform perforations. These perforations coalesce to form a large, second interatrial communication (ostium secundum) maintaining interatrial blood flow.
At this time, to the right of the first septum, an anterosuperior infolding of the atrial roof occurs and forms a second septal structure (septum secundum). It expands posteroinferiorly as a thick-walled muscular ridge to form an incomplete partition that overlies the ostium secundum. As atrial septation is accomplished, septum secundum forms the limbus of the fossa ovalis and septum primum forms the valve of the fossa ovalis. The channel for interatrial blood flow through the ostium secundum is known as the foramen ovale.

Concurrently with atrial septation, the left horn of the sinus venosus forms the coronary sinus, and the right sinus horn becomes a part of the right atrium. Infolding at the sinoatrial junction forms the right and left venous valves. Whereas the right venous valve is maintained and forms the rudimentary valves of the inferior vena cava (eustachian valve) and the coronary sinus (thebesian valve), the left venous valve becomes fused to the superior, posterior, and inferior margins of the fossa ovalis.

Defects at the level of the fossa ovalis presumably result from deficiency, perforation, or absence of the septum primum (the valve of the fossa ovalis); because the ostium secundum appears enlarged or unguarded, these defects are labelled as secundum type.

The secundum ASDs vary greatly in size and shape, but not directly involve major cardiac structures (vena cava, pulmonary veins, coronary sinus, or atrioventricular valves). However relationships to these structures, are important considerations for transcatheter closure. Tissue rims of the defect must be present and substantial enough to anchor the device. The anatomic margins (also referred to as “rims”) surrounding the atrial septum are defined below.

Aortic rim: rim related to the aorta that abuts the anterior-superior septum of the defect. It may also be called the anterior-superior rim, retro-aortic rim or retro-aortic mound.

Superior rim: the rim that abuts the superior wall of the atrium.

Atrioventricular Valve (AV) rim: rim that abuts the atrioventricular valves or crux of the heart, also called the inferior-anterior rim.
Inferior Vena Cava (IVC) rim: rim that abuts the inferior vena cava, also called the inferior-posterior rim.

Posterior rim: most rightward and posterior rim opposite the aortic rim, and anatomically related to the right upper pulmonary vein.

Superior Vena Cava (SVC) rim: posterior-superior rim which is bordered by the superior vena cava and is near the upper pulmonary vein.

**Effects of Atrial Septal Defect on the Heart**

Large interatrial communication, with chronic left-to-right shunt imposes volume overload on the right-sided cardiac structures and results in dilation of the right
atrium and right ventricle. As the right ventricle dilates, the ventricular septum begins to straighten, such that the two ventricular chambers become D shaped when viewed in the echocardiographic short-axis plane. The tricuspid and pulmonary annuli can be dilated, and the valves can be incompetent and mildly thickened. Dilation of the central pulmonary arteries also may occur. Dilation of the left atrium usually is mild. The wall thickness and mass of the left ventricle tend to be normal in patients with isolated secundum ASD.

Effects of moderate to large ASD on the Lungs

The chronic volume overload causes dilation of the entire pulmonary vascular bed. Microscopically, the arteries, capillaries, and veins are engorged. Medial hypertrophy is evident in the muscular pulmonary arteries and the pulmonary veins, although its extent is usually masked by vascular dilation. Muscularization of arterioles also may occur\(^\text{27}\). In a few patients with a secundum ASD, severe and irreversible hypertensive pulmonary vascular disease develops, and there is a striking female preponderance for this association\(^\text{28}\). Pulmonary vascular obstructive lesions include not only plexiform lesions but also thrombotic lesions\(^\text{29}\). In older patients, the coexistence of chronic pulmonary venous hypertension (owing to left ventricular hypertrophy or failure) or chronic hypoxic pulmonary hypertension (owing to chronic obstructive or interstitial pulmonary disease) may contribute to the pulmonary vascular disease associated with the interatrial communication and thereby add to risk of closure.

Physiology

The direction of blood flow is primarily is related to the relative compliances of the ventricles. As right ventricle is more compliant, results in less resistance to filling from the right atrium, causing left to right shunt. In infancy, RV is thick, stiff, and not very compliant. Therefore, there is a minimal amount of left-to-right shunting.

Most infants with isolated ASDs are asymptomatic. However, there have been reports of infants with ASDs who present with heart failure. Hemodynamic findings at cardiac catheterization in these infants have been no different from those in children who do not have heart failure. Thus, the pathophysiology for heart failure in these infants is not fully understood. Despite of increased pulmonary blood flow,
pulmonary artery pressure is slightly increased and in most patients, pulmonary resistance remains in the normal range. However, a wide spectrum of hemodynamic findings has been reported in ASD’s including pulmonary vascular obstructive disease occurring in patients as young as 3 months of age.\textsuperscript{30}

Steele et al\textsuperscript{31} reported their results of 702 patients found to have isolated ASDs of the ostium secundum or sinus venosus type at cardiac catheterization. Of these 702 patients, 40 (6\%) had pulmonary vascular obstructive disease, defined as a total pulmonary resistance of 7 U/m\textsuperscript{2}; there were 34 women (85\%) and 6 men. Interestingly, no patient younger than 19 years of age presented with an ASD and pulmonary vascular obstructive disease.

Haworth\textsuperscript{30} reported on 10 patients with ASDs and pulmonary vascular obstructive disease (pulmonary arteriolar resistance of 4 to 16 U/m\textsuperscript{2}). Four of the patients were younger than 6 months of age at the time of catheterization and presented with congestive heart failure and failure to grow. Five patients were evaluated at 2 to 9 years of age, and two had severe pulmonary vascular disease (pulmonary resistance of 16.5 and 15.5 U/m\textsuperscript{2}).

**Clinical Features**

Most infants with ASDs are asymptomatic, and the condition goes undetected. More recently, infants are referred earlier for murmur detected during routine clinical examination by paediatricians for echocardiographic evaluation so that the average age at which ASDs are being detected is about 6 months. Older children with a moderate left-to-right shunt often are asymptomatic. Children with large left-to-right shunts are likely to complain of some fatigue and dyspnoea. Growth failure is uncommon. Rarely, ASDs in infants are associated with poor growth, recurrent lower respiratory tract infection and heart failure.

A typical patient with isolated secundum ASD is often asymptomatic until the third and fourth decade of life. Typical symptoms include decreased exercise capacity, fatigue, syncope and palpitations. Patients with significant shunting may develop right ventricular failure, atrial tachycardia, pulmonary hypertension and embolic events all of which can lead to significant morbidity and potential mortality. The age
at which a patient becomes symptomatic is highly variable and does not correlate well with shunt size.

Left untreated over time, even small ASDs can develop increased left-to-right shunting due to progressive increase in left ventricular (LV) diastolic pressure with aging, which causes increased left atrial pressure. In patients who develop pulmonary hypertension (PHTN) due to flow through ASD, approximately 10% will progress to Eisenmenger’s syndrome\textsuperscript{32}.

**Clinical examination**

Inspection of the chest may reveal a precordial bulge and a hyperdynamic cardiac impulse, especially in large left-to-right shunts. Palpation reveals a prominent systolic impulse. There are three important auscultatory features: (a) a typical wide and fixed splitting of the second heart sound, (b) a soft systolic ejection murmur at the second left intercostal space and (c) an early to middiastolic murmur at the lower left sternal border. The term ‘fixed S2’ refers to the constant time interval between $A_2$ and $P_2$ throughout the respiratory cycle. A delay in $P_2$ is due in part to prolonged emptying of the right ventricle because of increased volume of blood to be ejected; and considerable vasodilation of the pulmonary vasculature delays intra-arterial pulmonary tension necessary to close the pulmonary valve. The increased flow of blood across the pulmonary valve produces a crescendo-decrescendo (ejection-type) systolic murmur, heard maximally over the upper left sternal border and transmitted into both lung fields. The increased volume of blood shunted and flowing across the tricuspid valve results in the early to middiastolic murmur, maximal along the lower left sternal border. When significant pulmonary hypertension develops, the above characteristic findings change because of a smaller or absent left-to-right shunt. The widely split $S_2$ can disappear, $P_2$ becomes louder, the systolic murmur becomes shorter, and the diastolic murmur disappears.

**Radiologic Features**

Usually there will be cardiomegaly with pulmonary vascular markings, and this finding becomes more prominent with age and the larger the left-to-right shunt. If pulmonary vascular obstructive disease develops, the main pulmonary artery
becomes quite large and the peripheral lung fields become clear or oligemic (peripheral pruning)

**Electrocardiographic Features**

Usually, rhythm is sinus; however in few patients (more in older) junctional rhythm or supraventricular tachyarrhythmia, such as atrial flutter, can occur\(^{33}\). In most patients, the mean frontal plane QRS axis is between +95 to +170 degrees. The P-R interval may be prolonged, especially in older patients, because of intra-atrial and sometimes H-V conduction delay, resulting in first-degree atrioventricular block \(^{34}\). In about half the cases, tall P waves reflect right atrial enlargement. There is usually some variant of the rSR pattern (incomplete right bundle branch block pattern) in lead V1, consistent with right ventricular volume overload. The duration of the QRS complex is < 0.10 second, and R in lead V1 is somewhat prolonged.

**Echocardiographic Features**

Echocardiography shows increased right atrial and right ventricular dimensions and the defect in the atrial septum. The subcostal examination is the most effective for diagnosis because it places the ultrasound beam nearly perpendicular to the plane of the atrial septum.

However, in older patients transesophageal echocardiography (TEE) has become the most accepted diagnostic examination. Associated partial anomalous pulmonary venous connection also can be diagnosed and characterized confidently using TEE. Peripheral contrast-enhanced echocardiography, in certain circumstances, can clarify the type and relative degree of left-to-right and right-to-left shunting\(^{35}\).

Using Doppler technology, one can reasonably determine the ratio of pulmonary (Qp) to systemic (Qs) blood flow (Qp:Qs). Using Doppler echocardiography, one can estimate right ventricular systolic pressure and pulmonary artery pressure from the tricuspid and pulmonary valve regurgitation Doppler velocity waveforms.
Cardiac Catheterization

Cardiac catheterization is unnecessary for the diagnosis of secundum ASD. Occasionally, questions about pulmonary vascular obstructive disease or associated cardiac defects arise that require catheterization. An ASD is suspected when the oxygen saturation in the right atrium is greater than that in the superior and inferior vena cavae. An increase in oxygen saturation of > 10% from the superior vena cava to the right atrium in one series of blood samples or an increase of 5% in two series of samples usually indicates an interatrial communication. However, a ventricular septal defect with tricuspid insufficiency, a left ventricular to right atrial shunt, or partial or complete atrioventricular septal defect may produce similar findings. Anomalous pulmonary venous connection to the right atrium or vena cava or systemic arteriovenous fistula will also produce increased oxygen saturation in the right atrium and may be mistaken for an ASD. Phasic and mean pressures in the right and left atria are equal with large defects. Generally, the right ventricular systolic pressures are slightly increased to 25 to 35 mm Hg.

Pulmonary artery pressure usually is normal to slightly increased; however, a small but significant number of patients may have moderate increases in the pulmonary artery pressure. In the usual situation, the pulmonary arteriolar resistance is <4.0 U/m². Angiography almost never is necessary for the diagnosis of ASDs because of good echocardiography techniques.

Natural History

The natural course of ASDs is relatively benign except for the largest openings and those associated with other cardiac defects. Typically, patients with ASDs remain active and asymptomatic through early childhood. Many patients have lived into even seventh decades with ASDs of moderate size before symptoms developed. Secundum ASDs can close spontaneously, remain open or enlarge. Spontaneous closure of isolated ASDs has been reported with some frequency. In 1983 Cocherham et al. reported results of 87 children who underwent cardiac catheterization at <4 years old because of significant secundum ASD. At follow-up they found that 15 of 87 (17%) had spontaneous closure. For those whose first study was done at <1 year old, spontaneous closure occurred in 22%. If the study
Review of Literature

was done between 1 and 2 years old, spontaneous closure occurred in 33%. If the first study was done between 2 and 4 years, the spontaneous closure rate was down to 3%. Their recommendation was to wait until after age 4 years for elective closure.

Natural history of ASD diagnosed in childhood has a variable course. ASD diameter when untreated increases in 65% of cases and 30% will have more than a 50% increase in diameter. Only 4% of ASDs close spontaneously\textsuperscript{38}

Using echocardiography, one has been able to evaluate more accurately the size and hemodynamic effects of an ASD. A prospective echocardiographic study suggested that as many as 24% of newborns have evidence of an opening (3 to 8 mm) in the atrial septum in the first week of life\textsuperscript{39}. However, by a little more than 1 year of age, 92% of the patients were found to have spontaneous closure of the opening, and in most patients, there was evidence of a valve like opening of the atrial septum that was believed to contribute to closure. It appears that spontaneous closure or a decrease in size is most likely to occur in ASDs <7 to 8 mm and with younger age at diagnosis. Radzik et al\textsuperscript{40} reviewed the results in 101 infants diagnosed at a mean age of 26 days with an average follow-up of 9 months. Spontaneous closure occurred in all 32 ASDs <3 mm in diameter, 87% of 3- to 5-mm ASDs, 80% of 5- to 8-mm ASDs, and in none of 4 infants with defects >8 mm. These authors concluded that no follow-up is necessary if a defect is <3 mm in diameter, but for those with a defect 3 to 5 mm or 5 to 8 mm, should be evaluated by the end of the 12\textsuperscript{th} and 15\textsuperscript{th} month respectively, by which time >80% of the defects will be closed.

Helgason and Jonsdottir\textsuperscript{41} reviewed the medical records of all patients in Iceland with a diagnosis of ASD born between 1984 and 1993. ASD was confirmed by 2-D echocardiogram and data only from patients with secundum ASDs were analyzed. A total of 84 children diagnosed at a mean age of 12 months were followed for 4 years. Spontaneous closure or decreased size was observed in 89% with a 4-mm ASD, 79% with a 5- to 6-mm defect, and only 7% with a defect >6 mm. Even infants with congestive heart failure can have spontaneous closure or a reduction in the size of the ASD years after the diagnosis\textsuperscript{42}. Occasionally, spontaneous closure will occur as late as 16 years\textsuperscript{27}. 

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Unfortunately, some ASDs can enlarge enough over time to require closure. McMahon et al. evaluated 104 children who were diagnosed with their ASDs at a mean age of 4.5 years and followed for a mean of about 3 years. The defects were defined as small (3 to <6 mm), moderate (6 to <12 mm), or large (>12 mm). Among the 34 patients with a small ASD, 7 increased to moderate size and 3 increased to large size. Among 40 patients with a moderate ASD, 8 became large, and for the 30 patients with large defects, all remained large. Overall the defects enlarged in 65% of patients and some to the extent (>20 mm) that they could not be closed by transcatheter techniques.

The presence and severity of functional limitation among patients with ASDs seem to increase with age. Congestive heart failure rarely is found in the first decade of life, but it can become common once the patient is older than 40 years of age. The onset of atrial fibrillation or less commonly atrial flutter can be a hallmark in the course of patients with ASDs. The incidence of atrial arrhythmias increases with advancing age to as high as 13% in patients older than 40 years of age and 52% in those older than 60 years of age.

Pulmonary vascular disease can occur in 5% to 10% of patients with untreated ASDs, predominantly in females. Usually it occurs after 20 years of age, although rare cases in early childhood have been recorded. Debate continues about what causes pulmonary vascular obstructive disease, which patients are at risk and at what age it occurs. It does not appear simply to be the magnitude of the shunt persisting for several decades. An untreated symptomatic ASD leads to significant morbidity and reduces life expectancy. Complications of an untreated ASD can include right ventricular failure, atrial arrhythmias, paradoxical embolism, pulmonary hypertension and cyanosis secondary to reversal of shunt from pulmonary vascular disease. Mortality rates for untreated ASDs are as high as 25%.

American Heart Association (AHA) guidelines recommend closure of secundum ASDs, either surgically or percutaneously, in patients with right atrial heart volume overload. [AHA/ACC – 2008 guidelines Closure of an ASD either percutaneously or surgically is indicated for right atrial and RV enlargement with or without symptoms. (Class I, Level of Evidence: B)]
Prior to the advent of interventional catheter procedures for major ASDs (Qp:Qs ratio >1.5:1) in children and young adults, surgical repair was the treatment of choice. Since most ASDs are well tolerated in infancy and may spontaneously close, elective repair frequently has been deferred until the child is at least 4 years of age. In some patients with very large ASDs, closure is done at younger ages. There is no advantage in delaying repair much beyond this age and there may be harm in delaying repair to the teenage years and beyond. Long-standing volume overload of the right atrium and ventricle causes certain irreversible changes in the right atrium and right and left ventricles that possibly contribute to atrial arrhythmias and premature death. Early operation has been recommended for those young children who have unremitting heart failure or associated pulmonary hypertension. Indications for closure of an ASD in adults have been controversial. In 1970, Campbell et al reported that adults with ASDs appeared to die at an earlier age than normal adults of the same age and gender.

In 1994, Shah et al reported on a selected group of 82 adults with uncomplicated ASDs and no pulmonary hypertension, only some of whom had undergone repair. All patients were older than 25 years of age at presentation and were older than 45 years of age at the time of study. The investigators found no difference in survival or symptoms between the medically and surgically treated groups.

There were no differences in incidence of new supraventricular arrhythmia (including atrial fibrillation), stroke, embolic phenomenon, or cardiac failure. Limitations of this study included non random assignment of medical versus surgical therapy, exclusion of patients with pulmonary hypertension and the proportion of patients lost to follow-up (22%). Long-term follow-up data from Murphy et al suggested that adults older than 41 years of age derived minimal benefit from operative closure.
Catheter Device Closure

Transcatheter techniques for closure of ASDs have been available for several years. Transcatheter closure of secundum ASDs has been demonstrated to be safe and effective in both children and adults, with similar success and complication rates to surgery, and the potential for decreased hospital stay.

In February 2012, Kutty et al. reported the results of a retrospective observational outcomes study published in the American Journal of Cardiology, comparing long-term results of transcatheter and surgical ASD closure. This study represents the longest reported duration of follow-up after transcatheter closure with a mean follow-up of 10 years. Overall complication rates observed in both groups were low and differed by less than 2%. All-cause mortality rates following transcatheter ASD closure compare favourably to surgical closure.

These results validate transcatheter secundum ASD closure as a safe and effective therapeutic option for patients in whom the need for defect closure is clearly indicated.

In 1976, King et al. reported the first transcatheter closure of a secundum ASD in humans with a double-umbrella device. It was successful in five of ten patients. Since then, devices have undergone several evolutionary changes in an attempt to improve the design and the technique of delivering the devices. Problems encountered using these devices included residual shunt, fractures of the hardware, embolization of the device, large delivery systems, and defects too large to close.

Masura et al. reported 4.7- to 9-year (median 6 years) follow-up of 151 patients (average age 12 years; average weight 36 kg.) who had transcatheter closure of ASDs from September 1995 to January 2000. Complete closure was seen at 3-year follow-up, and there were no deaths or significant complications. Erosion and thrombus formation have occurred with use of septal occluder devices. The incidence of device erosion was reported at 0.1%. Most occur in the first 72 hours, but one case occurred 3 years after implant. In a single-centre report of 407 patients where 9 different atrial septal occluder devices were used, thrombus formation in the right or left atrium was found in 1.2% of patients.
Review of Literature

Temporary atrioventricular block (first, second, and third) has been reported in a few patients, all resolving by 6 months after the procedure.

Extensive literature review to trace the long term outcome after transcatheter closure of ASD in adult and paediatric population showed that not many studies conducted in this regard. Few studies cited down are about adult population.

Jategonkar et al\textsuperscript{50} did an observational study on 96 patients with significant L to R shunt symptoms. Mean age was 69.9±5.3 years with follow up period of 3 months. Mean ASD size was 14.8±5.8mm. There were no major procedure related complications. They found that RV end diastolic volume was reduced from 38.9±8.7 to 32.3±8. 16 patients had new onset of atrial tachyarrhythmias on follow up. Clinical status/CV and cerebrovascular symptoms/PA pressures were not assessed on follow up. Tiny shunt was found in 16.9% on follow up.

Humengerer et al\textsuperscript{51} did an observational follow up study in 236 patients with ASD size of 22mm, volume overload features, age at procedure was 49±18 years with Qp:Qs 2.2:1. No major complications encountered during the procedure. On mean of 12 months follow up they found that no residual defects, RVEDV reduced from 43±7 to 34±6mm. 11 patients had new onset atrial fibrillation.

Giardini et al\textsuperscript{52} did an observational follow up study in 134 patients with ASD size of 18±5mm, volume overload features. Age at procedure was 39±19 years, with Qp:Qs >1.5:1. No major complications encountered during the procedure. On mean of 4.8±2.7 years follow up they found no residual defects, 8 patients had new onset atrial fibrillation.

Majunke et al\textsuperscript{53} did an observational follow up study in 650 patients with ASD size of 21.2±5.2 mm, volume overload features. Age at procedure was 45.8±16.2, with Qp:Qs ~1.9:1. No major complications encountered during the procedure. On mean of 8 years follow up they found that 3.6% residual defects, RVEDV reduced from 33.3±10.6 to 23.6±10.1mm. 4.3 % patients had new onset atrial fibrillation.

Knepp et al\textsuperscript{54} did a retrospective study in 65 patients with ASD size of 18mm, volume overload features. Age at procedure was 49.4±15.4 years. No major complications encountered during the procedure. On mean of 73 months follow up they found that no residual defects, 3 patients had new onset atrial fibrillation.
Review of Literature

Patel et al\textsuperscript{55} did a retrospective study in 113 patients with ASD size of 17.2±7.3mm, volume overload features, age at procedure was 57.9±11.6 years, with Qp:Qs 2.2:1. No major complications encountered during the procedure. On mean of 37 months follow up they found that 6% residual defects, RVEDV reduced from 33.3±7.6 to 23.8±6.6mm. 2 patients had new onset atrial fibrillation.

Jategonkar et al\textsuperscript{56} did a retrospective study also in 332 patients with ASD size of 14.7±5.3mm, volume overload features, age at procedure was 49.4±18 years. No major complications encountered during the procedure. On mean of 904±839 days follow up they found that 12.3% residual defects, RVEDV reduced from 36.4±7.9 to 29.4±6.8mm. 1.5% patients had new onset atrial fibrillation.
AIMS AND OBJECTIVES
Aims and Objectives

To assess the intermediate and long term clinical outcome after transcatheter closure of OS ASD in adult and paediatric patients

To assess the long term complications after device closure.

To assess the changes in ECG and Echo features post device closure
MATERIALS AND METHODS
Materials and Methods

It was a single centre, retrospective cross sectional observational study conducted at Sree Chitra Tirunal Institute For Medical Sciences and Technology, Thiruvananthapuram, Kerala

Study period and study population

This study was conducted over a period of 18 months from January 2012 to June 2013, on the follow up patients who have undergone ASD device closure on or before 30-06-2008 from Sree Chitra Tirunal Institute For Medical Sciences and Technology, Thiruvananthapuram, Kerala

Design

Cross sectional clinical and non-invasive evaluation

Eligibility criteria

All those patients who have undergone successful ASD device closure on or before 30-06-2008 from Sree Chitra Tirunal Institute For Medical Sciences and Technology, Thiruvananthapuram, Kerala and willing to participate in our study.

Methods

All of the eligible patients were called by post/mail/telephone to participate in the study. All subjects who responded and gave their consent to participate were included in the study.

Baseline characteristics (pre device closure) of all patients who participated in the study were traced out from the hospital records. Symptoms, clinical findings, CXR (Chest radiography), ECG (Electrocardiography), TTE (Transthoracic echocardiography), TEE (Transesophageal echocardiography) findings, hemodynamic data during device closure, age at procedure, immediate post procedure/in hospital complications were reviewed.

On follow up, all these patients, who participated in the study, were evaluated for Clinical outcome, ECG changes along with Transthoracic (TTE) and
Materials and Methods

transesophageal echocardiography (TEE) assessment. Patient population was classified into two groups according to their age at the time of device closure as > 12 years and <12 years groups.

Detailed history was elicited to evaluate the long term clinical outcome Primary outcome was defined as cardiovascular (CV) mortality, cerebrovascular accident/transient ischemic attack (CVA/TIA), Arrhythmias/palpitations

Secondary outcome parameters were incidence of recurrent headache/migraine, chest pain, dyspnoea and in paediatric patients growth pattern, recurrent infections.

Any of the non cardiac symptoms if complained were thoroughly evaluated by the concerned unit.

Thorough and complete clinical examination was conducted with special attention to cardiomegaly, second sound, flow murmur/new murmurs. Cardiomegaly was defined by the displaced apical impulse. Second sound was evaluated for nature of split, intensity of aortic and pulmonary components. Murmurs if any were described as new, persistent (comparing with predevice closure hospital records) along with their location, type, grade, character, duration, conduction, radiation and variation with dynamic exercises.

Standard 12 lead ECG was performed in all using MORTARA ELI 250 machine. Rhythm, QRS axis, PR interval, RBBB, RVH was assessed.

Transthoracic echo was performed in all using HD 11- PHILIPS and ENVISOR-PHILIPS machines. Residual defect, device related issues like obstruction/regurgitation of valves, RV size/function, LV function, TR and PAH, device position were specifically looked for. TTE was performed by 2 examiners and findings were confirmed. All the 4 standard views performed and modified views were performed whenever necessary.

RV size was measured in parasternal long axis view. TR and RVSP were assessed in apical 4 chamber view. Device position and residue along with any complications like regurgitation-obstruction were meticulously looked for in all standard and modified views.
Materials and Methods

TEE (Trans-esophageal echo) was performed in all those patients who gave consent for the same. PHILLIPS IE 33 was used for this purpose. 2% lignocaine was used for surface anaesthesia. Intracardiac anatomy and device status were evaluated in 0°, 30°, 45°, 60°, 90°, 120° along with modified views if required. TEE images were reviewed by 2 senior cardiologists. Residual defect, device position, device related valvular complications like obstruction/regurgitation, MR/TR/PS progression, Aortic erosion, thrombus or any complications were specifically looked for.

Long term intracardiac complications after device closure (TEE) looked for as described in the literature search were,

1. Device embolization
2. Device Arm Fracture
3. Device/intracardiac thrombosis
4. Erosion
5. Device related valvular dysfunction, outflow tract obstruction
6. Infective endocarditis

STATISTICAL ANALYSIS

SPSS 11.0 version was used to analyse the data.

Chi square test was used to compare categorical variables between two groups (>12 years and <12 years at the time of device closure). Student t test was used to compare mean values of quantitative variables between two groups. Paired t test was used to assess the average change in quantitative variables pre and post procedure (device closure); was analysed overall and separately in groups (> 12 years and > 12 years). Wilcoxin signed rank test was used to assess the change in graded variables between two stages (pre and post procedure).
OBSERVATIONS AND RESULTS
Between January-1998 and June 2008, 291 patients of OS (ostium secundum) ASD underwent successful transcatheter closure in SCTIMST. All of them were called by mail/telephone calls to participate in the study.

All subjects who gave their consent to participate in the study were included. 15 (5.2%) patients were lost to follow up after procedure. 19 (6.5%) patients had < 54 months of follow up. 23 (7.9%) patients were contacted on phone hence had incomplete data. 18 (6.2%) patients had follow up > 60 months, but did not give consent for Echo'/ECG hence had incomplete data (7.9%). Hence, 216 had complete data (74.3%).

Baseline characteristics of all patients who underwent procedure were traced out from hospital records. Symptoms, clinical findings, ECG, TTE, TEE findings, hemodynamic data during procedure, age at procedure, immediate post procedure/in hospital complications were reviewed. Patient population was classified into < 12 years and > 12 years (at the time of device closure) Mean age for < 12 y was 6.65 (SD 2.1) Mean age for > 12 years 29.7 (SD 13.01) Total follow up period was 79.3 ± 27.3 months. < 12 years group had mean follow up period of 77.2 ± 26.7 months. > 12 years group had mean follow up period 80.4 ± 27.6 months

Baseline characteristics

154 (71.3%) patients were females [55 in < 12 years (71.4%) group and 99 (71.2%) in > 12 years group respectively (p- 0.97).] 14 (6.5%) patients had history of failure to thrive [12 in < 12 years (15.6%) group and 2 (1.4%) in > 12 years group respectively p-<0.05]. 46 (21.3%) patients had history of recurrent respiratory tract infections [34 in < 12 years (44.2%) group and 12 (8.6%) in > 12 years group respectively p-<0.05). 120 (55.6%) had exertional dyspnoea [24 in < 12 years (31.2%) group and 96 (69.1%) in > 12 years group respectively p-<0.05]. 43 (19.9%) had exertional palpitations with no documented arrhythmias [3 in < 12 years (3.9%) group and 40 (28.8%) in > 12 years group respectively p-<0.0.5] 29 (13.4%) patients gave history of atypical chest pain [3 in < 12 years (3.9%) group
Observations and Results

and 26 (18.7%) in > 12 years group respectively p <0.05]. 9 (4.2%) patients had presyncope/syncope [1 in < 12 years (1.3%) group and 8 (5.8%) in > 12 years group respectively p <0.05] One (0.5%) was a case of post operative case with residue. One patient (0.5%) in > 12 years group had documented atrial fibrillation which was reverted with amiodarone prior the procedure. Baseline characteristics are shown in table 1 and Fig1 and 2.

Table 1 Baseline characteristics

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>&lt; 12 years</th>
<th>&gt; 12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>154 (71.3)%</td>
<td>55 (71.4)%</td>
<td>99 (71.2)%</td>
</tr>
<tr>
<td>FTT</td>
<td>14 (6.5)%</td>
<td>12 (15.6)%</td>
<td>2 (1.4)%</td>
</tr>
<tr>
<td>Recurrent LRTI</td>
<td>46 (21.3)%</td>
<td>34 (44.2)%</td>
<td>12 (8.6)%</td>
</tr>
<tr>
<td>Exertional Dyspnoea</td>
<td>120 (55.6)%</td>
<td>24 (31.2)%</td>
<td>96 (69.1)%</td>
</tr>
<tr>
<td>Exertional Palpitations</td>
<td>43 (19.9)%</td>
<td>3 (3.9)%</td>
<td>40 (28.8)%</td>
</tr>
<tr>
<td>Atypical Chest Pain</td>
<td>29 (13.4)%</td>
<td>3 (3.9)%</td>
<td>26 (18.7)%</td>
</tr>
<tr>
<td>Syncope /Presyncope</td>
<td>9 (4.2)%</td>
<td>1 (1.3)%</td>
<td>8 (5.8)%</td>
</tr>
<tr>
<td>Family H/o ASD</td>
<td>1 (0.5)%</td>
<td>1 (1.3)%</td>
<td>0 (0)%</td>
</tr>
<tr>
<td>Documented AF</td>
<td>1 (0.5)%</td>
<td>0 (0)%</td>
<td>1 (0.7)%</td>
</tr>
<tr>
<td>Post op residual ASD</td>
<td>1 (0.5)%</td>
<td>0 (0)%</td>
<td>1 (0.7)%</td>
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</table>
Fig 1  Symptoms at presentation (in > 12 years and < 12 years compared)

Fig 2  Symptoms at presentation (in > 12 years and < 12 years compared)
Clinical findings (from hospital records) at the time of device closure

179 (82.9%) patients had cardiomegaly [60 in < 12 years (77.9%) group and 119 (85.6%) in > 12 years group respectively p-0.15]. 215 (99.5%) had wide and fixed split second heart sound. P2 was loud in 96 (44.4%) patients [31 in < 12 years (40.3%) group and 65 (46.8%) in > 12 years group respectively p-0.45]. 211 (97.7%) patients had flow murmur [75 in < 12 years (97.4%) group and 136 (97.8%) in > 12 years group respectively p-0.84]. Clinical findings at presentation are shown in table 2 and Fig 3.

### Table 2 Clinical findings at presentation

<table>
<thead>
<tr>
<th>Clinical sign</th>
<th>Total</th>
<th>&lt; 12 years</th>
<th>&gt; 12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiomegaly</td>
<td>179 (82.9)</td>
<td>60 (77.9)</td>
<td>119 (85.6)</td>
</tr>
<tr>
<td>Wide Fixed Split S₂</td>
<td>215 (99.5)</td>
<td>77 (100)</td>
<td>138 (99.3)</td>
</tr>
<tr>
<td>P₂ loud</td>
<td>96 (44.4)</td>
<td>31 (40.3)</td>
<td>65 (46.8)</td>
</tr>
<tr>
<td>Flow Murmur</td>
<td>211 (97.7)</td>
<td>75 (97.4)</td>
<td>136 (97.8)</td>
</tr>
</tbody>
</table>
Observations and Results

Fig 3 Clinical findings at presentation (> 12 years and < 12 years compared)
Observations and Results

Baseline ECG pattern

215 (99.5%) were in sinus rhythm. One patient (0.5%) in > 12 years group had documented atrial fibrillation which was reverted with amiodarone prior the procedure. PR interval was normal in all patients. (151.6±19.6ms in < 12 years group and 165.7±25.2 in > 12 years group respectively). Mean QRS axis was 77.9±32.2 (76.7±31.1 in < 12 years group and 78.5±32.9ms in > 12 years group respectively with p-0.68). 174 (80.6%) patients had features of right bundle branch pattern [59 in < 12 years (76.6%) group and 115 (82.7%) in > 12 years group respectively p-0.47]. ECG findings pre-closure are shown in table 3

<table>
<thead>
<tr>
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<th>Total</th>
<th>&lt; 12 years</th>
<th>≥12 years</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus rhythm</td>
<td>215 (99.5)</td>
<td>77 (100)</td>
<td>138 (99.3)</td>
<td>-</td>
</tr>
<tr>
<td>PR</td>
<td>160.6 ± 24.3</td>
<td>151.6 ± 19.6</td>
<td>165.7 ± 25.2</td>
<td>-</td>
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<tr>
<td>AXIS</td>
<td>77.9 ± 32.2</td>
<td>76.7 ± 31.1</td>
<td>78.5 ± 32.9</td>
<td>0.68</td>
</tr>
<tr>
<td>IRBB</td>
<td>174 (80.6)</td>
<td>59 (76.6)</td>
<td>115 (82.7)</td>
<td>0.47</td>
</tr>
</tbody>
</table>

Baseline echocardiographic findings

All had good LV and RV functions. ASD size was 16±5.2 [13.1±3.5mm in < 12 years group and 17.6±5.3 mm in > 12 years group respectively p<0.05). 4 (1.9%) patients had multiple defects 2 in each group (2.6% in < 12 years group and 1.4% in > 12 years group respectively). 214 (99%) had normal pulmonary venous
Observations and Results

drainage. 2 (0.93%) patients (both in > 12 years group) had partial anomalous pulmonary venous drainage. Both had RUPV draining into to SVC. 33 (15.3%) patients had tricuspid regurgitation (grade 2 or more) [9 in < 12 years (11.7%) group and 24(17.2%) in > 12 years group respectively p-0.5). Mitral regurgitation (grade II or more) was present in 25 (11.6%) patients (3 in < 12 years (3.9%) group and 11(7.9%) in > 12 years group respectively). Right ventricular systolic pressure (PA pressure) by TR jet was 28.5±9.1mm Hg [28.5±7.2 in < 12 years group and 29.9±9.7 in > 12 years group respectively with p < 0.05]. MVP was present in 14 (6.5%) patients (4 in < 12 years (5.2%) group and 10 (7.2%) in > 12 years group respectively). Mild valvular pulmonary stenosis was present in 8 (3.7%) patients (4 (5.2%) in < 12 years group and 4 (2.9%) in > 12 years group respectively).

Enlarged RV (defined as > 50% of LV size in parasternal long axis view) was found in 176 (81.5%) patients [63 in < 12 years (81.8%) group and 113 (81.3%) in > 12 years group respectively p-0.92]

<table>
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<th>&lt; 12 years</th>
<th>≥ 12 years</th>
</tr>
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<tbody>
<tr>
<td>NORMAL PVD</td>
<td>214 (99.0)</td>
<td>77 (100)</td>
<td>137 (99.3)</td>
</tr>
<tr>
<td>Multiple ASD</td>
<td>2 (0.9)</td>
<td>0 (0)</td>
<td>2 (1.4)</td>
</tr>
<tr>
<td>RV size Enlarged</td>
<td>176 (81.5)</td>
<td>63 (81.8)</td>
<td>113 (81.3)</td>
</tr>
<tr>
<td>MVP</td>
<td>14 (6.5)</td>
<td>4 (5.2)</td>
<td>10 (7.2)</td>
</tr>
<tr>
<td>PS</td>
<td>8 (3.7)</td>
<td>4 (5.2)</td>
<td>4 (2.9)</td>
</tr>
</tbody>
</table>
Observations and Results

Cath data during device closure

168 (77.8%) patients had Qp:Qs ratio > 2:1 [57 in < 12 years (74%) group and 111 (79.9%) in > 12 years group respectively p-0.45]. 48 (22.2%) patients had Qp:Qs ratio between 1.5: to 2:1 [20 in < 12 years (26%) group and 28 (20.1%) in > 12 years group respectively p-0.54].

RA mean pressure was 6.9±3.4mm Hg (5.6±2.8 in < 12 years group and 7.6±3.5 in > 12 years group respectively with p-0.007). RVSP was 32.3±9 mmHg (29.2 ± 6.9 in < 12 years group and 34.1 ± 9.6 in > 12 years group respectively with p-0.009). RV end diastolic pressure was 7.5±3.1mm Hg (6.5 ±2.7 in < 12 years group and 8.1±3.2 in > 12 years group respectively with p-0.002). Average PA pressure was 29.2/13.6 (19.8) [25.8/12 (17.7) in < 12 years group and 30.9/14.4 (21) in > 12 years group respectively with p-0.001]

Device to ASD size ratio was 1.33:1 (1.34:1 in < 12 years group and 1.29:1 in > 12 years group respectively).

Device size used was 21.3±5.3mm (17.5±3.9mm in < 12 years group and 23.4±4.8mm in > 12 years group respectively). Device type used were AMPLANTZER (77 cases, 35.6%) HEART-R (63 cases, 29.2%). BLOCKAID (76 cases, 35.2%) respectively. In < 12YRS group device 28 AMPLANTZER (36.4%) 26 HEART-R (33.8%) 23 BLOCKAID (29.9%) devices were used. In >12YRS group device 49 AMPLANTZER (35.3%) 37 HEART-R (26.6%) 53 BLOCKAID (38.1%) devices were used.

LA approach 203 (94%) was used in 203 procedures [73 (94.8%) in < 12 years group and 130 (93.5%) > 12 years group respectively]. RUPV approach was used in 10 (94%) procedures [3 (3.9%) in < 12 years group and 7 (5%) > 12 years group respectively]. LUPV approach was used in 3 (1.4%) procedures [1 (1.3%) in < 12 years group and 2 (1.4%) > 12 years group respectively]. Cath findings during device closure (> 12 years and < 12 years compared) are showed in table 5 AND Fig 4.

2(0.9%) patients had tiny residue and 2(0.9%) had left alone additional ASD during closure.
### Observations and Results

**Table 5 Catheterization findings during device closure**

(> 12 years and < 12 years compared)

<table>
<thead>
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<th>Total</th>
<th>&lt; 12 years</th>
<th>&gt;= 12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Qp:Qs &gt;2:1</td>
<td>168 (77.8)</td>
<td>57 (74)</td>
<td>111 (79.9)</td>
</tr>
<tr>
<td>Qp:Qs 1.5:1 to 2:1</td>
<td>33 (15.3)</td>
<td>13 (16.9)</td>
<td>20 (14.4)</td>
</tr>
<tr>
<td>Qp:Qs ≤1.5:1</td>
<td>15 (6.9)</td>
<td>7 (9.1)</td>
<td>8 (5.8)</td>
</tr>
<tr>
<td>LA approach</td>
<td>203 (94)</td>
<td>73 (94.8)</td>
<td>130 (93.5)</td>
</tr>
<tr>
<td>RUPV approach</td>
<td>10 (4.6)</td>
<td>3 (3.9)</td>
<td>7 (5)</td>
</tr>
<tr>
<td>LUPV approach</td>
<td>3 (1.4)</td>
<td>1 (1.3)</td>
<td>2 (1.4)</td>
</tr>
</tbody>
</table>
Intraprocedure period and Hospital stay post procedure were largely uneventful. During procedure one child had transient SVT, and one > 12 years age group patient had transient ST change both not requiring any active intervention. One patient (> 12 years group) had transient atrial tachycardia requiring no active intervention during first 24 hrs of hospital stay.

Clinical outcome on follow up

Among 216 (74.2%) none had CVA/TIA/CV mortality.

All patients were free from dyspnoea/growth disturbances/recurrent respiratory infections. None (in both groups) had presyncope/syncope.

2 (0.9%) had exertional palpitations (both in > 12 years group) and evaluated further with 24 hours. Holter study – no arrhythmia documented. They were further evaluated for thyroid abnormalities and kept on medical follow up. 1 patient (0.5%) had asymptomatic atrial tachycardia (> 12 years group) and was put on oral anticoagulation. This patient was aged 59 years with systemic hypertension and
diabetes. She did not have any residue/pulmonary hypertension on evaluation. 1 (0.5%) patient (> 12 years group) was on permanent pacemaker (VVI) for sick sinus syndrome and was doing fine at follow up.

6 (2%) had atypical chest pain (all in > 12 years group), with normal left ventricular function, negative TMT. However 2 of them had already undergone coronary angiogram from elsewhere with normal findings.

1 (0.4%) had migraine (> 12 years group) with family history of migraine, put on prophylaxis and kept under follow up with Neurologist This patient had no residual defect or pulmonary hypertension.

On clinical examination one (> 12 years group) had cardiomegaly which was attributed to hypertensive heart disease. All patients in both groups had normal second sound. Systolic murmur found in 6 (2.8%) patients was attributed to the underlying mild valvular PS which did not progress on follow up. [2 (2.6%) in < 12 years group and 4 (2.9%) in > 12 years group]. Clinical outcome on follow up is shown in table 6.

**Table 6  Clinical outcome on follow up**

<table>
<thead>
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<th>Total</th>
<th>&gt; 12 years</th>
<th>&gt;= 12 years</th>
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<tbody>
<tr>
<td>CVA/TIA</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>CV mortality</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Recurrent LRTI</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
</tr>
<tr>
<td>Exertional Dyspnoea</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
</tr>
<tr>
<td>Exertional Palpitations</td>
<td>2 (0.9)</td>
<td>0 (0)</td>
<td>2 (1.4)</td>
</tr>
<tr>
<td>Migraine</td>
<td>1 (0.5)</td>
<td>0 (0)</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>1 (0.5)</td>
<td>0 (0)</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>Normal Second Sound</td>
<td>215 (99.5)</td>
<td>77 (100)</td>
<td>138 (99.3)</td>
</tr>
<tr>
<td>Murmur</td>
<td>6 (2.8)</td>
<td>2 (2.6)</td>
<td>4 (2.9)</td>
</tr>
<tr>
<td>Chest pain -- atypical</td>
<td>6 (2.9)</td>
<td>0 (0.0)</td>
<td>6 (1.4)</td>
</tr>
</tbody>
</table>
Observations and Results

ECG changes on follow up

214 (99%) patients were in sinus rhythm. 1 patient (0.5%) had asymptomatic atria tachycardia (> 12 years group). 1 (0.5%) patient (> 12 years group) was on permanent pacemaker (VVI) for sick sinus syndrome.

PR interval was normal on follow up (158.5 ± 20.6 msec) (152.7 ± 17.9 msec in < 12 years group and 161.8 ± 21.4 > 12 years group respectively). No significant changes in PR (p – 0.24) compared with pre closure.

QRS axis on follow up was 44.7±24.7 [47.3±22.7 in < 12 years group and 43.3±25.7 in > 12 years group respectively]. Overall axis shifted from 77.9 ± 32.2 to 44.7 ± 24.7 (< 0.05). In < 12 years group axis shift was from 76.7±31.1 to 47.3±22.7 p<0.005. In < 12 years group axis shift was from 78.9±32.5 to 43.3±25.7 p<0.004.

22 (10.2%) showed persistence of in IRBB [9 (11.7%) in < 12 years group and 13 (9.4%) in > 12 years group respectively]. 152 (70.4%) patients showed disappearance of RBBB. [50 (65%) in < 12 years group and 102 (73.3%) in > 12 years group respectively]. ECG findings on follow up showed in table 7 and Fig 5-8.

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>&lt; 12 years</th>
<th>≥12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus rhythm</td>
<td>214 (99%)</td>
<td>77 (100%)</td>
<td>137 (99%)</td>
</tr>
<tr>
<td>AT</td>
<td>1 (0.5)</td>
<td>0 (0)</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>Pacemaker Rhythm</td>
<td>1 (0.5)</td>
<td>0 (0)</td>
<td>1 (0.7)</td>
</tr>
<tr>
<td>RBBB</td>
<td>22 (10.2%)</td>
<td>9 (11.7%)</td>
<td>13 (9.4%)</td>
</tr>
<tr>
<td>PR</td>
<td>158.5 ± 20.6</td>
<td>152.7 ± 17.9</td>
<td>161.8 ± 21.4</td>
</tr>
<tr>
<td>AXIS</td>
<td>44.7 ± 24.7</td>
<td>47.3 ± 22.7</td>
<td>43.3 ± 25.7</td>
</tr>
</tbody>
</table>
Observations and Results

Fig 5 ECG findings on follow up

Fig 6 ECG compared Pre and post Device Closure (all patents)
Fig 7 ECG compared Pre and post Device Closure (< 12 years group)

Fig 8 ECG compared Pre and post Device Closure (> 12 years group)

Observations and Results
On Transthoracic echocardiography

All (both groups) had good RV/LV function. 17(7.9%) patients had suspicion of residual defect [4 patients (5.2%) in < 12 years group and 13 patients (9.4%) in > 12 years group].

RV size was 16.4±4.5mm [15.4±3.8mm in <12 years group and 17±4.8mm in >12 years group p-0.01]. Mean RV size reduced from 23.9±7.2 to 16.4 ± 4.5 (p < 0.05) However, RV size was still enlarged in 34 (15.7%) patients [14 patients (18.2%) in < 12 years group and 20 patients (14.4%) in > 12 years group p-0.01]

RVSP measured by TR jet velocity was 19.7±4.8 (18.4±3.8 Hg in <12 years group and 20±5.2mm Hg in >12 years group). RVSP by TTE reduced from 28.5 ± 9.1 to 19.7 ± 4.8 (p < 0.05)

8 (3.7%) patients had tricuspid regurgitation (grade 2 or more) (2 in < 12 years (2.6%) group and 6 (4.3%) in > 12 years group respectively) Mitral regurgitation (grade II or more) was present in 9 (4.2%) patients (4 in < 12 years (5.2%) group and 5(3.6%) in > 12 years group respectively). Significant decrease in regurgitation grade (p-0.01 for MR and p-0.03 for TR). No features of device related valvular obstruction/new regurgitation were observed. No progression of valvular stenosis/regurgitation were seen on follow up. TTE findings on follow up showed in table 8 and Fig 9-11.

Table 8  TTE findings on follow up

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>&lt; 12 years</th>
<th>≥12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>MR Grade II</td>
<td>9 (4.2)</td>
<td>4 (5.2)</td>
<td>5 (3.6)</td>
</tr>
<tr>
<td>Enlarged RV size</td>
<td>34 (15.7)</td>
<td>14 (18.2)</td>
<td>20 (14.4)</td>
</tr>
<tr>
<td>RESIDUAL ASD</td>
<td>17 (7.9)</td>
<td>4 (5.2)</td>
<td>13 (9.4)</td>
</tr>
<tr>
<td>RVOTO (Mild)</td>
<td>10 (4.6)</td>
<td>5 (6.5)</td>
<td>5 (3.6)</td>
</tr>
</tbody>
</table>
Observations and Results

Fig 9  TTE findings pre and post device closure (overall)

Fig 10  TTE findings comparison pre and post device closure
(< 12 years group)
On Transesophageal echocardiography

186 (85.7%) patients underwent TEE evaluation. Intracardiac anatomy and device status were evaluated in 0°, 30°, 45°, 60°, 90°, 120°; along with modified views if required.

16 out of 17 suspected residual defects on TTE were found to have IVC jet hitting the LA disc of the device and coming back to RA giving false impression of residual defect.

One (0.5%) had a residual defect with malalignment at aortic rim, with no features of volume overload. This patient was in sinus rhythm, had stable device position, no history of infective endocarditis. Hence, kept on medical follow up.

None of the patients had device embolization/unstable position. None of them had features of device erosion/disc fracture. None of them had evidence of device related valvular dysfunction (obstruction/regurgitation)

One lady was found to have milder form of Ebsteins anomaly with no RVOTO, normal RV function with no residual defect.
Observations and Results

None of them had definite e/o thrombus; however one had suspicion of LA thrombus. This patient was worked up for infective endocarditis (negative) and coagulation profile (normal prothrombin time/aPTT) negative d-dimer, normal platelet count, good LV/RV function. He received 3 months anticoagulation. TEE findings on follow up shown in Table 9 and fig 12-19.

It is important to note that another 41 patients (14.1%) (Apart from patients with complete data as described above) had incomplete data; Data was collected on phone call and from available hospital records (if f/u period is > 60 months)

None of them had primary outcomes. None had residue/PAH cardiomegaly/RV enlargement. Some patients’ present medical details (evaluated elsewhere by Cardiologists) were collected via conversations with the patient and/or treating Cardiologist, letter (questionnaire) returned by the patients.

Table 9 TEE findings on follow up

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>&lt; 12 years</th>
<th>≥12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEE Residue</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Malaligned at AO RIM)</td>
<td>1 (0.5)</td>
<td>1 (1.3)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Erosion</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
<td>0(0.0)</td>
</tr>
<tr>
<td>New onset LV/RV OTO</td>
<td>0 (0.0)</td>
<td>0 (0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Device arm fracture</td>
<td>0(0)</td>
<td>0(0)</td>
<td>0(0)</td>
</tr>
<tr>
<td>MR Grade II</td>
<td>10 (4.6)</td>
<td>4 (5.2)</td>
<td>6 (4.3)</td>
</tr>
<tr>
<td>Mild Ebsteins</td>
<td>1 (2.6)</td>
<td>0 (0)</td>
<td>1 (4.8)</td>
</tr>
<tr>
<td>? LA Thrombus</td>
<td>1 (2.6)</td>
<td>1 (5.6)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>
Observations and Results

Fig 12 TEE image [0°] in a patient with ASD device with no residue/complications

Fig 13 TEE image [30°] in a patient with ASD device with no residue/complications
Fig 14 TEE image [60°] in a patient with ASD device with no residue/complications

Fig 15 TEE image [90°] in a patient with ASD device with no residue/complications
Fig 16 TEE image [120°] in a patient with ASD device with no residue/complications
Fig 17 TEE images showing IVC jet hitting LA disc giving suspicion of residue on TTE
Fig 18 TTE/TEE images of a milder Ebsteins with ASD device in situ
Fig 19 TEE image in a patient with ASD device in situ with suspicion of thrombus.
DISCUSSION
This study not only evaluated the intermediate and long term clinical outcome after ASD percutaneous closure but also the ECG changes, RV size and PA pressure changes on echo, along with long term device related complications. TEE evaluation which is considered as the gold standard in case of ASD pre and post closure. The strength of this study was that 85.7% of the patients underwent TEE evaluation on follow up.

Baseline data was analysed with special reference to the age at presentation and at procedure. Analysis of baseline characteristics reveals that incidence of OS ASD is more in females with ratio ~2.5:1. Familial incidence of 0.5%, multiple ASDs in 2.8% cases was consistent with other studies. Associated MVP (6.5%) valular PS (3.7%) are in accordance with other studies1, 3, 4.

Contrary to the popular belief, children with ASD had recurrent respiratory infections (44.2%) and failure to thrive (15.2%) in our study. However, this may at least partly contributed by primary protein-energy malnutrition prevalent in our setup. Exertional dyspnoea (69.1% vs 31.2% p< 0.05) and palpitations (28.8 %vs 2.9% p< 0.05) were more common in age group > 12years as expected. Reason for this is the progressive dilatation of RV along with LV compliance related issues with advancing age. History of syncope/presyncope (5.8% vs 1.3% p < 0.05) and documented AF (1.4% VS 0) again signifies the need of early intervention in patients with ASD.

Cardiomegaly was found in 179 (82.9%) patients [60 in < 12 years (77.9%) group and 119 (85.6%) in > 12 years group respectively p-0.15] reflecting age independent effect of ASD shunt on cardiac size. However, heart tends to increase in size with age for a given amount of shunt. Nearly all patients with ASD will have wide and fixed split second heart sound 215 (99.5%). Loud P₂ in ASD with significant shunt (irrespective of age) may not necessarily indicate pulmonary hypertension as found in our study; 96 (44.4%) patients had loud P₂ [31 in < 12 years (40.3%) group and 65 (46.8%) in > 12 years group respectively p-0.45]. Nearly all ASDs with significant shunt (irrespective of age) will have patients flow murmur across the pulmonary and tricuspid valves as found in our study; 211 (97.7%) [75 in < 12 years (97.4%) group and 136 (97.8%) in > 12 years group respectively p-0.84].
Majority of ASDs will have sinus rhythm on ECG [215 (99.5%) in our study]. PR interval is usually normal. QRS axis tends to shift to the right in both children and adults with ASD; 77.9±32.2 (76.7±31.1 in < 12 years group and 78.5±32.9ms in > 12 years group respectively with p-0.68). Incomplete/complete right bundle branch pattern RBB incidence is high in patients with ASD 174 (80.6%); it does not have statistically significant difference in paediatric and adult population; but tends to be more in > 12 years group (82.7% vs 76.6% p-0.47) This finding is different from other studies mainly because of patient selection at appropriate/early age for closure of ASD.

ASDs generally will have normal RV and LV functions. ASD tends to increase in size with age [16±5.2 [13.1±3.5mm in < 12 years group and 17.6±5.3 mm in > 12 years group respectively p<0.05] However this may just be an observation, serial follow up in an untreated ASD will give us the information on this. Majority of ASDs are single and clinical presentation of multiple ASDs are same irrespective of age [4 (1.9%) patients had multiple defects 2 in each group (2.6% in < 12 years group and 1.4% in > 12 years group respectively]. Most ASDs will have had normal pulmonary venous drainage 214 (99%); but should be looked for anomalous pulmonary venous drainage [2 (0.93%) patients (both in > 12 years group) had partial anomalous pulmonary venous drainage. Both had RUPV draining into to SVC]. Enlarged RV (defined as > 50% of LV size in parasternal long axis view) is found in both children and adults with ASD [176 (81.5%) patients, 63 in < 12 years (81.8%) group and 113 (81.3%) in > 12 years group respectively p-0.92]. RV size RV as increases as age advances due to progressive volume overload. (mean 26.1mm in > 12 years group and 20mm in < 12 years group p<0.05). Pulmonary artery pressure increases with age in patients with untreated ASD. [mean 29.9 vs 25.5 p<0.0.5 in > 12 years age group].

Pressure data analysis during Cath study in > 12 years group and < 12 years group itself is enough to say that early intervention is indicated and is crucial in patients with OS ASD. [RA mean pressure in mm Hg (7.6 vs 5.6 p< 0.007) RVSP in mmHg (34.1 ± 9.6 vs 29.2 ± 6.9 p-0.009) RV end diastolic pressure in mm Hg (8.1±3.2 vs 6.5±2.7 p-0.002). RVEdp in mm Hg (mean 8.1 vs 6.5 p<0.002) PA diastolic pressures in mm Hg (mean 14.4 vs 12 p-0.003) Average PA pressure in mm Hg
Discussion

[30.9/14.4 (21) vs 25.8/12 (17.7) p-0.001] were suggestive of increase pressures with increasing age].

Qp:Qs is independent of age, but tends to increase with age if untreated. [168 (77.8%) patients had Qp:Qs ratio > 2:1 [57 in < 12 years (74%) group and 111 (79.9%) in > 12 years group respectively p-0.45]. 48 (22.2%) patients had Qp:Qs ratio between 1.5: to 2:1 [20 in < 12 years (26%) group and 28 (20.1%) in > 12 years group respectively-0.54]

On follow up, it was nice to find that all patients were free from dyspnoea; all children were growing well without significant respiratory infections. None had CVA/TIA or cardiovascular mortality/morbidity. This proves beyond doubt, the long term safety of percutaneous closure of ASD.

All were NYHA (New York Heart Association) class I on follow up. However, objective assessment (like TMT or 6min walk test) would have made more accurate assessment of functional capacity. One asymptomatic patient of age 57 years, hypertensive, was found to have paroxysmal atrial arrhythmia (atrial fibrillation during ECG and sinus rhythm during TEE) and put on anticoagulation and amiodarone. One patient required pacemaker insertion with diagnosis of sick sinus syndrome was doing fine on follow up. This finding is different from other studies. Probable explanation for this tremendous success is the timing of closure of ASD at appropriate age.

If we compare the incidence of palpitations pre and post closure, we can find that in < 12 years group there were 3 (3.9%) patients with palpitations and none had palpitations on post closure follow up. Again suggests the importance of early closure of ASD once the diagnosis is made. In > 12 years group incidence of palpitations reduced from 40 patients (28.8%) to 2 (1.4%) These 2 patients who had exertional palpitations were on further evaluation found to have non-arrhythmic palpitations with normal Holter study and no volume overload on Echo. These were counselled, evaluated for thyroid abnormality, and kept on follow up. This finding is different from other studies. Jategonkar et al50, 56 studied 96 patients of post ASD device closure (age 69.9±5.5 years) and found 16 to have paroxysmal AF on follow up of 3 months. Giardini et al52 al followed 134 patients (age 39±19) for 4.8±2.7 years and found 8 patients to have atrial arrhythmias. Humeberger et al51 followed 236
patients (age 49±8) for 12 months and found 12 patients to have atrial arrhythmias. As suggested by Silversides CK et al and many studies, device closure of an ASD before the onset of atrial arrhythmias may protect against the subsequent development of arrhythmia, particularly in patients less than 55 years of age. Reason for such a successful outcome in our study is probably early age at intervention.

One patient had migraine on follow up. This was diagnosed to be primary migraine with positive family history and responded to prophylaxis. This may not be related to device closure (as anybody can develop migraine especially with strong family history).

6 (1.4%) patients (all in > 12 years age group) complained of atypical chest pain on follow up with no residue, normal LV function, no device related complications. 2 had already undergone coronary angiogram from elsewhere with no lesions, other 4 patients underwent Exercise Stress Test with no inducible ischemia with good effort tolerance.

All except one (cardiomegaly due to hypertensive heart disease) had normal cardiac size, normal second sound. ECG evaluation showed no significant change in PR interval (p=0.24) with axis shifted from 77.9 ± 32.2 to 44.7 ± 24.7 (< 0.05) this axis was uniform across both groups. Significant no of patients (70.4%) showed disappearance of in IRBB. Persistent IRBBB (10.2%) was more in < 12 years group (11.7% VS 9.4% p0.25) On further analysis these patients were found to have lesser post device follow up period mean 60 months, suggesting RV remodelling may take some more time. Serial monitoring and analysis will give us the time required for RV remodelling post device closure. ECG changes are reversible after closure, due to RV remodelling without affecting PR interval. PR interval at the baseline was not prolonged, because of the case selection at the appropriate age. PR interval remaining unaltered indicates AV node function not being affected by device placement. Otherwise ECG changes on follow up were similar in both groups.

Transthoracic imaging on follow up showed features to suspect the presence of residual defect in 17 (7.9%) of the patients. [4 and 13 respectively in < 12 years and > 12 years group respectively] This is similar to other standard studies. Jategonkar
et al\textsuperscript{50} had 16.9\% residues on 3 months follow up. Young et al\textsuperscript{57} had 18.9\% residues on 15 months follow up, Swan et al\textsuperscript{58} had 16\% residues on 6 weeks follow up, Jategonkar et al\textsuperscript{66} had 12.3\% on 3 years follow up in another study. Some other studies showed fewer incidences of residues on follow up TTE evaluation. Balint et al\textsuperscript{9} had 4 \% residues on 15 months follow up, Majunke et al\textsuperscript{53} had 3.4\% residues on 8 years follow up, Patel et al\textsuperscript{55} had 5\% residue on 3 years follow up. We had 80 months follow up. Our follow up period was 79.3 ± 27.3 months.

MR grade in patients with associated MVP or otherwise and TR grade did not increase on follow up so was PS gradient. So to say mild PS do not progress at least on intermediate follow up and closure of ASD does not influence MR progression or PAH in patients with MVP. None of the patients developed significant aortic regurgitation on follow up. Not many studies were found on literature search to assess the progression of PS/MR after closure of ASD. 132 (84.3\%) patients showed reduction in RV size on follow up. RV size reduced from 23.9 ± 7.2 to 16.4 ± 4.5 (p < 0.05) However, 34 (15.7\%) patients still had RV enlargement, on further analysis all of them had preclosure shunt >2:1 and mean follow up period of 60 months. Probably RV remodelling may take some more time in some individuals (with > 2:1) after ASD closure. This persistent RV enlargement was similar in both groups. Our study when compared with other studies (Humenberger et al\textsuperscript{61}, Jatenberger et al\textsuperscript{68}, Swan et al\textsuperscript{57}, Patel et al\textsuperscript{53}) is different in the sense that other all studies were done in older individuals (all mean age > 49 years) with very large RVs (mean RVed > 39.9mm) But the reduction in RV size is comparable.

RVSP by TTE reduced from 28.5 ± 9.1 to 19.7 ± 4.8 (p < 0.05) suggesting the importance of ASD closure to prevent progression of PAH. This was uniform in both groups. Balint et al\textsuperscript{9} al showed reduction in PASP (58 to 44 mm Hg) on 31 months of follow up, Young et al\textsuperscript{57} found reduction from 30 to 25mmhg over 15 months of follow up, Majunke et al\textsuperscript{53} found reduction of 33.4 to 28.3 mm Hg on 8 years follow up. But our study is different because of the reasons mentioned in the previous paragraphs.

TEE was probably the most important tool of evaluation after ASD device closure in our study. 186 (85.7\%) patients underwent TEE evaluation. Out of 17 patients who
were thought to have residue on TTE were found to have no flow across the septum. All these had IVC flow hitting the LA disc and giving the false impression of residue. However the other one had malalignment at the aortic rim and found to have residue although tiny in size with no features of volume overload. This shows the importance of TEE on the evaluation of post ASD closure patients especially when TTE shows suspicion of residue. None of the patients had evidence of device related aortic erosion, disc fracture, device embolization, valvular dysfunction (obstruction/regurgitation) This success is probably due to the appropriate device size selection (ratio of ASD size to Device was 1.33:1, not too oversizing the device, not more than 5mm of the defect size) along with proper deployment technique for correct alignment during device closure. After closure of the defect with appropriate size device, the RV remodelling along with endothelialisation of the device will ensure no long term intracardiac complications. Small additional ASDs in 2(0.9%) which were left alone and tiny residues in 2(0.9%) during closure were closed on follow up TEE, indicating tiny residues/small ASDs close to the main defect even left alone during closure will close on long term follow up.

One of the patients was found to have suspicion of thrombus near LA disc on TEE. His further evaluation (d-Dimer, coagulation profile, platelet count, BNP, work up for infective endocarditis, LA appendage by TEE) was unremarkable. He was in sinus rhythm and asymptomatic as well. He received 3 months of oral anticoagulation with target INR of 2.0-3.0

One lady was found to have associated milder form of Ebsteins anomaly with no RVOTO, normal RV function, no residue, no PAH. This may not be sufficient to say that all Ebsteins anomalies with ASD can be considered for device closure, however worth considering for further RCTs to proceed further.

Among other patients who did not have complete data, but had clinical+TTE data on long term follow up (41 in number (14%) other than 216 with compete data) were found to have FC I clinical status, no CVA/TIA, dyspnoea/palpitations, no headache no recurrent infections. There TTE findings were normal RV function, normal valve function, with no PAH/RV enlargement/residue. Excellent outcome in available comparative data.
LIMITATIONS AND SCOPE
The status of 15 (5.2%) patients who did not have a follow up after device closure could not be assessed. 41 (14%) patients did not have complete data although all were free from primary and secondary outcome.

This was a single centre retrospective, cross sectional observational study. Serial follow up data was not analyzed, which would tell us the serial changes in ECG and Echo’ findings post percutaneous closure of ASD.

Outcome was not compared with controls/surgical closure patients. Objective assessment of functional class was not done. Only symptomatic patients underwent Holter study.

Device remodelling per se was not assessed.

Scope

Pre and post device closure assessment by 3D by TTE/TEE gives a better picture of RV size and function.

Pre and post device closure assessment of functional status (e.g. effort tolerance on treadmill) will be more objective, especially in adult population.

Serial follow up will tell us about the serial changes in ECG and Echo’ findings post percutaneous closure of ASD.

Pre and post device closure assessment of LA/RA discs by 3DTEE in relation with defect and rims will give better idea regarding the long term changes in the device per se (device remodelling). This will help in the proper selection of size of the device to avoid device related complications.
CONCLUSIONS
Transcatheter closure of ASD is a safe and effective procedure which is to be considered early in childhood.

Long term clinical outcome after percutaneous closure of AS is excellent.

Post device closure arrhythmias are rare; but can occur.

Post device closure QRS axis normalizes without affecting PR interval

Post closure, RV size and PA pressures come down significantly

If properly selected, ASD device closure patients will not have any long term complications related to the device.

Small additional ASDs/tiny residues left alone during device closure procedure will close on long term follow up.
BIBLIOGRAPHY
Bibliography


46. Kutty et al., Long-Term (5- to 20-Year) Outcomes After Transcatheter or Surgical Treatment of Hemodynamically Significant Isolated Secundum Atrial Septal Defect, American Journal of Cardiology, Feb 13 2012


Bibliography


APPENDICES
CONSENT FORM

I, the undersigned Mr/Mrs ............................................................
father/mother/son/daughter/self/relative of .............................. Hospital number
.................... aged.......... (years) hereby give consent for my ...................... to
participate in the study, “Long term follow up after ASD-device closure” conducted
by Dr. Sanjeev H Naganur under guidance of Dr Krishnamurthy K M and Prof J M
Tharakan in the department of Cardiology Shree Chitra Thirunal Institute of Medical
Sciences and Technology. During the study, participant will undergo clinical
examination, ECG, Chest radiograph, Echo’ (TTE/TEE) and Fluoroscopic
examinations if required. I am also aware of the fact that not participating in the
study will not alter the standard follow up care and medical advice. After
understanding the details of the study I give consent for my .............. to participate
in the above mentioned study.

Signature of participant/relative:

Name/Relation:

Signature of Doctor:

Dr. Sanjeev H Naganur/Dr Krishnamoorthy K M

Date:

Trivandrum
PROFORMA

Name:                       Sex :          Hospital number :                     Phone number :

Address:

Prior to device closure: (from MEDICAL RECORDS)

Symptoms: FTT/recurrent LRTI/Palpitations/dyspnoea/cyanosis/any other

Clinical Examination: Cardiomegaly/Second heart sound/Pulmonary sound (P2)/Flow murmur

Chest radiograph : Cardiomegaly/Vascularity/pulmonary bay/RPA size/PAH

ECG: Rhythm/PR/Axis/RBBB/RVH/any other

TT-Echocardiography: PVD/RV/LA/ASD size/TR/MR/LV function/RVSP/Other abnormalities:

TEE : ASD size/PVD/Rims/Other significant findings :

At the time of Device closure: (from MEDICAL RECORDS)

Age:                Height:  cm             Weight:   kg                MRD number:

TEE findings:

Cath' findings:        Qp/Qs :           PA pressure :

Device details:        Approach/Balloon assistance:

Any significant events during procedure:
At discharge/24 hours post procedure: (from MEDICAL RECORDS)

Hospital stay:

Symptoms: Examination:

ECG: rhythm/rate/PR/axis/RVH/RBB

Echo': Residual ASD/TR /RVSP/RVOTO/LVOT/MR/LV function/Other significant findings

At Follow up:

Time since Device closure:

Weight: kg Height: cm

Symptoms: FTT/recurrent LRTI/Palpitations/dyspnoea/cyanosis/any other

Clinical Examination: Cardiomegaly/Second heart sound/Pulmonary sound (P2)/Flow murmur

Chest radiograph: Cardiomegaly/Vascularity/pulmonary bay/RPA size/PAH

ECG: Rhythm/PR/Axis/RBB/RVH/any other

TT-Echo': PVD/RV/LA/ASD size/TR/MR/LV function/RVSP/RVOTO/LVOTO/Other abnormalities:

TEE: Any significant findings

Fluoroscopy:
Letter/questionnaire sent to the patients

Name: 
Hospital Number: 

[If you are unable to come in person for detailed evaluation as part of study being done in SCTIMST, Trivandrum, Kerala, please fill the details as asked below and post back at the earliest]

Device closure was done on: 

Current phone no: 
Age at present: 

Last visit to SCTIMST, Trivandrum on: 

Contact address if changed: 

Please tick yes/no for the questions asked below: 

Breathing difficulty : yes/no 
Palpitations : yes/no 
Presyncope : yes/no 
Syncope : yes/no 
Chest pain : yes/no 
Stroke : yes/no 
Any other symptom : 

Any medications you are taking: 

Relevant health related tests done recently (PLEASE SEND A COPY) 

Any medical queries: 

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