

SHORT TERM OUTCOMES FOLLOWING PULMONARY VALVE REPLACEMENT WITH FLOMERO BIOPROSTHESIS: A PILOT STUDY

Dr. Kaushik R

PDF IN PAEDIATRIC CARDIOVASCULAR &
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**SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL
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**Dept. of Science and Technology,
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A THESIS SUBMITTED BY

Dr. Kaushik R

TO

**SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES
AND TECHNOLOGY, TRIVANDRUM.**

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श्री चित्रा तिरुनाल आयुर्विज्ञान और प्रौद्योगिकी संस्थान, त्रिवेन्द्रम
तिरुवनन्तपुरम - ६९५०११, केरल, इंडिया
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM
Thiruvananthapuram - 695 011, Kerala, India
(An Institute of National Importance under Govt. of India)

Grams : Chitramet, Phone : +91-471-2443152, Fax : +91-471-2550728 / 2446433, E-mail : sct@sctimst.ac.in, Website : www.sctimst.ac.in

DECLARATION BY STUDENT

CERTIFICATE

I, Dr. Kaushik R, hereby certify that I personally carried out the work depicted in the thesis titled
**SHORT TERM OUTCOMES FOLLOWING PULMONARY VALVE REPLACEMENT
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CERTIFICATE BY THE RESEARCH GUIDE

Name of the guide: Dr Baiju.S.Dharan

Department: Department Of Cardiovascular And Thoracic Surgery

This is to certify that Dr KAUSHIK R., Post Doctoral Fellow Paediatric in the Department of Cardiovascular and Thoracic Surgery of this institute has fulfilled the requirements prescribed for **PDF IN PAEDIATRIC CARDIOVASCULAR AND THORACIC SURGERY** degree of Sree Chitra Tirunal Institute For Medical Sciences And Technology, Trivandrum

The thesis entitled “**SHORT TERM OUTCOMES FOLLOWING PULMONARY VALVE REPLACEMENT WITH FLOMERO BIOPROSTHESIS: A PILOT STUDY.**” No part of the thesis was submitted for the award of any degree or diploma before this date.

Clearance was obtained from the Institutional Ethics Committee for carrying out the study.

Signature

Dr Baiju.S.Dharan

Professor

Department of Cardiovascular and Thoracic Surgery

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APPROVAL OF THE THESIS

The thesis entitled.

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REPLACEMENT WITH FLOMERO BIOPROSTHESIS: A PILOT**

STUDY Submitted by

Dr. Kaushik R

for the degree of

POST DOCTORAL FELLOWSHIP PAEDIATRIC

of

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND

TECHNOLOGY, TRIVANDRUM

is evaluated and approved by

(Name & Signature of the Guide)

Dr.Baiju .S.Dharan

Professor

Dept of CVTS, SCTIMST

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Dr. Kaushik R

Post Doctoral Fellow

Department of Cardiovascular and Thoracic Surgery.
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LIST OF ABBREVIATIONS

SR NO	ABBREVIATION	FULL FORM
1)	CHD	Cyanotic heart disease
2)	TOF	Tetralogy of fallot
3)	PA	Pulmonary artery
4)	VSD	Ventricular septal defect
5)	RVOTO	Right ventricular outflow tract obstruction
6)	PVR	Pulmonary valve replacement
7)	MRI	Magnetic resonanace imaging
8)	PR	Pulmonary regurgitation
9)	CABG	Coronary Artery Bypass Grafting
10)	VSD	Ventricular septal defect
11)	CPB	Cardiopulmonary Bypass
12)	RV	Right ventricle
13)	EHA	European Heart Association
14)	HVR	Heart Valve Replacements
15)	IABP	Intra-Aortic Balloon Pump
16)	PI	Pulmonary Insufficiency

17)	RVESV	Right ventricle end-systolic volume
18)	RVEDV	Right Ventricle end-diastolic volume
19)	LV	Left Ventricle
20)	LVEF	Left Ventricular Ejection Fraction
21)	MI	Myocardial Infarction
22)	MCS	Mechanical Circulatory Support
23)	NYHA	New York Heart Association
24)	PA	Pulmonary Artery
25)	PCI	Percutaneous Coronary Intervention
26)	PIVSR	Postinfarction ventricular septal rupture
27)	RV	Right Ventricle
28)	TPA	Tissue Plasminogen Activator
29)	VSD	Ventricular Septal Defect
30)	VSR	Ventricular Septal Rupture
31)	VT	Ventricular Tachycardia





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1. INTRODUCTION

The most prevalent kind of cyanotic CHD is TOF (Tetralogy of Fallot) Fallot's tetralogy, described by Fallot himself as stenosis of the PA (Pulmonary artery), a Ventricular septal defect (VSD), hypertrophy of the right ventricle (RV), and rightward deviation of the origin of the aorta.¹

TOF patients often get their VSD corrected and the Right ventricular outflow tract obstruction (RVOTO) relieved within the first six months of life.² Surgical palliation is also performed before full repair when clinically necessary. Following RVOT (Right ventricular outflow tract) surgery, the patient develops progressive RVOT impairment. RVOT dysfunction is manifested by severe pulmonary insufficiency that results in a volume-loaded RV, progressive pulmonary stenosis that results in a pressure-loaded RV (seen in patients with an RVOT conduit), or mixed disease with both pulmonary insufficiency and stenosis.²

PR (Pulmonary Regurgitation) is a typical consequence after TOF repair and is particularly severe in those who have a transannular patch. Since stroke volume is maintained and RV dilatation is not initially linked with a decrease in RV ejection fraction³, patients traverse a prolonged preclinical compensated period during which they hardly ever complain of any symptoms. Finally, RV compensatory adaptations fail and RV contractile dysfunction develops in TOF patients with severe PR⁴.

RV dysfunction can be reversed for a while with treatment that eliminates PR, but gradually the damage progresses leading to irreversible myocardial injury.⁵

Pulmonary valve replacement (PVR) with a bioprosthetic valve is a current standard treatment for symptomatic patients with pulmonary insufficiency (PI) after cardiac surgery for adults and children.⁶

Pulmonary valve replacement aims to abolish PR and reverse the damage done and/or to prevent further damage. Although many factors impact late outcomes after TOF repair, chronic PR is widely recognized as a key pathophysiologic driver.⁷ Optimal timing for surgery is still a matter of debate with newer modalities like cardiac MRI quantifying RV function providing important inputs for the same.⁸ Surgical options involve open repair, Minimally invasive surgery, and percutaneous pulmonary valve implantation.

The first-line treatment for congenitally diseased PVs is almost always repair to preserve the native tissue. However, PV replacement is often inevitable, such as when valvuloplasty or valvectomy eventuates in significant pulmonary regurgitation. A range of heart valve replacements (HVRs) have been introduced since the 1950s, including mechanical valves, bioprosthetic valves homografts, and autografts.⁹

Bioprosthetic valves are constructed from biological tissues, often in combination with metallic or polymeric supports, and demonstrate more physiological flow profiles and reduced susceptibility to thrombosis than mechanical valves. Bioprosthetics are commercially available for surgical or transcatheter delivery and are treated with glutaraldehyde to preserve structural integrity and reduce immunogenicity. Various bioprosthetics have historically been used as PV replacements, including the Hancock II (a stented porcine aortic valve), the Carpentier-Edwards

PERIMOUNT (a stented bovine pericardial valve), and the Freestyle (a porcine aortic root)¹⁰

Recent bovine-derived bioprosthetics include the FLOMERO bioprosthesis, which is less immunogenic and lessens the requirement for post-operative anticoagulants. The study's objective is to evaluate the short-term effects of employing the FLOMERO bioprosthesis for PVR

LITERATURE REVIEW

General review of literature

Tetralogy of Fallot is the most common form of cyanotic congenital heart disease, and one of the first to be successfully repaired by congenital heart surgeons. Since the first procedures in the 1950s, advances in the diagnosis, perioperative and surgical treatment, and postoperative care have been such that almost all those born with tetralogy of Fallot can now expect to survive to adulthood. The startling improvement in outcomes for babies born with congenital heart disease in general—and for those with tetralogy of Fallot in particular—is one of the success stories of modern medicine.

Tetralogy of Fallot was first described by Niels Stenson in 1671, although its precise anatomical description was elegantly illustrated by William Hunter at St Georges Hospital Medical School in London in 1784: "...the passage from the right ventricle into the pulmonary artery, which should have admitted a finger, was not so wide as a goose quill; and there was a hole in the partition of the two ventricles, large enough to pass the thumb from one to the other. The greatest part of the blood in the right ventricle was driven with that of the left ventricle into the aorta, or great artery, and so lost all the advantage which it ought to have had from breathin". His description of a large outlet ventricular septal defect together with subpulmonary and pulmonary valve stenosis, and its resulting physiology, was refined by Etienne-Louis Fallot in 1888 in his description of *L'anatomie pathologique de la maladie bleu*, but the term tetralogy of Fallot (a tetrad of (i) ventricular septal defect with (ii) over-

riding of the aorta, (iii) right ventricular outflow obstruction, and (iv) right ventricular hypertrophy) is attributed to Canadian Maude Abbott in 1924.

We now regard tetralogy as a family of diseases, all characterised by a similar intracardiac anatomy (figure1), but highly variable in terms of pulmonary artery anatomy, associated abnormalities, and outcomes. Here, we focus on the most common form, in which the heart has normal segmental anatomical structure, the right ventricular outflow tract is patent at birth, and no other major intracardiac abnormalities, such as atrioventricular septal defect, exist.

About 3·5% of all infants born with a congenital heart disease have tetralogy of Fallot, corresponding to one in 3600 or 0·28 every 1000 livebirths, with males and females being affected equally.¹³ Its precise cause is unknown, as for most congenital heart diseases. Most cases seem sporadic, although the risk of recurrence in siblings is about 3% if there are no other affected first-degree relatives. However, a strong and increasingly recognised genetic substrate to tetralogy can affect the outcome after surgical repair.¹⁴

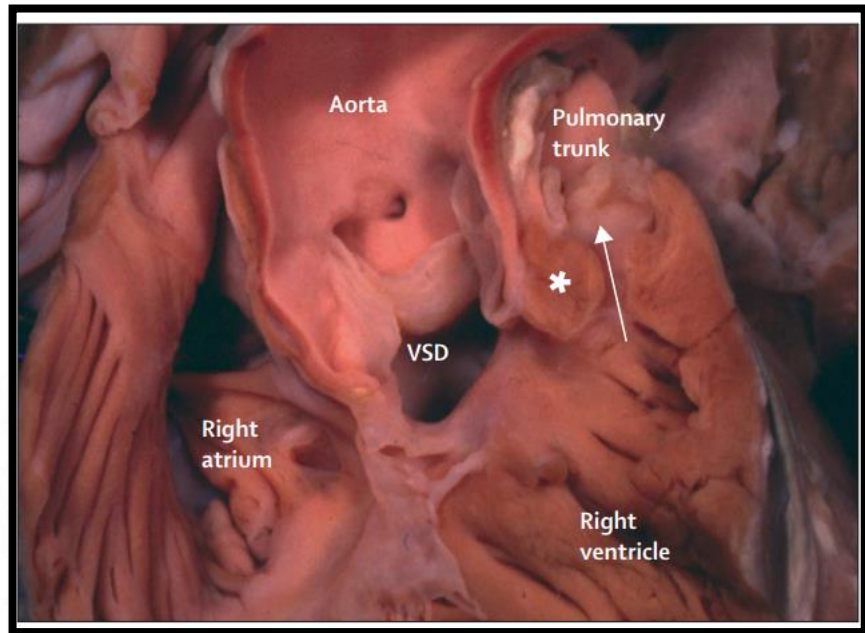


Figure 1: Morphological features of tetralogy of Fallot. The subpulmonary narrowing (arrow) is formed between the malaligned muscular outlet septum (asterisk), which is deviated anterocephalad relative to the limbs of the septomarginal trabeculation and the hypertrophied septoparietal trabeculations. There is a large ventricular septal defect with over-riding of the aorta, which is partly committed to the hypertrophied right ventricle. Note the dysplastic and stenotic pulmonary valve. VSD=ventricular septal defect. Image kindly provided by Robert H Anderson.

Pathophysiology

The ventricular septal defect is almost always large and non-restrictive in tetralogy of Fallot, ensuring that the pressure is equal in the two ventricles. Consequently, the loud systolic murmur typical in affected infants originates from the dynamic narrowing of the right ventricular outflow tract. The direction and magnitude of flow through the defect depends on the severity of the obstruction of the right ventricular outflow tract. If

obstruction to right ventricular outflow is severe, or if there is atresia, a large right-to-left shunt with low pulmonary blood flow and severe cyanosis requiring intervention at birth are present.¹⁵ However, most patients have adequate pulmonary blood flow at birth but develop increasing cyanosis during the first few weeks and months of life. In countries with well developed paediatric cardiac services, severe cyanosis, recurrent hypercyanotic spells, squatting, and other consequences of severely reduced pulmonary blood flow are nowadays rare because diagnosis is seldom delayed and infants undergo palliative procedures, or frequently complete repair within the first few days, weeks, or months of life. Temporary treatment with propranolol, which decreases right ventricular hypercontractility and heart rate and increases systemic vascular resistance, is sometimes used to reduce the incidence of hypercyanotic spells before surgery.

Diagnosis

Similar to many complex congenital heart diseases, tetralogy of Fallot is frequently diagnosed during fetal life. For those with severely obstructed pulmonary blood flow, fetal diagnosis allows better planning of perinatal management and facilitates early prostaglandin therapy to maintain ductal patency, thus avoiding life-threatening cyanosis in the early newborn period. Nonetheless, most children present with the condition after birth. Although an experienced paediatrician or cardiologist usually suspects the diagnosis clinically, transthoracic cross-sectional echocardiography provides a comprehensive description of the intracardiac anatomy (figure 3). With the exception of patients with major aortopulmonary collateral

arteries and rare cases in whom echocardiographic assessment is incomplete, any other diagnostic investigations (eg, cardiac catheterisation) are now rarely done before palliative or corrective surgery.

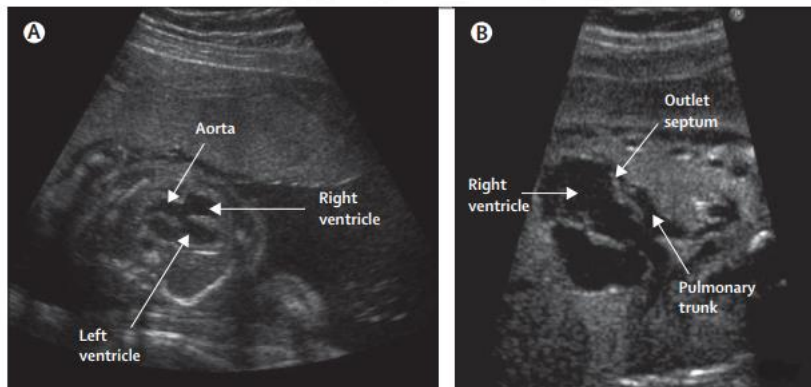


FIG 2 Prenatal diagnosis of tetralogy of Fallot The long-axis view of the fetal echocardiogram (A) shows a large ventricular septal defect with over-riding of the aorta. The typical anterocephalad deviation of the outlet septum is seen (B), causing obstruction to the flow into the pulmonary trunk. Image kindly provided by Edgar Jaeggi

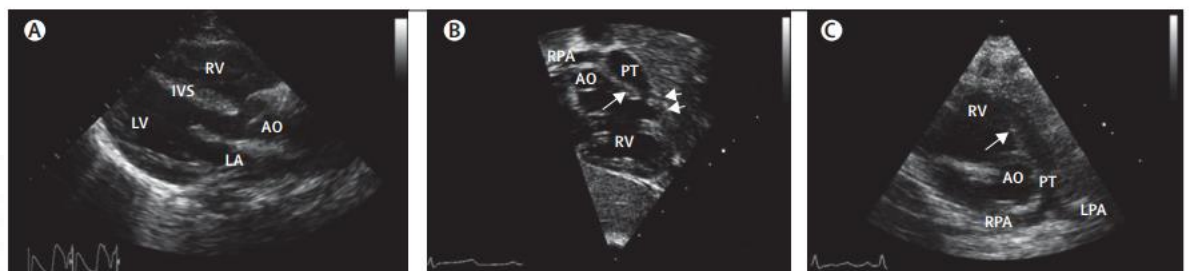


FIG 3 Postnatal diagnosis of tetralogy of Fallot by transthoracic echocardiography (A) The parasternal long-axis view shows the aortic valve over-riding the crest of the ventricular septum (IVS) and severe hypertrophy of the right ventricular myocardium. (B) Subcostal right oblique view of the obstruction of the subpulmonary infundibulum due to the anterocephalad deviation of the malaligned outlet septum (long arrow) and the abnormal arrangement of the septoparietal trabeculations (short

arrows). (C) Parasternal short-axis view shows small peripheral pulmonary arteries (RPA and LPA) with supra-valvular narrowing of the pulmonary trunk (PT). LV=left ventricle. RV=right ventricle. AO=over-riding aorta. LA=left atrium

Management

Before the advent of surgical intervention, about 50% of patients with tetralogy of Fallot died in the first few years of life, and it was unusual for a patient to survive longer than 30 years.¹⁶ Nowadays, almost all those born with this disease in all its variants can expect to survive surgical correction and reach adult life. Since the first reported intracardiac repair of tetralogy in 1955,¹⁷ the age of patients receiving primary corrective surgery has gradually decreased, with some units advocating surgery at diagnosis, even within the first few days of life. Most centres prefer to operate on children aged 3–6 months, reserving earlier open-heart surgery for those presenting with severe cyanosis or hypercyanotic spells. Some centres continue to offer surgical palliation by construction of a systemic-to-pulmonary arterial shunt, balloon dilation, or placement of a stent in the right ventricular outflow, in neonates and young infants, thereby deferring intracardiac repair.¹⁸ Potential disadvantages of this staged approach include long-lasting pressure overload of the right ventricle and persistent cyanosis. Long-term hypoxaemia contributes to cardiomyocytic degeneration and interstitial fibrosis, which have been implicated in myocardial dysfunction and ventricular arrhythmias.¹⁹ However, the opponents of early palliation point to the frequent need for aggressive outflow tract procedures, the adverse effects of early bypass surgery on the neonatal brain,²⁰ the often complicated and lengthy postoperative recovery in small infants, and implications of all these factors for late

adverse outcomes. The best age for repair has been previously discussed in a review²¹ of results obtained at the Hospital for Sick Children in Toronto (Canada) during the transitional period towards a policy of primary complete repair. Between 1993 and 1998, 227 consecutive children underwent complete repair, with the incidence of previous palliation with a systemic-to-pulmonary artery shunt falling from 38% to 0% during that time. The overall mortality was only 2.6%, but this also fell with transition to primary repair, becoming 0% in 1996–98. Nonetheless, primary repair in babies younger than 3 months of age has been associated with longer intensive care and hospital stay than in those older than 3 months, suggesting that the optimum age of elective repair is 3–6 months of age. Whenever done, reparative surgery should ideally result in complete closure of the ventricular septal defect, preservation of right ventricular form and function, with an unobstructed right ventricular outflow tract incorporating a competent pulmonary valve. Unfortunately, the nature of the subpulmonary obstruction rarely makes this possible. Surgical repair has made consistent progress over the past 50 years. Early techniques included repair of the ventricular septal defect via a large right ventriculotomy and extensive resection of the right ventricular outflow musculature and pulmonary valve leaflets. Improvements of the transatrial–transpulmonary approach have benefited early and middle-term outcomes by avoiding right ventriculotomy and its associated scarring and dysfunction.²² Furthermore, in the past 20 years a shift from the need for complete relief of obstruction²³ towards a policy to preserve the pulmonary valve, even at the expense of a modest residual stenosis, has occurred.²⁴ This shift might keep adverse late effects of pulmonary incompetence to a minimum and retain the integrity of the outflow tract,

avoiding late aneurysmal dilation. Changes in the management of children born with tetralogy of Fallot in the 21st century are being guided by results of surgery done in the second half of the 20th century. Indeed, the evolution of complications in adult life, their careful cataloguing, and the new understanding of their mechanisms provide ample evidence that follow-up of patients with congenital heart disease needs to be a continuous process, not only because of the burgeoning needs of adult survivors but also as a responsibility to the children requiring treatment today. Understanding the causes of complications during the early postoperative period and the late postoperative period after repair of tetralogy of Fallot has led to the description of unique pathophysiological changes and development of novel treatments that have implications for cardiovascular diseases as a whole, and are important for the progress of research on adult congenital heart disease as its own subspecialty

Complications

The early postoperative period Most children undergoing complete repair have an uncomplicated postoperative recovery and are discharged within a week of surgery. For a minority, the early postoperative course is complicated by a low cardiac output syndrome despite an apparently adequate repair with preserved biventricular systolic function. Echocardiographic doppler studies in these patients often show evidence of what is known as restrictive right ventricular physiology.²⁵ Occurrence of restrictive physiology is related to the degree of myocardial damage that takes place during repair. Chaturvedi and colleagues²⁶ showed that the development of restrictive physiology was associated with significantly

increased troponin concentrations on release of the aortic cross clamp and throughout the early postoperative period. Interestingly, it does not seem to be related to age at operation, but is more common on follow-up of patients in whom a transannular patch had been inserted across the ventriculopulmonary junction.^{26,27} Early postoperative restrictive physiology requires a longer duration of inotropic support, longer stay in an intermediate care station, and higher doses of diuretics.²⁸ However, it is a transient phenomenon, usually resolving within 72 h, although reappearance in the later postoperative follow-up period can occur.^{29 30}

Pulmonary incompetence

Not long ago, residual pulmonary incompetence was regarded as an inevitable, but unimportant, late sequel of repair. Much emphasis was placed on the need for complete relief of obstruction, often at the expense of a freely regurgitant and ever-dilating outflow tract. Although data for the relation between residual outflow tract obstruction and early postoperative mortality were concerning,¹⁹ they are not anymore. Indeed, during the past decade the degree of residual pulmonary incompetence has been related to the most severe adverse outcomes of progressive exercise intolerance, right heart failure, ventricular arrhythmia, and sudden death. The misguided assertions of investigators that pulmonary incompetence was an unimportant late outcome of tetralogy can be understood when one considers the time course of the effects of postoperative pulmonary regurgitation. The problems of tetralogy occur decades after repair and might be incompletely defined because of the expected decades of further survival, even for the earliest cohorts of survivors of surgical repair. This prolonged time course also illustrates the need for continuous and vigilant

follow-up of all patients in whom intracardiac repairs have been done in childhood. Even so, there was circumstantial evidence more than two decades ago that the late outcome of tetralogy might be adversely affected by the degree of pulmonary incompetence.

In 1984, Shimazaki and colleagues²⁷ showed that symptom-free survival was decreased in patients with isolated (ie, no other major cardiac lesions) pulmonary incompetence. Interestingly, almost no complication was seen during the first 30 years of life, but thereafter a rapidly progressive condition of right heart failure, exercise intolerance, and death evolved, all of which resonate with late problems of tetralogy. In the 1970s and 1980s, right heart dysfunction was known to be more likely if an outflow tract patch was needed at the time of repair,²⁸ as was the fact that exercise dysfunction was related to the cardiothoracic ratio (as a surrogate of right heart dilation) on chest radiograph.²⁹ Nonetheless, only in the past 10 years has direct quantification of pulmonary regurgitation by the gold standard cardiac magnetic resonance been possible (figure 4). A clear relation between the amount of pulmonary regurgitation and right ventricular dilation was established with one of the first quantitative methods— videodensitometry—described in 1981 by Falliner and colleagues.²⁹ In 1988, we described a technique to quantify pulmonary regurgitation from right ventricular pressure–volume loops measured with angiograms,³⁰ or with a conductance catheter technique. We showed a correlation between the volume of pulmonary regurgitation during isovolumic relaxation, and right ventricular volumes and exercise dysfunction.

In another study,³¹ we used the uniquely dynamic nature of conductance catheter recordings to show the relation between increased right

ventricular afterload and the degree of pulmonary regurgitation. These data strongly support the notion that substantial branch pulmonary artery stenosis, especially in the setting of free pulmonary regurgitation, should be treated aggressively by balloon dilation with or without implantation of an endoluminal stent. Although restrictive physiology, characterised by antegrade diastolic flow in the pulmonary artery throughout the respiratory cycle, is associated with reduced cardiac output and slowed early postoperative recovery in children,²¹ its presence as a primary phenomenon in adults is mainly beneficial. In the first study of restrictive physiology, in adults 15–35 years after repair, Gatzoulis and colleagues³⁴ showed that those with a restrictive right ventricle had a smaller cardiothoracic ratio on chest radiograph and better exercise performance (both of which might be entirely normal) than those without restrictive physiology. This is because the poorly compliant right ventricle prevents adverse remodelling (dilation) of the right ventricle that occurs in response to pulmonary regurgitation when diastolic function is less abnormal. In turn, in many studies the amount of pulmonary regurgitation has been directly related to right heart dilatation and exercise performance. Although these findings have been confirmed by others,^{35,36} additional studies, mainly using magnetic resonance, have failed to show a consistent relation between restrictive physiology, as evidenced by antegrade diastolic flow, and either right ventricular volumes or exercise performance.^{37,38} Another important observation in the study by Gatzoulis and colleagues³⁴ was that QRS duration on the electrocardiogram in those with restrictive physiology was shorter than in those without restrictive physiology. We also showed a relation between the duration of the QRS and both right ventricular volumes and propensity

to symptomatic arrhythmia and sudden death.³⁹ We proposed the term mechanoelectrical interaction to describe the relation between the degree and type of right ventricular remodelling and its electrophysiological properties, and showed a threshold for QRS prolongation of 180 ms, and the occurrence of symptomatic ventricular tachycardia and sudden death in a single centre analysis. This phenomenon has been confirmed by us and others,^{40–42} albeit with different thresholds of QRS duration, and improved methods of describing the relation between mechanical and electrical function. However, the most comprehensive assessment comes from a multicentre study⁴³ that showed in 793 patients from five centres that pulmonary regurgitation was the most important haemodynamic determinant of symptomatic arrhythmia. Furthermore, not only was absolute duration of QRS an important predictor, but also rate of change of duration (>3 ms per year over the 10-year assessment period) was strongly associated with poor outcomes. Ventricular dysrhythmia is not the only problem that characterises the late electrophysiological outcome of these patients. Although often less dramatic in terms of haemodynamic sequelae, atrial arrhythmias are almost equally as frequent.^{43–45} Although attention has been focused on the right ventricle, there is emerging awareness of the effect of biventricular dysfunction on late outcomes. The right ventricle is anatomically integrated with the left ventricle through subepicardial bundles of aggregated myocytes that run from the free wall of the right ventricle to the anterior wall of the left ventricle. Moreover, the ventricles share the septum and are enclosed in the same pericardial cavity. Interaction of the two ventricles results in alterations of both diastolic and systolic function.⁴⁶ Experimental studies have shown that part of the external mechanical work generated by the

right ventricle is a direct consequence of left ventricular contraction or contraction of shared myocytes, and conversely that dilation of the right ventricle undermines left ventricular systolic performance. Therefore, a strong correlation between right and left ventricular ejection fractions exists in patients after repair of tetralogy of Fallot.

Furthermore, those with substantial coexisting left ventricular dysfunction have a high risk of sudden death late after repair.⁵¹ The mechanisms for this interaction are incompletely understood but might in part be related to increasing dys-synchrony between contraction of the two ventricles. Indeed, D'Andrea and colleagues⁵² showed that patients with the longest delay between onset of contraction of the two ventricles had worse exercise performance and a high incidence of ventricular arrhythmia. Although pacing of the right ventricle might improve the intraventricular desynchrony of right ventricular contraction,⁵³ biventricular resynchronisation might be valuable in postoperative tetralogy patients with overt interventricular dyssynchrony. We reported extraordinary functional improvement after biventricular pacing under these circumstances.⁵⁴ Thus, follow-up of adults and teenagers late after repair of tetralogy of Fallot focuses on the assessment of the degree of pulmonary regurgitation, its secondary effects on ventricular remodelling, and risk stratification for arrhythmia and sudden death. Having almost ignored the possibility of needing pulmonary valve replacement in the past, the assessment of need and optimum timing of pulmonary valve implantation is one of the present hot topics in this research area. Timing and effects of pulmonary valve replacement Pulmonary regurgitation as an important determinant of many late complications of early repair set the scene for various important studies reporting the effectiveness, or

otherwise, of surgical pulmonary valve replacement. Although most would agree that those with new onset symptomatic sustained ventricular tachycardia and those with overt symptoms of exercise intolerance or right heart failure are almost all candidates for surgery, many more potential candidates who do not fulfil such criteria exist. In 2000, Therrien and colleagues⁵⁵ reported the effects of pulmonary valve replacement in 25 consecutive patients attending the Toronto congenital cardiac centre for adults. The investigators concluded that operations had taken place too late. In these adults with grossly dilated ventricles, pulmonary valve replacement did not have any effect on right ventricular volumes or ejection fraction. A subsequent study⁵⁶ from the same group but in younger patients with lower degree of right ventricular dilation suggested a threshold for adequate reverse remodelling, measured by cardiac magnetic resonance, of 170 mL/m² for end-diastolic volume and 85 mL/m² for end-systolic volume. Similar results, but with somewhat different thresholds, have been obtained by other groups.^{57,58} Recovery of function is less likely to happen above a certain degree of right ventricular dilation. However, many questions remain unanswered. For example, what should we recommend to asymptomatic patients with a borderline-sized right ventricle? Although at low risk, surgery is not entirely favourable and long-term viability of implanted valves is inconsistent. What is the role for formal exercise testing in the timing of surgery? The lack of a relation between symptoms and measured performance is one of the most pervasive dilemmas in the field of adult congenital heart disease, but the usefulness of formal exercise testing remains to be defined. And finally, is there a threshold above which surgery is too risky or futile? One of the reasons why these questions remain mainly unanswered is that too

little is known about functional responses to surgery. Exercise performance might improve by valve replacement and seems unrelated to the degree of ventricular remodelling,^{59–61} but there are no definitive data on which to base preoperative recommendations and decision making.

Pulmonary valve replacement after repaired Tetralogy of Fallot

The first Blalock–Taussig shunt operation in 1945 improved life expectancy from a few months to years. Lillehei et al. followed with the first successful intracardiac repair in 1954. The current early survival of TOF is excellent, at approximately 90–98% through the first two decades of life. While early mortality has improved, studies of adult survivors with repaired TOF showed that late complications included exercise intolerance, right heart failure, arrhythmias and sudden death. Many of these complications are related to the hemodynamic effects of pulmonary regurgitation (PR) and associated chronic right ventricular (RV) volume overload. Patients with PR can remain asymptomatic for many years with a gradual deterioration of exercise performance inversely related to PR severity. Because echocardiography is often inadequate for evaluating early RV dysfunction, cardiac magnetic resonance (CMR) imaging has become an integral tool in evaluation of RV volumes, severity of PR, and biventricular function in following these patients later in life. It has been shown that Pulmonary valve replacement (PVR) alleviates symptoms,

normalizes RV volumes, and improves RV function. It is unclear whether performing PVR decreases the risk of ventricular tachycardia (VT) or sudden death. There is strong evidence that elimination or significant reduction of PR after PVR is associated with symptomatic improvement, decrease in RV end-diastolic volume (RVEDV) and RV endo-systolic volume (RVESV), and no significant change in RV ejection fraction using CMR imaging

Pulmonary regurgitation

After intracardiac repair of TOF, the patient nearly always had residual right ventricular outflow tract (RVOT) pathology, most commonly PR. For many years, PR after repaired TOF was considered

gn⁵⁵ Young patients appeared to tolerate PR well and were subsequently lost to follow-up. It was later found that as these patients reached adulthood, the hemodynamic burden of chronic PR presented as late morbidity including exercise intolerance, right heart failure, and arrhythmias. The most common cause of mortality was sudden death. These adverse long-term outcomes in patients with TOF are demonstrated by a significant decrease in their survival curve in the second decade following repair. RV systolic function in chronic PR is initially preserved. This stage may last for years and many patients remain relatively free of symptoms. As PR progresses, however, several studies have shown a close relationship between the severity of PR and RV end-diastolic volume. Severe PR is also known to impair biventricular function. Davlouros et al. demonstrated that left ventricular (LV) systolic dysfunction correlated with RV dysfunction in adults with repaired TOF, suggesting an unfavorable ventricular–ventricular interaction. This study also showed

three independent predictors of LVEF: length of time that patient remained palliated, aortic regurgitation fraction, and RVEF; the first two correlated inversely with LVEF. The mechanism that links RV dysfunction to LV dysfunction, however, is incompletely understood in repaired TOF.

Arrhythmias

Arrhythmias are also common sequelae of repaired TOF. Atrial arrhythmias may be present in approximately one third of patients and are a major source of morbidity. Although the prevalence of sustained ventricular tachycardia (VT) is low, it is believed to be responsible for an increased incidence of sudden death in patients with TOF. Gatzoulis et al. in a multicenter study of 793 patients with repaired TOF at a mean of 8.2 ± 8 years, demonstrated that an older age at TOF repair and QRS duration >180 ms were independent predictors of sudden death. Khairy et al. in a multi-institutional study in 556 patients with repaired TOF reported a prevalence of atrial tachyarrhythmias (20.1%) and ventricular arrhythmias (14.6%) that markedly increased after 45 years of age. These arrhythmias were associated with the number of cardiac surgeries, QRS duration, and left ventricular diastolic dysfunction. Targeted intraoperative cryoablation procedures during PVR appear to decrease the incidence of arrhythmias, at least in the short term. In patients with risk factors for VT and sudden death, preoperative electrophysiological study and implantation of a cardioverter–defibrillator should be considered.

Evaluation of pulmonary regurgitation and ventricular function

For many years, echocardiography has been used as the primary non-invasive imaging modality in the follow-up of patients with repaired TOF. The complex geometry of the RV makes determining volume and function

difficult using echocardiographic assessments. Doppler echocardiographic assessment of PR is also dependent on the observer's experience, and is limited by post-operative structural changes. CMR imaging has become the reference standard for assessment of biventricular size and function and offers a reliable method for the longitudinal assessment of PR severity and RV dilation in patients with repaired TOF [10, 20, 21]. Furthermore, CMR imaging is non-invasive and does not use ionizing radiation. Echocardiography and CMR imaging can be used in concert as a combined non-invasive imaging study in patients with repaired TOF. Yamasaki et al. demonstrated that cardiac computed tomography (CCT) can also assess RV function and pulmonary regurgitant fraction (PRF), compared with CMR imaging in patients with repaired TOF [22]. CCT is more useful than CMR for the patients with an implanted pacemaker/defibrillator, patients with claustrophobia or any clinical condition that prohibits a long CMR examination.

Indication and timing for PVR

PVR can be accomplished with a low early mortality in many centers, but should be performed before there is irreversible RV dysfunction and the increased propensity for VT and sudden death [23, 24]. The optimal management of patients who may have clinical or physiological indications for PVR has been widely researched and debated. Identification of risk factors for adverse clinical events is important for determining the optimal timing of PVR. There is agreement that PVR is indicated in symptomatic patients with significant PR, heart failure, or new arrhythmias. In the absence of these findings, physicians should consider a broad range of clinical data and longitudinal imaging modalities when considering PVR in the individual patient.

Several CMR imaging based studies have found that RV volumes return to normal after PVR if the pre-operative RVEDVI is <82–90 ml/m². Biventricular dysfunction and RV hypertrophy are associated with poor outcome in patients with repaired TOF.

Valve selection

Regarding the selection of prosthetic valve, the stented bioprosthetic valve is preferred for isolated PVR in adult patient, but structural valve deteriorations are unavoidable in the long term. A biological valve offers the advantages of not needing anticoagulation, whereas future replacement could be performed by transcatheter valve-in-valve procedures. A younger age at implantation with biological valve is associated with higher rates of valve deterioration [28]. Controversy still remains with respect to the best bioprosthesis to implant. Fiore et al. reviewed the performance of the mosaic porcine, bovine pericardial, and homograft prostheses in a cohort of patient who underwent PVR at a mean of 15.3 years after initial outflow tract reconstruction and reported that all three prostheses performed similarly for 3 years and PR developed more frequently in homografts [29]. Use of a mechanical valve could be an alternative especially for patients with high surgical risk including multiple prior surgery and patients already using anticoagulation agents. Pragt et al. demonstrated that freedom from valvular thrombosis was 86% at 10 year post-PVR and freedom from reoperation 91% at 10 years at a median follow-up duration of 4.26 years (range, 0–27 years) in 364 patients (69.8% with cardiac diagnosis of TOF and mean age at time of

implantation was 27.2 ± 12.2 years) ^[30]. Propensity for thrombosis was 1.7% per patient year and incidence for reoperation was 1.0% per patient year. They concluded that mechanical PVR is associated with a limited risk of valvular thrombosis and thrombolysis was an effective treatment with a success rate up to 88%. Fleling et al. also demonstrated a 10% incidence of thrombosis or pannus valve-related complications at a mean follow-up duration of 5.5 years in 66 patients following mechanical PVR (77% with cardiac diagnosis of TOF and mean age at time of implantation was 35 ± 13 years). Mechanical valve in the pulmonary position seems not to do worse. Mechanical valve is suitable for the selected patients. These selected patients, we think, are who had multiple prior operations and ventricular dysfunction or who need left-sided valve surgery with mechanical valve simultaneously. Further data of both biological valve and mechanical valve in the long term will be necessary

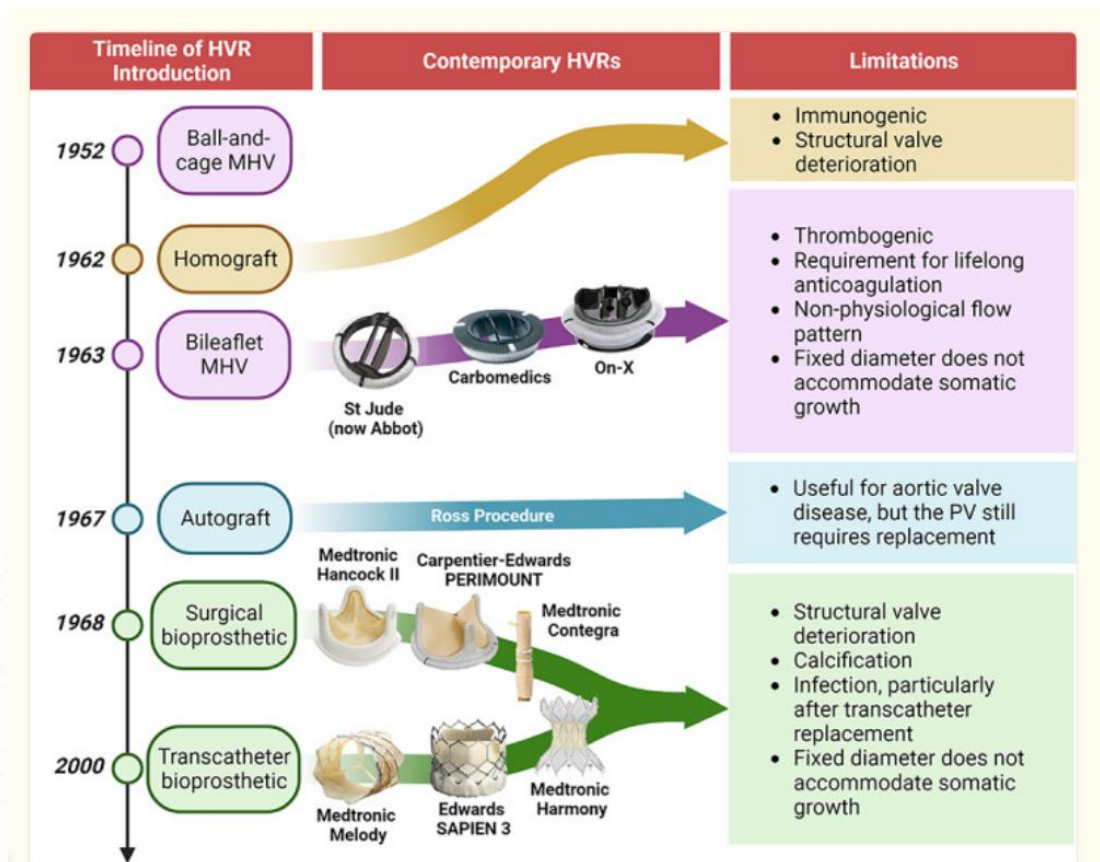


FIG 4 Timeline of HVRs

3.AIM AND OBJECTIVE

Pulmonary valve replacement is a follow up procedure in nearly all corrected TOF associated with lot of perioperative complications. This is one of the first studies to evaluate short term outcomes after using the flomero bioprosthetic valve.

PRIMARY OBJECTIVE:

- Mortality Rate

SECONDARY OBJECTIVE

To evaluate

- Need for re intervention
- Cross clamp time
- Need for post operative anticoagulants
- Post procedural pressure gradient
- Duration of ventilation (hrs)
- Duration of ICU stay (hrs),

MATERIALS AND METHODS:

STUDY DESIGN: Retrospective, non-blinded, single-center, observational study.

STUDY SETTING: The study is performed at a tertiary care centre, SCTIMST, where 600 paediatric cardiac surgery cases are done per year.

The study was conducted in the department of Cardiovascular and Thoracic Surgery at Sree Chitra Tirunal Institute For Medical Sciences And Technology , Trivandrum

SUBJECT/PARTICIPANT SELECTION

Inclusion criteria:

Patients who had undergone intracardiac repair for TOF or associated spectrum scheduled for elective PVR.

Exclusion criteria:

- Associated complex Congenital Heart disease
- Incomplete data.
- Lost to follow up at 2 months.

Ethical consideration:

Before the study's conduction, relevant permission was taken from the Institutional Ethics Committee (IEC) of the Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum (SCTIMST).

Approval from authorities:

Permission was obtained for conducting a study in the cardiovascular and thoracic surgery department from the institutional ethics committee. The same were informed to the authority of the cardiovascular and thoracic surgery department in SCTIMST.

i. Designing of Patient, consent form and data collection sheet:

The following forms were designed for study:

1) Consent form- not applicable

2) Data collection sheet- Data was obtained from the institutional electronic medical records. Data collection was done, and details of the patient information such as Age, Gender, Preoperative parameters, Co-morbidities, Intra-operative parameters and postoperative parameters, along with follow-up, was compiled. (Annexure-Other).

SAMPLE SIZE DETERMINATION:

There was a total of 48 patients who underwent surgery for pulmonary valve replacement (38 of PVR with Flomero ,10 who underwent PVR using livanova,perimount magna ease ,epic) between 2021-2023, all of them were included in the study.

Hence, data were analyzed with those numbers

CLINICAL PROCEDURE AND EXAMINATION:

After obtaining permission from the IEC, Data was collected from electronic medical records. Preoperative, intra-operative and post-operative data was collected, and the principal investigator and co-investigators performed retrospective data analysis. After the procedure, the principal investigator kept the data and patient details confidential.

Interventions: This is a retro-prospective study, and no intervention is required.

Outcomes parameters: Evaluation of the study objectives was the outcome parameters.

Method: All patients who underwent surgical repair of post-infarction ventricular septal rupture from 2021- 2023 who meet the inclusion and exclusion criteria were

included in the study.

The investigators collected the details from electronic medical records. The scanned copies of old files, discharge summary and OPD records were used to collect the required information.

STATISTICAL PROCEDURE:

The statistical procedures were carried out in two steps:

1. Compilation and presentation of data:

Compilation of data was done systematically. Using a Microsoft Excel worksheet (Microsoft, USA, version 8.1), A master table was made; accordingly, the data was subdivided and distributed, presented in the form of individual tables and graphs.

2. Statistical Analysis:

Statistical analysis using IBM Statistical Package for Social Science (Statistics for Windows, version 21.0, Armonk, NY: IBM Corp.) was done, and the data was compared by applying statistical tests to find the statistical significance of the results.

Eligible patients' data was entered and analysed using SPSS version 11 statistical software (SPSS Inc., Chicago, IL). Descriptive analysis was carried out as mean and standard deviation for quantitative variables, and frequency and proportion for categorical variables.

Mean values between the two groups were compared using an independent sample t-test. Categorical variables was studied using Chi-square test.

For non-normally distributed parameters, median and interquartile range (IQR) were compared between both groups using Mann Whitney test. Pre operative and post operative outcomes were compared using Wilcoxon's signed rank test. A value of $p < 0.05$ was considered statistically significant.

RESULTS

DEMOGRAPHIC VARIABLES

VARIABLE (FLOMERO)	FREQUENCY (n, %)
AGE IN CATEGORY	
<15 Years	6 (15.7%)
15 – 30 Years	25 (65.7%)
>30 Years	7 (18.4%)
GENDER	
MALE	20(52.6%)
FEMALE	18(47.3%)

VARIABLE (OTHER VALVES)	FREQUENCY (n, %)
AGE IN CATEGORY	
<15 Years	1(10%)
15 – 30 Years	5 (50%)
>30 Years	4 (40%)
GENDER	
MALE	5 (50%)
FEMALE	5 (50%)

TABLE 1 Demographic variables in both the groups

Distribution of study patients according to age: Out of the total 48 study patients, the largest proportion, 65.7% (n=25), fell within the age range of 15 to 30 years. This was followed by 18.4% for the 15-30 age group and 15.7% for lesser than 15 years age group.

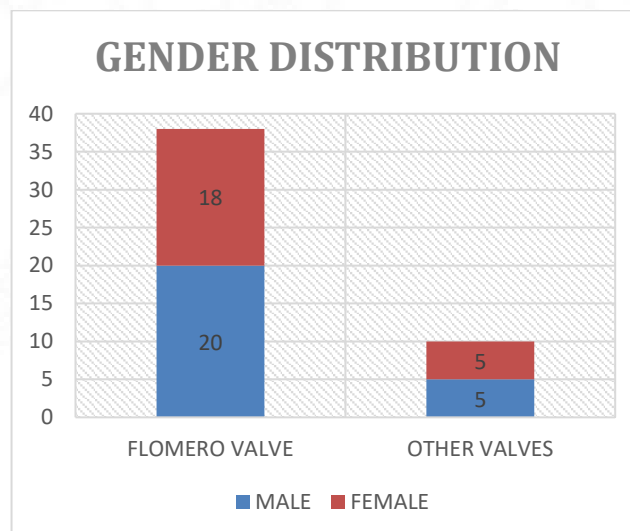
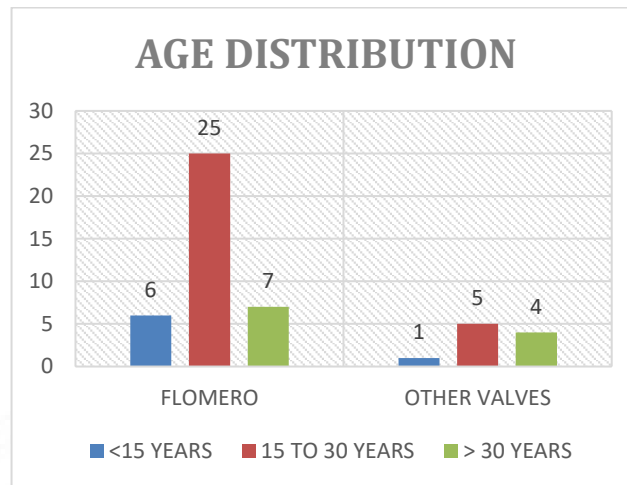


Figure 1. Distribution of study patients according to their age and gender

PRE OPERATIVE PARAMETERS

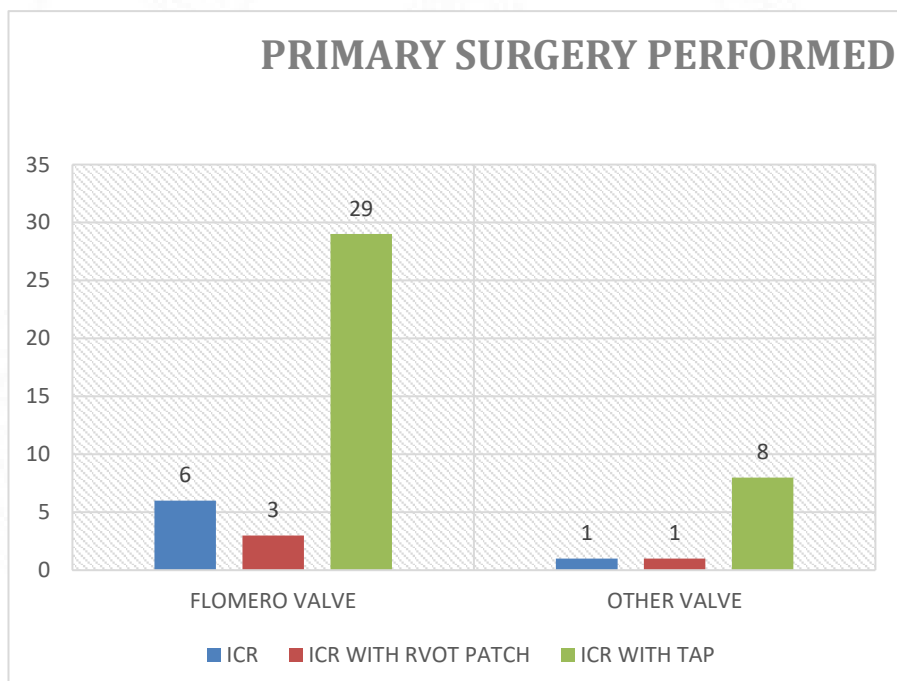
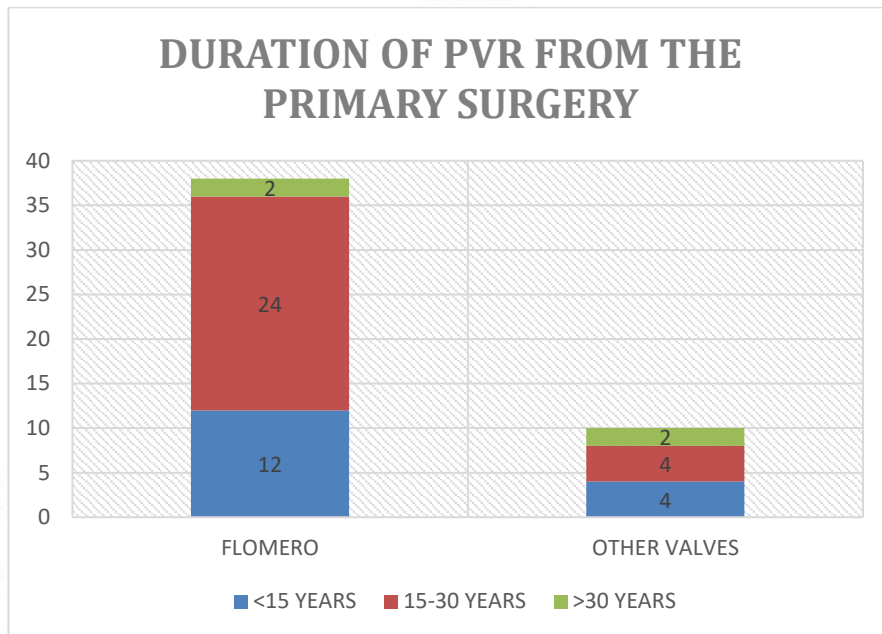
DURATION OF PVR FROM PRIMARY SURGERY <15 Years 15 – 30 Years >30 Years	12(25%) 24(63.1%) 2(5.2%)
PRIMARY SURGERY PERFORMED 1. ICR 2. ICR WITH RVOT PATCH 3. ICR WITH TAP	7(18.4%) 3(7.8%) 29(76.3%)

FLOMERO VALVES

OTHER VALVES

DURATION OF PVR FROM PRIMARY SURGERY <15 Years 15 – 30 Years >30 Years	4 (40%) 4 (40%) 2 (20%)
PRIMARY SURGERY PERFORMED 1. ICR 2. ICR WITH RVOT PATCH 3. ICR WITH TAP	1 (10%) 1 (10%) 8 (80%)

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PARAMETERS	FLOMERO VALVE(I)	OTHER VALVES (II)
	MEAN ± SD	
RVEDV	174.3±44.08	185±59.01
RVESV	95.6±88	109.5±45.17
RVEF	45.61±9.3	41.88±7.1
LVEDV	266.1±96.6	230.63±92.2
LVESV	145.5±85.46	130.25±61.8
LVEF	56.55±8.4	54.13±7.62
PR FRACTION	52.3±13.2	52.75±8.97

OUTCOME MEASURE	FLOMERO VALVE	OTHER VALVES (LIVANOVA, PERIMOUNT MAGNA EASE ,EPIC)
MORTALITY	0 (100% survival)	1

PRIMARY OUTCOME MEASURES

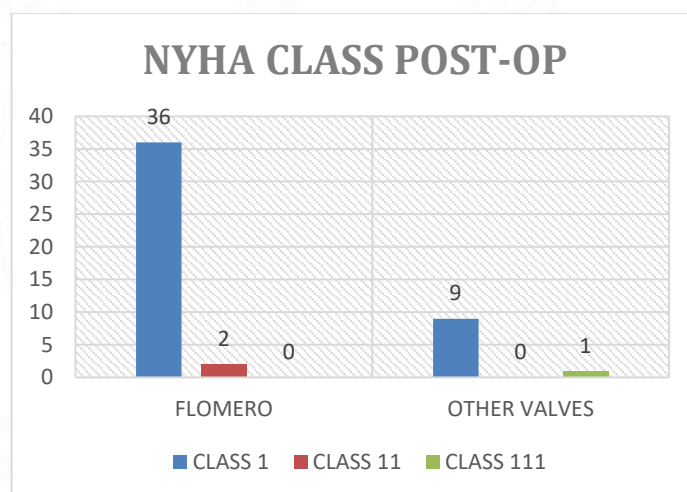
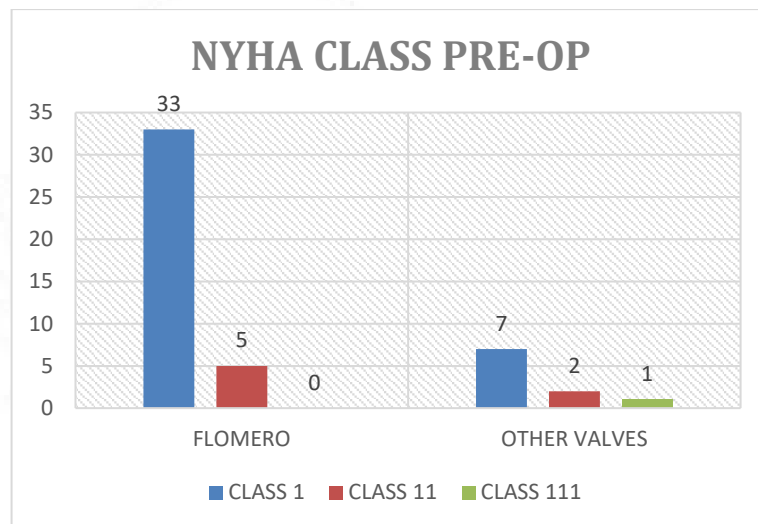
INTRAOPERATIVE PARAMETERS

PAREMETERS	FLOMERO VALVE	OTHER VALVES
PROCEDURE EXECUTED		
REDO PVR	23(60%)	7 (70%)
REDO PVR WITH RVOT PATCH	1(3%)	
REDO PVR WITH TV PLASTY	2 (5%)	2 (20%)
REDO PVR WITH FEM-FEM BY PASS	1 (3%)	
REDO PVR WITH VSD CLOSURE	3 (8%)	
REDO PVR WITH AV REPAIR	1(3%)	
REDO PVR WITH PA PLASTY	5 (13%)	
MICS	2 (5%)	
CPB TIME	141.89±58.19	184.7±150.2
CROSS CLAMP TIME	69.25±30.3	126.2±89.5

NHYA CLASS

NHYA CLASS	FLOMERO VALVE	OTHER VALVES
PRE-OP		
CLASS I	33 (86.8%)	7 (70%)
CLASS II	5 (13.1%)	2 (20%)
CLASS III	0	1 (10%)
POST-OP		
CLASS I	36 (94.7%)	9 (90%)
CLASS II	2(5.2%)	0

CLASS III		1 (10%)



DISCUSSION

Long-standing chronic PVI in patients previously submitted to TOF or pulmonary stenosis repair can result in RV dilation and failure, increasing tricuspid regurgitation, impaired exercise performance, and supraventricular or ventricular arrhythmias. A timely reoperation of PVR may prevent these consequences, with a complete RV function recovery.

The percutaneous approach now offers a less invasive treatment that may potentially reduce the number of patients for surgery and shift the indications toward earlier intervention. Unfortunately, not all patients, such as the patients presented in this report, are good candidates for transcatheter pulmonary valve implantation for RVOT morphology, associated cardiac anomalies, or both, and the number of surgical patients still is high.

The type of valve to be inserted into the RVOT still is debated. Several different types of prosthesis have been used for PVR, but all are prone to failure and likely will require reintervention. Currently, the options include both mechanical and several biologic valves.

Bioprosthetic valves (including homografts, xenografts, prosthetic valved conduits, and bioprosthetic valves) perform well hemodynamically but are prone to structural degeneration that results in multiple reoperations. Mechanical valves lead to a persistent need for anticoagulation therapy, and despite some positive reports in the literature, generally have been associated with pulmonary thromboembolic complications.

Consequently none of these valves seems to be ideal because they all present advantages and disadvantages at the same time. Homograft implantation of both xenograft valves and prosthetic valved conduits require extensive dissection of the pulmonary arteries to avoid kinking or external compression. These pulmonary valve substitutes have a length that needs to be implanted a complete pulmonary artery dissection to avoid technical complications. This is an important point to be discussed because dissection can be difficult and dangerous for patients who have undergone several previous surgical procedures. Also, extreme care must be taken during implantation because any twisting, kinking, or external compression can easily lead to early failure.

Currently, bioprosthetic valves probably are the most widely used for pulmonary valve replacement because they are readily available, do not need permanent anticoagulation therapy, and are easy to implant.

Shinkawa et al. analyzed the outcome and performance of bovine pericardial valves in the pulmonary position. Freedom from pulmonary valve reoperation was 100 % at 1 year, 97.7 % at 3 years, and 97.7 % at 5 years, similar to our results described in this report.

Kanter et al. reported their experience with the stented porcine valve in the pulmonary position and found that the actuarial freedom from reoperation was 100 % at 8 years. Fiore et al. reported that late pulmonary valve dysfunction was lower with the bovine and pericardial valve than with a homograft.

According to stratification by age, it appears that bioprostheses implanted into patients older than 10 years and with TOF have excellent durability in terms of reoperation.

The stentless porcine valves have been used extensively in the left ventricular outflow tract of adults, but information on their durability in the RVOT has been limited. Hawkins et al. reported good midterm results with the use of these valves, but freedom from redo PVR for stentless porcine valves was found to be significantly lower than that for stented porcine and stented bovine pericardia

Recent bovine-derived bioprosthetics include the Flomero bioprosthesis, which has been shown to be less immunogenic and lessen the requirement for post-operative anticoagulants. The current study reports the short-term outcomes of pulmonary valve replacement using the flomero bioprosthetic valve which is a stented bovine pericardial valve.

This is one of the first studies to report the short term outcomes following the use of flomero bioprosthetic valve in patients with previous TOF ICR. Post-procedural serial echo immediately, 1 month postoperatively, 6 months, and 1 year after surgery were assessed and the results were statistically significant ($p < 0.005$) as compared to pre-operative severity of pulmonary regurgitation.

In conclusion, this single institution retrospective study showed that the stented bovine bioprosthetic valves in the pulmonary position had excellent early outcomes. A longer follow-up period is necessary for further evaluation of the way this prosthesis performs with regard to longevity and durability.

CONCLUSION

In conclusion, this single institution retrospective study showed that the bovine bioprosthetic valves in pulmonary position had excellent early outcomes. A longer follow-up period is necessary for further evaluation of the way this prosthesis performs with regard to longevity and durability.

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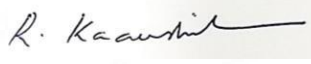
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ANNEXURES

CV of PRINCIPAL INVESTIGATOR

R	KAUSHIK	
Last Name	First Name	Middle Name
Date of Birth (dd/mm/yy) 28/04/1990		Sex: Male
Study Site Affiliation : Principal Investigator		
Professional Mailing Address (Include Institution name)		Study Site Address (Include Institution name)
Senior Resident Cardiothoracic and Vascular Anesthesia SCTIMST Trivandrum		Senior Resident Cardiothoracic and Vascular Anesthesia SCTIMST Trivandrum
Mobile Number: 9003123566		Email: kaushikramachandran90@gmail.com
Academic Qualifications (Most recent qualification first)		
Degree/Certificate	Year	Institution, Country
Mch CTVS	2021	SRI JAYADEVA INSTITUTE OF CARDIOVASCULAR SCIENCES
MS Gen Surgery	2018	Sri Ramachandra institute of higher education and research
MBBS	2011	Sri Ramachandra institute of higher education and research
Details of professional registration : 2023 TCMC Registration Number :91937 Year: 2023		
Current and previous positions (most recent position first)		
Month and Year	Title	Institution/Company, Country
Jan 2023	Senior Resident	SCTIMST
DEC 2021	Assistant professor	SRI JAYADEVA INSTITUTE OF CARDIOVASCULAR SCIENCES

Brief summary of relevant research experience:
Current project/s at hand:

Signature: 	Date: 04/09/2023 Place: TRIVANDRUM	IEC/Ver3/09
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CV of Guide

Last Name - Dharan	First Name – Baiju	Middle Name S
Date of Birth: 28/05/1970		Sex - Male
Study Site Affiliation Co - Investigator		
Professional Mailing Address (Include Institution name)		Study Site Address (Include Institution name)
Professor and Head Dept of CVTS, SCITMST Trivandrum 695001		Professor and Head Dept of CVTS, SCITMST Trivandrum 695001
Telephone (Office): -		Mobile Number: 9995019925
Telephone (Residence): 0471 2917026		Email: baijUSD@sctimst.ac.in
Academic Qualifications (Most recent qualification first)		
Degree/Certificate	Year	Institution, Country
MCH (CVTS)	2002	SCTIMST, Trivandrum, India
MS (General Surgery)	1999	Calicut Medical College, India
MBBS	1995	TD Medical College, Allepey, India
Details of professional registration: TCMC Reg no – 23256 (1995)		
Current and previous positions (most recent position first)		
Month and Year	Title	Institution/Company, Country
	Professor	SCTIMST
2010	Associate Professor, CVTS	SCTIMST
2006	Assistant Professor, CVTS	SCTIMST
2003	Assistant Professor, CVTS	Amrita Institute, Cochin
Brief summary of relevant research experience: More than 15 publications as Principal Investigator / Co investigator		

Current project/s at hand: Currently heads the division of Congenital Heart Surgery in the dept of IEC/Ve CVTS	
 Signature:	Date: 04/09/2023 Place: SCTIMST

APPENDIX – APPROVALS & PERMISSIONS

Institutional Ethics Committee (IEC) Approval Letter


ശ്രീ ചിത്ര തിരുനാലി അനുവിജ്ഞാൻ ക്ലിനികൽ പരീക്ഷണ, തിരുവനന്തപുരം
തിരുവനന്തപുരം - 695 011, കേരളം, ഇന്ത്യ
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM
Thiruvananthapuram - 695 011, Kerala, India
(An Institute of National Importance under Govt. of India)
Grams : Chitramet, Phone : +91-471-2443152, Fax : +91-471-2550726/2446433, E-mail : sct@actimal.ac.in, Website : www.actimal.ac.in

Institutional Ethics Committee
CDSCO Registration No: ECR1189InsUKL/2013/RR-21
DHR Registration No: ECINEWINST/2022/2775

SCT/IEC/2134/ER/SEPTEMBER-2023. 26 10 2023

Dr. Kaushik R
Senior Resident
Department of CVTS
SCTIMST, Thiruvananthapuram

Dear Dr. Kaushik,

The Institutional Ethics Committee reviewed your project titled "SHORT TERM OUTCOMES FOLLOWING PULMONARY VALVE REPLACEMENT WITH FLOMERO BIOPROSTHESIS: A PILOT STUDY (IEC/2134)" on 26th October, 2023.

Principal Investigator	Dr. Kaushik R, Senior Resident in CardioThoracic and Vascular Surgery, SCTIMST
Co-Principal Investigator(s)	Senior Prof. Dr. Raju Dharan, HOD Cardiothoracic and Vascular Surgery, Paediatric division, SCTIMST
Duration of the study	3 years from AUGUST 2020 - AUGUST 2023

The following documents were reviewed:

Checklist	Remarks
1. Checklist Form	1. Checklist Form
2. Covering letter addressed to the Chairman, IEC, SCTIMST	2. Covering letter addressed to the Chairman, IEC, SCTIMST
3. Project Proposal	3. Project Proposal
4. Protocol	4. Protocol
5. Covering letter addressed to the Chairman, IEC, SCTIMST regarding consent	5. Covering letter addressed to the Chairman, IEC, SCTIMST regarding consent
6. IEC Application Form	6. IEC Application Form
7. Declaration Form	7. Declaration Form
8. CV of Principal Investigator	8. CV of Principal Investigator

IEC Decision

The IEC approved the conduct of the retrospective study titled "SHORT TERM OUTCOMES FOLLOWING PULMONARY VALVE REPLACEMENT WITH FLOMERO BIOPROSTHESIS: A PILOT STUDY" in the present form.

IEC approval is issued on the basis of retrieval of secondary data from medical/laboratory records without involving human participants directly. The approval of this proposal is valid for a period of 3 years only. If, in any case the study is to be converted to a prospective nature, the approval given will be void and a fresh application has to be filed stating the reason for changing the design of the study.

Remarks:

The Institutional Ethics Committee expects to be provided a copy of the final report/publication.

There was no member of the study team who participated in voting / decision making process. The ethics committee is organized and operated according to the requirements of Good Clinical Practice and the requirements of the Indian Council of Medical Research (ICMR).

Sincerely,


Dr. G. Srinivas
Member Secretary, IEC

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE (IEC)
SCTIMST, THIRUVANANTHAPURAM



APPENDIX – SUPPLEMENTARY TABLES

MASTER CHART (DATA ENTRY)

HOSP NO	D.O.S	AGE	SEX	DIAGNOSIS
269412	ABHILASH J	02-01-2022	14 M	TOF POST ICR +TAP SEVERE PR
241371	ARAVIND B	04-04-2022	17 M	TOF ICR RVOT PATCH + VALVOTIC
318401	ABHIRAM SA	15-07-2022	12 M	TOF ICR MPA PLASTY
8809141	DEEPA TREESA JOSEPH	26-07-2022	37 F	TOF ICR + TAP
237839	MOHAMMED FARZEEN	01-08-2022	20 M	TOF ICR + TAP
268475	MOHAMMED SHAMIL	20-09-2022	17 M	TOF ICR + TAP + UNIFOCALIZATIO
243378	YOGESH VISWAS R	12-10-2022	19 M	TOF ICR + TAP
9803119	SAHALA PK	25-10-2022	25 F	TOF ICR + TAP
471379	NITHYA SRI	16-11-2022	12 F	TOF ICR + TAP
181589	ELSA MARY MATHEW	21-11-2022	22 F	TOF ICR + TAP
273608	SHAJU ML	13-12-2022	39 M	TOF ICR + TAP
272085	SATHYAVRUTHAN M	21-12-2022	27 M	TOF ICR + TAP
196416	ADIL MUHAMMED VS	24-01-2023	22 M	TOF ICR + TAP
9908388	HARIKRISHNAN G	31-01-2023	24 M	TOF ICR + TAP
188886	AISWARYA SRUTHI M	22-02-2023	24 F	TOF ICR + TAP
365347	FATHIMA HADIYA VK	01-03-2023	9 F	TOF ICR + TAP
196739	MONISHA M	17-03-2023	23 F	TOF ICR + TAP
317260	AMAL RUBEESHA	20-03-2023	12 F	TOF ICR + TAP
209148	SANOOJA MOHAMMEI	21-03-2023	26 F	TOF ICR + RVOT PATCH
207508	MOHAMMED MUSTAF	28-03-2023	21 M	TOF ICR + TAP
9105090	RAMYA P	24-04-2023	36 F	TOF ICR + TAP
307510	SNEHA	08-05-2023	15 F	TOF ICR + TAP+ MONOCUSP
9308727	DEEOU RAJ M	23-05-2023	30 M	TOF ICR + TAP
255777	MUNEER KHAN M	26-05-2023	18 M	TOF ICR + RVOT PATCH
37527	BINDU TR	30-05-2023	47 F	TOF ICR + TAP
207093	RAJESH KANNAN S	13-06-2023	32 M	TOF ICR + TAP
9503775	NIDHIN JOSE	14-06-2023	30 M	TOF ICR
295573	BHAGYA LEKSHMI MS	22-06-2023	16 F	TOF ICR
9007993	SAJEER P	05-07-2023	31 M	TOF ICR
533067	DANI PA	11-07-2023	25 M	TOF ICR TAP
9608319	ANOOP A	19-07-2023	30 M	TOF ICR + TAP
9902338	MUBEENA KV	20-07-2023	30 F	TOF ICR + TAP
417002	ASMINA NASRIN CK	24-07-2023	17 F	TOF ICR + TAP
239434	BHAVANA R	03-08-2023	19 F	TOF ICR + TAP
180132	AMAL RAJ RL	08-08-2023	24 M	TOF ICR + TAP
315349	ABHIJITH M	10-08-2023	13 M	TOF ICR
8901276	SAJI REDOLF	24-08-2023	52 F	TOF ICR
9603230	JESSENTHA PG	30-08-2023	28 F	TOF ICR + TAP



APPENDIX – SUPPLEMENTARY TABLES
MASTER CHART (DATA ENTRY)

243831 FATHIMA HUSNA	08-04-2021	16 F	TOF ICR + RVOT PATCH
11463 SEETHA	15-04-2021	40 F	TOF ICR + TAP
282049 MOHAMMED ADNAN	21-04-2021	16 M	TOF ICR + TAP
18425 SURESH BABU TC	17-05-2021	43 M	TOF ICR + TAP
8807514 GNANA JEGAN RAJ D	02-07-2021	39 M	TOF ICR + TAP
296585 HAFIS J	11-10-2021	12 M	TOF ICR + TAP
493508 MERIN BABU	19-11-2021	20 F	TOF ICR + TAP
516736 RUCHIKA JAIN	29-06-2022	44 F	TOF ICR
273885 MUHAMMED RIHAN	14-11-2022	15 M	TOF ICR + TAP



APPENDIX – SUPPLEMENTARY TABLES
MASTER CHART (DATA ENTRY)

YEAR OF P	PREVIOUS NYHA CLASS	PR	TR GRADE	RV EDV	RV ESV	RV EF	LV EDV	LV ESV
2009	1 I	PS GRADIE	3	71	36	49	77	39
2007	1 I		3	2	183	89	51	330
2013	1 I		4	3	167	87	47	164
1998	1 I		4	2	164	86	47	271
2005	1 I		4	2	162	80	50	259
2008	1 I		4	3	144	82	42	287
2006	1 I		4	1	141	64	54	205
2002	1 I		4	2	164	65	59	240
2010	1 I		4	2	192	116	39	173
2004	1 I		4	2	141	103	26	170
2008	1 I		3	2	170	74	56	305
2010	1 I		4	2	148	71	51	217
2002	1 I		4	2	158	102	35	313
2007	1 I		4	2	157	83	46	269
2001	1 I		4	2	232	136	41	314
2013	2 I		4	2	190	90	52	143
2002	1 I		4	3	178	104	41	295
2012	1 II		4	2	162	75	54	190
2003	1 I		4	3	167	73	56	254
2003	1 I		4	3	173	85	50	285
1997	1 II		4	2	200	137	31	373
2012	1 I		4	3	150	75	49	184

APPEDIX - Plagiarism Check Report



PATIENT INFORMATION PROFORMA

Case No:

- Age
- Gender

PRE-OPERATIVE PARAMETERS

- Hypertension
- Diabetes
- Dyslipidemia
- POAD
- Chronic kidney disease
- Chronic obstructive lung disease
- Cardiogenic shock
- Inotropic support
- Type of MI
- Left ventricular dysfunction
- Right ventricular dysfunction
- Multi vessel disease
- Previous PCI / CABG
- Mitral regurgitation
- Left ventricular ejection fraction
- VSR size
- Time interval between MI and VSR repair

INTRA-OPERATIVE PARAMETERS

- VSR Location
- Type of repair
- Concomitant Procedure
- CPB Time
- Aortic cross clamp time
- IABP
- ECMO

PATIENT INFORMATION PROFORMA

POST OPERATIVE PARAMETERS

- Ventilation time
- Reexploration
- ICU stay
- Hospital stay
- Post-op Ventricular function
- Residual VSR
- Sepsis
- Arrhythmia
- AKI
- Adverse Neurological outcome
- Renal replacement therapy
- Tracheostomy
- In-hospital Mortality

FOLLOW UP

- NYHA class at 3 months, 6 months & 1 year
- MI Post discharge
- 1 year survival



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An Institution of National Importance established by an Act of the Indian

Parliament (Act No.52 of 1980)

**Dept. of Science and Technology,
Govt. of India**