MODIFIED BENTALL PROCEDURE-LONG TERM SURVIVAL AND SHORT AND LONG TERM OUT COMES- A SINGLE CENTRE EXPERIENCE

Thesis Submitted By

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DECLARATION

I, **Dr. Rashida Hyderabadwala**, hereby declare that this thesis titled "**Modified Bentall Procedure-Long Term Survival And Short And Long Term Out Comes- A Single Centre Experience**" has been prepared by me under the capable supervision and guidance of **Dr. Vivek V Pillai, Additional Professor** and **Dr. Bineesh K Radhakrishnan: Associate professor, Department of CVTS** Department of Cardiovascular and Thoracic Surgery, Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram.

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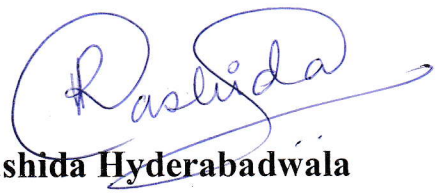

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We hereby certify that this thesis titled "**Modified Bentall Procedure-Long Term Survival and Short and Long Term Out Comes- A Single Centre Experience**" is the bonafide work of **Dr. Rashida Hyderabadwala** MCh CVTS resident, done under our guidance at Department of cardiovascular and thoracic surgery at Sree Chitra Tirunal Institute for Medical Sciences & Technology, Thiruvananthapuram.

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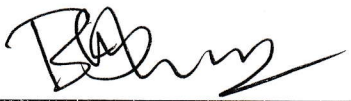

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LIST OF ABBREVIATIONS

AV	– Aortic Valve
AR	– Aortic Regurgitation
LV	– Left Ventricle
NYHA	– New York Heart Association
FC	– Functional Class
BSA	– Body surface area
LVEF	– Left ventricular Ejection fraction
LVEDD	– Left ventricular End diastolic diameter
LVESD	– Left ventricular End systolic diameter
AVR	– Aortic Valve Replacement
TEE	– Trans Esophageal Echocardiography
VT	-Ventricular Tachycardia
SD	– Standard Deviation
AF	– Atrial Fibrillation
CVA	– Cerebrovascular Accident
INR	– International Normalized Ratio

TCA	- Total circulatory arrest
CPB	- Cardiopulmonary bypass
ACC	- Aortic cross clamp
DHCA	-Deep hypothermic circulatory arrest
POD	-Postoperative day
HTK	- Histidine, Tryptophan and Ketoglurate
IRAD	-International Registry of Acute Aortic Dissection (IRAD)
MODS	-Multiorgan Dysfunction Syndrome
CUSUM	-Cumulative sum analysis

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INTRODUCTION

Aortic root replacement with the reattachment of the 2 main coronary arteries was originally described by Bentall De Bono[1]. This operation has undergone many modifications in the last 3 decades[2]. Notable adaptations have been creation of an aortic cuff around the coronary ostia and extensive mobilization and attachment of the coronary buttons. Although the operation was originally designed to treat patients with Aortic root aneurysms the indications for radical root replacement have expanded to a variety of other conditions including aortic dissections and infective endocarditis with aortic root abscess. [3-5]

Although the Modified Bentall procedure is a durable operation with a low ascending aorta and valve reoperation rate, the replacement of the aortic valve with a mechanical valve carries a significant life time risk of both thromboembolism and major hemorrhagic complications3. Biological valve conduits including stented, stentless, allograft and autograft are available and avoid the risk of thromboembolism and hemorrhagic complications, with superior coronary perfusion and lower myocardial oxygen demands than their mechanical counterparts do [6]

The choice of which operation to perform for Aortic root disease is multifaceted and influenced by both the surgeon and patients. The young patient (<40 years) who wishes to avoid anticoagulation or has contraindications for the same will benefit from valve sparing procedures if he or she has a competent aortic valve and minimal valve deformity 6. Patient > 70 years are like to benefit from aortic root replacement with a biological valve which has a low probability of rapid structural deterioration in older patients with low rate of stroke and reoperation [7].

Over the last decades, there has been a documented considerable improvement in mortality rates in patients submitted to the Modified Bentall procedure. Early and

hospital mortality range between 0. 7% to 12% in most studies [8-10]. The disproportionate percentages in early mortality among several reports could be explained by different patient populations, concomitant procedures, and modifications in operative techniques or postoperative management strategies.

Since there is no national registries to study the outcomes of Bentall in different institutions in our country, the individual institutional results remains the bench mark for comparing results of Modified Bentall with one's own experience. Our institute has a well-organized team comprising Cardiothoracic and Vascular Surgeon, Interventional Radiologist and Cardiologists. This study aims at assessing the outcomes of the procedure done since 2008 in our institute.

OBJECTIVES

Primary Objectives

1. To assess short term and long term outcomes and long term survival after Modified Bentall procedure

2. To evaluate factors determining the outcome of the procedure

Secondary Objectives.

- To assess the survival of Modified Bentall procedure post 10 years of surgery
- To evaluate quality of life of patient / Functional class post Modified Bentalls procedure
- To evaluate complications post-surgery-immediate post-operative and long term
- To evaluate the LV mass regression in patients with AS and volume regression in patients with AR post-surgery

REVIEW OF LITERATURE

Surgical Anatomy of the Aortic Valve

The aortic valve has three cup-shaped leaflets or cusps: the noncoronary cusp, the left cusp, and right cusp. These spring from three crescent-shaped valvular annuli within the expanded sinuses of Valsalva. The plane of the aortic annuli marks the line of demarcation between the left ventricular cavity and the aorta. Attachments of the aortic valve to the left ventricular outflow tract are both muscular and membranous in nature (Fig. 1. 0). The three fibrous annuli are all associated with somewhat different structures. The noncoronary annulus is singular in that it does not give rise to a coronary artery and is attached to the left ventricle only by membrane. Adjoining halves of the left and noncoronary annuli and the small area beneath the intervening commissure, the fibrous subaortic curtain, are continuous with the anterior leaflet of the mitral valve. Below the noncoronary and right coronary annuli and the intervening commissure lie the central fibrous body and the membranous septum, which are divided into atrioventricular and interventricular segments by the contiguous attachment of the nearby tricuspid valve. This membrane usually circles under the noncoronary annulus and merges with the anterior leaflet of the mitral valve. The Bundle of HIS passes into the muscular ventricular septum just below the membranous septum before dividing into left and right bundle branches. These travel downward and inferiorly along the medial side of the left ventricular outflow tract. This conduction tissue is, therefore, close to portions of the noncoronary and right coronary annuli. Behind the noncoronary sinus, and in direct opposition to it, are the interatrial groove and parts of the left and right atria. Part of the right coronary annulus, as mentioned earlier, is directly attached through the central fibrous body to the muscular septal wall. It courses along the right ventricular outflow tract, merging at its commissure with the left coronary annulus adjacent to the pulmonary valve annulus. The right coronary artery originates from the upper part of the right coronary sinus of Valsalva and courses down the right atrioventricular sulcus. The left or anterior segment of the left coronary annulus underlies the only part of the aortic root not related to any of the cardiac chambers. The right or posterior half of the left coronary annulus is in opposition to the left atrium. The left main coronary artery arises from the upper part of the left sinus and runs a short but variable distance behind it before dividing into its branches. It is important to understand the functional anatomy of the aortic valve when considering valve repair or valve

preserving aortic root procedures. The aortic root consists of four components: the aortic annulus, the aortic cusps, the sinuses of Valsalva, and the sinotubular junction. The aortic annulus is attached to the interventricular septum and fibrous structures along 55% of its circumference, with the remaining 45% being attached to the ventricular myocardium. The aortic cusps have a semilunar shape and the length of the base is normally 1. 5 times the length of the free margin. The commissure is the highest point where two cusps meet, which is just below the sinotubular junction. The annulus has a scalloped shape, and the diameter of the annulus in younger individuals is normally 15% to 20% larger than the diameter of the sinotubular junction. In older patients, these two diameters are nearly equal. The average length of the free margin of an aortic cusp is 1. 5 times the diameter of the sinotubular junction. In general, the noncoronary cusp is slightly larger than the other two, and the left is the smallest.

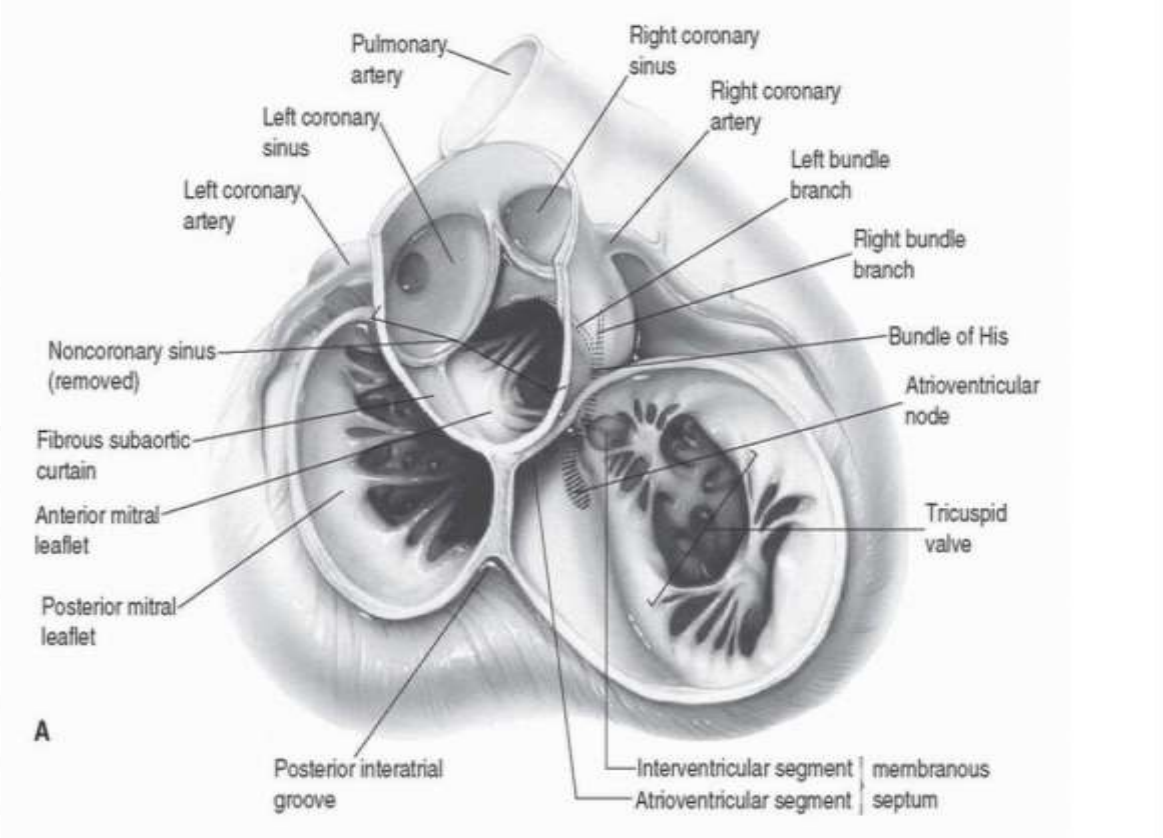


Figure 1: Posteroanterior view of the aortic valve with PA transected and atria removed. [13]

RELATIONS OF THE AORTIC ROOT:

RIGHT CORONARY SINUS:. The entire right coronary sinus lies adjacent to the

RVOT. The central part lies adjacent to the crista supraventricularis, and the left part is adjacent to the area of the RVOT in the angle between the crista supraventricularis and the pulmonary valve. The, posterior (noncoronary) part of the right coronary sinus is related to the area of the right ventricle posteroinferior to the crista supraventricularis. Inferiorly, the entire right coronary sinus is related to the interventricular septum; the muscular septum lies under the central and left parts, while either membranous or muscular septum may lie under the posterior part of the right coronary sinus.

NONCORONARY SINUS. The atrial chambers with the intervening atrial septum lie adjacent to the noncoronary sinus. The right and central parts of the noncoronary sinus are related to the right atrium and the interatrial septum, whereas the left part is related to the left atrium. Inferiorly, the right part, like the posterior part of the right coronary sinus, may be related either to the membranous or the muscular septum depending on the size of the membranous septum. However, beneath the central part of the noncoronary sinus, the membranous septum is a constant structure. The left part of the noncoronary sinus inserts into the anterior mitral leaflet and, along with the posterior part of the left coronary sinus, is the only part of aortic wall not connected to the ventricular wall.

LEFT CORONARY SINUS. The posterior part of the left coronary sinus shares the same relationship as the left part of the noncoronary sinus, that is, it is related to the left atrium posteriorly and to the anterior mitral leaflet inferiorly. The central part of the left aortic sinus is the only part of the aortic root that is not related to a cardiac chamber; it is adjacent to the epicardium only. The right part of the left coronary sinus lies adjacent to the pulmonary trunk at the level of the left pulmonary sinus. Inferior to it lies the muscular interventricular septum.

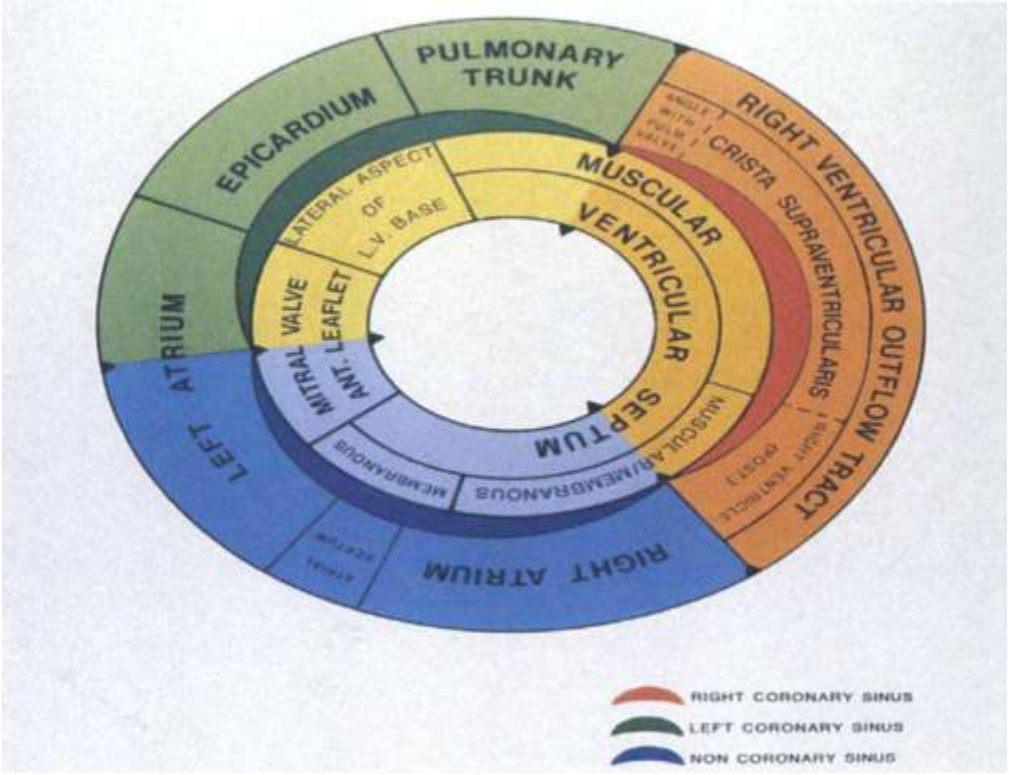


Figure 2: Anatomical relationship of the Aortic root sinus [14]

The most critical function of the aortic root is to perfuse the coronaries. Typically there are two coronary ostia that perfuse the left and right coronary arteries, respectively. Multiple ostia for both coronaries, however, is not rare and knowledge of ostia anatomical variation is crucial during an aortic root reconstruction and coronary angioplasty. [15-16] In a study by Pejkovic et al, ostia were located 2-10mm inferior to the sinotubular junction in 90% of cases. Additionally, separate conal ostia from the right sinus of Valsalva were found in 33% of cases. The pathologic significance of left and right coronary arteries originating from only one ostia (from either the left or right sinus of Valsalva) has a noted correlation with sudden death at a young age. This anomaly is exceedingly rare.

DISEASES OF THE AORTIC ROOT:

1. Congenital

Bicuspid aortic valves are present in approximately 0. 5-2% of the population. [17] Rather than a simple failure of fusion of two cusps, embryology studies with animals portray a complex interaction between intracellular pathways and between individual stem cells. [17] Multiple formations of bicuspid valves have been described in addition to variable surface sizes. The most common bicuspid formation is anterior-posterior in

nature with the left and right coronary ostia sharing the raphe of anterior sinus of Valsalva. [20] Bicuspid aortic valves, and the associated aortopathy, can lead to valvular stenosis and regurgitation, as well as ascending aneurysms and dissections. One-fourth of patients with bicuspid valves will have normal valvular function and, in one natural history study, required no medical or surgical intervention at 20 years of follow-up. [21, 22].Figure 2 shows a normal valve in the open and closed compared to a bicuspid valve. Fusion of two leaflets is noted in the closed bicuspid valve positions. The relative frequency of each morphological abnormal leaflet fusion is depicted. [18] Unicuspid and quadricuspid valves also exist but are less common. Unicuspid valves occur in approximately 1 of 10, 000 individuals and patients seem to have similar valve and aortic pathology as compared to patients with bicuspid valves. [23, 24] The prevalence of unicuspid aortic valves are so rare that the risk of aortic root disease cannot be quantified by clinical studies; only case reports and summaries exist. Likewise, quadracusp valves are rare occurring in 1-10 patients per 100, 000. It usually leads to insufficiency at an early age. [25]. Anecdotall, authors recommend stress testing prior to undergoing valve replacement. [25]. Congenital bicuspid valve as seen from the root position, in vivo. Bulky calcifications cover the luminal surface of the valve.

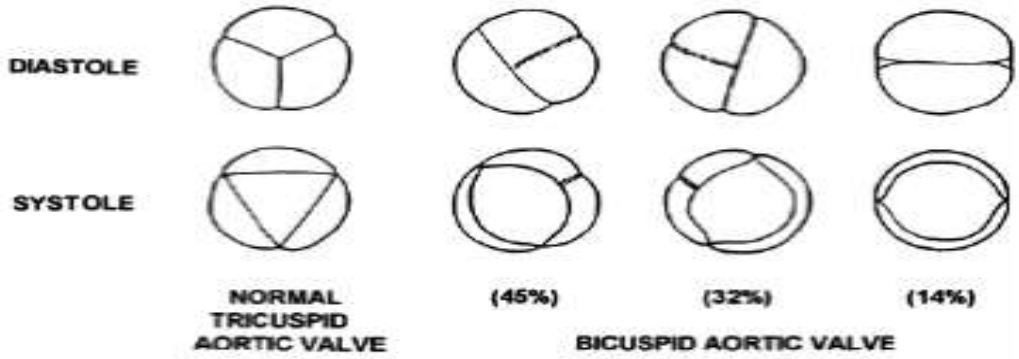


Figure 3: Fusion of two leaflets noted in bicuspid valves and frequency of abnormal form

*2. **Acquired***

The most common acquired condition of the aortic valve is calcific valvular disease. [26] This typically leads to aortic stenosis but can also cause a mixed pathology of both stenosis and regurgitation. During 2009 in the United States alone, over 40, 000 patients underwent aortic valve replacement (AVR) with or without coronary artery bypass grafting. [27] Isolated infection of the leaflets typically leads to regurgitation. Usually both of these conditions are not considered "root" problems as they can be treated with

surgical replacement of the aortic valve, however they can evolve into root problems when calcium deposition in the aortic wall becomes severe or the infection forms a root abscess, as will be discussed.

3. Aneurysmal disease of the root

Since the ascending aorta begins at the level of the sinotubular junction it is frequently involved with any aneurismal root pathology. Hence, any discussion of root pathology often involves the ascending aorta as well. The ascending aorta is considered to be aneurysmal if the diameter is greater than 3. 5cm. [28] the aortic root, however, is not considered aneurysmal until it is greater than 4cm. [29] Aneurysms of the root and ascending aorta have multiple etiologies including genetic, inflammatory, acquired and infectious. Disorders that cause degenerative changes in the root wall are most common. Aortic root aneurysms are common, accounting for roughly 70% of all thoracic aneurysms. [30] The risk of fatal complications of these aneurysms strongly correlates with aneurysm size. In one natural history study, the risk of death, dissection, or rupture in patients with aneurysms >6 cm had an incidence of 15% per year. [22] Reports on growth of the aorta are variable with some reports showing little growth while others report growth of up to 0. 2 cm/year in patients with aortic stenosis and a bicuspid valve. [22]

4. Genetic

Marfan's Syndrome, Loeys-Dietz syndrome, Ehlers-Danlos syndrome as well as others are known genetic causes of aneurismal dilatation. [23, 24, 25] These disorders cause deficits in the formation of the aortic wall during embryogenesis and lead to flow abnormalities. This eventually can result in aneurysm formation. Marfan's syndrome is a well-characterized, autosomal-dominant disorder that causes cystic necrosis within the media layer of the aortic wall. These patients have mutations in a single fibrillin gene, FBN1. Prevalence of Marfan's ranges from 1 in 10, 000 to 20, 000 people. [26]

Although aortic root problems have the most dramatic sequelae in patients, other systems are adversely affected by this single gene mutation including the lungs, bones, muscles, and the central nervous system. Aortic dissection and subsequent rupture is the most common cause of sudden death in Marfan's patients. [27] Patients with Marfan's associated aortic root dilatation are recommended to undergo surgical repair if the diameter of the aorta is >4. 5cm. [23] Diagnosis of the disorder at a young age is crucial to prevent catastrophic aortic complications, yet 24% have the initial operation in an

emergency setting. [28] Loeys-Dietz Syndrome is an autosomal-dominant disorder that became known for having an association with aortic root aneurysms. The disorder was first discovered in 2005. [29] The baseline aortic diameter in these individuals are small, yet have a tendency to dissect or become aneurysmal at a young age. When ascending aneurysms are identified in these patients, one group of authors recommended fixation when the size reached 4. 2 cm. [23]. Ehlers-Danlos Syndrome is an autosomal-dominant disorder with many subtypes and each subtype typically leads to specific end-organ pathology. Vascular type (Type 4) Ehlers- Danlos Syndrome is prone to cause dissection without aneurysm formation. [29, 30]

Surgical results in these patients have been poor. [30] Patients with a bicuspid aortic valve are predisposed to root aneurysms because of the associated aortopathy. An exact inheritance pattern for bicuspid disease has not been determined, rather, it's believed that most cases of bicuspid valves are due to multiple genes that interact causing abnormal root structure. [31]

Researchers believe that pooling the genetic and histologic changes identified in bicuspid valve patients ultimately leads to aortic root dilatation. Root enlargement is described at a younger age in patients with bicuspid valves and therefore the risk of root disease is higher in these patients. [32]

5. Inflammatory

A variety of inflammatory disorders affect aortic compliance leading to aneurysm formation and dissection, prompting the need for surgical repair. Giant cell arteritis (GCA) causes inflammation of the endothelium typically involving the temporal arteries leading to malaise, frequent temporal headaches, fevers, and jaw claudication. Infrequently patients will have complete visual loss. The gold standard of diagnosis remains temporal artery biopsy. The temporal artery is found to be involved in approximately 50% of specimens while the proximal aorta and immediate branches have less frequent involvement, 10-15%. [33]

In a study of autopsy specimens, 4 in 1000 specimens had giant cell arteritis while 1. 5 per 1000 were found to have dissection. [34]

Takayasu's arteritis is a form of large vessel vasculitis characterized by granulomatous inflammation in the aortic wall leading to intimal fibrosis and narrowing. Early symptoms are\ non-specific including malaise, fevers and rigors while late phase symptoms are ischemic in nature consisting of syncope, angina, and visual disturbance.

[35]

In rare cases, rupture of the aorta and proximal branches is caused by aneurismal disease of the vessels. Survival with rupture of a lesion due to Takayasu's disease is exceedingly rare. The mainstay of treatment is systemic corticosteroids. [36] In cases of aneurysm formation, surgical intervention should be delayed until the acute inflammatory phase has resolved. [37, 38].

Other inflammatory disorders account for a minority of aortic root pathology. Reiter's syndrome is an autoimmune inflammatory disease that is characterized by reactive arthritis.

There are rare cases of ascending aneurysms and severe aortic regurgitation in patients with longstanding inflammatory responses in severe cases of Reiter's. [39] Ankylosing spondylitis is an inflammatory disease that has a strong association with HLA-B27 and is characterized by joint pain involving the axial skeleton. Nearly 20% of patients with ankylosing spondylitis required aortic valve replacement in one case control study. [40]

6. Infectious

Infections of the aortic valve that are uncontrolled can lead to spread to contiguous structures, i. e. spread to the aortic wall causing dehiscence and formation of root abscesses. Left unchecked, the infection can erode further leading to involvement of the mitral and tricuspid valves as well as fistulisation to atria and right ventricle. The need for surgery in the management of cardiovascular complications of syphilis in the past fifty years has been exceedingly rare. Patients in need of surgery because of these complications are usually not diagnosed until after fixation. When surgical correction is required, ascending aortic involvement is diffuse, starting at the sinotubular junction proximally and extending distal to the arch. Grossly and histologically the aortic wall is comparable to those patients with GCA or ankylosing spondylitis. [41]

7. Calcific atherosclerosis

Calcific atherosclerosis of the coronaries is well characterized in the literature. However, within the past decade implications of a heavily calcified aortic root have also become evident, especially in association with calcific aortic stenosis. This may make aortic valve replacement complicated and necessitate root replacement. [42-44]

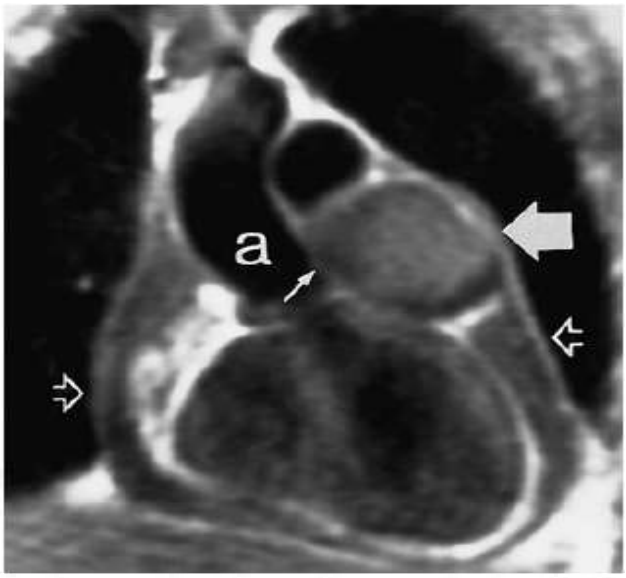


Figure 4. A sinus of Valsalva aneurysm highlighted by the large bold arrow + the inflow tract to the aneurysm.

8. Sinuses of Valsalva aneurysms

Aneurismal disease of the sinuses of Valsalva occurs between the aortic valve annulus and the sinotubular junction (Figure 3). Relative to the spectrum of other aortic root pathology, sinus of Valsalva aneurysms are very rare. Studies of large patient series show that the rate of these aneurysms found in all cardiac operations is roughly 0. 5%, and more so in Eastern populations. Most have extended adjacent to the left ventricle by the time of surgery. [45, 46]. The sinus most commonly involved is the right coronary, followed by the non-coronary and left coronary sinus. [47] Indications for surgery include rupture, infection, and flow impedance of the coronary ostia. The goal of surgery, regardless of the specific technique, is to close the defect of the wall, resect the fistula if present, and resect the aneurysm sac. [50]

9. Aortic root trauma

Traumatic injury to the aortic root requiring operative management is rare, yet one needs to be aware of the injury pattern and understand indications for operative repair. Blunt thoracic aortic traumatic injury usually occurs at the level of the ligmentum arteriousum just distal to the branch point of the left subclavian artery. [49] A minority of injuries, <10%, occur at the level of the ascending aorta. [49] When aortic injuries are identified, surgery can often be delayed until other traumatic injuries are corrected according to Mattox et al. [50] those patients who are at highest suspicion of aortic injury need CT angiography. The sensitivity of CT is typically high enough to use for screening,

however, the assessment of the aortic root is currently regarded as inadequate. [51] Well designed studies in the last two decades sought to provide evidence that trans-esophageal echocardiogram (TEE) was a reasonable screening test, however, it was no better than CT with regard to all thoracic injuries. [52-54] When sensitivity, cost utilization, and quality of life on follow-up are given equal consideration, it is advocated that chest radiograph and aortography continue to be the best diagnostic tools to assess for proximal aortic and root injury[55]. Patients with root injuries often have other major injuries requiring management prior to the root and aorta. [56] When surgical repair is indicated it is frequently for contained rupture of the aortic wall. Because the injury is often distal to the sinotubular junction, surgical fixation is feasible. [57] Injuries to the aortic valve leaflets, sinuses, and coronary ostia have also been reported, but only in case reports due to the lack of prevalence. [58, 59]

PATHOPHYSIOLOGY AND PRESENTATION OF AORTIC ROOT DISEASE

1. Aortic stenosis

The aetiology can be divided into three separate categories including post inflammatory scarring, senile calcific stenosis, and calcific stenosis of the congenitally deformed valve. [60]. Rheumatic fever accounts for less than 10% of all cause aortic stenosis and continues to decline in modern society but is still very common in underdeveloped countries. Regardless of the aetiology of aortic stenosis, all have the potential to progress to left heart failure if left untreated. Grossly, calcific disease of the aortic valve is a heaped up mass of calcium that usually projects into the sinuses. [61] Only recently is this process of calcium deposition being understood as an active regulatory process rather than degenerative. Calcium deposition on the valves is the result of a complex interaction between interstitial cells via paracrine signals. [62] Valvular sclerosis eventually leads t o a pressure gradient between the left ventricular outflow tract and aortic lumen. The left ventricle attempts to compensate and overcome this pressure gradient to maintain perfusion by concentric hypertrophy of the myocardium. [62]

Clinically this corresponds to the three hallmarks of aortic stenosis including angina, congestive heart failure (CHF), and syncope. Symptom severity directly correlates with prognosis, as 50% of patients with CHF will die in 2 years without intervention. [63]

2. Aortic regurgitation

Regurgitation of flow into the left ventricle occurs during the diastolic phase. Causes of this reverse flow are numerous, however, the predominant causes of include calcific stenosis and a dilated aortic root. [61] Calcific stenosis leads to stiff leaflets that stay in a fixed open position, even in the diastolic phase, and this allows for reflux into the left ventricle. [61] Aortic root disease causing valvular regurgitation is due to tension on and retraction of the cusps. [14] The Starling principle demonstrates the stretch of the myocardium is increased due to volume expansion from the regurgitant blood. [64] Cardiac contractility is increased due to an added volume at end diastole. This creates a vicious cycle of increased output due to contractility, yet there is also gradually increasing regurgitant flow as left ventricular output increases. Chronically, the forces of volume and pressure overload in addition to increased contractility lead to eccentric hypertrophy. Hypertrophy leads to increased myocardial wall tension causing to fibrosis and ischemia. [65] Chronic reflux of flow back into the left ventricle causes a combination of pressure and volume overload. [65]

Signs and symptoms are not noted until the patient develops congestive heart failure. Patients without significant predisposing factors (Marfan's or bicuspid valve) however may have progressive regurgitation for decades without symptoms. [66] The first symptoms to develop are disguised as primarily pulmonary complaints such as exertional and nocturnal dyspnoea. Some patients complain of vague thoracic pain or headaches. Angina is a late finding that signifies end stage left ventricular function. [67]

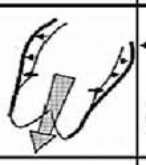
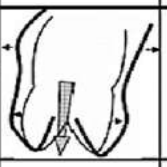
AI Class	Type I Normal cusp motion with FAA dilatation or cusp perforation				Type II Cusp Prolapse	Type III Cusp Restriction
	Ia	Ib	Ic	Id		
Mechanism						
Repair Techniques (Primary)	STJ remodeling *Ascending aortic graft*	Aortic Valve sparing: *Reimplantation or Remodeling with SCA*	SCA	Patch Repair *Autologous or bovine pericardium*	Prolapse Repair *Plication Triangular resection Free margin Resuspension Patch*	Leaflet Repair *Shaving Decalcification Patch*
(Secondary)	SCA		STJ Annuloplasty	SCA	SCA	SCA

Figure 5:El Khoury functional classification for aortic regurgitation (SCA: Subcommissural annuloplasty is used much less frequently now than in the past

3. Type A dissection

A dissection occurs when there is a tear of the intima and a tunnelled pathway is made between the media and adventitia parallel to the lumen of the blood vessel. This dissection flap that is created diverts blood flow through true and false lumens with the false lumen created by the dissection. [68] Type A dissections are located in the ascending aorta and are known for having a high mortality. [69] With respect to the aortic root, patients with dissection can have dilated sinuses, aortic regurgitation, and acute pericardial tamponade and therefore repairing the dissection may also include root replacement or modification. A number of factors predispose certain populations to getting Type A dissection including genetic and acquired diseases. [70] Once the dissection flap is made, the false lumen diameter expands and there is elongation of the false lumen. It is hypothesized that the false lumen enlarges and true lumen collapses over time for two reasons. First, the relative overabundance of elastin within the wall of the true lumen causes it to be more compliant and compressible. Second, the pressure within the false lumen is higher causing the dissection flap to collapse the true lumen. [70] Symptomatically, Type A dissection is characterized by what is often described by patient as being "ripping" or "tearing" chest pain. Because dissections are known to travel retrograde, patients may have profound hypotension if the dissection involves the pericardium or aortic valve. Pericardial tamponade complicates approximately 20% of Type A dissections. [71]

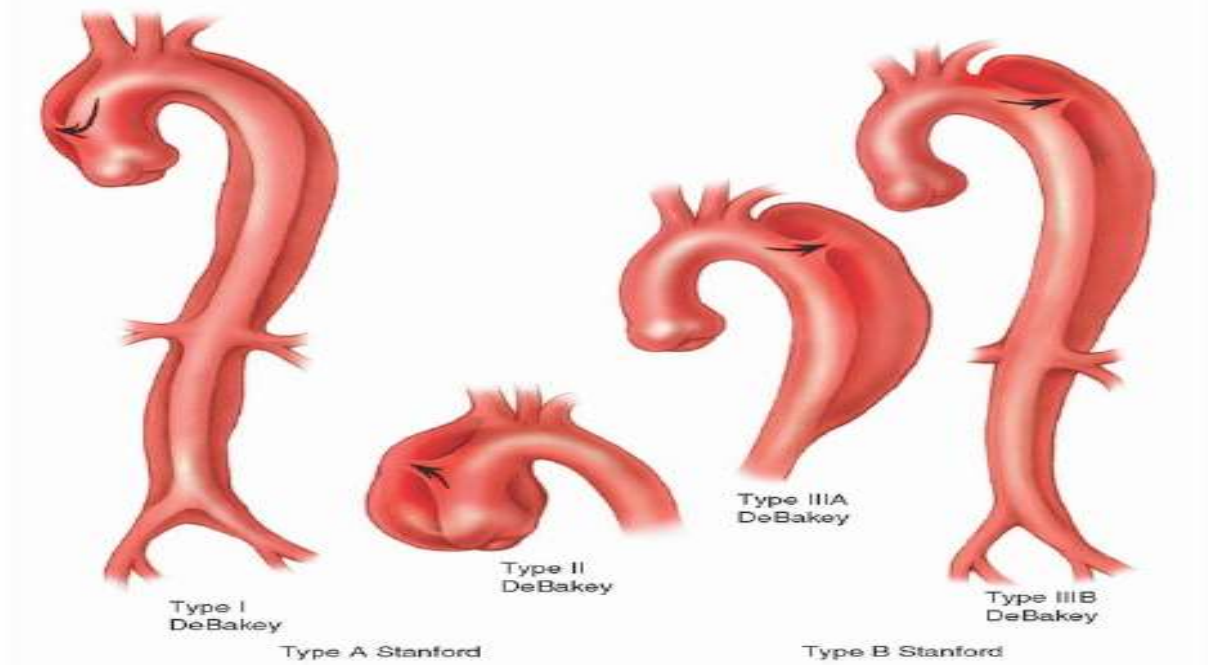


Figure 6: Classification of Aortic dissections

4. Ascending aorta & root aneurysms

The majority of patients with ascending aneurysms have inherent tissue abnormalities that result in a weak aortic wall. The most well described disorders associated with proximal aortic aneurysms of patients with a bicuspid valve and Marfan's. Both abnormalities cause cystic medial necrosis by replacement of normal elastic mesenchymal cells with mucoid degenerated cells. Patients with Marfan's and those with bicuspid aortic valves also have degenerative changes in the media. [72] The aortic roots have variable amounts of elastin and larger baseline aortic root diameters than the general population. [73-75] the underlying genetic association has yet to be determined.

Imaging of the aortic root

Imaging modalities most readily available for assessment of aortic root pathology include, echocardiography, both, transthoracic (TTE) and trans-oesophageal (TEE), computed tomography (CT), angiography, and magnetic resonance imaging (MR). Each has advantages and disadvantages when analysing abnormalities and planning for surgical repair. Echocardiography can assess aortic root and valve anatomy and function however, it does not give good views of the distal aorta. ECHO is also very useful for imaging other heart valves and ventricular size and function, all important for operative planning. While TTE is known to give accurate measurements of aortic root structures, it is not able to adequately detect dissection locations or extent of dissection with accuracy. [76] TEE, has proven to be safe and effective in the pre and post-operative assessment of patients with aortic dissection. [77, 78]

Computed tomography is an attractive means of assessing the ascending aorta when pathology such as dissection, aneurysm formation, ulceration, and intramural hematoma are suspected. Arterial wall enhancement with contrast is necessary for this technology to be utilized and patients with renal dysfunction or contrast allergy may have a contraindication. Low volume contrast studies have recently been used safely in patients with renal dysfunction. [80] Most series report the sensitivity for Type A dissection to be >90%. [81] Due to the varying degrees of signal enhancement, CT is able to distinguish between the false and true lumens in addition to the presence of thrombosis or communication of the false lumen. Similarly, the assessment of ascending aneurysms is accurate because of CT's ability to determine size, relative assessment of flow, and the aneurysms relationship to surrounding vital structures. [80] With regard to valve pathology, multidetector CT is able to provide an accurate depiction of aortic annulus

size, valve calcification, and degree of stenosis as compared to preoperative TEE and MR. [82] CT scans may be used to image the coronary arteries, heart and other thoracic structures. Indeed, heart surgery has been done safely without coronary angiogram in patients with normal coronaries on CT angiogram. [83, 84] Sensitivity Specificity-Echo (TEE) 95-99% 92-97%, Helical CT 96-100% 87-99%, MRI 95-99% 95-100%

	Sensitivity	Specificity
Echo (TEE)	95-99%	92-97%
Helical CT	96-100%	87-99%
MRI	95-99%	95-100%

Table 1: Mea-analysis by Shiga et al comparing sensitivity and specificity of CT TEE and MRI in Aortic dissection

Result of meta-analysis by Shiga et al describing the sensitivities and specificities of TEE, CT, and MRI for detecting thoracic aortic dissection. [79]. Use of MR angiography is typically an adjunct form of imaging used with echocardiography in patients with complex anatomy. At some institutions MR angiography is replacing CT as the primary imaging modality for assessment of diseases involving the thoracic aorta due toits decreased risk of radiation exposure. MR angiography (CE MR) provides improved diagnostic accuracy of thoracic vascular pathology when compared to other imaging. It has demonstrated a higher sensitivity and specificity than other forms of MR imaging and echocardiography. [85] Emergency use of MR is limited. Steady state free procession MR is a newer technology that allows for better visualization of structures by decreasing surrounding interference without the use of contrast. [83] This method has demonstrated success in the accurate visualization of diseases such as aneurysm, intramural hematoma, dissection, and ulceration of the native aorta as well as assessment of postoperative graft placement. [88] It is particularly attractive for patients who have a contrast allergy. Coronary angiography remains the gold standard for evaluation of the coronary arteries. Aortography can demonstrate aortic insufficiency and enlargement of the aorta, although we use CT and echo as it is much more accurate and less invasive. Ventriculography may also be done, however with severe aortic stenosis it may be difficult to cross the valve and may not be indicated because the risk of emboli. [87] Right and left heart pressures may also be obtained at the time of catherization.

SURGICAL MANAGEMENT OF THE AORTIC ROOT

In 1968, Bentall and Bono published the case of a patient with an ascending aortic aneurysm that involved the root and included coronary involvement. [88] In their case, a composite aortic graft was sewn to the annulus with a mechanical Starr valve. The coronaries were attached via an inclusion technique into the wall of the new prosthetic aortic root. Currently, the Kouchoukos modification with direct coronary button modification is the standard for root replacement today. Typically, the aortic valve tissue is removed, all abnormal aortic tissue in the sinuses and the ascending aorta is removed, and buttons of the right and left coronary artery are created. The root is then replaced with one of the following: a valved-conduit (either mechanical or biologic), a stentless valve as a root, a homograft, or a pulmonary autograft. There is a proximal suture line at the level of the left ventricular outflow tract, a distal suture line where the pathology of the aorta usually ends, and suture lines for re-implantation of both the coronary buttons.

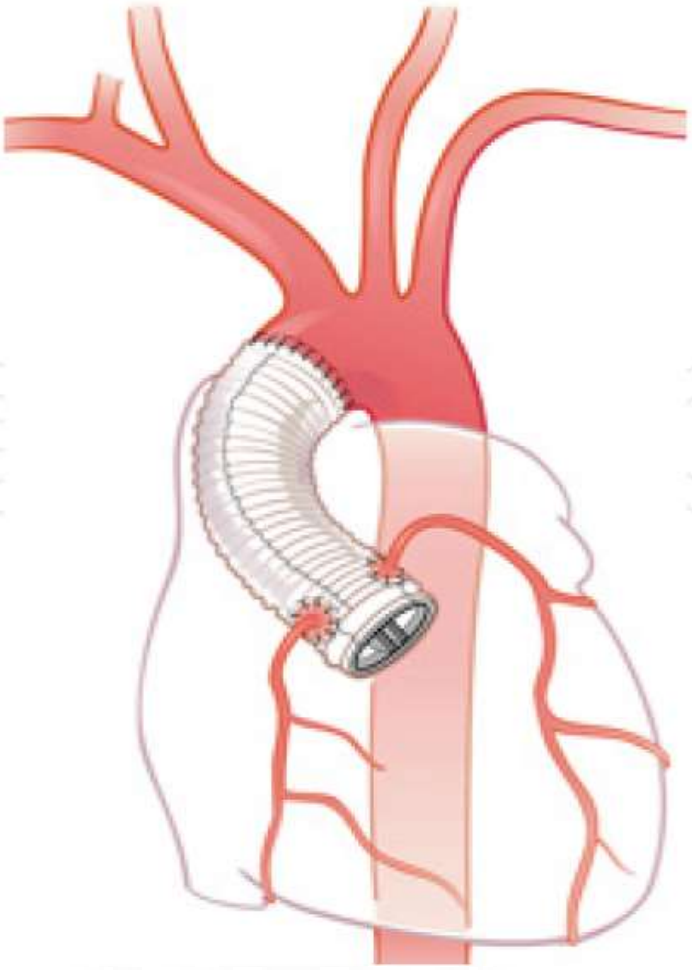


Figure 7: Graphic description of the Modified Bentall procedure

Prior to the description of this technique, which Bentall and DeBono published first, and which is now referred to as the Bentall procedure, aortic root pathology was a vexing problem for the thoracic surgeon and was addressed by a variety of techniques. All the previous methods of repair, such as aneurysm banding, plication, and supracoronary aortic replacement, were characterized by incomplete removal of diseased aortic tissue. This led to a high postoperative complication rate. Operations were most often performed on an emergency basis as a last resort, and consequently operative mortality in these patients was high. Since the introduction of the Bentall technique in 1968, aortic root replacement has become a safer procedure. This has prompted more frequent elective repair of ascending aortic lesions, with a much lower morbidity and mortality, allowing the further expansion of operative indications for elective composite aortic root replacement. Figure 6, an illustration from Mr. Bentall paper with Mr. De Bono, [89] shows the essentials of the Classic Bentall: use of a composite graft with the valve anchored in the aortic annulus; coronary ostia sutured to two small holes in the graft; and the graft wrapped with aortic wall.

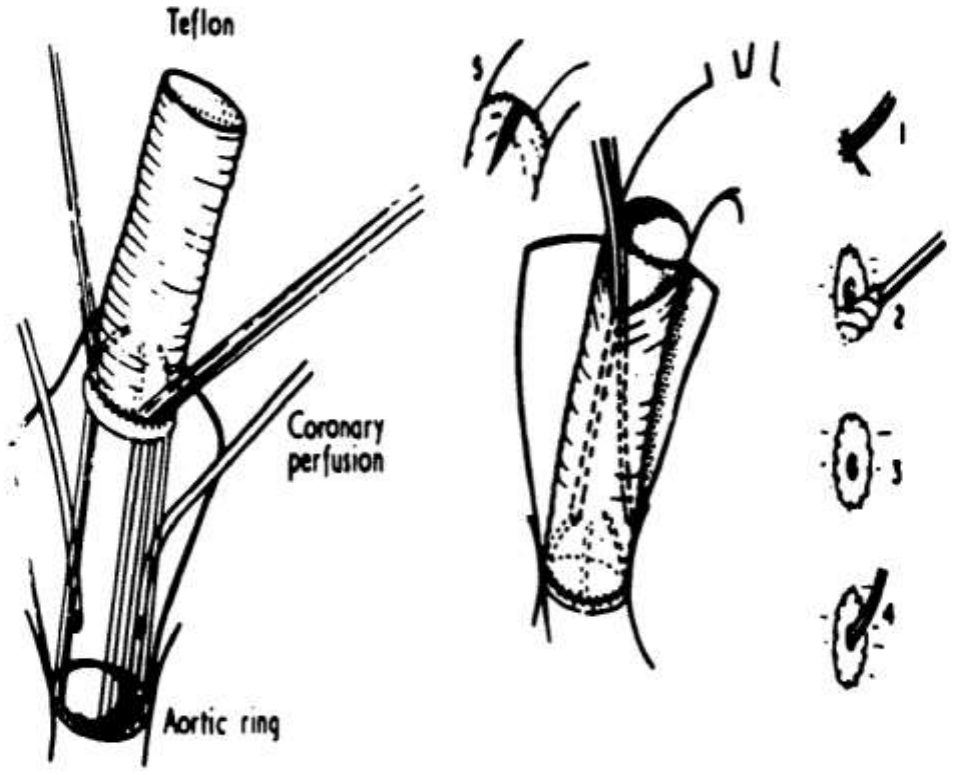


Figure 8: The Classical Bentall procedure from the original article by Bentall De Bono

In Paris, Professor Cabrol et al. [90] developed an operation for the ascending aorta and aortic valve which we refer to as the Cabrol Bentall procedure. There is a valve containing conduit, but the valve is displaced slightly superior to the annulus in Dr. Cabrol's original work. The important difference is that a separate graft is utilized to restore flow to the coronaries, usually an 8- or 10-mm Dacron graft. This technique was designed to overcome what Cabrol believed to be drawbacks in the Classic Bentall operation: poor visibility, and tension on the coronary anastomosis. Cabrol also believed that this modification would decrease the incidence of pseudo aneurysm formation at the coronary anastomoses. The Cabrol technique can also be performed easily even if the coronary ostia are not displaced cephalad, which is almost a prerequisite for safe performance of the Classic Bentall. Professor Cabrol was also responsible for introducing the idea of shunting the wrap of the native aorta to the right atrium in order to auto transfuse in cases of severe coagulopathy. We refer to this as a Cabrol fistula to distinguish it from the Cabrol treatment of the coronary arteries. Another modification that has been widely used was popularized by Kouchoukos et al. [91] and involves the complete resection of all diseased aortic tissue except for small buttons of aorta surrounding the coronary ostia. These buttons are directly anastomosed to the composite graft, and there is no use of the so-called "inclusion" technique, in which the native aorta is wrapped around the graft at the conclusion of the procedure. We refer to this technique as the Button Bentall. The Button Bentall allows excellent visualization of the coronary anastomoses, and avoids the use of an interposition graft, which may have a predisposition toward kinking and thrombosis. This procedure can also easily be performed in patients whose coronary ostia are not displaced cephalad. Over the past decade, modern series-[92-98] have reported each of these procedures to have a relatively low morbidity and mortality. As each technique has its advantages and drawbacks, there is a need to tailor their use to the individual case.

Retrospective review of 140 consecutive patients who underwent Bentall operations between October 1986 and March 1994, using three different anastomotic techniques: Classic, n = 30; Button, n = 95, and Cabrol, n = 15. Overall hospital mortality was 5%. In univariate analysis, acute type A dissection, rupture, new preoperative neurological symptoms, and the Cabrol technique were associated with a higher hospital mortality, but by multivariate analysis no independent risk factors were demonstrated. Overall

rates of reoperation did not differ among the three techniques. The actuarial freedom from reoperation was 87% at 5 years. The 5-year actuarial survival for all patients was 79% (Classic 85%, Button 82%, Cabrol 52%): the poorer results with the Cabrol modification are likely due to patient selection, complicated by a higher early mortality in this small group of patients. The presence of dissection was associated with a higher mortality in Marfan's patients (50% vs 8%, p = 0. 03). The rate of aortic valve-related complications was 3. 6%/patient per year. Actuarial event-free survival was 67% at 5 years. In patients with BAV and a diameter of the aortic sinuses or ascending aorta of ≥4.0 cm, lifelong serial evaluation of the size and morphology of the aortic sinuses and ascending aorta by echocardiography, CMR, or CT angiography is reasonable, with the examination interval determined by the degree and rate of progression of aortic dilation and by family history. In asymptomatic patients, or symptomatic patients with BAV and diameter of the aortic sinus or the ascending aorta >5.5 cm, operative intervention to replace the same should be undertaken

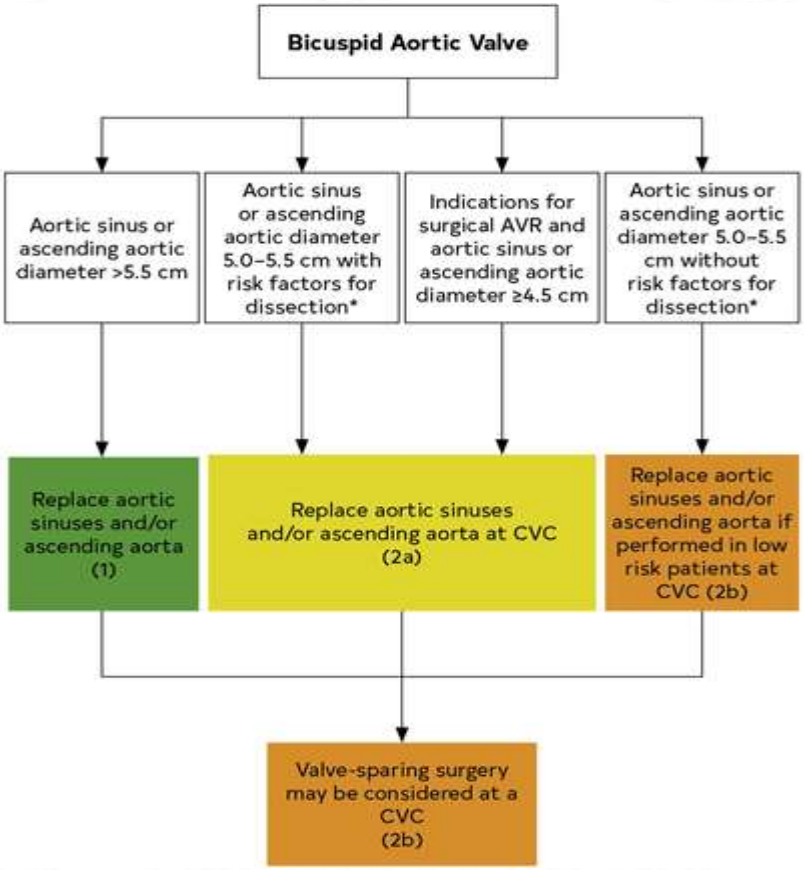


Figure 8 :Intervention for replacement of the aorta in patients with a BAV

The routine procedure of choice is the Button Bentall technique. With the Classic Bentall and the Cabrol variation reserved for use under special circumstances.

In total, 46 studies with 7, 629 patients (mean age, 50 years; 76% men) were selected. Pooled early mortality was 6% (422 patients). During a mean follow-up of 6 years (49, 175 patient-years), the annual linearized occurrence rate for late mortality was 2. 02% (1. 77%– 2. 31%; 892 patients), for aortic root reoperation it was 0. 46% (0. 36%–0. 59%), for hemorrhage it was 0. 64% (0. 47%–0. 87%), for thromboemboli it was 0. 77% (0. 60%– 1. 00%), for endocarditis it was 0. 39% (0. 33%–0. 46%), and for major adverse valve-related events it was 2. 66% (2. 17%–3. 24%). Operations performed in more recent years were associated with lower rates of aortic root reoperation (beta [–0. 452; p [0. 015). The systematic review illustrates that rates of aortic root reoperation after the Bentall procedure have decreased over the years. However, late mortality, major bleeding, and thromboembolic complications remain a concern. This report may be used to benchmark the potential therapeutic benefit of novel surgical approaches, such as valve-sparing aortic root replacement.

The disproportionate percentages in early mortality among several reports could be explained by different patient populations, concomitant procedures, and modifications in operative techniques or postoperative management strategies. Benke et al., on a total of 147 patients who underwent aortic root reconstruction reported an overall early mortality rate of 3. 4% [11]. The authors recognized concomitant surgery (CABG and mitral valve) as independent predictor of early complication but this however, was not translated as a risk factor for mortality in their study

Etz et al, studied a series of 597 consecutive studies showing overall hospital mortality was 3. 9%with biological valves versus 2. 8%with mechanical valves. In patients 50 to 70 years, age greater than 65 years (relative risk: 3. 3), clot (relative risk: 2. 5), coronary artery disease (relative risk:3. 5[P<. 0001]), and degenerative etiology (relative risk: 0. 4) were independent risk factors for long term survival (after postoperative day 30); there was no difference in long-term survival between biological and mechanical valves (relative risk: 0. 9) [12].

From August 1996 to October 2013, 110 consecutive patients underwent the modified Bentall technique. The procedure used Dacron composite graft with a mechanical valve (St. Jude Medical®) for aortic root replacement. Total bleeding after the operation was 450 ± 105 mL. The mean duration of intensive care unit and hospital stay were 2 ± 1 and 5 ± 2 days, respectively. Sixty-six patients (60 %) were discharged from the surgical intensive care unit on the first postoperative day, 34 patients (30. 9 %) were discharged

on the second day and tenpatients (9. 1 %) needed more time to stay in the intensive care unit due to haemodynamic or respiratory problems. 5-years follow up; survival rate was 97 %. In the three deceased patients, causes of death were mediastinitis, sepsis and myocardial infarction. No operation-related complications such as anticoagulant-related haemorrhage, valve or graft thrombosis, or coronary pseudo aneurysm were occurred during follow-up. The proposed modification of the Bentall technique seemed to minimize late intra-operative blood loss, improves homeostasis, shortens the operation time and is associated with excellent long-term outcomes in patients undergoing composite graft replacement of the aortic root. [99]

One hundred forty-two elective patients younger than 65 years without concomitant procedures who underwent replacement of the thoracic aorta and aortic valve between 1989 and 2000 were studied by Departments of Cardiothoracic Surgery and Biomathematics, Mount Sinai School of Medicine, New York, New York, 85% were men, and the median age was 46 years (range, 13 to 64 years). There were no intraoperative deaths. Two patients had a stroke postoperatively, one of which was fatal. Complications during follow-up included 2 cases of endocarditis, 1 peripheral thromboembolic event, and 10 instances of significant bleeding (requiring hospitalization or transfusion). Surgery for distal aortic segments was performed in 4 patients, but no patient required reoperation in the proximal aorta. Kaplan-Meier curves show overall survival is 0. 95 (95% confidence intervals, 0. 9 to 0. 99) at 5 years and 0. 93 (95% confidence intervals, 0. 86 to 0. 99) at 8 years, and event-free survival is 0. 85 (95% confidence intervals, 0. 78 to 0. 92) at 5 years and 0. 78 (95% confidence intervals, 0. 68 to 0. 88) at 8 years. It was concluded that the button Bentall procedure could be performed with excellent short-term and long-term results in relatively uncomplicated elective patients in whom aortic valve disease is combined with dilatation of the ascending aorta. Results of this traditional operation are the standard against which the long-term outcome of newer approaches, such as valve-sparing operations, should be compared. [100]

Between January 2006 and December 2013, 100 patients underwent aortic root replacement using rigid tilting disk TTK Chitra heart valve as the composite graft valve. This study includes patients between the age group 19–62 years who had Chitra valve in the composite graft. A single surgeon was involved during the entire period. The early operative mortality was 2% (two patients). The late mortality was 3% ($n = 3$).

Significant left ventricle (LV) remodelling was noted in 85% (n = 85) of the patients. Midterm survival is good. . Bentall procedure can be done safely with intra-operatively prepared composite graft with any existing valve. The Bentall procedure using intra-operatively prepared composite graft using TTK Chitra valve is safely used in many countries, and it offers excellent midterm results. The results are comparable to other series of composite graft study making it an effective, accepted, safe, and cost-effective root replacement prosthesis. [101]

A systematic review and metaanalysis of characteristics of and long-term outcome after the Bentall procedure with a mechanical valve prosthesis included in total, 46 studies with 7, 629 patients (mean age, 50 years; 76% men) were selected. Pooled early mortality was 6% (422 patients). During a mean follow-up of 6 years (49, 175 patient-years), the annual linearized occurrence rate for late mortality was 2. 02% (1. 77%– 2. 31%; 892 patients), for aortic root reoperation it was 0. 46% (0. 36%–0. 59%), for haemorrhage it was 0. 64% (0. 47%–0. 87%), for thromboembolic it was 0. 77% (0. 60%– 1. 00%), for endocarditis it was 0. 39% (0. 33%–0. 46%), and for major adverse valve-related events it was 2. 66% (2. 17%–3. 24%). Operations performed in more recent years were associated with lower rates of aortic root reoperation (beta [–0. 452; p [0. 015). This systematic review illustrated that rates of aortic root reoperation after the Bentall procedure have decreased over the years. However, late mortality, major bleeding, and thromboembolic complications remain a concern. [102]

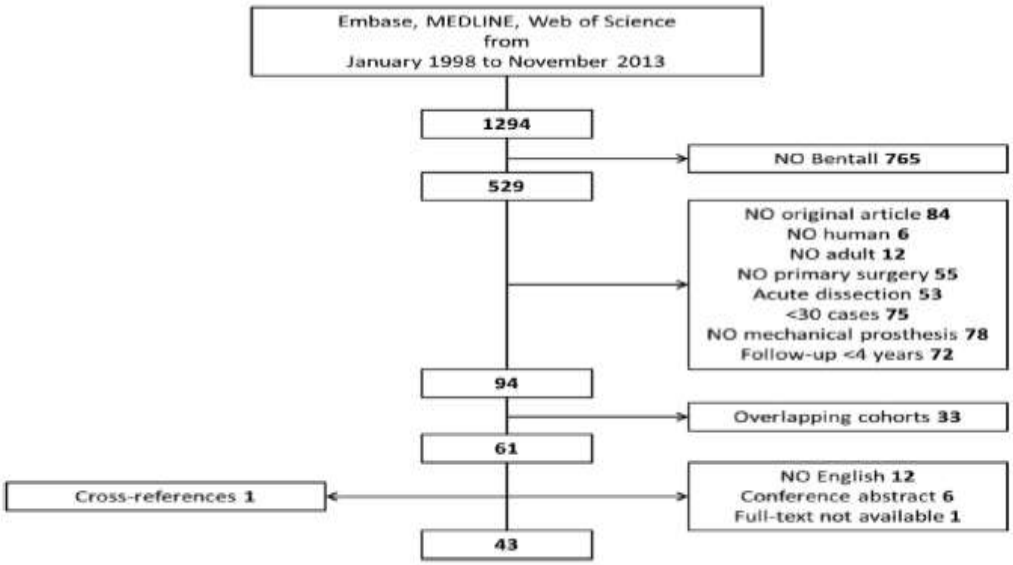


Figure 9: Flow Chart of the literature review and analysis

METHODS AND METHODOLOGY

Study Type - A retrospective observational study

Study population – All patients who underwent Modified Bentalls procedure in SCTIMST during period of 01/01/2008 to 31/12/2019

Data collection procedure – Retrospective analysis was performed by principal investigator after going through medical records. All the patients underwent follow up after the procedure and routinely as per departmental protocol. Follow up involved Trans thoracic ECHO, ECG, Chest X-ray and INR monitoring. Telephonic interview was done by principal investigator with a translator. Patients were asked about symptoms in the follow up period. Data so collected from the medical records and telephonic interview was analyzed. Procedure DID NOT involve banking of biological samples, HIV testing, genetic testing

Eligibility Criteria

Inclusión Criteria –

Symptomatic and asymptomatic Ascending Aortic and valvular pathology who underwent and Modified Bentalls procedure during period of 01/01/2008 to 31/12/2019

Exclusion Criteria –

- Patients under the age of 18
- Patients not willing for follow up.
- Patients who underwent additional procedures like CABG, Mitral valve repair/replacement. ASD/VSD closure
- H/O previous cardiac surgery.

Opérative techniques

All 104 patients underwent aortic root replacement with a composite valve graft made with prosthetic valve and graft. Most commonly used was TTK Chitra valve (TTK Health Care Limited, Thumba, Trivandrum, and Kerala India). 5 patients had bioprosthetic valve (Carpentier Edwards Perimount Magna, pericardial Aortic bioprosthesis, EDWARDS LIFESCIENCES LLC. One Edwards Way, Irvine ,CA 92614) and 1 patient received a Medtronic open pivot valve (The Medtronic Open Pivot AP360®). The button Bentall technique, a modification of the original technique described by Kouchoukos and coworkers. Cardiopulmonary bypass (CPB) was established by femoral cannulation in 6 patients, and the rest 98 patients by routine aorto-right atrial cannulation.

Standard hypothermic (28° to 32 °C) cardiopulmonary bypass techniques were used. Myocardial protection was achieved by administration of cold, blood cardioplegic solution, tepid blood cardioplegia and HTK solution with cardioplegia delivery system directly into the ostia. After commencing CPB the aorta was opened. Aneurysmal or dissected part/ ruptured aorta was excised with preservation of coronary buttons. Aortic valve cusps were then excised. After sizing of the aortic annulus with the valve sizer, a series of 2-0 pledget-supported polyester mattress sutures were on the annulus (either from LV to Aorta / Aorta to LV) for fixation of the composite graft. Preparation of the composite graft (Fig. 7) after the adequate size of the valve was determined, a tube graft (woven collagen-coated polyester vascular prosthesis) was selected mostly one size above the selected valve. A TTK Chitra sewing ring is not bulky, and hence, the ideal size was one size higher than the graft (e. g., if 21-size TTK Chitra is used, then a 22-mm coated Dacron graft is ideal). The sized TTK Chitra heart valve (TTK Health Care Limited, Thumba, Trivandrum, and Kerala India) is placed inside the tube graft and

sutured to the graft using 4-0 polypropylene continuous sutures making it a composite graft. {The tube grafts used are (a) InterGard: InterVascular MAQUET Cardio-vascular, La Ciotat, France; (b) Unigraft (K-DV) B/Braun Vascular System: Am Aesculap-Platz, 78532 Tuttlingen, Germany; and (c) BARD (collagen impregnated polyester vascular graft: 1625 West 3rd Street, Tempe, AZ 85281, USA)}. The chief surgeon takes aortic annular sutures while the composite graft is prepared by assistants to save CPB time. Then, the sewing ring suture bites in the composite graft was taken in such a way that it included the graft, giving reinforcement for the graft onto the valve. The composite graft was seated to the annulus and fixed. Button holes were created in the graft for coronary anastomosis. Holes in the graft for re-implantation of coronaries were made by initially making a nick with No. 11 blade and fashioning the coronary ostia using Potts scissors. The adequate length of the coronary arteries was mobilized, and the coronaries were implanted in an end-to-side fashion with 5-0 continuous polypropylene sutures. After the coronary buttons were sutured, the distal end was anastomosed to the proximal end of the resected ascending aorta or to the proximal arch. In case of aortic dissection with intimal flaps, arch was assessed under a brief total circulatory arrest (TCA) (cooled to 18°). 8 patients required TCA during the distal anastomosis, with mean TCA time of 3 minutes +/- 1 minute. One patient was put on DHCA with temperatures of 18 degree centigrade.

We then did the distal graft anastomosis after reapplying the cross-clamp as high as possible on the ascending aorta. The mean cardiopulmonary bypass time was 131. 22+/- 31. 08 minutes and the mean aortic cross-clamp time was 96. 8 +/- 18. 15 minutes for the entire cohort of patients. Routine weaning from bypass is done. Hemorrhage is the most common complication after Bentall procedure. Precise suturing with adequate tightening of each bite, less CPB time, using a felt for distal anastomosis, and using

fibrin sealant were the techniques adopted for adequate haemostasis.

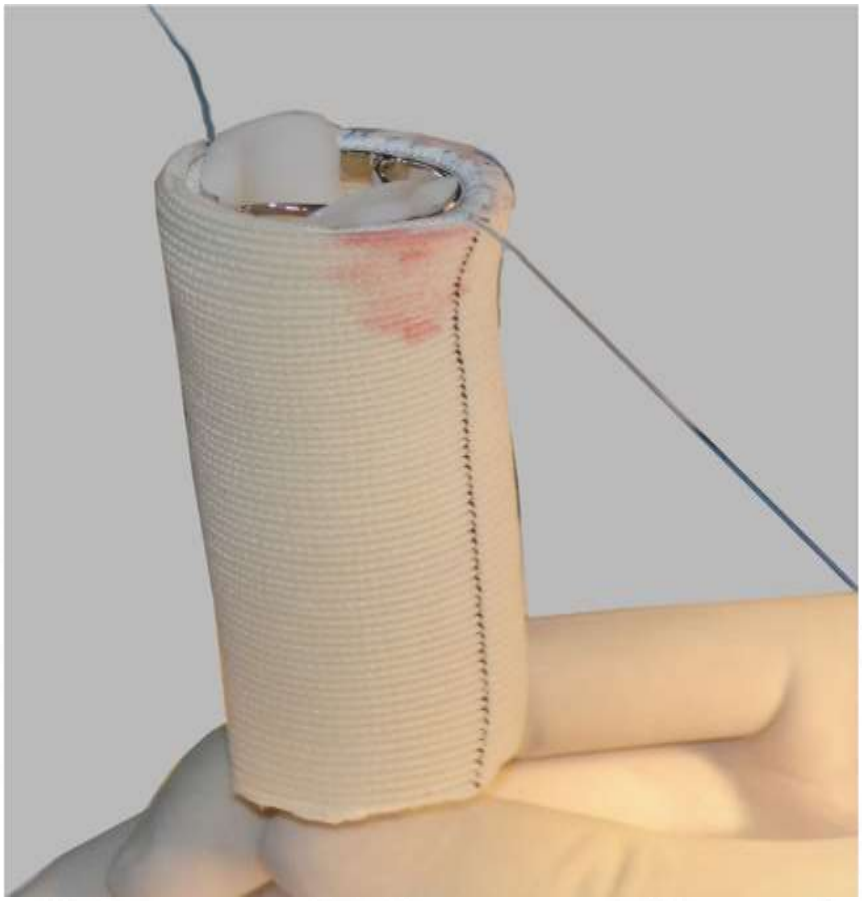


Figure 10: This composite graft has been made with composite TTK Chitra valve a tube graft. Valve held with holder from inside

Statistical Analysis

Data were entered in SPSS windows for data description and analysis. Percentages, medians, or means and standard deviations were calculated. Follow-up was measured from the date of the operation to either the earlier of the date of death or the last contact alive. Early death was defined as death during the initial hospitalization regardless of the number of days or death within 30 days of the procedure among patients discharged alive. Long-term follow-up was calculated for survivors, commencing 6 months after the procedure and terminating at the earlier of either death or last contact alive for survival estimates. For long-term adverse events, follow-up time was taken at the point of its

occurrence of adverse event. Functional class of symptoms at the time of follow-up and via telephonic interview was enquired and grades as per NYHA classification. Pre-operative echocardiographic values of systolic LV internal diameter (LVESD), diastolic LV internal diameter (LVEDD) and inter-ventricular septal thickness in diastole (IVSD) were documented in millimeters (mm). Trans-valvar mean and peak gradients were measured in millimeters of mercury (mmHg). LV ejection fraction was also documented.

Immediate post-op echocardiogram data of trans-valvar mean and peak gradients as well as post-surgery values of LVEDD, LVESD, IVSD, and LV ejection fraction were collected.

LV mass was calculated using the equation proposed by Devereux et al. [103].

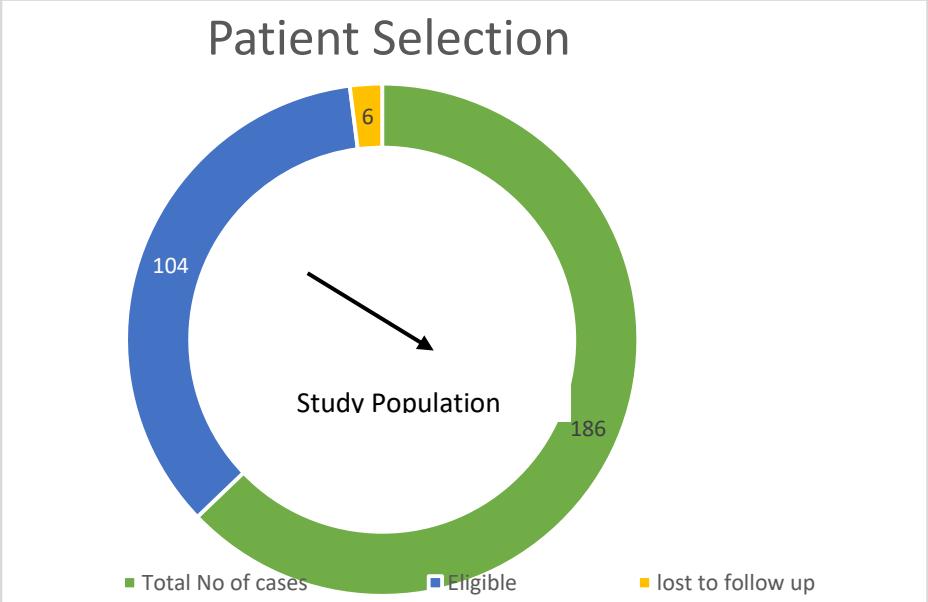
LV mass (g) = 0. 8{1. 04 [([LVEDD + IVSD + PWD] 3 − LVEDD3)]} + 0. 6.

Means between groups were compared using independent sample t tests. Preoperative and postoperative comparison of means was done using paired t tests. Percentages were compared by Fisher exact test. P values <0. 05 were treated as significant.

RESULT

Patient Sélection

Total of 186 patients underwent Modified Bentall procedure from January 1 2008 to December 31st 2019. 104 patients fulfilled inclusion and exclusion criteria of our study and were considered for follow up of which 6 were lost to follow up making the sample size of 98.



Graph 1: Patient Selection and Study Population

Pre – Operative Patient Characteristics:

Of the 98, there were 83 males and 15 females.

Gender	Frequency	Percent
Male	83	84. 7
Female	15	15. 3
Total	98	100

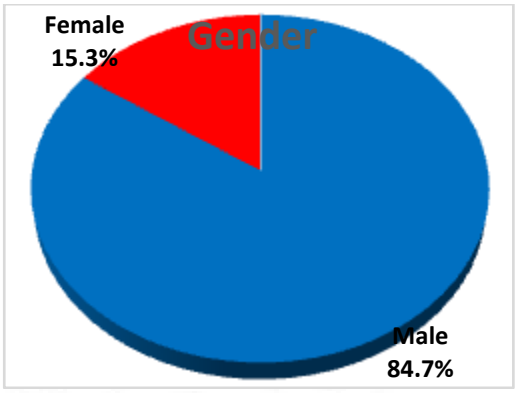


Table 2 and Graph 2: Pre-Operative Patient Characteristics

Pre-Operative Risk factors

Risk factors	Frequency	Percentage
HTN	51	52
DM	25	25. 5
CAD	6	6. 1
COPD	13	13. 3
RENAL DISEASE	8	8. 2
NEUROLOGICAL	7	7. 1
PVD	5	5. 1
CTD/MARFANS	37	37. 8
OTHER	29	29. 6
SMOKING	25	25. 5
EMERGENCY	4	4. 1

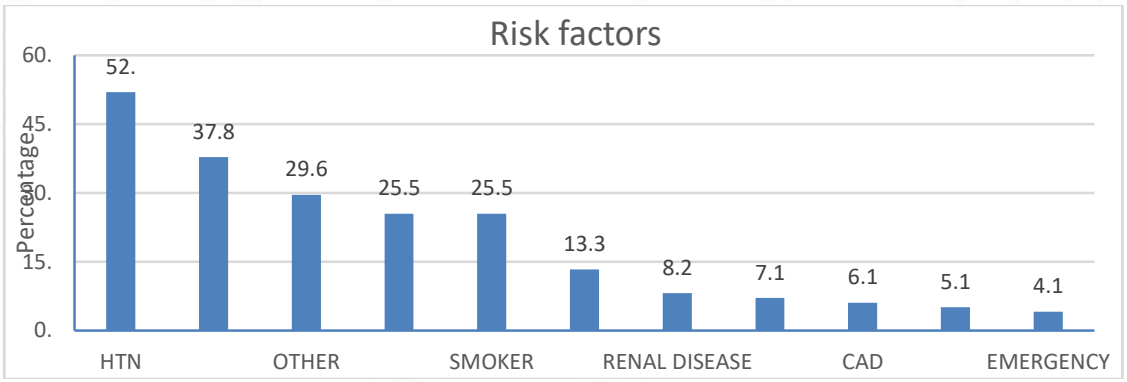


Table 3 and Graph 3: Pre-Operative Risk factors

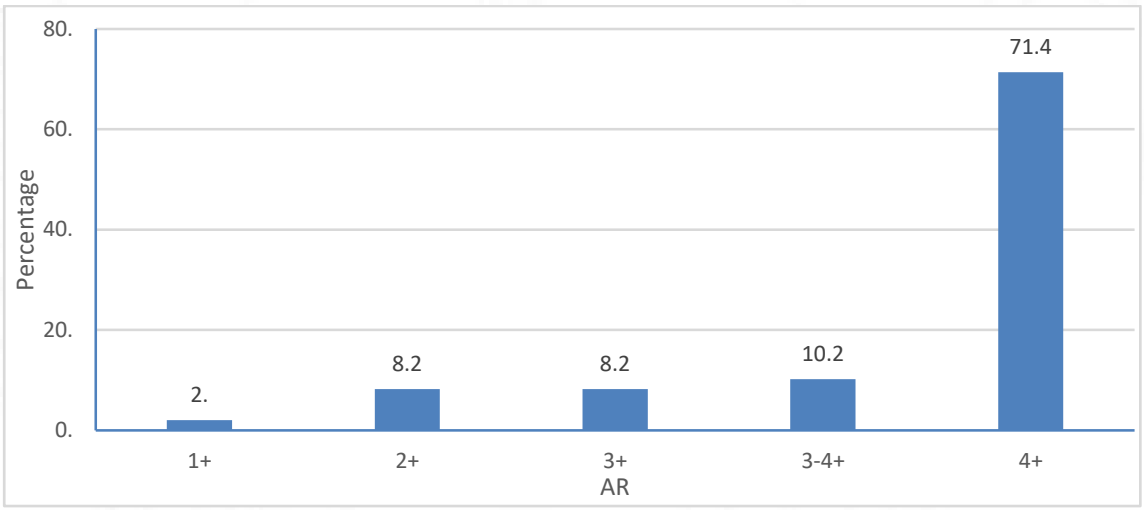
The most common comorbidity was Hypertension seen in 51 patients which is (52%)

followed by Diabetes mellitus in 25 % of the population. 6% had coronary artery disease on medical management. 8% had renal dysfunction preoperatively –reflected in Serum creatinine value of >1. 5 mg/dl. 7%had neurological dysfunction preoperatively with H/O CVA with no deficits preoperatively, one patient with seizure disorder. 37% had connective had Marfan's syndrome. Other comorbidities were seen in 29% of the population most commonly Hypothyroidism. One patient had neurofibromatosis and one had H/O Aortic fenestration done 3 years prior in view of Type B aortic dissection. 25 % of the patients were smokers and 13 % had respiratory disease most commonly Bronchial Asthma. 4% patients were taken up for the surgery on emergency basis, remaining were semi emergency /elective

Pre-Operative Pathophysiology:

Most common pathophysiology involved severe Aortic regurgitation seen in 78 % of the patients, 21. 2 % had aortic stenosis with Bicuspid aortic valves. `

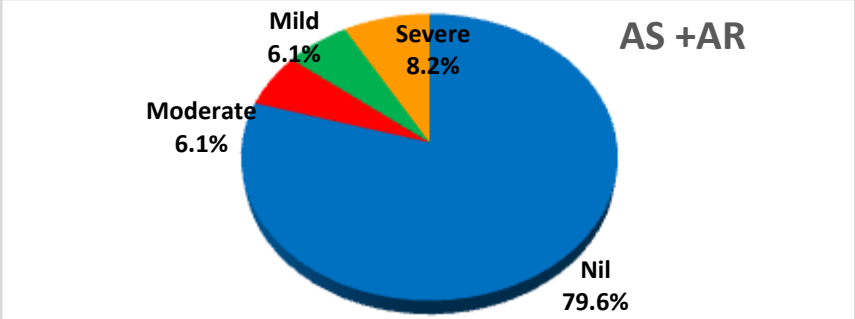
Aortic stenosis with regurgitation was seen in 20% of the patients with 8% having severe aortic stenosis, 6% having mild and 6% having moderate stenosis.



Graph 4(a) : Population distribution according to indication of surgery

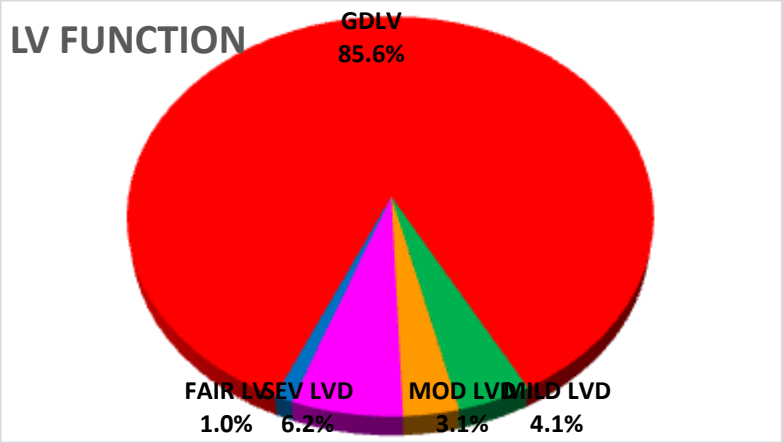
AR	Frequency	Percent
1+	2	2
2+	8	8. 2
3+	8	8. 2
3-4+	10	10. 2
4+	70	71. 4
Total	98	100

Table 4 and Graph 4(b): Severity of Aortic regurgitation



AS +AR	Frequency	Percent
Nil	78	79. 6
Mild	6	6. 1
Moderate	6	6. 1
Severe	8	8. 2
Total	98	100

Table 5 and Graph 5: Distribution of combined lesions of Aortic stenosis with regurgitation



Graph 6: Distribution of the study population based on LV function

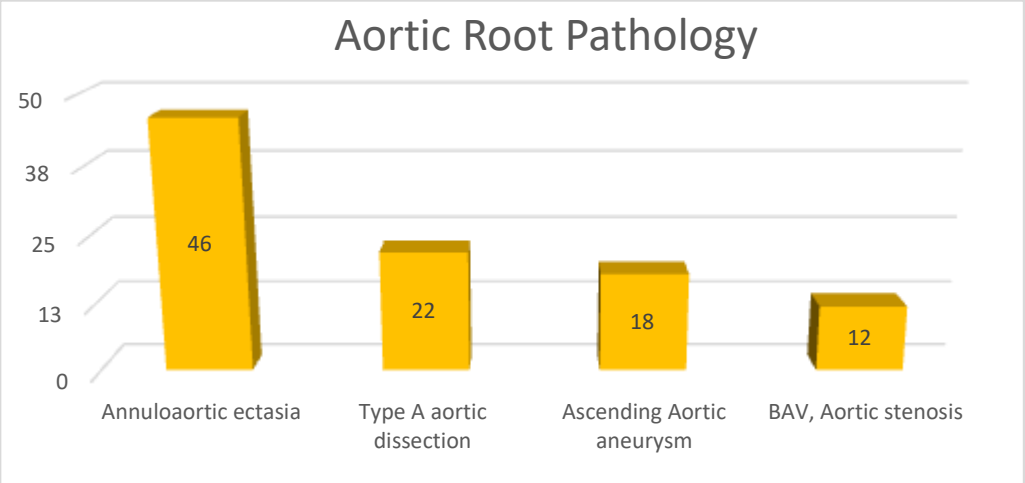
6. 2 % of the patients had severe LV dysfunction preoperatively and it was found to be associated with poor outcomes.

	Mean	SD
AGE	48. 98	13. 33
HT(CM)	168. 44	14. 01
WT(KG)	67. 09	15. 68
SINOTUBULAR JN	52. 54	9. 92
ASCENDING AORTA	56. 01	11. 63
AO ANNULUS	26. 80	4. 65
LV MASS	449. 85	155. 19
LV MASS(POST OP)	288. 39	128. 18
LV VOLUME	2688. 58	1042. 33
LV VOLUME(POSTOP)	1973. 60	1241. 71
HB	13. 22	1. 61
ALBUMIN	4. 46	4. 14
S. CREAT	1. 00	0. 30
ESR	15. 16	7. 61
CPB TIME	131. 22	31. 08

	Mean	SD
AORTIC CROSS CLAMP	96. 80	18. 15
TEMPERATURE	23. 17	1. 98
AORTIC VALVE SIZE	25	22
ALBOGRAFT	26	26
ICU STAY	3. 74	2. 63
HOSPITAL STAY	9. 72	3. 74

Table 6: Mean and standard deviation of preoperative, post-operative, intraoperative And postoperative variables

The mean cardiopulmonary bypass time was 131. 22+/- 31. 08 minutes and the mean aortic cross-clamp time was 96. 8 +/- 18. 15 minutes for the entire cohort of patients. 8 patients required TCA during the distal anastomosis, with mean TCA time of 3 minutes +/- 1 minute. The average ICU stay was 3.74 days and average hospital stay was 9.72 days



Graph 7: Distribution of the study population based on Aortic root pathology (Diagnosis)

The most common diagnosis was Annuloaortic ectasia with severe AR seen in 46 patients.22 patients had Type A aortic dissection, of which one had chronic healed

dissection and one had type B dissection with sudden progression into type A. 18 patients had Ascending aortic aneurysm and 12 patients had bicuspid aortic valve with AS and dilated aortic root.

The maximum root/ascending aorta diameters at the time of surgery was 56. 1 ± 11. 3 mm. patients were shifted from the operation theater with a small dose of inotropic supports. Seventeen patients required atrial/ventricular pacing. Inotropes were discontinued after 48h in 21 patients. Six patients had both inotropic supports and pacing. Pacing was discontinued after 6 hours in two patients and after 16–21 h in two patients. Ninety patients were extubated within12–18h. The mean intensive care unit stay was 3. 74 + 2. 63, and the mean hospital stay was 9. 72 + 3. 61 days.

	Alive (n=89)		**Death(n=9)**		**t**	**p**
	Mean	**SD**	**Mean**	**SD**		
AGE	48. 33	13. 12	55. 44	14. 52	1. 537	0. 127
LV MASS	443. 62	150. 49	511. 48	195. 47	1. 254	0. 213
LV MASS	281. 82	126. 86	367. 30	126. 45	1. 713	0. 090
LV VOLUME	2673. 20	1005. 90	2840. 67	1419. 29	0. 457	0. 648
LV VOLUME	1986. 13	1288. 95	1823. 29	341. 35	0. 332	0. 741
HB	13. 29	1. 65	12. 60	1. 10	1. 22	0. 225
ALBUMIN	4. 52	4. 34	3. 88	0. 42	0. 441	0. 660
S. CREAT	0. 99	0. 30	1. 18	0. 19	1. 928	0. 057
ESR	15. 11	7. 86	15. 56	4. 75	0. 165	0. 870
CPB TIME	129. 78	22. 80	145. 56	75. 76	1. 46	0. 148
AORTIC CROSS CLAMP	95. 24	15. 67	112. 22	31. 60	2. 766	0. 007
ICU STAY	3. 42	1. 03	7. 00	7. 67	4. 229	0. 01
HOP STAY	9. 28	2. 72	14. 11	7. 99	3. 968	0. 01

Table 7: Comparative variables between the alive and expired population

The above table comparative variables between the alive and expired population. Longer ICU stay and hospital stay was noted in the expired population with a p value < 0. 05 that is statistically significant. Also longer cross clamp time was found to be associated with mortality with a p value of 0. 007

Post – Operative Patient Characteristics

5 patients were lost to follow up of our hospital and 98 patients were followed up during mean 10 year follow up.

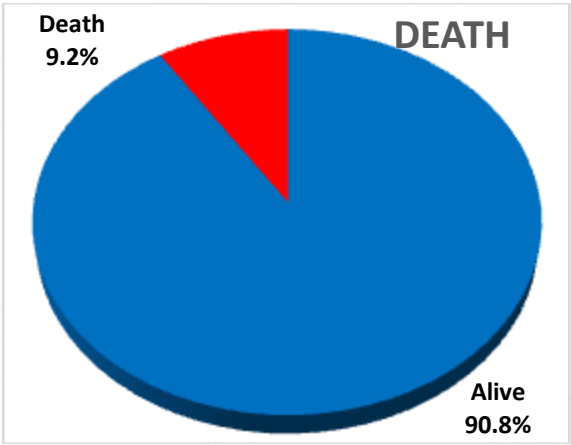
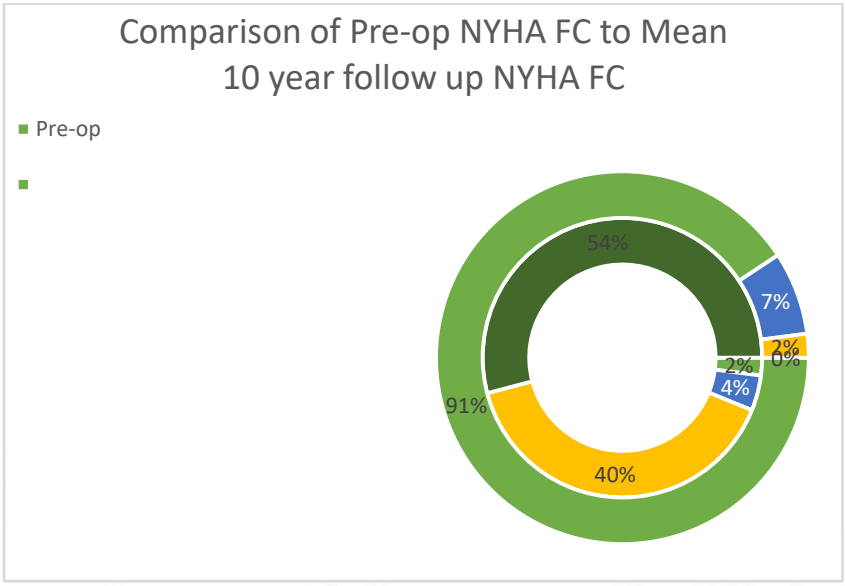


Table 8 and Graph 7: Post - Operative Outcomes

DEATH	Frequency	Percent
Alive	89	90. 8
Death	9	9. 2
Total	98	100. 0

Table 8.Post - Operative Outcomes

Comparison of Pre-op NYHA FC to Mean 10 year follow up NYHA FC



Graph 8: Comparison of Pre-op NYHA FC (Inner ring) to Mean 10 year follow up NYHA FC (Outer ring)

Results showed there was significant improvement in NYHA Functional Class at 10 year mean follow up period. 91 % of patients ($p < 0.01$) of study population remained in NYHA FC I during 10 year mean follow up period, which was at 2 % when compared to pre-operative period.

The most common symptom of presentation included shortness of breath. 3 patients complained of shoulder pain.

In the telephonic interview 91% patients were found to be able to carry on with routine activities without significant symptoms. 2 patients were symptomatic due to development of other valvular heart lesion-mainly mitral regurgitation.

90 % paints were compliant with anticoagulation and had minor OAC related complications like bruising and minor gum bleed. 7 patients had H/O hospitalization for anticoagulation related complication-anemia and melena and required FFP transfusion.

Complications post Modified Bentall Procedure:

	Frequency	Percentage
CARDIAC EVENT	17	17. 3
BLEEDING	7	7. 2
RENAL DYS	6	6. 1
NEUROLOGICAL	4	4. 1
WOUND INF	1	1
POST OP PNEUMONIA	3	3. 1
SEPSIS	4	4. 1
CARDIAC EVENT	13	13. 4
CVA(I/H)	7	7. 5
REOPERATIONS	10	11. 1
ENDOCARDITIS	3	3. 3

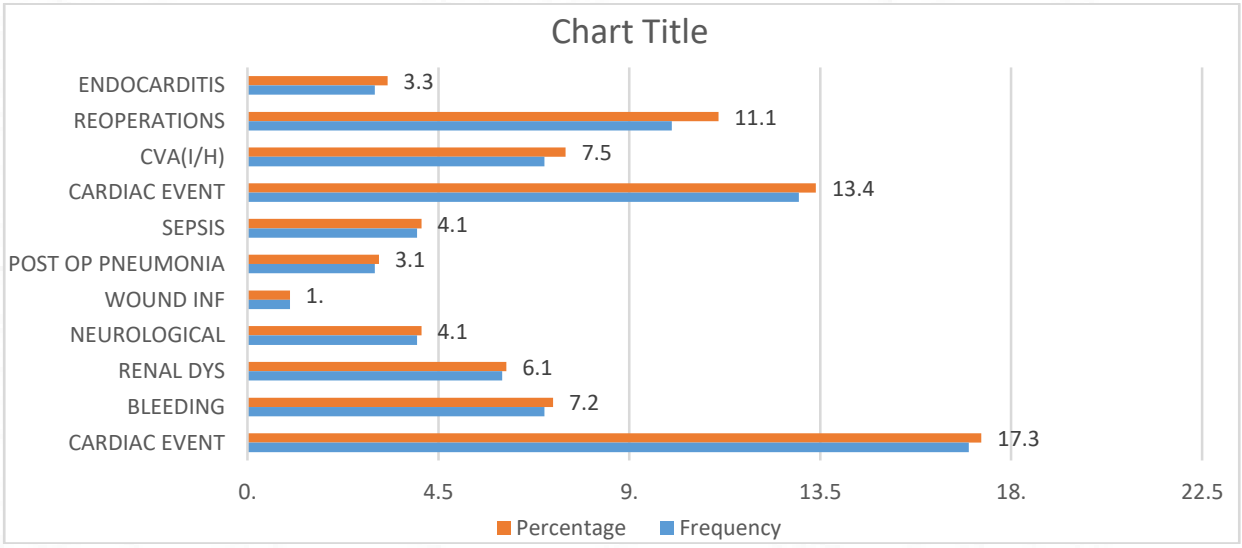


Table 9 and Graph 9: Complications post Modified Bentall procedure

Bleeding

7. 2 % of the population developed significant bleeding –drainage of > 800ml on POD .5 patients were given mediastinal wash bedside for the same. 2 patients were re-explored on POD1 for the same.

Cardiac Rhythm

Majority of patients in study were in Sinus Rhythm prior to surgery and also during post op follow up period. They received Warfarin / Acitrom lifelong. New onset of AF was diagnosed during ICU stay and follow up period. 16 patients developed AF of which 14 reverted to sinus rhythm with Amiodarone and DC version. One patient continued to be in AF follow up as well. 3 patients developed Ventricular tachycardia on Postop day 0, reverted with DC version. One patient developed complete heart block and required PPI on POD 4.Transvenous leads placed under GA with rate set at 90 BPM, with pulse generator in the left pectoral fossa, no post procedural complications noted.

Neurological

7. 5 % of the study population had adverse neurological outcomes in the post-operative period. 5 patients had CVA within one month after surgery with resolution of deficits on follow up. One patient developed Encephalitis on POD 13, improved prior to discharge. One patient had CVA 5 years after surgery with cerebellar infarct.3 patients of these 7 had preoperative H/O CVA.

Renal dysfunction:

6. 1 % developed renal dysfunction with deranged S. creatinine levels of more than 1. 5mg/dl of which 3 patients underwent dialysis for the same.

Endocarditis

3. 3 % developed Endocarditis. One patient was culture negative, managed with antibiotics for 6 weeks. One patient was admitted with pyrexia of unknown origin 1 month post to surgery and later diagnosed to have Scrub typhus with healed vegetation. One patient developed Endocarditis with *E.Coli* as the causative organism, treated with culture sensitive antibiotics for 6 weeks.

Sepsis

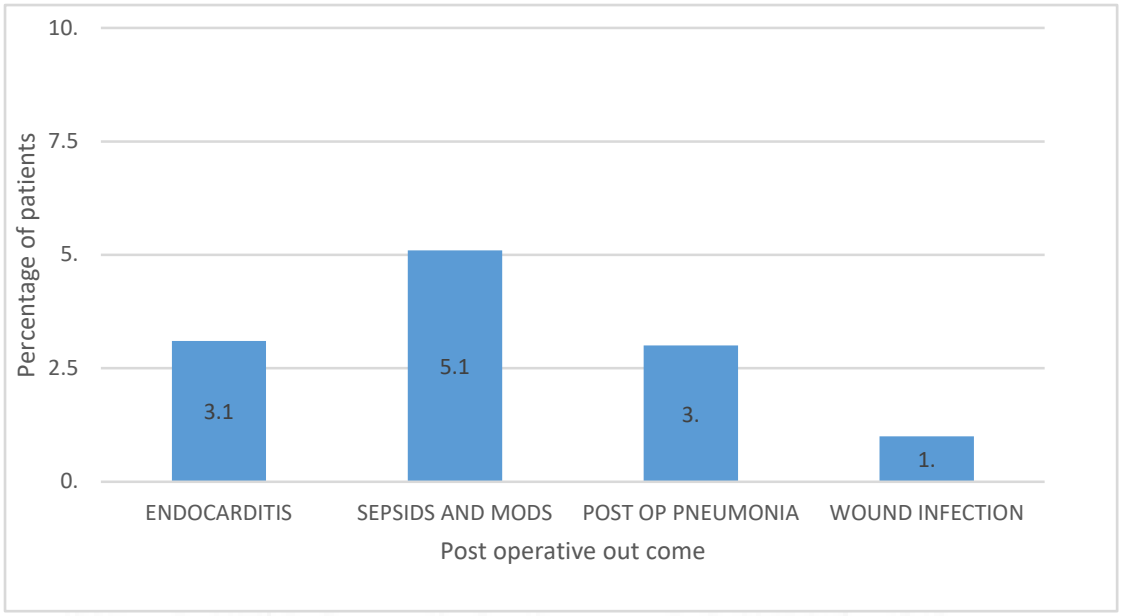
5. 1 % developed sepsis of which 4 patient succumbed to MODS and sepsis. One patient recovered and discharged on POD 23. 2 Patients had blood cultures positive for

Klebsiella pneumonia treated with IV Meropenem for 14 days +IV Colistin for 14 days.1 patient had blood culture positive for *Klebsiella pneumonia* and *E.Coli* treated with IV Meropenem for 21 days and IV Amikacin for 7 days. One patient had Sputum culture positive for *Pseudomonas aeruginosa* on POD 3 and blood cultures positive with same organism on POD 7 treated with IV Colistin and Colistin nebulistaion, but patient developed ARDS and MODS with mortality on Day 25.One patient had urine and blood cultures positive for *E Coli* treated with IV Meropenem for 14 days and oral Nitrofurantoin for 14 days and discharged on POD 23.

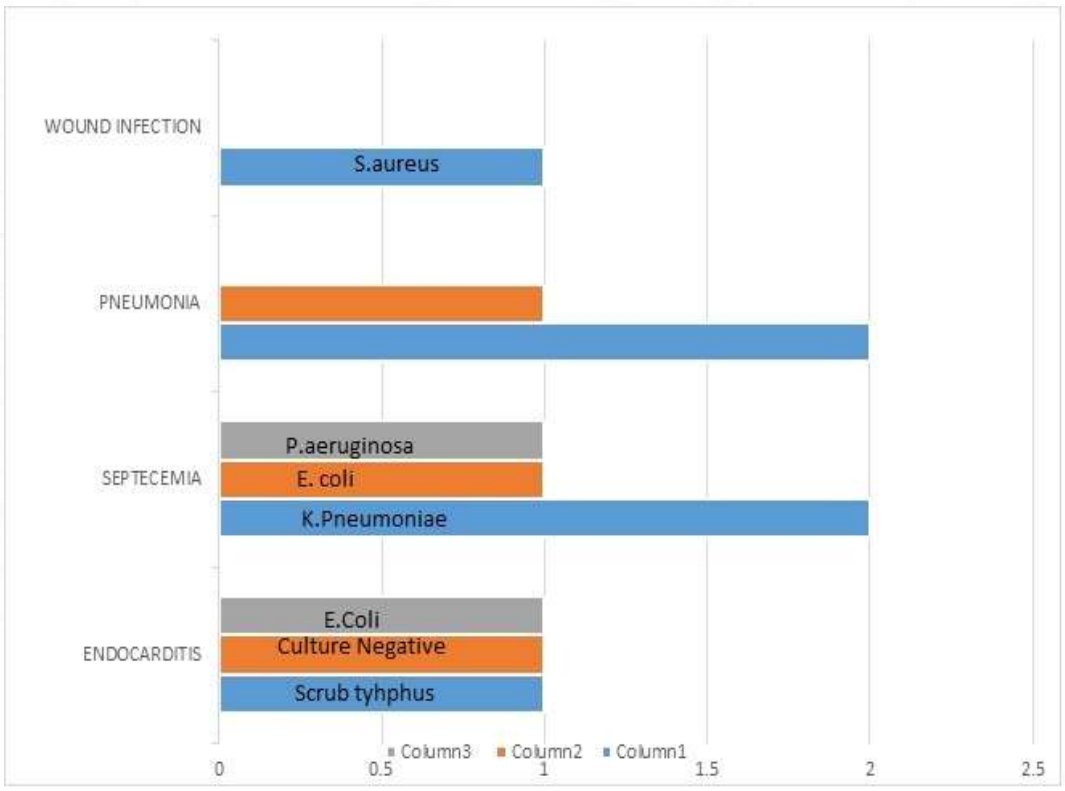
Postoperative Pneumonia and Wound infection

3 patients developed postoperative pneumonia, 2 recovered with conservative management of antibiotics, chest physiotherapy. Both patients were re-intubated and later tracheostomy was done for toileting of secretion. One patient developed sepsis with mortality, included in the above group.

One patient developed wound infection-presented to OPD with wound discharge 2 weeks after surgery, Pus Culture showed Methicillin sensitive *Staphylococcus aureus*. Patient was hospitalized, and regular dressing was daily for 3 days and IV Vancomycin given for 7 days and secondary suturing done after healthy granulation tissue appeared. Patient was discharged after 10 days. On OPD follow up wound was healthy with no discharge.



Graph 10: Range of infections in the postoperative period



Graph 11 : Causative organisms in the various infections

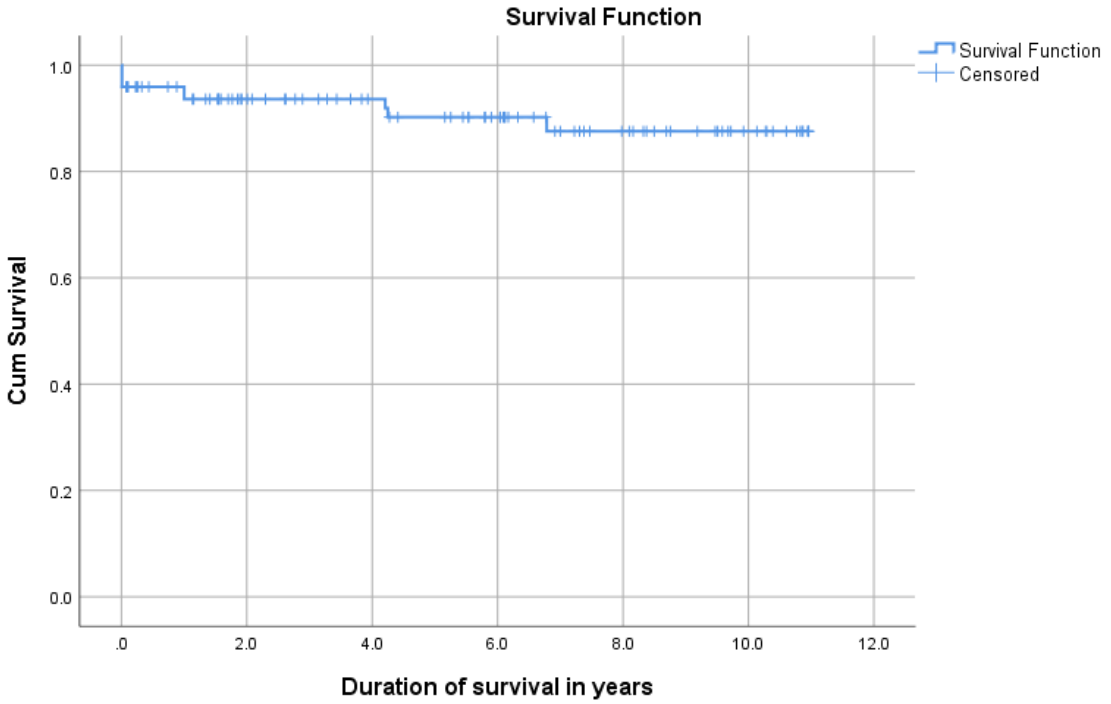
Reoperations:

11. 1 % underwent reoperations within the 1st year of primary surgery. 8 underwent PE drainage –subxiphoid and 2 patient underwent Redosternotmy+ MV repair for severe MR

	Alive		Death		Total		$\chi 2$	df	p
	N	%	N	%	N	%			
CARDIAC EVENT	13	14. 6	4	44. 4	17	17. 3	5. 075	1	0. 024
BLEEDING	5	5. 7	2	22. 2	7	7. 2	3. 336	1	0. 068
RENAL DYS	4	4. 5	2	22. 2	6	6. 1	4. 469	1	0. 035
WOUND INF	1	1. 1	0	0	1	1	0. 102	1	0. 749
POST OP PNEUMONIA	1	1. 1	2	22. 2	3	3. 1	12. 26	1	0
SEPSIS	2	2. 3	2	22. 2	4	4. 1	8. 219	1	0. 004
CVA(I/H)	3	3. 5	4	50	7	7. 5	22. 69	1	0
REOPERATIONS	9	10. 7	1	16. 7	10	11. 1	0. 201	1	0. 654
ENDOCARDITIS	1	1. 2	2	28. 6	3	3. 3	15. 2	1	0

Table 10: The above outcomes were compared between the alive and death population

The above outcomes were compared between the alive and death population and presence of cardiac events, bleeding, renal dysfunction, CVA, postoperative pneumonia, sepsis and Endocarditis were associated with higher mortality-p <0. 05



Survival time in years			
		95% Confidence Interval	
mean	se	Lower Bound	Upper Bound
9. 944	. 321	9. 315	10. 574

Table 11: The Average survival in 10 years was found to be 9.94 years

The average survival at 10 years was found to be 9. 94 years.

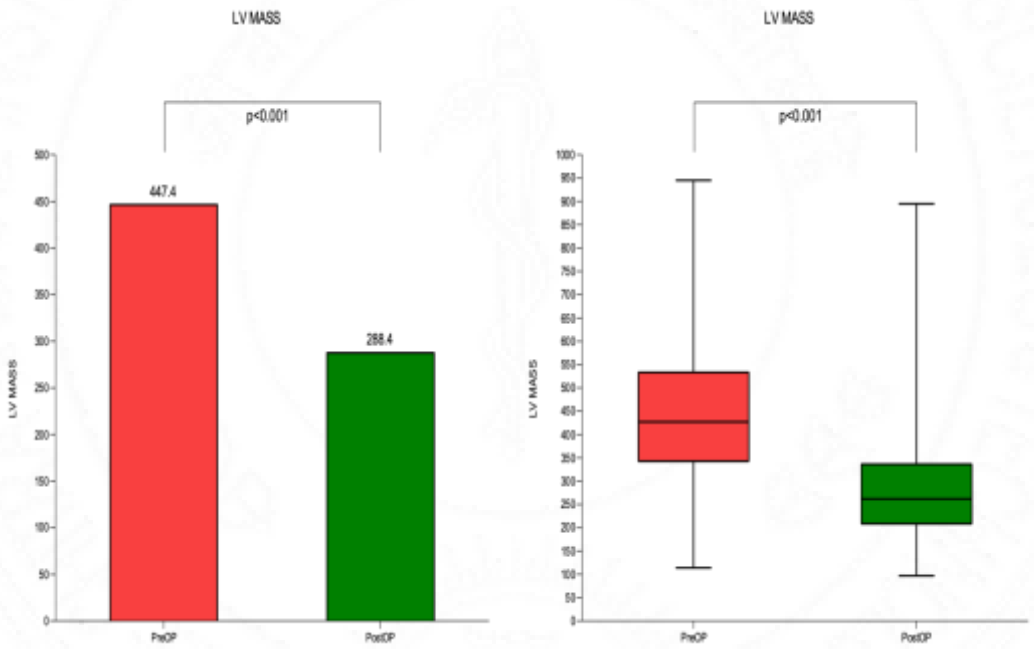
Early mortality defined as mortality within the 1st month of surgery was 5 %. Late mortality was 4 %. All-cause mortality was 9 %. Of the 9 deaths, 1 death was in the immediate post op period on day 0 from Bleeding with hypotension and arrhythmias. 4 patients died in the first month after surgery due to sepsis with MODS. One patient died 5 years after surgery due to intra-cerebral bleed. One patient developed hepatic failure in 2nd year post surgery. One mortality was following aspiration pneumonia secondary to

Pharyngeal muscle weakness due to CVA with cerebellar infarct 5 years after surgery. One patient expired following multiple injuries sustained after Road traffic accident-non cardiac related.

Kaplan Meier curve for survival is shown above, showing average survival of 9 .94 years after Modified Bentall procedure at 10 years follow up.

Table 12: LV mass regression postoperatively

	N	LV MASS		Paired difference		Paired t test	
		Mean	SD	Mean	SD	t	p
Pre op	91	447. 4	153. 9	159. 0	174. 7	8. 7	<. 001
Post op	91	288. 4	128. 2				



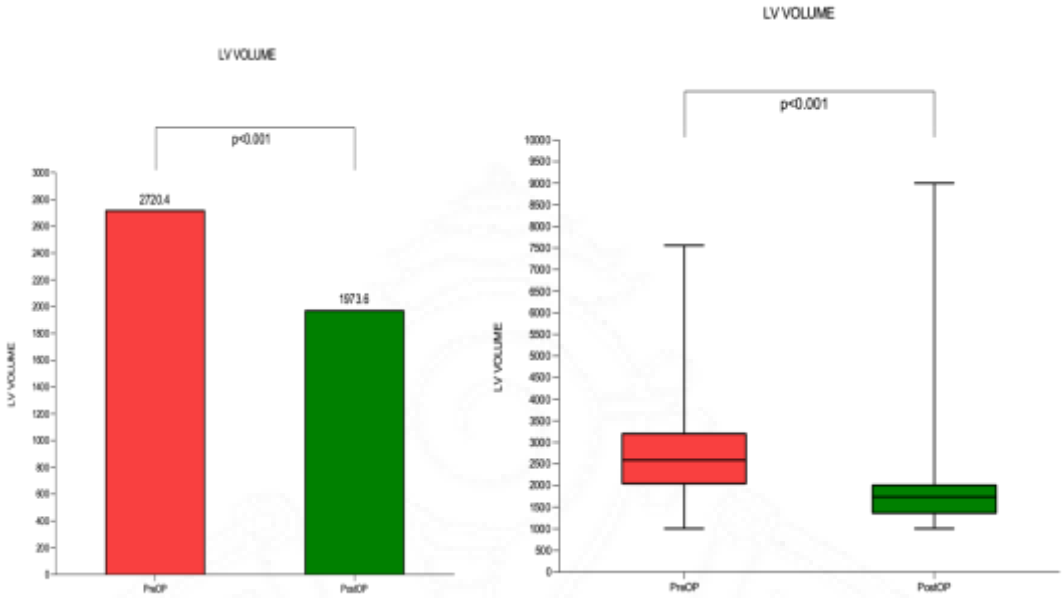
Graph 12 : LV mass regression postoperatively

	N	LV MASS			Wilcoxon signed rank test p
		Median	Q1	Q3	
Pre op	91	427	341	535	<0. 001
Post op	91	262	207	338	

The LV mass was calculated based on the LVIDD, LVISD, Septal thickness and posterior wall thickness and compared in the preoperative ECHO and the post- operative ECHO of the latest follow up period. The median preoperative LV mass was 427 gram and postoperatively it was reduced to 262 gram with indicating regression of the LV mass and reverse remodeling as seen above based on Wilcoxon Signed rank test and Paired T test. The regression was statistically significant.

Table 13: LV volume regression postoperatively

	N	LV VOLUME		Paired difference		Paired t test	
		Mean	SD	Mean	SD	t	p
Pre op	91	2720. 4	1059. 0	746. 7	1639. 6	4. 3	<. 001
Post op	91	1973. 6	1241. 7				



Graph 13: LV volume regression postoperatively

	N	LV VOLUME			Wilcoxon signed rank test p
		Median	Q1	Q3	
Pre op	91	2590	2028	3220	<0. 001
Post op	91	1738	1345	2028	

The LV volume was calculated based on the LVIDD, LVISD, Septal thickness and posterior wall thickness and compared in the preoperative ECHO and the post-operative ECHO of the latest follow up period. There was significant regression of the LV volume as seen above based on Wilcoxon Signed rank test and Paired T test. The preoperative volume had a mean value of 2590 ml and postoperatively it was found to be 1738ml. The regression was statistically significant.

DISCUSSION

Various studies have been done with Chitra valve assessing the longevity and short- and long-term adverse effect and has come up with excellent results in terms of survival and hemodynamic profile when used for various valve replacement procedures [104, 105]. Mean prosthetic valve gradient in our study was found to be 22 +/-12. 2. The hemodynamics is modified with root replacement, as the artificial conduit may alter the blood flow dynamics this may affect valve longevity when compared to studies related to valve replacement alone. These formed the basis of our study. This assessment required a long-term study. Since there is no national registries to study the outcomes of Bentall in different institutions in our country, the individual institutional results remains the bench mark for comparing results of Modified Bentall with one's own experience

This study aimed at assessing the outcomes of the procedure done since 2008 in our institute

The disproportionate percentages in early mortality among several reports could be explained by different patient populations, concomitant procedures, and modifications in operative techniques or postoperative management strategies. The Modified Bentall procedure with Chitra valve showed satisfactory results.

Benke et al., studied a total of 147 patients who underwent aortic root reconstruction reported an overall early mortality rate of 3.4% analyzed over 25 years between 1998 and 2013. [11].

The Kaplan-Meier estimated overall survival rates for the 147 patients were 91.8 ± 2.3 %, 84.3 ± 3.1 %, 76.3 ± 4.9 % and 59.5 ± 10.7 % at 1,5,10 and 20 years, respectively. Their 10 year survival was 76.3 % as opposed to our study population, with a survival of 91 % at 10 years .Concomitant procedures were performed in 39 patients (27 %);

included mitral valve surgery in 11, coronary artery bypass grafting (CABG) in 12, hemi- and total arch replacement in 10. As the demographic data in both studies are similar with similar distribution of preoperative risk factors, concomitant surgery (CABG and mitral valve) can be considered as independent predictor of early complication but this however, was not translated as a risk factor for mortality in their study. Our study had an early mortality of 5.1 %.The main cause of death in both studies was sepsis. Their operative mortality was 2 %. The overall early mortality rate, defined as death within 30 days of initial hospitalization, was 3.4 % .We did not have any mortality intra-operative and early mortality was 5.1 %.The difference in mortality could be attributed to higher incidence of sepsis in our patients. The authors also divided the study population on the basis of diagnosis and found no statistically significant survival advantage in the Non Marfan's population.Our study showed cardiac events, bleeding, renal dysfunction, CVA, postoperative pneumonia, sepsis, endocarditis, longer CPB and cross clamp time, longer ICU and hospital stay were associated with higher mortality-p <0. 05. Since the above study had follow up period of 25 years, surgeons experience was also evaluated as a variable determining the outcome of the procedure and they found that the beginning the CUSUM curve started with an upward inflection, indicating the major learning curve effect. Based on their findings after 27 operations the risk of the early complications started to reduce. 25–30 operations are necessary to acquire better results on survival. After that, they observed a downward inflection, indicating better results and an increase in the experience of the surgeon with respect to the predicted complication rates.

Nezafati et al [99] studied 110 consecutive patients underwent the modified Bentall technique from August 1996 to October 2013. The procedure used Dacron composite graft with a mechanical valve (St. Jude Medical®) for aortic root replacement. To avoid

intra-operative complications, no mobilization of coronary ostia was done. Additionally, the tubular aorta was kept minimally unchanged .Our operative technique involved mobilization of the coronary ostia. The CPB time was comparable in both studies of 131 minutes. Total bleeding after the operation was 450 ± 105 mL. The mean duration of intensive care unit and hospital stay were 2 ± 1 and 5 ± 2 days, respectively, which was lower than in our study with mean ICU stay of 3.4 days and hospital stay of 9.72 days. The longer hospital stay in our study could be due to unit protocol of discharging patients only after the INR is > 1.8, at which only oral anticoagulation is considered to be adequate. The patients requiring longer stay in the intensive care unit was due to hemodynamic or respiratory problems in both studies.5-years follow up; survival rate was 97 %. In the three deceased patients, causes of death were mediastinitis, sepsis and myocardial infarction. No operation-related complications such as anticoagulant-related hemorrhage, valve or graft thrombosis, or coronary pseudo aneurysm occurred during follow-up. They concluded their proposed modification of Bentall technique seemed to minimize late intra-operative blood loss, improves homeostasis, shortens the operation time and is associated with excellent long-term outcomes .However we noted similar freedom from intraoperative complications with standard Modified Bentall technique.

One hundred forty-two elective patients younger than 65 years without concomitant procedures who underwent replacement of the thoracic aorta and aortic valve between 1989 and 2000 were studied by Departments of Cardiothoracic Surgery and Biomathematics, Mount Sinai School of Medicine, New York, New York, 85% were men, and the median age was 46 years (range, 13 to 64 years). There were no intraoperative deaths. Two patients had a stroke postoperatively, one of which was fatal. Complications during follow-up included 2 cases of endocarditis, 1 peripheral thromboembolic event, and 10 instances of significant bleeding (requiring

hospitalization or transfusion). Surgery for distal aortic segments was performed in 4 patients, but no patient required reoperation in the proximal aorta. Kaplan-Meier curves show overall survival is 0.95 (95% confidence intervals, 0.9 to 0.99) at 5 years and 0.93 (95% confidence intervals, 0.86 to 0.99) at 8 years, and event-free survival is 0.85 (95% confidence intervals, 0.78 to 0.92) at 5 years and 0.78 (95% confidence intervals, 0.68 to 0.88) at 8 years. It was concluded that the button Bentall procedure could be performed with excellent short term and long-term results in relatively uncomplicated elective patients in whom aortic valve disease is combined with dilatation of the ascending aorta. They have longer survival in their study with reduced postoperative complications, particularly neurological as compared to our study with 7.5 % adverse neurological outcomes. This could be attributed to the lower mean age of their study population 46 years, as opposed to our study with mean age group of 62 years and also absence of CVA preoperatively in their study group as opposed to our study in which the preoperative incidence of CVA was 7.1 %.. The other adverse outcomes post-surgery are similar in both studies. [100]

Another study was conducted in our institute between January 2006 and December 2013,in which 100 patients underwent aortic root replacement using rigid tilting disc TTK Chitra heart valve as the composite graft valve in our institute. This study included patients between the age group 19–62 years who had Chitra valve in the composite graft. A single surgeon was involved during the entire period. The early operative mortality was 2% (two patients). The late mortality was 3% (n = 3). Significant left ventricle (LV) remodeling was noted in 85% (n = 85) of the patients. Midterm survival at 5 years was 97%.The mean aortic cross-clamp time was 103.01 + 19.72 min, and the mean cardiopulmonary bypass time was 136.11+ 24.84 min. The early operative mortality was 2% (two patients). The late mortality was 3%.This was comparable to our study.

Significant left ventricle (LV) remodeling was noted in 85% (n = 85) of the patients. It was concluded that the Modified Bentall procedure can be done safely with intra-operatively prepared composite graft with any existing valve. The Modified Bentall procedure using intra-operatively prepared composite graft using TTK Chitra valve is safely used in many countries, and it offers excellent midterm results. Their results were comparable to other series of composite graft study making it an effective, accepted, safe, and cost-effective root replacement prosthesis. The study population between this and our study was similar. However their study also analyzed patient who underwent concomitant procedures like CABG and MV repair which were excluded in our study. Also the LV remodeling was by comparison the preoperative and postoperative LV dimensions alone with calculation of LV mass and LV volume. Their study analyzed midterm survival at 5 years, whereas our study had a longer follow up of 10 years. [101]

Although a small percentage of patients with critical aortic stenosis do not develop left ventricle hypertrophy, increased ventricular mass is widely observed in conditions of increased afterload. There is growing epidemiological evidence that hypertrophy is associated with excess cardiac mortality and morbidity not only in patients with arterial hypertension, but also in those undergoing aortic valve replacement. Valve replacement surgery relieves the aortic obstruction and prolongs the life of many patients, but favorable or adverse left ventricular remodeling is affected by a large number of factors whose specific roles are still a subject of debate. Age, gender, hemodynamic factors, prosthetic valve types, myocyte alterations, interstitial structures, blood pressure control and ethnicity can all influence the process of left ventricle mass regression, and myocardial metabolism and coronary artery circulation are also involved in the changes occurring after aortic valve replacement. In the above mentioned studies only our study analyzed LV remodeling in the postoperative period with regression of LV mass and LV

Volume.

Treibel et al.[106] present their data on reverse LV remodeling in 116 patients with severe, mostly symptomatic AS who were undergoing aortic valve replacement (AVR) with or without coronary artery bypass grafting, 19% showed reduction in indexed LV mass 1 year post-AVR. This reduction in mass was caused by a 22% reduction in cellular volume (i.e., myocyte regression) and a 16% decrease in matrix volume. There was also an associated improvement in exercise capacity, left atrial pressure, N-terminal pro–B-type natriuretic peptide, and LV volumes following afterload reduction post-AVR. After extensive research not many studies could be found showing LV mass and volume regression in the post Modified Bentall population. Our study compared mean LV mass and mean LV volumes of the study population in the preoperative and latest post-operative ECHO which showed statistically significant LV remodeling post Modified Bentall procedure.

In older reports (before1990), the mortality for a mechanical valve Bentall procedure was 9. 1%. This has consistently decreased over years with improved surgical techniques, increased surgical experience, and advances in perfusion techniques, intensive care, and other technical aspects. Recent studies shows an overall in-hospital mortality as low as <5%. With increased awareness and patient counseling, there is also a significant reduction in valve-related complication. Recently, studies with both mechanical and bioprosthetic valves have shown early mortality of 1–4% and late mortality of 8–12%. High-risk patients have an increased mortality as seen in the centers participating in the IRAD, with early mortality reported up to 25% with proximal aortic dissections [105]. In our long-term study, we have an all-cause mortality of 9. 2 % with early mortality of 5 %. The early mortalities was seen in high-risk patients. The late mortality n 4% was from non-cardiac causes. This long term study has demonstrated

good postoperative and hemodynamic profiles with significant LV remodeling with significant improvement in LV mass index and minimal valve-related adverse events.

CONCLUSION

Modified Bentall procedure can be done safely with intra-operatively prepared composite graft with any existing valve. Composite graft using TTK Chitra valve is safely used in many countries for Modified Bentall procedure with good results. These mechanical composite roots have shown acceptable gradients and significant ventricular reverse remodeling during long term follow up with acceptable prevalence of prosthetic-related complications. The results are comparable to other series of composite graft study making composite graft with TTK Chitra effective accepted, safe, and cost-effective root replacement technique.

The long term survival is 9. 3 years with early mortality of 1 % comparable with other studies.

LIMITATIONS

As the study is retrospective lapses in data recoding can produce error in the results.

Also Echocardiography parameters may vary between observers producing a confounding factory in the study.

The above study excluded patients who underwent concomitant procedures.

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PATIENT INFORMATION PROFORMA

PROFORMA No:

AGE/SEX-

DATE OF SURGEY-

ANTHROPOMETRY
- Height-
- Weight
- BMI-

RISK FACTORS:
- Hypertension
- Diabetes Mellitus
- Coronary Artery disease
- COPD
- Renal disease
- Neurological
- Peripheral Arterial disease
- Marfans/CTD
- Smoker
- Emergency/Elective surgery

INDICATION FOR SURGERY

PREOPERATIVE IMAGING
- ECHO scan –AR/AS/AS+AR-
  - Ascending aortic dimension-sinus and STJ
  - Aortic Annulus
  - Left ventricular function
- Cardiac CT- Ascending Aortic dimensions
- Coronary Angiogram > 40 years –annulo aortic ectasia

PREOPERATIVE WORK UP
- Hemoglobin
- Albumin
- Serum Creatinine
- ESR/CRP

INTRAOP
- Cardioplegia
- CPB Time
- Aortic cross clamp time
- Surgery- Valve anatomy
- Albograft

Extent of operation: Ascending/hemiarch/Ascending and arch
TCA
DHCA
Temperature

IMMEDIATE POST OP
Cardiac Event
Bleeding
ECHO
Renal Dysfunction
Neurological
Re exploration
Uncontrolled Hypertension/hypotension
Wound Infection
Duration of ventilation
Reason if prolonged> 24 hrs
Post operative pneumonia –
Sepsis-
`
Positive cultures-
ICU stay
Total hospital stay post operatively

Follow Up-

Yearly:
Cardiac event
CVA (Ischemic/ hemorrhagic)
Mortality
Reoperations
Anticoagulation:
Peripheral embolization
Endocarditis:
ECHO-Aortic gradient- AR- Paravalvular leak: LVDysfunction LV Remodelling

If Death –
Post operative day-
Cause-Cardiac /Non cardiac

Quality of life-
Symptomatic
Physical activity/NYHA (Classification)

MODIFIED BENTALL'S PROCEDURE-LONG TERM SURVIVAL AND SHORT AND LONG TERM OUTCOMES- A SINGLE CENTRE EXPERIENCE:A RETROSPECTIVE OBSERVATIONAL STUDY

Telephone Recruitment Script

Hello, my name is Dr Rashida Hderabadwala. I'm calling from Sree Chitra Institute of Medical Sciences and Technology(SCTIMST) about a research study. Am I speaking to _____ (name of recruit) or his/ her parent?

If "no," wait for recruit to pick up, leave a message, or ask for a time to call back.

If "yes":

I got your phone number from the hospital records. Is this a good time to talk?

Arrange to call at another time, if appropriate.

I'm calling about a research study of outcomes of an operation that you or your child has underwent called "MODIFIED BENTALL PROCEDURE-LONG TERM SURVIVAL AND SHORT AND LONG TERM OUTCOMES- A SINGLE CENTRE EXPERIENCE:A RETROSPECTIVE OBSERVATIONAL STUDY". The purpose of this research study is to learn more about the long-term results of this operations and your present condition. Joining a research study is completely voluntary. If it's alright with you I'd like to take about 2-3 minutes to explain the basic idea of the study and to see if you would be interested in taking part.

If you agree to participate, we will ask you to come into the clinic, where we will discuss the study with you in more detail, and you can decide if you want to participate. There will be no risks for the participants because of participation in the study. No specific intervention will be done. The participants are evaluated in detail for any cardiac cause for functional impairment. A follow up examination and evaluation may be helpful in identification of any risk factors for poor outcomes or functional deterioration. And this is part of routine assessment.

We will do our best to keep your information confidential by not mentioning your identity and keeping the records on a password-protected computer. You don't have to answer these questions, and you can choose to stop at any time without penalty. If you have questions about the study, you can call me at 9900897621. If you have questions about your rights as a research subject or technical clarifications, you can call Dr. Mala Ramanathan, Member Secretary, IEC, SCTIMST and Additional Professor, AMCHSS, SCTIMST (Email: iec.mem.sec@sctimst.ac.in, Phone no. 0471-2524234)

If accepting: Document eligibility response and make appointment, if appropriate.

If the patient has expired and relative replies that "He/ She is no more/ has expired"

I'm sorry to hear about his/her demise. Can you tell me the time of death and the details about the cause of death? If you are agreeable, can we go through the hospital records of this person?

If yes: Thank you. The details might be helpful in preventing similar complications in other people. Thank you for your time.

If *no*: That's perfectly understandable. Thank you for your time.

പരിഷ്ക്കരിച്ച ബെന്റാൾ നടപടി – ദീർഘകാലത്തെ അതിജീവനവും ഇടക്കാലത്തെയും ദീർഘകാലത്തെയും നേട്ടങ്ങളും– ഏക കേന്ദത്തിലെ അനുഭവം: മുൻകാലാധിഷ്ഠിതമായ ഒരു നിരീക്ഷണ പഠനം.

ടെലിഫോൺ വഴി പങ്കാളികളെ ഉൾപ്പെടുത്താനുള്ള കുറിപ്പ്

ഹലോ, എന്റെ പേര് ഡോ. റാഷിദാഹെഡ്റബാവാല. ഞാൻ ശ്രീചിത്ര ഇൻസ്റ്റിറ്റ്യൂട്ട് ഫോർ മെഡിക്കൽ സയൻസസ് ആന്റ് ടെക്നോളജി (SCTIMST) യിൽ നിന്ന് ഒരു ഗവേഷണ പഠനത്തിനായാണ് വിളിക്കുന്നത്. ഞാൻ ……..…. (പങ്കെടുപ്പിക്കാനുദ്ദേശിക്കുന്നയാളുടെ പേര്) നോടോ അദ്ദേഹത്തിന്റെ മാതാപിതാക്കളോടോ ആണോ സംസാരിക്കുന്നത്?

അല്ലെങ്കിൽ പങ്കെടുപ്പിക്കാനുദ്ദേശിക്കുന്നയാൾ ഫോണെടുക്കുന്നതുവരെ കാക്കുക, ഒരു സന്ദേശം നല്കുക, അല്ലെങ്കിൽ തിരിച്ചുവിളിക്കാൻ സമയം ചോദിക്കുക.

ആണെങ്കിൽ
എനിക്ക് ആശുപത്രി രേഖകളിൽ നിന്നാണ് താങ്കളുടെ ഫോൺ നമ്പർ കിട്ടിയത്. ഇത് സംസാരിക്കാൻ പറ്റിയ സമയമാണോ?

അനുയോജ്യമെങ്കിൽ, മറ്റൊരു സമയത്ത് വിളിക്കാൻ ഏർപ്പാടു ചെയ്യുക.

'പരിഷ്ക്കരിച്ച ബെന്റാൾ നടപടി – ദീർഘകാലത്തെ അതിജീവനവും ഇടക്കാലത്തെയും ദീർഘകാലത്തെയും നേട്ടങ്ങളും– ഏക കേന്ദത്തിലെ അനുഭവം: മുൻകാലാധിഷ്ഠിതമായ ഒരു നിരീക്ഷണ പഠനം' എന്നതു സംബന്ധമായി താങ്കൾ/താങ്കളുടെ കുട്ടി വിധേയമായ ഒരു ശസ്ത്രക്രിയയുടെ ഫലത്തെപ്പറ്റി അറിയാനാണ് ഞാൻ വിളിക്കുന്നത്. ഈ തരം ശസ്ത്രക്രിയകളുടെ ദീർഘകാല ഫലങ്ങളെപറ്റിയും താങ്കളുടെ ഇപ്പോഴത്തെ അവസ്ഥയെപ്പറ്റിയും കൂടുതലറിയുക എന്നതാണ് ഈ പഠനത്തിന്റെ ഉദ്ദേശം. ഗവേഷണന പഠനത്തിൽ പങ്കെടുക്കുന്നത് പൂർണ്ണമായും സ്വമേധയായാണ്. താങ്കൾ പഠനത്തിൽ പങ്കെടുക്കാൻ താത്പര്യപ്പെടുന്നുണ്ടോ എന്നറിയാൻ, പഠനത്തിന്റെ അടിസ്ഥാന ആശയം വിശദീകരിക്കാൻ ഞാൻ 2–3 മിനിട്ട് എടുക്കുന്നതിൽ കുഴപ്പമില്ലല്ലോ.

താങ്കൾ പങ്കെടുക്കാൻ സമ്മതിക്കുകയാണെങ്കിൽ, താങ്കളോട് ക്ലിനിക്കൽ വരാൻ ആവശ്യപ്പെടുകയും അവിടെ വെച്ച് താങ്കളോട് പഠനത്തിന്റെ കൂടുതൽ വിശദവിവരങ്ങൾ ചർച്ചചെയ്യുകയും താങ്കൾ പഠനത്തിൽ പങ്കെടുക്കുന്നോ എന്ന് തീരുമാനിക്കുകയും ചെയ്യാം. പഠനത്തിൽ പങ്കെടുക്കുന്നതുകൊണ്ട് അപായമൊന്നുമുണ്ടാകില്ല. താങ്കളിൽ ശാരീരികമായ ഇടപെടലുകളില്ലാതെ പ്രത്യേകമായ പരിശോധനകളൊന്നുമില്ല. പങ്കെടുക്കുന്നവരിൽ പ്രവർത്തന സംബന്ധമായ തകരാറുകൾക്ക് കാർഡിയാക് കാരണങ്ങൾ ഉണ്ടോയെന്ന് വിശദമായി വിലയിരുത്തും. പ്രവർത്തനപരമായ ശേഷിക്കുറവുണ്ടാകുന്നതിൽ എന്തെങ്കിലും അപായസാദ്ധ്യതകളുണ്ടോയെന്ന് കണ്ടെത്താൻ ഒരു തുടർ പരിശോധനയും വിലയിരുത്തലും സഹായകമായേക്കാം.. ഇത് പതിവായുള്ള വിലയിരുത്തലിന്റെ ഭാഗമാണ്.

താങ്കളുടെ വ്യക്തിവിവരങ്ങൾ രേഖപ്പെടുത്താതെ രേഖകൾ പാസ്വേർഡിനാൽ സംരക്ഷിക്കപ്പെട്ട കമ്പ്യൂട്ടറിൽ സൂക്ഷിച്ച് താങ്കളെപ്പറ്റിയുള്ള വിവരങ്ങൾ രഹസ്യമാക്കി വയ്ക്കാൻ ഞങ്ങൾ പരമാവധി പരിശ്രമിക്കും. താങ്കൾ ഈ ചോദ്യങ്ങൾക്ക് ഉത്തരം പറയണമെന്നില്ല, കൂടാതെ താങ്കൾക്ക് ഏതു സമയത്തും പങ്കാളിത്തം അവസാനിപ്പിക്കാം. പഠനത്തെപ്പറ്റി ചോദ്യങ്ങളുണ്ടെങ്കിൽ താങ്കൾക്ക് എന്നെ 9900897621 എന്ന നമ്പറിൽ ബന്ധപ്പെടാം.

ഗവേഷണ പങ്കാളിയെന്ന നിലയിലുള്ള താങ്കളുടെ അവകാശങ്ങളെപ്പറ്റിയുള്ള ചോദ്യങ്ങൾക്കോ, സാങ്കേതീക വിശദീകരണങ്ങൾക്കോ താങ്കൾക്ക് ബന്ധപ്പെടാൻ ഡോ. മാല രാമനാഥൻ, മെമ്പർ സെക്രട്ടറി, IEC, SCTIMST, പ്രൊഫസർ, AMCHSS, SCTIMST. ഫോൺ നമ്പർ 04712524234, ഇമെയിൽ iec.mem.sec@sctimst.ac.in

സമ്മതിക്കുന്നെങ്കിൽ, അനുയോജ്യമാണെങ്കിൽ യോഗ്യത രേഖപ്പെടുത്തുക,

രോഗി മരിക്കുകയും ബന്ധു "അദ്ദഹം മരിച്ചു" എന്നു മറുപടി നൽകിയാൽ അദ്ദേഹത്തിന്റെ മരണത്തിൽ ഞാൻ അനുശോചിക്കുന്നു. താങ്കൾക്ക് മരണമടഞ്ഞ സമയം പറയാനാകുമോ, മരണ കാരണത്തിന്റെ വിശദാംശങ്ങൾ നൽകാനാകുമോ?

താങ്കൾ സമ്മതിക്കുമെങ്കിൽ ഈ വ്യക്തിയുടെ ആശുപത്രി രേഖകൾ പരിശോധിക്കാമോ? സമ്മതമെങ്കിൽ നന്ദി. മറ്റുള്ളവരിൽ സമാനമായ സങ്കീണ്ണതകൾ തടയാൻ വിശദാംശങ്ങൾ സഹായിച്ചേക്കാം.

താങ്കളുടെ സമയത്തിന് നന്ദി
വേണ്ടായെങ്കിൽ. അത് മനസ്സിലാക്കാനാകും. താങ്കളുടെ സമയത്തിന് നന്ദി

PLAGARISM CHECK

Curiginal

Document Information

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Sources included in the report

SA	**Axén Sofie.pdf** Document Axén Sofie.pdf (D20359074)	1
SA	**Sree Chitra Tirunal Institute, Thiruvananthapuram / Sai Suraj Thesis edit.pdf** Document Sai Suraj Thesis edit.pdf (D78220926) Submitted by: vtp@sctimst.ac.in Receiver: vtp.sctims@analysis.urkund.com	7
W	URL: https://academic.oup.com/icvts/article-abstract/6/Supplement_1/S1/669254 Fetched: 10/11/2019 8:16:30 AM	1
W	URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC101074/ Fetched: 5/6/2021 8:50:14 AM	1

Technical Advisory Committee (Clinical Studies)
SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES & TECHNOLOGY
THIRUVANANTHAPURAM – 695011, INDIA

Date: 19.11.2020

TAC Registration No: SCT-/S/2020/979

Project title: MODIFIED BENTALL'S PROCEDURE-LONG TERM SURVIVAL AND SHORT AND LONG TERM OUTCOMES- A SINGLE CENTRE EXPERIENCE:A RETROSPECTIVE OBSERVATIONAL STUDY

Principal Investigator:	
Dr. Rashida Hyderabadwala, Senior Resident, Department of Cardiothoracic and Vascular Surgery, Department of CVTS, SCTIMST	Degree: MBBS, MS
Co-Principal Investigator(s)	
Dr. Vivek Pillai, Additional Professor, Department of CTVS, SCTIMST	Degree: MS , MCh CTVS
Dr. Bineesh K Radhakrishnan, Assistant Professor, Department of CTVS, SCTIMST	Degree : MS , MCh CTVS

IEC had the following comments on the application:

1. The study is referred to as retrospective case series and prospective observation alternatively. Please clarify
2. Clarify if there is going to be any telephonic follow up. If there is, there is a need for consent processes to accompany this part of the data collection.
3. Explain the duration of follow up for this study and how the documentation will be done.
4. The objectives seem to be about abdominal aortic aneurysms which is total different from the title indicated.
5. Explain how the details of the six minute walk test will be obtained. If patient is going to be required to revisit the hospital, the cost involved should be mentioned.

In response to the IEC comments, the investigators have submitted their responses for TAC review.

The response, examined by TAC, is satisfactory and recommended for approval by IEC since the revised proposal does not involve the six minute walk test. The PI has communicated that inclusion of this test was a technical error and the other details of the proposal remaining the same as in the previous approval version.

MEMBER SECRETARY
TAC (Clinical Studies)
SCTIMST

Dr Srinivas G

ANNEUXURE

SI NO	NAME	HOSP NO	PHONE NO	AGE/SEX	DATE OF SURG	ANTHOPOMETRY		
						HT(CM)	WT(KG)	BSA
1	ASOK K	9500536	9446705436/9446190833	50Y/M	04-12-2019	181CM	87	
2	DAS K	476752	9446004695	55Y/M	29-11-2019	180CM	78	
3	SHASHI DEVAN	478398	9447496842	62Y/M	27-11-2019	170CM	58.5	
4	RATHEESH KUMAR K	8803688	9349772663	55Y/M	21-11-2019	168	73	
5	MOOSA K	473809		65Y/M	10-10-2019	158CM	54.5	
6	RAJENDRAN C	472945	8903859356	59Y/M	04-10-2019	159 CM	62	
7	RADHA A	473238	9567473046	67Y/F	26-09-2019	159 CM	63	
8	SUBHASH KUMAR DINODIA	4711859	9656114999	44Y/M	03-09-2019	160CM	63	
9	MUHAMMED MUSTAFA	465585	9846161314	34/M	25-07-2019	186 CM	67.5	
10	MUBARAK	9502786	9847861551	35Y/M	05-04-2019	165	66	
11	SREENIVASAN K	463895	9048070999	63Y/M	12-02-2019	171CM	80	
12	LENIN ANDREWS	438904	8547528415	32Y/M	13-11-2018	175CM	76	
13	SEBASTIAN KC	456785	9961331575	44Y/M	05-11-2018	178CM	60	
14	MAHADEVAN S	453319	9003457435	52Y/M	07-09-2018	170CM	56	
15	PADMAVATHY S.	4480627	9447840627	65Y/F	28-08-2018	140CM	39	
16	NAVEEN M J	448791	8086242498	41Y/M	03-08-2018	183CM	78.4	
17	BHASKARAN K N	351793	9947949248	66Y/M	20-06-2018	170	70	
18	JAYESH J.	450605		37Y/M	14-06-2018	178 CM	63	
19	AISWARYA BABU	448712		31Y/F	12-06-2018	175	72	
20	SANFEER	451080	811942814/9846261075	33Y/M	31-05-2018	186.5CM	64.5	
21	MANOJ GN	449026	9656783411	45Y/M	18-04-2018	184	86	
22	VAREEDHA JT	445109	7902507458	58Y/F	26-03-2018	157	50	
23	MAHIBALAN	442730	7502034600	28Y/M	09-02-2018	183	73	
24	SAMSHUDEEN KUNJU	441427	9388598484	65Y/M	23-02-2018	170	75	
25	BINEESH VK	440713	7356878012	34Y/M	30-11-2017	179	67.5	
26	ALIAS AL	434988	9645822862	58Y/M	14-09-2017	187	65	
27	SAJEER	431739	9645382715	35Y/M	18-05-2017	166	70	
28	MANEESH MATHAI	426901	9447245495	37Y/M	10-02-2017	175	78	
29	ABDUL KHADER	426522	9605737574	49Y/M	24-03-2017	165	61	
30	SAMSY S	418245	91595945522	65/Y/M	23-05-2017	170	65	

RISK FACTORS								
HTN	DM	CAD	COPD	RENAL DISEASE	NEUROLOGICAL	PVD	CTD/MARFANS	OTHER
N	N	N	N	N	N	N	N	HYPOTHYROID/PAROXYSMAL AF
Y	N	N	N	N	N	N	Y	N
N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	N	N
N	N	N	Y	N	N	N	N	N
N	N	N	N	N	N	N	N	N
Y	Y	Y	N	N	N	N	N	N
N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	Y	N
N	N	N	N	N	N	N	N	N
Y	Y	N	Y	N	N	N	N	N
N	N	N	N	N	N	N	N	N
Y	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	N	N
N	N	N	N	N	Y	N	N	HEARING IMPAIRMENT
Y	N	N	N	N	N	N	Y	N
Y	N	N	N	N	Y	Y	Y	N
N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	Y	N
N	N	N	N	N	N	N	N	N
Y	Y	N	N	N	N	N	N	N
N	N	N	N	N	N	N	MARFANS	N
Y	Y	Y	Y	N	N	N	N	MILD LV DYSFUNCTION
N	N	N	N	N	N	N	N	N
Y	N	N	Y	Y-NEPHROTIC SYN	N	IRAAA	MARFANS	N
Y	Y	N	N	N	SEIZURE DIS	N	N	N
Y	N	N	Y	N	N	N	N	N
Y	Y	Y	N	N	N	Y	MARFANS	S/P PCI
Y	Y	N	N	N	N	N	MARFANS	N

SMOKER	EMERGENCY	INDICATION OF SURGERY	AR	AS	AS +AR
N	N	SEV AR,AORTIC ROOT DILATATION	3+		
N	N	SEV AR, DIL LV, LVH DLP	3-4+		
N	N	ASC AOR ANEURYSM, MOD-SEV AR	4+		
N	N	SEV AS, MOD AR,DILATED ASC AORTA	3+		
N	N	SEV AR, ANNULO AORTIC ECTASIA	4+		
N	N	ASC AOR ANEURYSM, MILD AR	2+		
N	N	ASC AOR ANEURYSM, MOD-SEV AR,CAD	3-4+		
N	N	ASC AOR ANEURYSM,SEV AR	4+		
N	N	BAV WITH SEV AS	2+	100/88	SEV AS
N	N	BAV,SEV AR, DIL ASC AORTA	4+		
N	N	ASC AOR ANEURYSM, MILD AR	2+		
N	N	DIL AORTIC ROOT,SEV AR, DIL LV	4+		
N	N	SEV AR,ASC AORTIC ANEYRYSM	3-4+		
N	N	BAV,DIL AA, SEV AR,MILD AS	4+		
N	N	BAV,MILD AR, MOD AS	1+		
N	N	ASC AOR ANEURYSM, SEV AR	4+		
N	N	AOR ROOT ANEURYSM, SEV AR	4+		
N	N	ASC AOR ANEUR,SEV AR	4+		
N	N	ASC AOR ANEURYSM,MILD AR	4+		
Y	N	ASC AAA, BAV,SEVERE AR	3-4+		
N	N	ANNULOAORTIC ECTASIA ,SEV AR	3+		
N	N	CHRONIC HEALED AORTIC DISS,SEV AR	4+		
N	N	ANNULOAORTIC ECTASIA,ASC AO ANEURYSM	4+		
Y	N	ASC AORTIC ANEURYSM	4+		
N	N	BAV,SAC AO ANEURSM	4+		27 mild as
Y	N	ANNULOAORTIC ECTASIA, SEV AR	4+		
Y	N	TYPE A AORTIC DISSC,SEV AR	4 +		
N	N	ASC AO ANEUR,SEV AR,	4+		
Y	N	ANNULOAO EC,ASC SO ANEURYSM,SEV AR	4+		
N	N	ASC AORTIC ANEU,BAV ,SEV AR	4+		

		ECHO						
AS AORTA-SINUS	Asc Aorta	AO ANNULUS	LV FUNCTION	LVIDD/IS	SEPTUM	PW	LV MASS	LV VOLUME
45	52	24	GDLV	60/48	12/18	10/12	427.4	2423
59	26	28	GDLV	58/47	12/15	12/14	386.14	2261
56	73	22	GDLV	59/45	12/18	10/12	416.3	2341
55	37	29	GDLV	53/27	18/20	9/11	369.9	1881
48	52	25	GDLV	61/42	13/11	8/12	340.95	2506
41	47	23	GDLV	45/30	13/9	9/11	198.1	1345
48	60	20	GDLV	63/44	11/16	9/11	399.1	2676
66	70	23	GDLV	70/44	12/16	10/12	498.3	3316
24	46	24	GDLV	47/32	12/15	8/11	237.9	1471
48	66	24	GDLV	57/40	12/16	8/11	339.65	2182
46	65	24	GDLV	62/51	9/11	9/12	313.26	2590
47	56	29	GDLV	77/51	9//11	10/12	452.8	4024
52	45	30	GDLV	76/56	10/12	12/14	518.3	3919
56	55	31	GDLV	71/53	9/8	11/13	372.04	3413
67	45	21	GDLV	34/24	12/11	7/10	114.01	7558
80	82	47	GDLV	62/43	18/18	11/16	538.4	2590
72	58	29	GDLV	60/37	16/14	12/14	427.4	2423
65	67	28	GDLV	56/40	12/15	10/12	330.2	2104
66	70	30	GDLV	58/34	11/16	9/11	349.2	2261
60	43	35	GDLV	65/45	14//6	12/14	441.3	2852
50	62	26	GDLV	59/40	8/14	10/12	340.7	2341
50	50	21	GDLV	59/46	8/10	12/14	305.45	2341
60	77	33	GDLV	66/44	12/10	11/15	430.6	2942
58	56	24	SEV LVD	77/64	14/10	12/14	583.9	4024
44	49	37	GDLV	80/49	15/20	11/13	775.1	4349
48	57	30	GDLV	73/51	10/7	12/14	436.3	3611
58	69	21	GDLV	62/43	14/10	10/12	369.3	2590
50	77	34	GDLV	85/61	12/10	12/14	627.5	4918
60	55	24	GDLV	56/33	15/14	12/14	365.44	2104
51	38	26	GDLV	71/49	12/8	9/11	350.7	3413

	CARDIAC CT				PREOP			
SINUS	ASC AORTA	ANNULUS	ANGIOGRAM-AOROANNULAR ECTASIA	HB	ALBUMIN	S.CREAT	ESR	
	56	54	31 N		15	44.4	1.13	6
58/63/39	30x29mm	32x24mm	Y		15.8	4.9	1.01	12
	58 77X72		30 Y		13.1	3.3	1.1	8
	45 64x60		33 Y		15.1	4.7	0.83	4
	46 40X42	28X22	N		15.4	5	1.2	18
41X45			N		15.1	4.3	0.95	13
41X40	43X41	31X30	Y		13.4	4.6	0.97	27
52X53	55X55	30X32	N		14.3	4	1	35
45X38.5	49X50	36X28	N		14.8	4.3	1.1	32
53X48	46X42	42X34	N		13.6	3	0.91	13
	51	64	N		12.8	4.5	1	17
42X44		40X33	N		12.6	4	0.9	22
	54	62	33 N		14	3.8	0	20
	56	64	30 Y		14.3	4.2	1.05	20
	58	70	28 Y		11.4	3.7	0.85	13
	67	77	30 N		11.7	3.2	0.89	35
	55	67	32 N		13.7	3.5	1.01	7
45	70		34 N		12	4	0.9	12
58	67		28 N		12	4.2	0.87	16
53.5X56.3	58.8X55.6	40.6X34.9	N		14.3	4.7	0.91	6
49X46	61X59	33X22	CT ANGIO		16.6	8.5	1.35	10
69X35	69X40		22 N		12.8	4	1.15	8
45X45	66X50		33 N		14.9	4.4	0.87	11
55X51	73X68	32X26	Y		12.5	4.31	1.18	8
48x44	40x37	45x44	N		14.5	4.7	0.95	10
54X53	37X34	36X27	Y- ECTATIC VESSEL		11.9	3.8	1.7	13
	55	60	24 Y		13.5	4	1.3	10
	77	72	39.7 Y		14	4	1.3	12
47X48	40X41	28X21	Y-SVD		11.8	4.1	1.28	10
50X45	36X34	26.6X22	Y-NC	13	4.1	0.72	7	

INTRAOP CARDIOPLEGIA	CPB TIME	AORTIC CROSS C	VALVE ANT	ALBOGRAFT	EXTENT	TCA	DHCA	TEMPERATURE
CUSTODIAL	98	65	#27 CHVP	#28	AS AORTA	N	N	28
CUSTODIAL	110	76	#25CHVP	#26	AS AORTA	N	N	28
CUSTODIAL	126	97	#23PM MAGNA	#28	AS AORTA	N	N	28
CUSTODIAL	178	116	#29CHVP	#30	aortic clamp	N	N	28
CUSTODIAL	102	66	#23CHVP	#26	AS AORTA	N	N	28
CUSTODIAL	101	69	#21 CHVP	#24	aortic clamp	N	N	28
CUSTODIAL	139	82	#23CHVP	#26	AS AORTA	N	N	28
CUSTODIAL	110	79	#23CHVP	#24	aortic clamp	N	N	28
CUSTODIAL	112	81	#27CHVP	#28	AS AORTA	N	N	28
CUSTODIAL	112	80	#29CHVP	#30	AS AORTA	N	N	30
CUSTODIAL	121	90	#27CHVP	#28	AS AORTA	N	N	28
CUSTODIAL	120	80	#25CHVP	#28	AS AORTA	N	N	28
CUSTODIAL	123	83	#26 CHVP	#28	AS AORTA	N	N	27
CUSTODIAL	105	78	#27 CHVP	#30	AS AORTA	N	N	28
CUSTODIAL	126	84	#19CHVP	#22	AS AORTA	N	N	28
CUSTODIAL	127	94	#29CHVP	#30	AS AORTA	N	N	28
CUSTODIAL	120	92	#21PM MAGNA	#24	AS AORTA	N	N	27
CUSTODIAL	121	90	#24 CHVP	#26	AS AORTA	N	N	28
CUSTODIAL	123	92	#26	#28	AS AORTA	N	N	28
CUSTODIAL	108	77	#27CHVP	#28	AS AORTA	N	N	29
CUSTODIAL	99	71	#25 CHVP	#28	AS AORTA	N	N	30
CUSTODIAL	123	91	#21CHVP	#20 DACRON	AS AORTA	N	N	26
CUSTODIAL	103	74	#25 CVHP	#26	AS AORTA	N	N	28
CUSTODIAL	114	83	#23 CHVP	#26	AS AORTA	N	N	26
CUSTODIAL	136	100	#27CHVP	#28	AS AORTA	N	n	28
CUSTODIAL	130	98	#29CHVP	#30	AS AORTA	N	N	28
COLD BLOOD	190	130	#25CHVP	#26	AS AORTA	Y-10MINS	N	20.5
CUSTODIAL	186	90	#29	#30	AS AORTA	N	N	28
CUSTODIAL	107	75	#21	#22	AS AORTA	N	N	28
CUSTODIAL	112	83	#21 PM TFX	#24	AS AORTA	Y-3 MINS	N	28.7

				IMMEDIATE POSTOP			
CARDIAC EVENT	BLEEDING	ECHO	RENAL DYS	NEUROLOGICAL	UNCONT HTN	WOUND INF	DURATION OF VENT
N	N	GDLV	N	N	N	N	1 DAY
N	Y-REEX	GDLV	N	N	N	N	1DAY
Y-SVT	N	GDLV	N	N	N	N	1 DAY
N	N	MOD LVD	N	N	N	N	2 days
N		GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	2 DAYS
N	N	MILD LVD	N	N	N	N	1DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	FAIR LV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	FAIR LV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAT
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	y-wash given	GDLV	N	N	N	N	2 DYAS
N	N	TRIVIAL PE	N	N	N	N	1 DAY
N	y-wash given	GDLV	N	HEADACHE, N CT	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
VF-DC VERTED	N	MOD LVD, RV DYSF	N	N	N	N	1 DAY
n	N	MILDPE	N	N	N	N	1 day
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	2 DAYS
N	N	GDLV	N	N	N	N	2DAYS
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	2DAYS

POST OP PNEUMONIA	SEPSIS	ICU STAY	HOP STAY	CARDIAC EVENT	CVA(I/H)	MORTALITY	REOPERATIONS
N	N	3 DAYS	9DAYS	N	N	N	N
N	N	3 DAYS	9 DAYS	N	N	N	N
N	N	9 DAYS	15 DAYS	YES- AF	N	N	N
N	N	5DAYS	10 DAYS	N	N	N	N
N	N	3 DAYS	8 DAYS	N	N	N	N
N	N	3 DAYS	8 DAYS	N	Y-I	N	N
N	N	3 DAYS	7 DAYS	N	N	N	WOUND DEBRIDEMENT
N	N	3 DAYSS	8 DAYS	N	N	N	N
N	N	3 DAYS	11 DAYS	N	N	N	N
N	N	3 DAYS	8 DAYS	N	N	N	N
N	N	5 DAYS	9 DAYS	N	N	N	N
N	N	3 DAYS	8 DAYS	N	N	N	N
N	N	3DAYS	9DAYS	N	N	N	N
N	N	3DAYS	3DAYS	Y CARDIAC ARREST	N	Y	
N	N	3 DAYS	9 DAYS	N	N	N	N
N	N	3 DAYS	13 DAYS	N	N	N	N
N	N	3 DAYS	12 DAYS	N	N	N	PE
N	N	4DAYS	9 DAYS	N	N	N	N
N	N	3DAYS	10DAYS	N	N	N	N
N	N	3 DAYS	7 DAYS	N	N	N	N
N	N	3 DAYS	8 DAYS	N	N	N	N
N	N	3 DAYS	5 DAYS	N	N	N	N
N	N	3 DAYS	5 DAYS	N	N	N	N
N	N	3 DAYS	8 DAYS	N	N	N	N
n	N	3 days	8 days	N	N	N	N
N	N	3 DAYS	9 DAYS	N	N	N	N
N	N	5 DAYS	2 DAYS	N	N	N	N
N	N	5DAYS	7 DAYS	N	N	N	N
N	N	2 DAYS	7 DAYS	N	N	N	N
N	N	3DAYS	9DAYS	N	N	N	N

1 ST YEAR

ANTICOAGULATION	PERP EMBOLIZATION	ENDOCARDITIS	ECHO AORTIC GR	AR	PV LEAK	LV DYSFUNCTION	LVIDD/IS
REGULAR	N	N	20/12	N	N	GDLV	45/39
REGULAR	N	N	13/7	N	N	GDLV	46/34
REGULAR	N	N	19/9	N	N	GDLV	55/43
REGULAR	N	N	20/12	N	N	GDLV	46/29
REGULAR	N	N	13/15	N	N	GDLV	55/48
REGULAR	N	N	24/22	N	N	GDLV	65/45
& SUTURING			20/11	N	N	GDLV	54/46
REGULAR	N	N	18/6	N	N	GDLV	55/37
REGULAR	N	N	22/12	N	N	GDLV	57/43
REGULAR	N	N	16/14	N	N	MOD LVD	54/44
REGULAR	N	N	22/15	N	N	MILD LVD	47/33
REGULAR	N	N	34/18	N	N	GDLV	47/34
REGUALAR	N	N	36/18	N	N	GDLV	55/48
REGULAR	N	N	22/15	N	N	MILD LVD	60/46
REGULAR	N	N	16/18	N	N	GDLV	48/35
REGULAR	N	N	37/21	N	N	GDLV	48/29
REGULAR	N	N	22/12	N	N	GDLV	54/45
REGULAR	N	N	12/9	N	N	MILD LVD	46/39
REGULAR	N	N	13/6	N	N	GDLV	45/29
REGULAR	N	N	23/14	N	N	GDLV	43/28
REGULAR	N	N	44/23	N	N	GDLV	37/25
REGULAR	N	N	12/7	N	N	GDLV	50/35
REGULAR	N	N	10/5	N	N	MOD LVD	46/32
REFULAR	N	N	20/12	N	N	GDLV	51/36
REGULAR	N	N	12/4	N	N	MOD LVD	68/39
REGULAR	N	N	16/10	N	N	MILD LVD	56/40
REGULAR	N	N	12/6	N	N	GDLV	65/40
REGULAR	N	N	18/12	N	N	GDLV	42/31
REGULAR	N	N	22/11	N	N	GDLV	40/20

	LV REMODELLING				DEATH		
SEPTUM	POSTERIOR WALL	LV MASS	LV VOLUME		POD	CAUSE	SYMPTOMATIC
11/10	11/10		175.02	1345			
12/10	12/10		205	1407			
12/11	11/9		257	2028			
12/10	12/14		230.16	1407			
13/15	14/16		355.3	2028			
15/12	12/10		420	2852			
14/12	9/11		279.8	1954			
12/10	12/15		320.9	2028			
16/18	13/15		453.05	2182	N		N
18/16	12/16		436.6	1954	N		N
16/15	10/12	265.2	1471		N		N
15/13	8/12		251.37	1471	N		N
12/10	12/16		337.9	2028			
					Y	CARDIAC ARREST	
13/15	12/16		447.9	2423	N		
12/10	16/14		273.8	1536	N		N
15/13	12/13		273.8	1536	N		N
11/9	11/9		234.8	1954			
15/11	12/14		270.6	1407			
15/11	11/9	222.57	1345				
16/5	11/8	219.83	1225				
8/5	10/7	96.88	8999				
10/9	12/12	207.14	1669				
14/4	14/11	256.76	1407				
12/16	16/12	365.96	1738				N
15/6	15/13	522.1	3126				Y
12/8	12/10	280.47	2104				
10/12	10/7	320	2852				N
15/15	12/14	236.74	1168				N
10/8	13/11	155.4	1056		N	N	N

PHYSICAL QUALITY-NYHA

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31	DAVID RAJ M	384218	9446956127	29Y/M	09-11-2016	165		69
32	JOSIE JOHN	357624	7510203040	33Y/M	29-01-2018	165		70
33	SALINI REJI	272796	9142533601	19Y/F	28-12-2017	160		68
34	APPUKUTTAN C	411482	979292766	69Y/M	20-09-2016		166	70
35	JAMUNA KUMARI A	414488	76394114869	40Y/F	26-07-2016		154	55
36	ABHILASH CV	265763	9947076288	36Y/M	03-03-2016		172	64.5
38	GANGADHARAN	358662	9495148271	71Y/M	06-05-2016		161	65
39	RAJASEKHARAN K	408919	9947299553	52Y/M	27-01-2016		172	64
40	PALIANI SAMY V	403728	8608175446	61Y/M	17-10-2015		162	49
41	SIDHARTHAN P	399213	9645581134	57Y/M	23-09-2015		161	63.5
43	BENSON	396834	9946660151	53Y/M	04-08-2015		191	87
44	MUHAMMED ASHRAF	378727	9447320360	43Y/M	24-11-2014		188	59
45	APPU N	374393	9895855395	63Y/M	04-11-2014		151	50.7
46	SADASIVAN	380568	9496105791	60Y/M	01-10-2014		176.5	63.5
47	AJITHA S	293448	9387380897	44Y/F	22-07-2014		162	41
48	JOY K	375648	9895168567	41Y/M	20-06-2014		167	56
49	SAIFUDEEN P K	306228	9634223290	31Y/M	19-06-2014		170	67
50	BENY JOHN	9103511	9447057761	53Y/M	18-03-2014		173	61
51	ISMAIL FAARIH	374312	9846188656	18Y/M	14-03-2014		164	46
52	NAGULU V R	373603	9488717716	30Y/M	07-02-2014		191	58
53	JOSEPH THOMS	9105562	9447566518	66Y/M	19-12-2013		170	66
54	AMBILI MV	361399	8089860320	59Y/M	17-12-2013		157	47
55	PRAKASH KUMAR K	366974	446112122	48Y/M	29-11-2013		164	67.7
56	SUNIL KUMAR A G	2836	9447019695	49Y/M	20-11-2013		166	57
57	SURENREAN V	365692	8893364389	63Y/M	19-11-2013		170	88
58	NIZAMUDDIN H	286641	9947604698	60Y/M	01-01-2013		170	95
59	SUKUMARAN V	303162		67Y/M	31-10-2013		170	77
60	VASANTHA KUMARI MV	339595	9744401359	45Y/F	05-09-2013		169	67
61	HAWW A REEFA	366590	9603317468	29/F	27-08-2013		165	56
64	MANIKANDAN T C	249710	4712573369	47Y/M	05-06-2013		175	78
65	AYSHA BEEVI CP	357386	9745449405	53Y/M	26-03-2013		172	66
66	SHAMSUDDIN V	358992	9946418178	46Y/M	21-03-2013		180	78
67	INDIRA P K	316701	9961560365	39Y/M	01-02-2013		170	57

N	N	N	N	N	N	N	MARFANS	N
N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	N	N
Y	N	N	N	N	N	N	MARFANS	N
Y	Y	N	N	N	N	N	N	N
N	N	N	N	N	N	N	MARFANS	N
Y	N	N	N	Y- CREAT - 1.7	N	N	N	N
Y	N	N	N	N	N	N	N	N
Y	Y	N	Y	N	N	N	N	SEV LVD
Y	Y	N	N	N	N	N	N	MINOR CAD
Y	Y	N	N	N	N	N	MARFANS	EYE SURGERY,SEVERE LVD
Y	N	N	N	N		N	MARFANS	
N	N	N	N	N	N	N	N	LBBB WITH 1 DEGREE AV BLOCK
Y	Y	N	N	N	N	N	N	
Y	N	N	N	N	N	Y	MARFANS	REPAIR
Y	Y	N	N	N	N	Y	MARFANS	N
Y	N	N	N	N	N	N	MARFANS	HOCM,RVOTO,PAROXYSMAL AF
Y	N	N	N	N	N	N	MARFANS	N
Y	N	N	NN	N	N	N	MARFANS	N
N	N	N	N	N	N	N	MARFANS	MILD LVD,ACHLASIA CARDIA
N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	N	HYPOTHYROIDISM
Y	N	N	N	N	N	N	MARFANS	HYPOTHYROIDISM
N	N	N	N	N	N	N	N	N
Y	N	N	N	N	N	N	N	N
Y	Y	N	N	N	N	N	N	N
Y	N	N	Y	Y- CREAT - 1.7	N	N	N	N
Y	Y	Y	N	N	N	N	N	SEVERE LVD
Y	Y	N	N	N	N	N	MARFANS	SEV LVD,G6PD DEFICIENCY,S/P THY
Y	Y	N	Y	N	OLD CVA	N	N	HEALED AORTIC ROOT ABCESS
Y	Y	N	N	N	N	N	MARFANS	HYPOTHYROIDISM
Y	N	N	N	N	N	N	N	N
Y	Y	N	N	N	N	N	N	N

N	N	BAV SEV SEV AR,DIL ASC AO	4+	64/40		SEV AS
N	N	ANNULOAORTIC ECTASIA ,SEV AR	4+			
N	N	ANULOAORTIC AORTIC,SEV AR	4+			
N	N	SEV AR ,ASC AO DILATATION	4+			
N	N	SEV AR, UNRUPTURED SOV ANEURYSM	4+			
N	N	BAV, SEV ASC AOR ANEURYSM	4+	36/21		MOD AS- 36/21
N	N	BAV,SEV AS,ASC AO ANEURYSM	3-4+	37/15		MOD AS-
N	Y	ACUTE ASC AORTIC DISSECTION,ASC AOR ANEUR	4+			
N	Y	ASC AO +ARCH ANEURYSM ,CONTAINED RUPTURE	3+			
N	N	SEV AS +ASC AORTIC ANEURYSM	1+	83/50		SEV AS
N	N	ASC AO ANEURYSM, SEV AR	4+			
N	N	SEV AR, ANNULOAORTIC ECTASIA	4+			
N	N	SEV AS ,BAV ,ASC AORTIC ANEURYSM	2+	89/53		SEV AS
Y	N	ASC AORTIC ANEURYSM, SEV AR	4+		15	MILD AS
N	N	TYPE B AORTIC DISSECTION,SEV AR,DILA ASC AO	4+			
N	N	AORTIC ROOT ANEURSYM,MOD AR,MOD MR	4+		36	MILD AS
N	N	ANNULOAORTIC ECTASIA	4+			MILD AS
Y	N	BAV,SEV CALCIFIC AS,MILD AR,ASC AO ANEURYSM	2+	66/45		SEV AS
N	N	ANNULOAORTIC ECTASIA	4+			
N	N	SEV AR,ASC AORTIC ANEYRYSM,DISSCETION+ TYPE A	4+			
N	N	BAV, ASC AO DILATED	4+	33/10		MIL D AS
N	N	BAV,SEV AS, DILATED ASC AORTA	2+	130/90		SEV AS
Y	N	ASC AORTIC ANEURYSM, SEV AR	4+	45/26		MILD AS
N	N	BAV,MOD AS, MOD AR, ASC AO ANEURYSM	3+	46/26		MOD AS
N	N	ASC AOR ANEURYSM,SEV AR,GDLV,SR	4+	69/45		
N	N	BAV MOD AR,MOD AS,GDLV,SR	3+	49/20		MOD AS
N	N	SEV CALCIFIC AS, DILATED ASC AOR ANEURYSM	3-4+	44/22		MOD AS
N	N	ASC AORT ANEURYSM	4+			
N	N	TYPE A AORTIC DISSECTION,SEV AR	4+			
Y	N	BAV,AAANEURSYM,1ST DEGREE AV BLOCK	3-4+	51/33		MOD AS
N	N	TYPE A AORTIC DISS	4+			
N	N	ASC AO ANEURYSM.GDLV	4+			
N	N	TYPE ASC AORTIC ANEURYSM	4+			

	55	42	26	GDLV	61/40	12/19	10/12	459.6	2506
	56	45		GDLV	62/43	10/12	9/11	313.26	2590
	58	44		GDLV	60/44	11/14	10/12	350.1	2423
	45	47	23	GDLV	63/41	14/18	12/14	505.9	2676
34X28	52X34		21	GDLV	42/25	13/19	9/11	249.5	1168
	41	52	23	GDLV	69/48	12/18	10/11	510.4	3220
	40	52	24	GDLV	48/37	14/22	12/14	401.7	1536
	50	69	23	GDLV	64/43	12/27	9/11	665.4	2763
	45	71	30	MOD LVD	84/70	15/22	11/14	945.2	4802
	52	50	25	GDLV	50/30	14/20	12/14	389.7	1669
	52	68	26	SEVERE LVD 21	74/66	14/13	9/11	470.7	3712
	65	39	28	MOD LVD	89/71	9/13	12/11	614.6	5399
	55	41	31	GDLV	50/45	12/21	9/11	355.34	1669
	45	43	23	SEV LVD	69/58	11/19	12/14	510.4	3220
	50	54	18	GDLV	61/45	13/18	9/11	418.3	2506
	37	35	23	GDLV	55/35	8/14	11/14	337.9	2028
	52	57	36	GDLV	39/23	23/25	17/19	427	1002
	45	45	22	GDLV	45/21	15/23	19/21	511.3	1345
effaced	64x67		26	GDLV	63/40	12/15	9/14	440.4	2676
	60	62	23	MILD LVD	64/53	8/14	8/16	473.5	2763
	46	51	26	GDLV	54/35	16/19	15/20	538.8	1954
	30	46	22	GDLV	43/27	13/15	13/17	285.45	1225
	64	67	23	GDLV	69/47	13/12	12/18	534.6	3220
	49.5	39	27	GDLV	52/23	12/13	9/11	248.84	1809
	50	52	27	GDLV	69/45	10/12	7/12	396.5	3220
	46	44	28	GDLV	60/42	11/14	12/15	407.4	2423
	50	56	28	GDLV	72/51	13/21	12/18	828	3511
	48	55	22	SEVERE LVD 21	66/57	13/22	12/20	809.5	2942
	46	50	24	SEVERE LVD 21	56/55	13/10	8/15	347.6	2104
	32	56	26	GDLV	65/50	7/10	7/9	265.24	2852
	52	56	24	GDLV	54/32	12/15	11/16	380.54	1954
	48	56	24	GDLV	56/35	12/10	22/20	461	2104
	28	55	22	MILD LVD	48/28	16/14	16/9	334.6	1536

51X42	52X50	31X28	N	18.2	4.7	1.0	2
54	59	28	N	12	4	0.97	12
56	62	26	N	14	4.2	0.88	10
44.X42.6	53.3X51.6	29.8X22.8	Y	13	4.1	0.92	33
38X23	52X23 3 ANEUR	23	Y	13.3	4.1	.90	23
54X49	56X55	42X41	N	15	3.8	0.8	10
40X45	47X40	40X36	Y	15.6	0.8	1.4	12
51X50	69.5X67.6	24X 23	N	15	4.0	1.00	12
53	52X47	ANE-16X9	N	10.4	3.4	1.5	12
53	60		Y-MINOR CAD	12.9	4.1	0.5	6
58X56	64X66	22	N	12.6	4.4	0.80	13
67	30	35	Y	11.9	4.2	1.3	15
52.5	62	30	N	13.6	4.0	1	6
45.3	58.9	26	N	11.7	3.4	1.3	53
56	30	30	N	11.5	3.8	0.9	18
39.8	38	34	N	9.9	3.9	0.4	12
			N	10	3.9	0.57	20
			N	14.6	4	0.90	16
				13.6	4	0.8	1.1
62	64	33	N	14	4.1	1.10	17
41.8	54	32.6	N	12	4	0.7	12
27	50	30X32	N	12.4	4	0.8	13
46	63	32	N	11.5	4.8	1.2	9
31	58	29	N	12.8	4.2	0.8	23
44	52x48	32	n	14	4.2	0.9	11
50X47	60X48	65X44	N	11.4	4.2	1.1	12
66X60	47X46	30	N	13.9	3.9	.17	12
52	65	33	MID LAD 40%	13	4.0	1.1	12
55	59	32	N	12	4	1.0	12
51	62	28	N	12.8	4.2	0.8	13
52	56	30	DISS FLAP+	12.8	4	1.0	22
48	55	32	FLAP AT STJ TOWARDS POST WALL OF ASC	13.2	4	1.1	12
46	73	33	N	9.6	3.3	0.9	12

CUSTODIAL	108	75	#25CHVP	#30	AS AORTA	N	N	29
CUSTODIAL	130	90	26	28	AS AORTA	N	N	27
CUSTODIAL	114	87	24	26	AS AORTA	N	N	26
CUSTODIAL	112	88	#23 PM TFX	#26	ASC AORTA	N	N	29
CUSTODIAL	131	87	#21 CVP	#22	ASC AORTA	N	N	29
CUSTODIAL	128	96	#29 CHVP	#30	ASC AORTA	N	N	29
TEPID BLOOD	130	101	#23CHV[	#28	ASC AORTA	N	N	29
COLD BLOOD	106	83	#25 CHVP	#26	PROX ARCH	N	N	29
CUSTODIAL +CALFIORI	270	160	#25 CHVP	# 28	HEMI ARCH	Y-22 MINS	N	18
COLD BLOOD	147	117	#25 CHVP	#28	ASC AORTA	N	N	28
COLD BLOOD	97	76	#27 CHVP	#28	ACS AORTA	N	N	28
COLD BLOOD	114	86	#29 CHVP	#30	ASC AORTA	N	N	30
TEPID BLOOD	130	98	#27	#28	ACSC AORTA	N	N	28
TEPID BLOOD	127	90	#25 CHVP	#20	ASC AORTA	N	N	28
TEPID BLOOD	112	87	#23 CHVP	#24	ASC AORTA	N	N	28
TEPID BLOOD	187	133	#25 CHVP	#24	ASC AORTA	N	N	28
TEPID BLOOD	133	91	#27 CHVP	#28 collagen coa	ASC AORTA+sep	N	N	28
TEPID BLOOD	155	133	#25 CHVP	#26	ASC AORTA	N	N	27
TEPID CARDIOPLEGIA	118	91	#23	#24	ASC AORTA	N	N	28
TEPID	173	114	#25	#26- WRAP AOR	ASC	N	N	27
TEPID	153	105	#27CHVP	#28	ACS	N	N	27
TEPID BLOOD	154	117	#19CHVP	#20	ACS	N	N	27
TEPID	113	94	#23	#24	ACS	N	N	28
TEPID BLOOD	116	97	#25	#26	ACS	N	N	28
TEPID BLOOD	127	96	#25	#26	ACS	N	N	28
TEPID BLOOD	117	88	#25	#26	ACS	N	N	27.6
TEPID BLOOD	123	98	#23 PM MAGNA	#26	ACS	N	N	28
TEPID BLOOD	156	88	#23 CHVP	#24 ALBO	ACS	N	N	28
TEPID BLOOD	13	100	#25	#26	ACS	N	N	29
TEPID BLOOD	146	136	#27	#28	ACS	N	N	28
TEPID BLOOD FA	140	118	#25CHVP	#26	ACS	30S	N	28
TEPID BLOOD FA	200	118	#23	#24	ACS	N	N	28
TEPID BLOOD	199	106	#25	#26	ACS	26	N	29

N	N	GDLV	N	N	N	N	2DAYS
N	N	GDLV	N	N	N	N	2 DAYS
N	N	GDLV	N	N	N	N	1 DAY
Y AF-CONSERVATIVE	N	GDLV	N	N	N	N	1 DAY
N	N	3 MM PE	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
VF- REVERTED DC 20J	N	MOD LVD	Y	N	N	N	1 DAY
N	N	MOD LVD	N	N	N	N	2 DAYS
Y- ECTOPICS	Y-CONSE	MOD LVD	N	Y-VERTIGO	N	N	4 DAYS
N	N	GDLV,PE	N	N	N	N	1 DAY
N	N	SEV LVD	N	N	N	N	2 DAYS
N	N	MOD LVD, PE-5	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	2 DAYS
N	N	GDLV ,MILD PE	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	2 DAYS
N	N	GDLV	N	N	N	N	2 DAYS
AF	Y-CONSE	PE	N	N	N	N	2 DAYS
N	N	GDLV	N	N	N	N	2DAYS
N	N	GDLV	N	N	N	N	2 DAYS
N	N	GDLV	N	N	N	N	2 DAYS
N	N	GDLV	N	N	N	N	2 DAYS
N	N	GDLV, MILD PE	N	N	N	N	2 DAYS
N	N	GDLV,MILD MR	N	N	N	N	2 DAYS
RBBB	N	GDLV	N	N	N	N	2DAYS
N	N	GDLV	N	N	N	N	2 DAYS
N	N	GDLV	N	N	N	N	2 DAYS
AF-SLOW RATE	N	N	Y-CREAT-2	N	N	N	2 DAYS
VT DC 20J	N	SEV LVD	N	N	N	N	2 DAYS
N	N	SEV LVD	N	N	N	N	2 DAYS
N	N	MOD LVD	N	N	N	N	2 DAYS
N	N	MILD LVD	N	N	N	N	2 DAYS
N	N	MILD LVD,PE	N	N	N	N	2DAYS
VF- REVERTED DC 20J	Y-PE DRA	MILD LV	N	N	N	N	2 DAYS

N	N	2 DAYS	10 DAYS	N	VT- REVERTED	N	N
N	N	4 DAYS	9DAYS	N	N	N	N
N	N	3 DAYS	9 DAYS	N	N	N	N
N	N	3 DAYS	9 DAYS	N	N	N	N
N	N	3 DAYS	8 DAYS	N	N	N	N
N	N	3 DAS	12 DAYS	N	N	N	N
N	N	4 DAYS	12 DAYS	AF	N	N	N
N	Y	3 DAYS	20 DYAS	ATRIAL FLUTTER	N	N	N
N	N	6 DAYS	20DAYS	AF	N	N	N
N	N	4DAYS	7 DAYS	N	N	N	N
N	N	5 DAYS	10 DAYS	N	N	N	N
N	N	3 DAYS	9 DAYS	N	N	N	N
N	N	3 DAYS	10 DAYS	N	N	N	N
N	N	3 DAYS	10 DAYS	N	N	N	N
N	N	2 DAYS	7 DAYS	LFU			
N	N	2DAYS	7DAYS	N	N	N	N
N	N	4DAYS	8DAYS	N	N	N	Y-PE DRAINAGE
N	N	3DAYS	9 DAYS	N	N	N	N
N	N	3 DAYS	8 DAYS	N	N	N	N
N	N	3 DAYS	10 DAYS	N	N	N	N
N	N	4 DAYS	8 DAYS	N	N	N	N
N	N	3 DAYS	9 DAYS	N	N	N	N
N	NN	3DAYS	9 DAYS	N	N	N	N
N	N	3 DAYS	9DAYS	N	N	N	N
N	N	3 DAYS	9DYAS	N	Y	N	Y-PE DRAINAGE
N	N	3 DAYS	7 DAYS	N	N	N	
N	N	3 DAYS	9 DAYS	Y-AV BLOCK	N	N	N
N	N	3 DAYS	10 DAYS	LFU			
N	N	3 DAYS	12 DAYS	N	N	N	N
N	N	3 DAYS	9 DAYS	N	N	N	N
N		3 DAYS	7 DAYS	N	N	N	Y-PE DRAINAGE
N	N	3 DAYS	10DAYS	N	Y	Y	Y -PEDRAINAGE
N	N	6 DAYS	14DAYS	LFU			

REGULAR	N	N	12/7	N	N	GDLV	56/32
REGULAR	N	N	10/6	N	N	GDLV	55/46
REGULAR	N	N	21/17	N	N	GDLV	56/40
REGULAR	N	N	6/2	N	N	GDLV	53/36
REGULAR	N	N	12/6	N	N	GDLV	33/21
REGULAR	N	N	9/7	N	N	GDLV	47/33
REGULAR	N	N	21/13	N	N	GDLV	55/40
REGULAR	N	N	17/8	N	N	GDLV	45/29
COAGULOPATHY-HEMATURIA	N	N	25/12	N	N	GDLV	55/36
REGULAR	N	N	12/6	N	N	GDLV	40/27
REGULAR	N	N	33/20	N	N	SEV LVD	74/62
REGULAT	N	N	34/18	N	N	MILD LVD	56/45
REGULAR	N	N	15/6	N	N	GDLV	42/27
REGULAR	N	N	16/7	N	N	MOD LVD	59/46
REGULAR	N	N	12/8	N	N	GDLV	59/45
REGULAR	N	N	10/8	N	N	GDLV	44/38
REGULAR	N	N	16/12	N	N	GDLV	40/38
REGULAR	N	N	19/10	N	N	GDLV	44/28
REGULAR	N	N	12/10	N	N	GDLV	45/36
REGULAR	N	N	8/12	N	N	GDLV	45/35
REGULAR	N	N	30/19	N	N	GDLV	43/26
REGULAR	N	N	22/11	N	N	GDLV	54/37
REGULAR	N	N	14/8	N	N	GDLV	46/21
REGULAR	N	N	18/10	N	N	GDLV	45/28
REGULAR	N	N	16/8	N	N	GDLV	55/37
REGULAR-EPISTAXIS	N	N	36/22	N	N	GDLV,SEVERE MR	57/34
REGULAR	N	N	20/12	N	N	GDLV	55/45
REGULAR	N	Y	10/5	N	N	GDLV	56/42
REGULAR	N	N	12/16	N	N	GDLV	55/33
REGULAR	N	N	22/10	N	N	GDLV	56/48

12/8	9/11	234.3	2104	N	N	N
12/10	9/12	272.4	2028			
11/9	12/10	264.7	2104			
12/10	14/11	286.94	1881			N
12/10	13/11	133.03	7106	N	N	N
13/10	11/10	212	1471	N	N	N
13/10	9/11	272.4	2028	N	N	N
12/10	11/13	210.16	1345	N	N	YES-FEVER, SCRUB
12/10	12/16	337.9	2028		NC	
12/10	11/10	155.4	1056	N	N	N
14/11	9/11	470.7	3712			Y
12/10	12/10	280.47	2104	Y	NON CARDIAC	
12/10	9/13	189.2	1168			N
12/10	9/11	288.46	2341	N	N	N
11/10	9/8	224.55	2341	N	N	N
10/12	10/12	191.33	1285	N	N	N
10/12	9/8	136.2	1056	N	N	N
10/12	10/8	168.92	1285	N	N	N
8/12	8/12	198.1	1345	N	N	N
8/10	8/12	175.02	1345	N	N	N
8/10	8/10	142.5	1225	N	N	N
8/12	9/10	234.8	1954	N	N	N
8/8	10/10	137.72	1407	N	N	N
18/14	15/12	319.6	1345	N	N	N
12/10	11/10	257	2028	N	N	N
27/20	24/12	894.6	2182	N	N	N
20/12	12/11	410.05	2028	Y		
12/20	14/16	502.4	2104	N	N	N
20/22	18/20	621.5	2028	N	N	N
12/10	17/11	365.44	2104		Y	

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TYPHUS	
NCARDIAC CAUSE OF DEATH	
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3	2017-VETEBROBASILAR STROKE, 2019- STROKE -HEMORRAGIC, RECOVERED AKI
1	
3	SEVERE MR,
	DEATH DUE TO HEPATIC FAILURE 1 YEAR AFTER CARDIAC SURGERY
1	1ST YEAR-FEVERE, CULTURE NEG ENDO
1	
	IC BLEED-DEATH-CEREBELLAR HEMATOMA

68	SAIDALAVI M	351026	9895063542	63Y/M	18-12-2012	170	60
69	SASI C	335609	9037894630	60Y/M	12-12-2012	67	174
70	CHELLAPA S	348245	9677506389	41Y/M	11-10-2012	170	67
71	RAMAKRISHNAN P	345338	9961766921	48Y/M	11-09-2012	168	55
72	RAMESH K	344756	9943923118	42Y/M	16-08-2012	160	70
73	JOHN DN	290922	4712210593	68Y/M	31-07-2012	157	65
74	SURESH K	335965	9746787748	56Y/M	10-01-2012	161	63.5
75	PRASEEN S DAS	336730	9605252989	28Y/M	27-11-2011	179	67
76	SUBAIDA SYED	9500256	4942652987	54Y/M	13-07-2012	161	63.5
78	MUNIYAMMA I	313641	9843368204	46Y/F	04-11-2011	154	59
79	RAMYA P	327573	9626467689	35Y/F	06-09-2011	175	80.5
81	GOPALKRISHNAN NAIR	319739	9446904653	77Y/M	31-08-2011	170	67.5
82	YESHODA KV	320379	9846826518	59Y/M	18-08-2011	146	40
83	THIRUMURAN A	328889	9600420588	24Y/M	05-07-2011	170	48
84	SREEKALA SI	323264	9446196248	46Y/F	25-04-2011	174	86
85	MUJEEB RAHMAN V.P	324640	9947501515	30Y/M	01-04-2011	191	70
86	SHOUKHAT K.P	317065	9495324627	35Y/M	27-10-2010	162	44
87	BINDU O	298662	9895054785	43Y/M	14-07-2010	170	56
88	ALPONSA JOHNNY	296201	2744299425	53Y/F	30-06-2010	180	55
89	SHAHUBANATH A	312623	9495151934	37Y/F	03-06-2010	167	70
90	RATHINASAMY S	309595	9842331171	60Y/M	30-04-2010	165	65
91	AMBILI B	308815	9526264085	47Y/F	14-04-2010	160	70
92	RAVEENDRAN N	303706	9446551185	60Y/M	30-01-2010	170	66
93	RAMESHAN B	301109	9847603990	49Y/M	13-11-2009	177	72
94	CHACKO V M	299048		66Y/M	24-09-2009	165	53
95	NOUSHAD S	281384	9387692830	43Y/M	18-09-2009	170	86
96	MURALIDHARAN NAIR K	297617	9447105325	68Y/M	13-08-2009	169	60
97	KODHER MYDEEN	286083	9965406659	63Y/M	27-05-2009	167	68
98	AYYAPPAN P K	257046		55Y/M	06-03-2009	177	72
99	HARIKRISHNAN N	291818	9367713017	34Y/M	27-03-2009	170	70
100	JAYAKUMAR K	290258	9946368737	55Y/M	24-02-2009	167	67
101	SURESH N D	285164	9388809776	4Y/M	18-02-2009	170	73
102	JUDEX PV	288058	4842430870	42Y/M	23-01-2009	180	89

Y	Y	N	N	N	N	N	N	N
Y	N	N	N	Y	N	N	N	N
Y	N	N	N	N	N	N	N	VPC+
Y	Y	Y	Y	Y	N	N	N	POLYCYSTIC KIDNEY DISEASE
Y	N	N	N	N	N	N	MARFANS	N
Y	Y	N	N	N	MCA INFARCT	N	N	HBSAG+,PREOP INOTROPES-ADR 0.
Y	N	N	Y	N	N	N	N	MNG,S/P THYROIDECTOMY
Y	N	N	N	N	N	N	MARFANOID	N
Y	N	N	N	N	N	N	N	S/P THYROIDECTOMY
Y	N	N	N	N	N	N	N	N
Y	Y	N	Y-BA	N	N	N	MARFANS	N
Y	Y	Y	N	N	N	N	N	SVD
N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	MARFANS	N
N	N	N	N	N	N	N	MARFANS	SCOLIOSIS
N	N	N	N	N	N	N	MARFANS	FAMILY H/O
N	N	N	N	N	N	N	MARFANS	
N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	N	N
N	N	N	Y ASTHMA	N	N	N	N	N
N	N	N	N	N	N	N	MARFANS	N
N	N	N	N	N	ANXIETY DISORDEI	N	MARFANOID	ANXIETY,ALCOHOLIC
Y	N	N	N	N	N	N	MARFANS	PERICARIAL EFFUSION,AF
N	N	N	N	N	N	N	MARFANS	N
Y	Y	N	N	N	N	N	N	N
N	N	N	N	Y 1.79	N	N	N	N
N	N	N	N	N	Y-RT MCA CVA	N	N	N
N	N	N	N	N	N	N	MARFANS	N
N	N	N	N	N	N	N	N	N
N	N	N	Y ASTHMA	N	N	N	MARFANS	N
N	N	N	N	N	N	N	N	
N	N	N	N	y	N	N	MARFANS	S/P AORTIC FENESTRATION- TYPE 1

N	N	SEV CAL AS,ASC AO ANEURYSM	3+	73/49	SEV AS
Y	N	TYPE A AO DISSECTION,SEV AR,	4+		
Y	N	BAV,SEV AS, MILD AR,ASC AORTIC ANEURYSM	2+	90/70	SEV AS
Y	N	SEV AR,DIL AORTIC ROOT,OLD AWMI	4+		
Y	N	ASC AORTIC ANEURYSM,SEV AR	4+		
Y	Y	BAV,AC-TYPE A,R MCA STROKE,AKI	4+		
N	N	ANNULOAORTIC ECTASIA, SEVAR	4+		
Y	N	TYPE A AORTIC DISSECTION,SEC AR	4+		
N	N	ANNULOAORTIC ECTASIA,SEVERE AR	4+		
N	N	SEV AR,ANNULOAORTIC ECTASIA,DILATED ASC AORTA,FAIR L	4+		
N	N	AS AO DILATATION,BAV,MOD AR	3+		
Y	N	ASC AORTIC ANEURYSM,SEVAR	4+		
N	N	SEV AR,ASC AORTIC ANEURYSM	4+		
N	N	ANNULOAORTIC ECTASIA,SEV AR,HEALED DISSECTION,MARF	4+		
N	N	ANNULOAORTIC ECTASIA,MILD AR,MILD MR,	4+		
N	N	ANNULOAORTIC ECTASIA,MOD -SEV AR	4+		
N	N	ASC AORTIC ANEURYSM,SEVAR	4+		
N	N	BAV,SEV AR,ASC AO ANEURYSM	2+	98/84	
N	N	ANNUOAORTIC ECTASIA,MOD MR,SEV AR	4+		
Y	N	ANNULOAORTIC ECTSIA,SEV AR	3-4+		
Y	N	ANNULOAORTIC ECTASIA ASC AO ANEURYSM	4+		
N	N	ANNULOAORTIC ECTASIA,SEVERE AR	4+		
N		ANNULOAORTIC ECTASIA,SEV AR,GDLV	4+		
Y	Y	ASC AORTIC ANEURYSM,SEV AR	4+		
N	N	ASC AORTIC ANEURYSM,SEV AR	4+		
Y	N	ASC AORTIC ANEURYSM,SEV AR	3-4+		
N	N	ANNULOAORTIC ECTASIA,SEV AR,GDLV	4+		
Y	N	ANNULOAORTIC ECTASIA,SEV AR,GDLV	4+		
N	N	ASC AORTIC ANEURYSM,SEV AR	4+		
N	N	ASCENDING AORTIC ANEURYSM ,SEV AR,GBVF	4+		
Y	N	MARFANS,ANNULOAORTICECTASIA,SEV AR,MILD LVD	4+		
Y	N	ANNULOAORTIC ECTASIA,SEV AR,GDLV	4+		
Y	N	TYPE 1 DISSECION,ASC AO ANEURYSM,,MOD LVD	4+		

	40	58	26	GDLV	42/34	13/16	14/17	289.96		1168	
	49	65	28	GDLV	61/37	11/17	13/18	547.9		2506	
	39	43	24	GDLV	49/30	20/22	20/22		571.9		1602
	58	60	22	MILD LVD	66/40	19/22	20/24		930.3		2942
	65	49	26	FAIR LV	76/55	14/17	10/14	654.6		3919	
	48	50	24	SEV LVD	58/38	22/12	13/15		574.4		2261
	50	49	33	GDLV	62/35	21/22	14/16		634.3		2590
	48	50	24	GDLV	65/57	20/22	12/20	791.3		2852	
	45	49	28	GDLV	62/35	14/17	11/17	538.4		2590	
	69	70	43	GDLV	72/53	14/18	12/18	738.1		3511	
	77	30	25	GDLV	50/27	12/14	9/14	291.4		1669	
	62	68	25	GDLV	52/28	22/12	10/14	450.6		1809	
	57	58	24	GDLV	49/32	10/12	12/14	253.7		1602	
	70	76	28	GDLV	71/47	8/12	16/18	560		3413	
EFFACED		26	28	GDLV	51/23	10/15	9/12	285.06		1738	
	67.5	62	32	GDLV	52/38	10/14	12/14	309.6		1809	
	68	72	32	GDLV	65/49	11/16	9/16	531.5		2852	
	56	70	28	GDLV	65/46	1012	12/14	399.1		2852	
	45	56	24	GDLV	86/66	7/11	7/9	462.3		5036	
	56	65	28	GDLV	76/36	12/14	13/14	571.3		3919	
	62	58	36	GDLV	65/41	11/14	11/14	441.3		2852	
	57	60	26	GDLV	60/34	10/14	14/16	427.4		2423	
	45	56	24	GDLV	81/58	9/11	11/15	577.7		4460	
	56	66	28	GDLV	50/30	12/14	10/12	261.83		1669	
	47	56	24	GDLV	54/34	10/12	12/14	295.6		1954	
	56	67	28	GDLV	47/33	12/16	13/17	340.2		1471	
	55	62	27	GDLV	55/36	20/14	22/14	337.9		2028	
	56	67	28	GDLV	55/35	5/18	5/15	429.2		2028	
	55	65	27	GDLV	56/33	12/10	14/10	313.2		2104	
	50	67	28	GDLV	75/50	10/16	12/16	668.8		3815	
	54	66	28	MILD LVD	69/56	10/12	8/12	396.5		3220	
	60	66	28	GDLV	60/45	10/14	10/13	368.76		2423	
	40	77	33	MOD LVD	59/49	11/15	10/15	416.3		2341	

40		55	28	N	12.7	3.8	1.2	22
40		60	33	N	13.3	4.3	1.4	12
40	39X50		28	N	17.2	4.5	1	
57X56	56X55		30	PROX LAD <50, RCA < 50	14	4	1.6	22
40.7		43.1	42	NC	14.7	4	1	20
48.9X53.7	54X54		28	CT-CAG- LAD ,50%	12.6	3	1.2	12
39.5		33	29	N	13	3.7	0.6	16
50	56	30	N	12	4.2	1.0	12	
39.5	55.7	29.5	N	13.1	3.8	0.8	22	
	71X68	67X60	N	10.3	3.5	0.8	22	
55	55X27	28	N	13	4	0.5	13	
55	55	33	MINOR CAD	13	4	0.9	23	
4X70	57	38	N	10.8	4.2	1.3	17	
80x86	78	30		14.5	4.7	0.60	14	
60X62	66	32	N	14.5	4.0	0.7	22	
55.5	70	27	N	14.9	4.5	0.8	22	
67	73	32	N	12.8	4.7	0.9	10	
65	68	30	N	14	3.9	1.0	12	
59	47	34	N	13.1	4.6	0.8	12	
56	66	29	N	12	3.8	1.0	12	
55	77	23	N	13.8	4.0	0.9	22	
			N	13.5	4.9	1.0	13	
			N	12.9	4	1.20	16	
66	70	35	N	16	4	1.30	14	
55	65	30	N	14	4	1.00	14	
48	54	30	N	13	3.9	0.9	17	
55	46	32	N	15.7	4	1.70	13	
60	77	30	N	13.4	4	1.00	10	
56	66	28	N	14.6	3.5	1.00	12	
44	78	44	N	9.4	3	1.50	22	
45	57	30	N	10.6	3	1.0	22	
65	70	34	N	12.6	3.8	1.4	11	
45	77	35	N	11.8	4	1.90	22	

TEPID BLOOD		123	96	#25 CHVP	#26	ACS	N	N			28
TEPID BLOOD FA		204	132	#23 PM MAGNA	#26	ACS		17	N		18
TEPID BLOOD		119	81	#23 MEDTRONIC	#24	ACS	N	N			26
TEPID BLOOD-		130	107	#25CHVP	#26ALBO	ACS	N	N			24
COLD BLOOD		125	87	#29	#30	ACS	N	N			27
TEPID BLOOD-FA		181	158	#23 PM MGNA	#26	AORTIC ARCH	2 MIN	N			22
TEPID BLOOD-FA		137	112	#23CHVP	#24	ACS	N	N			25.3
TEPID-FA	138	107		#29CHVP	#30	ACS	N	N		26.6	
TEPID BLOOD FA	137	112		#23CHVP	#24	ACS	N	Y		23.5	
TEPID BLOOD	154	115		#2CHVP	#24	ACS	N	N		27	
TEPID BLOOD	117	82		#27CHVP	#28	ACS AO	N	N		28	
HYPERKALEMIC	100	82		#25 SJM	#25	ASC AO	N	N		26	
HYPERKALEMIC	120	96		#19 CHVP	#20 COATED CO	ACS	N	N		28	
TEPID BLOOD	126	88		#25 CHVP	28MM	ASC AO	N	N		26	
TEPID ROOT+OSTIAL	132	103		#27CHVP	#28	ASC AO	N	N		28	
TEPID BLOOD	135	101		#27SJM	28	ASC AO	N	N		26	
ANTEGRADE HYPERKA	162	126		#23 CHVP	#28	ASC AO	Y-3MINS	N		28	
TEPID BLOOD	120	96		#27	28	ASC AO	N	N		27	
TEPID BLOOD	100	82		#25	*26*	ASC AO	N	N		28	
TEPID BLOOD	110	97		#29	#30	ASC AO	N	N		27	
TEPID BLOOD	119	96		#23	#24	ASC AO	N	N		26	
TEPID BLOOD	130	106		#23	24	ASC AO	N	N		26	
TEPID BLOOD	120	91		#29	#30	ASC AO	N	N		26	
TEPID BLOOD	147	118		#27	28	ASC AO	N	N		26	
TEPID BLOOD	148	107		#25	26	ASC AO	N	N		27	
TEPID BLOOD	145	103		#27	28	ASC AO	N	N		27	
HYPERKALEMIC	136	107		#25 CHVP	26	ASC AO	N	N		26	
TEPID BLOOD	178	116		#27CHVP	#28	ASC AO	N	N		26	
TEPID BLOOD	154	108		#28	30	ASC AO	N	N		26	
TEPDI BLOOD	145	98		#25CHVP	#26	ASC AO	N	N		26	
TEPID BLOOD	107	83		#27	#28	ASC AO	N	N		26	
TEPID BLOOD	114	89		#27	#28	ASC AO	N	N		26	
TEPID BLOOD	175	121		#27	#28	ASC AO	N	N		26	

N	N	MILD LVD	N	N	N	N	2 DAYS
VF- REVERTED DC 20J	N	MOD LVD	Y-PD	Y-ENCEPHELOPATH	N	N	4 DAYS
BIGEMINI	N	GDLV-7MM PE	N	N	N	N	1DAY
N	N	GDLV	N	N	N	N	2 DAYS
N	N	N	N	N	N	N	2DAYS
AF,DC VERTED	YES-1630	MOD LVD	Y	Y-NEW LEFT OCC IN	N	N	REINTU
N	N	PE-3MM	N	N	N	N	2 DAY
N	N	GDLV	N	N	N	N	2 DAYS
VENTRICULAR BIGEMINI	N	GDLV,TRIVIAL PE	N	N	N	N	2 DAYS
N	N	GDLV, 3MM PE	N	N	N	N	2 DAYS
N	N	N	N	N	N	N	1 DAY
ST ELEVATION, CKMB N	N	GDLV,NO RWMA	N	N	N	N	REINTUBATED
N	N	GDLV ,PE 7M	N	N	N	N	1 DAY
N	N	GDLV.PE	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	MILD LVD, MILD MR	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAYS
N	N	MILD LVD	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1 DAY
AF	N	GDLV.MILD PE	N	N	N	N	2 DAYS
N	N	GDLV	N	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	1DAY
N	N	GDLV	Y CREAT-2	N	N	N	1 DAY
N	N	GDLV	N	N	N	N	N
N	N	GDLV	N	N	N	N	2 DAYS
N	N	GDLV	N	N	N	N	1DAY
N	N	GLOBAL HYPOKINESIA	N	N	N	N	2 DAYS
N	N	GDLV.MILD PE	N	N	N	Y	1 DAY
N	N	GDLV,MILD PE	N	N	N	N	1DAY

N	N	3 DAYS	10 DAYS	Y-AV BLOCK	Y IC BLLED	Y	
Y	Y	14 DAYS	20 DAYS	N	IC BLLED	Y	N
N	N	2 DAYS	10DAYS	LFU			
Y-LEFTLOWER LOBE	Y	7 DAYS	19 DAYS	N	N	N	N
N	N	3 DAYS	12 DAYS	N	N	N	N
Y-KLEBSIELLA	S	25DAYS	30DAYS				
N	N	3 DAYS	10DAYS	N	N	N	N
N	N	3 DAYS	12 DAYS	N	N	N	N
N	N	3 DAYS	10 DAYS	N	N	N	N
N	N	3 DAYS	11 DAY	N	N	N	N
N	N	3 DAYS	12 DAYS	N	N	N	N
N	N	3 DAYS	13 DAYS	N	Y VETEBROBAS-2016	N	N
N	N	3 DAYS	7 DAYS	N	N	N	N
N	N	4 DAYS	7 DAYS	N	N	N	N
N	N	3 DAYS	8 DAYS	N	N	N	N
N	N	3DAYS	7DAYS	N	N	N	N
N	N	4 DAYS	9 DAYS	N	N	N	N
N	N	4 DAYS	10 DAYS	N	N	N	N
N	N	4 DAYS	7 DAYS	N	N	N	N
N	N	4 DAYS	7DAYS	N	N	N	N
N	N	3 DAYS	7 DYS	N	N	N	N
N	N	3 DAYS	9 DAYS	N	N	N	N
N	N	3DAYS	10DAYS	N	N	N	N
N	N	5 DAYS	10 DAYS	N	N	N	PPI
N	N	5 DAYS	8DAYS	N	N	N	N
N	N	3DAYS	7DAYS	N	N	N	N
N	N	3 DAYS	10DAYS	N	N	N	N
N	N	3DAYS	9 DAYS	N	N	N	N
N	N	3 DAYS	9DAYS	N	N	N	N
N	N	4DAYS	10 DAYS	N	N	N	Y-PERICARDIOSTOMY
N	N	4 DAYS	8 DAS	N	N	N	N
N	N	4 DAYS	20DAYS	N	N	N	N
N	N	3DAYS	10 DAYS	N	N	N	N

	N	Y-ECOLI	16/12	N	N	SEV LVD,HYPOECHOIC PE	5/52
		Y	15/10			GDLV,VEG+	45/32
REGULAR	N	N	48/52	N	N	GDLV	52/58
REGULAR	N	N	16/7			GDLV	50/26
REGULAR	N	N	19/10	N	N	GDLV	46/32
REGULAR	N	N	23/12	N	N	GDLV	48/34
REGULAR-HEMOPTYSIS	N	N	19/10	N	N	GDLV	46/32
REGULAR	N	N	12/7	N	N	GDLV	48/33
REGLAR	N	N	12/5	N	N	GDLV	36/22
REGULAR	N	N	12/8	N	N	GDLV	45/22`
REGULAR	N	N	19/9	N	N	GDLV	35/20
RE	N	N	12/10	N	N	GDLV	60/38
REGULAR	N	N	16/7	N	N	GDLV	50/31
REGULAR	N	N	16/7	N	N	GDLV	51/36
REGULAR	N	N	9/5	N	N	GDLV	39/24
REGULAR	N	N	20/10	N	N	GDLV	45/39
REGULAR	N	N	20/12	N	N	MOD LVD	52/34
REGULAR	N	N	15/11	N	N	GDLV	45/28
REGULAR	N	N	20/10	N	N	GDLV	45/27
REGULAR	N	N	33/19	N	N	GDLV	46/25
REGULAR	N	N	14/7	N	N	GDLV	70/52
REGULAR	N	N	7/9	N	N	GDLV	45/28
REGULAR	N	N	8/10	N	N	GDLV	50/34
REGULAR	N	N	17/12	N	N	GDLV	45/38
REGULAR	N	N	12/10	N	N	GDLV	51/28
REGUALAR	N	N	12/10	N	N	GDLV	55/28
REGULAR	N	N	10/8	N	N	GDLV	45/28
REGULAR	N	N	11/6	N	N	GDLV	55/30
REGULAR	N	N	10/6	N	N	GDLV	50/45
REGULAR	N	N	22/12	N	N	GDLV	55/39
REGULAR	N	N	11/6	N	N	GDLV	45/28

12/22	18/22	619.7	1809	Y	SEPSIS	
12/22	10/12	335	1345	Y	MODS	
19/21	15/17	574.4	1809	N	N	N
12/20	14/17	444.5	1669	N	N	N
				Y	SEPSIS	
20/22	15/13	395.34	1407	N	N	Y-SHOULDER PAIN
12/20	10/12	334.6	1536	N	N	Y-CHEST PAIN
11/16	11/13	270.6	1407	N	N	N
11/10	12/14	232.25	1536	N	N	N
12/10	12/14			N	N	N-CHEST PAIN
10/12	9/14	222.57	1345	N	N	N
9/12	10/14	153.78	8024	N	N	N
10/12	12/14	350.1	2423	N	N	N
7/6	12/10	169.92	1669	N	N	N
12/10	9/12	241.23	1738	N	N	N
9/12	12/14	179.73	1002	N	N	Y-DOE
9/10	7/11	163.98	1345	N	N	N
9/11	8/10	207.28	1809	N	N	N
10/12	12/14	222.57	1345	N	N	N
9/11	8/10	163.98	1345	N	N	N
18/14	16/13	345.6	1407	N	N	N
8/10	10/12	321.75	3316	N	N	N
12/16	10/12	248.45	1345	N	N	N
10/16	8/10	261.83	1669	N	N	N
12/10	9/11	186.4	1345	N	N	N
15/13	20/14	419.4	1738	N	N	N
9/10	9/10	213.18	2028	N	N	N
12/10	9/10	175.02	1345	N	N	N
8/12	9/12	272.4	2028	N	N	N
8/10	9/12	207.14	1669	N	N	N
18/15	18/15	489.1	2028	N	N	N
11/14	7/10	198.1	1345	N	N	N

1
1
SEPSIS
1
2
1
1
2
2 DEATH IN 2016- PHARYNGEAL MUSCLE DYSFUNCTION, ASPIRATION-KLEBSIELLA PNEUMONIA
1
1
1 PROPTOSIS? THYROID PTHALMOPATHY 2011
1
2
1
1
1
1
1
1 DEPRESSIVE EPISODE +
1 PPI IN 2016 -CHB
1
1
1 N
1 N
1
1 PE DRAINAGE -1 ST YEAR-TAMPONADE
1
1 PARAVALVULAR AR2+
1 N

103	BALAKRISHNA PILLAI	279723	9562292311	66Y/M	17-01-2009	165	67
104	SAHADEVAN K	287879	4772128290	62Y/M	16-01-2009	165	87

N	N	N	N	N	N	N	N	NEUROFIBROMATOSIS
N	N	N	N	Y	N	N	N	N

Y	N	ANNULOAORTIC ECTASIA, MOD AR,ASC AORTIC ANEURYSM,	3-4+
N	N	BAV,SEV AR,ASC AO ANEURYSM	4+

54	56	28 GDLV	66/33	12/15	13/18	568.7	2942
52	58	32 GDLV	75/53	10/15	12/15	612.7	3815

60	66	33	N	14	4	1.2	20
58.4	48	30	N	11	3.8	1.5	12

TEPID BLOOD	106	87	#27	#28	ASC AO	N	N	26
TEPID BLOOD	105	86	#29CHVP	#30	ASC AO	N	N	28

N	N	GDLV,MILD PE	Y CREAT-2	N	N	N	1 DAY
Y-VENTRICULAR BIGEMINI	N	GDLV,MILD PE	N	N	N	N	1 DAY

N	N	3 DAYS	9DAYS	Y-CHB+PE	N	N	Y-PEDRANAIGE+PPI
N	N	4 DAYS	10DAYS	Y-VENTRICULAR ECTOPIC	N	N	Y-EMER PE DRAINAGE-2

REGULAR	N	N	12/10	N	N	MOD LVD	55/43
REGULAR	N	N	10/5	N	N	GDLV	61/43

10/12	12/14	304.33	2028	N	N	Y-DOE
9/12	12/14	359.6	2506	N	N	N

2	PE DARIANGE+PPI, WOUND INF,PNEUMONIA-KLEBSIELLA SEPSIS
1	PE DRAINAGE- SAME YEAR