



# Tricuspid Valve-A Biomedical Engineering Challenge

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## Abstract:

Tricuspid Valve Surgery is one of the difficult surgery that a surgeon may encounter. Results are variable. A cardiothoracic surgeon's operative skill and calibre is not sufficient but also in depth knowledge and application of Biomedical Engineering is also required for both diagnostic and therapeutic managements. In this article in depth knowledge of anatomy and pathophysiology and its implementation in biomedical engineering is discussed in depth.

## I. INTRODUCTION

Cardiovascular Disease (CVD) is considered one of the big killers already. It already kills more people in Europe than any other single cause of death. Heart diseases, or cardiovascular diseases, accounts for 29 percent of deaths worldwide according to the World Health Organization, not to mention the millions of people treated in hospitals for CVD every year which costs the economy billions of Euros annually<sup>1</sup>. Although valvular heart disease (VHD) is less frequent than coronary disease or heart failure (HF), it causes significant mortality and morbidity. An estimated 20,891 patients died because of VHD in the United States in 2007<sup>2</sup>. Tricuspid valve (TV) disease affects 0.8% of the general population in the United States<sup>3,4</sup>. Such data regarding the Indian subcontinents are lacking. The cardiac valves are responsible for moving the blood in the right direction through the heart. The tricuspid valve, also called the right-sided atrioventricular valve, regulates the opening between the right atrium (RA) and the right ventricle (RV) (Figure 1 & 2). As the right ventricle relaxes during diastole, and the pressure within the right ventricle is lower than the pressure in the right atrium, the tricuspid valve opens, and blood will flow from the right atrium to the right ventricle. Afterwards, the right atrium will contract allowing more blood to eject into the right ventricle. After contraction of the atrium, the tricuspid valve closes, whereby preventing a backwash of blood ("regurgitation") into the right atrium during the systolic phase of the heart cycle. The mitral valve (MV) regulates the opening between the left atrium and the left ventricle (LV) in a similar manner as the TV. When the pressure in the ventricles during systole becomes higher than the pressure in the pulmonary artery and aorta, the pulmonary and aortic valve open and guide the blood to the lungs and the rest of the body, respectively. After contraction of the ventricles, the pressure drops and the pulmonary and aortic valve close. The tricuspid valve is often ignored by cardiologists and surgeons because of its unique characteristics. Except for infective endocarditic, it is rarely affected in isolation. Most often, the prominent impact of other diseased valves minimizes its importance. Located at the entrance of the heart, its symptoms are primarily extra cardiac and often silent. Its behaviour is closely related to the function of the right ventricle; in most cases, tricuspid regurgitation is secondary to right ventricular failure. It follows the dictates of the mitral valve; resolution of the mitral valve problem is often followed by improvement in the degree of tricuspid regurgitation. Because it works in a low-pressure system, it is difficult to evaluate its preoperative importance

and to assess the value of different surgical techniques. These characteristics often lead cardiologists and surgeons to ignore the tricuspid valve with apparent impunity. However, recent developments in diagnostic tools and two-dimensional colour Doppler echocardiography have increased the awareness of this valve, which has been called the "Cinderella of all cardiac valves".

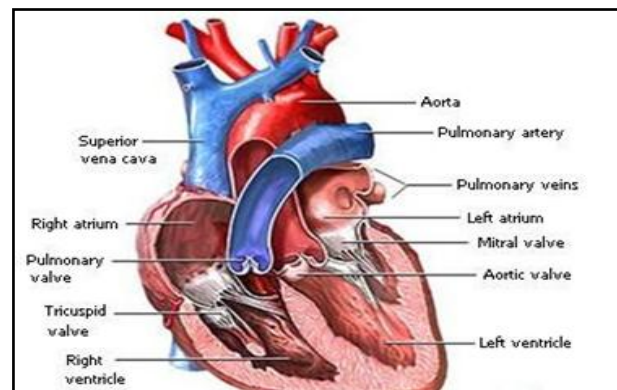


Figure.1. Anatomy of Heart

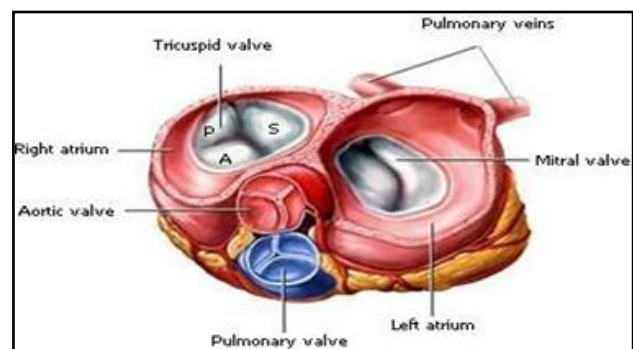


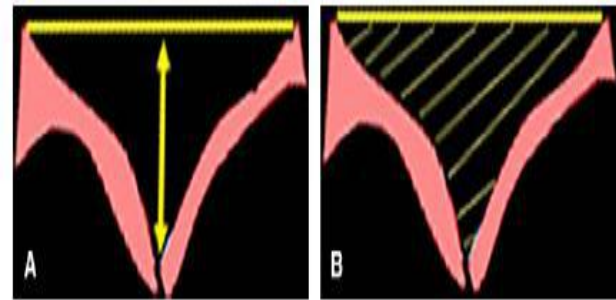
Figure.2. Anatomy & Relationship with Tricuspid Valve

## Anatomy

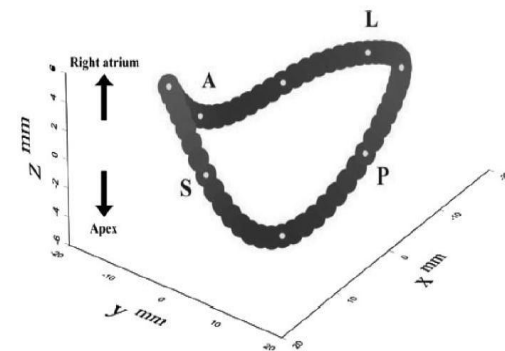
The Tricuspid Valve is composed of three leaflets (Anterior, Posterior and a septal leaflet), the chordae tendinae, two discrete papillary muscles, the fibrous tricuspid annulus, and the right atrial and right ventricular myocardium. The anterior leaflet and the septal leaflet are larger, whereas the posterior leaflet is notable for the presence of multiple scallops. TV commissures represent the border between the different leaflets (i.e. antero-septal, antero-posterior, and postero-septal). Because the septal wall leaflet is fixed, there is little room for movement if the free wall of the right ventricular/tricuspid

annulus should dilate<sup>7</sup>. Dilatation of the tricuspid annulus therefore occurs primarily in its antero-posterior (mural) aspect, which can result in significant dysfunction of the valve because of leaflet malcoaptation<sup>8,9</sup>. Leaflet coaptation represents the proper joining together of the different leaflets when the valve closes. The area of the annular plane to the atrial surface of the leaflets is called the tethering area, this term is often used together with the tethering/coaptation depth which stands for the distance from the annular plane to the begin point of leaflet coaptation (Figure 3)<sup>10</sup>. The tricuspid annulus has a complex 3-dimensional structure, which differs from the more symmetric “saddle-shaped” mitral annulus. Healthy subjects normally have a nonplanar, elliptical shaped tricuspid annulus, with the postero-septal portion being “lowest” and the antero-septal portion the “highest” (Figure 4). Patients with functional TR generally have a more planar annulus, which has dilated primarily in the antero-posterior direction, resulting in a more circular shape as compared with the elliptical shape in healthy subjects. Whereas the base of the anterior and posterior leaflets is attached to the free wall of the right ventricle, the septal leaflet is inserted into the base of the interventricular septum. This line of leaflet attachment, known as the tricuspid valve annulus, is more a landmark than an actual fibrous ring. This absence of an encircling fibrotic structure explains the large changes in the tricuspid valve’s orifice during the cardiac cycle and its easy dilation in disease. The mobility and size of the tricuspid valve orifice are dependent on the transversely oriented myocardial fibres that surround the atrioventricular valves. Tsakiris and associates found in a canine model that the size of the tricuspid valve orifice changed continuously during the cardiac cycle. The orifice area contracted (from its maximal diastolic size) by 20% to 30%. Tei and associates<sup>3</sup> confirmed these findings in humans by use of echocardiography. In their laboratory, they analysed an ovine model for the changes in the normal tricuspid valve orifice during the cardiac cycle as detected by the changes in distance between ultrasound crystals placed around the line of insertion of the leaflets<sup>4,5</sup>. The tricuspid valve orifice area expands and contracts twice during the cardiac cycle. Orifice contraction begins during the iso-volumic relaxation phase of the cardiac cycle and continues through the first half of diastole. Starting with the beginning of iso-volumic contraction, a second contraction occurs during ejection, which reduces the tricuspid valve orifice to its minimum area. This contraction corresponds to closure of the valve completed at the end of iso-volumic contraction. The reduction in orifice perimeter is not uniform. The segment of the annulus corresponding to the septal leaflet shortens by 12%, the anterior segment by 15%, and the posterior segment by 17%.<sup>4</sup> The notion that the length of the septal portion of the annulus can be used to determine the size of an annuloplasty device should be revised. This narrowing of the tricuspid valve orifice is due not only to contraction of its perimeter but, more important, to changes in the shape of the annulus. During contraction, the orifice becomes more elliptical because of displacement of the anteroposterior commissure toward the septum and the bulging of the septum. As in the mitral valve, the “annulus” is not in a single plane. In fact, the tricuspid valve annulus is saddle shaped, with its horn or pommel corresponding to the area of the antero-septal commissure and its cantle to the midpoint of the base of the posterior leaflet. This saddle shape or hyperbolic paraboloid, well known to architects as an ideal design to reduce building tension, has been shown in the mitral valve to significantly reduce peak leaflet stress.<sup>6</sup> Furthermore, the increase in the saddle shape during contraction has a *folding*, reducing effect on the normal

tricuspid valve orifice. In cases of tricuspid regurgitation, besides an increase of tricuspid valve annulus area, there is an increase in planarity or flattening of the normal annulus, which induces leaflet tethering.<sup>7</sup> Rigid structures, such as stented prostheses and rigid annuloplasty rings, destroy this configuration and (most likely) have a negative impact on the function of the right ventricle.<sup>8-20</sup>



**Figure 3. T.v tethering/coaptation depth (a) tethering area (b)** <sup>(10)</sup>



**Figure 4. Three dimensional shape of tricuspid annulus, (11) anterior, septal, posterior & lateral represented by a, s, p & l.**

### Pathophysiology

Tricuspid Valve Regurgitation (TR) or insufficiency implies a backflow of blood from the right ventricle to the right atrium during contraction of the right ventricle (Figure 5&6). TR is a common echocardiographic finding that is present in 80 to 90% of normal individuals<sup>3</sup>. The cause of TR is more often functional (secondary) rather than morphological (primary). In morphological TR, as the name already predicts, the leaflets themselves are morphologically affected which results in a dysfunctional tricuspid valve<sup>15</sup>. Congenital defects, trauma, carcinoid heart disease, toxic effects of chemicals, tumours, myxomatous degeneration, rheumatism, or endocarditis, possibly together with rupture of chordae or papillary muscles are the main causes of morphological TR.<sup>4-28</sup> A variety of etiologic factors can induce organic regurgitation, but today, the most frequent cause of organic tricuspid valve disease in urban populations is infective endocarditis. Tricuspid valve endocarditis used to be relatively rare, with an incidence of only 5% to 10% of patients with infective endocarditis. However, its frequency has dramatically increased with the spread of intravenous drug abuse.<sup>13</sup> In this population, the tricuspid valve usually has no pre-existing pathologic change. The lesions vary from isolated vegetation to destruction of the valve, including the annulus. *Staphylococcus aureus* remains the most common organism found in drug addicts, followed by gram-negative organisms and *Candida*. Fungal infections are also increasing because of longer periods of invasive monitoring of patients with multiorgan failure in intensive care units. In the developing world, rheumatic fever is the primary cause of organic valvular heart disease. Typical lesions show

varying degrees of leaflet thickening and (most often) commissural fusion. In severe cases, the thickened leaflets become diaphragm-like, with a central circular orifice. The sub valvular apparatus is seldom affected, and calcifications are rare. Although tricuspid valve stenosis is the classic lesion, predominant insufficiency is just as common. In a series of 253 patients with rheumatic heart disease who underwent tricuspid valve surgery, it has been found that organic involvement was present in 45% of the cases and that 45% of them also had annulus dilation.<sup>14</sup> In a classic study of 100 post-mortem hearts with rheumatic disease, Gross and Friedberg<sup>15</sup> found microscopic evidence of inflammation in the annulus of all four valves (in the acute rheumatic attack). Rheumatic tricuspid valve disease is always associated with rheumatic mitral valve or mitral-aortic valve lesions.

The incidence of chronic rheumatic tricuspid valve disease associated with rheumatic mitral valve disease varies widely, from 6% in an echocardiographic study<sup>16</sup> to 33% in an anatomic series<sup>17</sup> and 11% in a series of 1052 patients undergoing rheumatic valvular surgery.<sup>14</sup> In a Mayo Clinic surgical pathology study of excised tricuspid valves at the time of valve replacement,<sup>18</sup> post-inflammatory aetiology was responsible for 53% of the 363 valves studied. However, this frequency had diminished from 79% during the period from 1963 to 1967 to 24% during 1983 through 1987, reflecting the reduction in the incidence of rheumatic fever in the United States.

Leaflet tears and total or partial avulsion of a papillary muscle head occur after closed chest trauma. They are occasionally diagnosed at surgery and only classified postoperatively as traumatic by the patient, who recalls an old accident when prompted by the surgeon.<sup>19-21</sup> An occasional cause of traumatic tricuspid regurgitations that induced by the biotope during a right myocardial biopsy in transplanted patients. Leaflet tears or chordal avulsion results in severe regurgitation that requires urgent surgery.<sup>22</sup> Degenerative tricuspid regurgitation associated with mitral valve prolapsed is being increasingly observed. This double valve lesion is particularly frequent in Marfan syndrome as a manifestation of a fibrilopathy that also involves the aortic valve and ascending aorta. The reported frequency of tricuspid valve involvement among patients with mitral valve myxomatous disease oscillates between 21% and 52%.<sup>7,23,24</sup> Less common causes include organic tricuspid valve lesions secondary to carcinoid syndrome and appetite-suppressant drugs.<sup>25</sup>

In both cases, the leaflets are encased by a fibrous sheath that reduces their mobility, resulting in stenotic and regurgitant lesions. Functional TR is often called secondary TR because it results most commonly from a left-sided heart disease, right ventricular volume and pressure overload<sup>9,14,15</sup>. Functional or secondary tricuspid regurgitation can be defined as the incompetence of the TV in the absence of any structural leaflet disease<sup>11</sup>. This incomplete leaflet closure is thought to be caused by the dilatation of the tricuspid annulus (TA) and tethering of the tricuspid leaflet, after right ventricular dilatation and dysfunction which is associated with papillary muscle displacement<sup>16,17</sup>.

Functional TR is often unrecognized, being only apparent during periods of increased preload and afterload. Therefore, the preload, the afterload and the right ventricular (systolic) function in addition to tricuspid dilatation very important deterministic factors of TR. This may explain why TR is difficult to accurately assess because these factors can interfere

regarding the severity of TR under different conditions. The preload can be seen as the end-diastolic volume, associated with the ability of ventricular filling, during relaxation of the heart (diastole). The afterload corresponds with the force against which the ventricle must eject its contents during contraction of the heart (systole). The right ventricle's afterload depends (partly) on the mean pulmonary arterial pressure. Functional tricuspid insufficiency is understood to be exclusively due to annulus dilation and dysfunction. The leaflets, chords, and papillary muscles are otherwise normal. Because of the lack of an anatomic fibrous annulus, the tricuspid valve's annulus follows the dilation of the right ventricle.

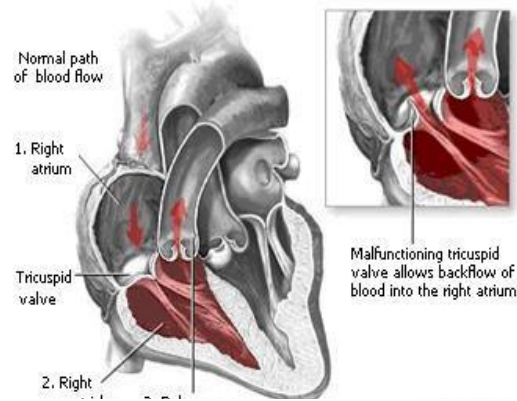
The total perimeter of the normal annulus is approximately 100 to 120 mm. In cases of functional tricuspid regurgitation, the circumference of the annulus can reach 150 to 170 mm.<sup>27,28</sup> This annulus dilation is nonhomogeneous. In a post-mortem study that included normal controls and hearts with rheumatic or myxomatous tricuspid valve disease, Carpentier and colleagues<sup>11,21</sup> showed that the anterior and posterior segments of the annulus dilated far more than the septal portion of the annulus. This report formed the basis for all annuloplasties that selectively reduce the whole annulus except at the level of the septum.

In congestive heart failure, functional tricuspid regurgitation is a predictor of poor survival<sup>22</sup> and may be an independent risk factor for the development of cardiac cachexia and protein-losing enteropathy.<sup>23</sup> Koelling<sup>24</sup> studied a total of 1436 patients with left ventricular systolic dysfunction (ejection fraction <35%). Mitral regurgitation was moderate in 30% and severe in 19%. Moderate tricuspid regurgitation was present in 23% and severe in 12%. Patients with severe mitral regurgitation were more likely also to have tricuspid regurgitation. Advances in echocardiography have revealed another sub-valvular mechanism that applies to both mitral and tricuspid functional regurgitations. Originally observed in functional ischemic mitral regurgitations, it also applies to the ischemic tricuspid valve and most probably to all functional insufficiencies. Right (or left) ventricular remodelling induces a lateral displacement of one or more papillary muscles that apically pulls (through the basal chords) the body of the corresponding leaflet.

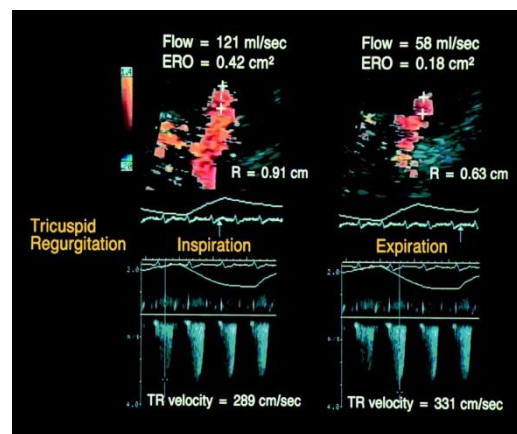
The leaflet becomes tethered down, losing its coaptation.<sup>7,25</sup> Functional tricuspid regurgitation is an expression of right ventricular failure with a generalized distortion of ventricular geometry. Although annuloplasty can reduce or abolish functional tricuspid regurgitation (by reducing the dilated annulus), it does not address the altered sub-valvular geometry. Awareness of this important mechanism is giving rise to new and exciting surgical approaches directed toward redressing the geometric distortion.<sup>26</sup> Generally, the symptoms of the left-sided heart disease predominate in those patients with secondary tricuspid valve disease. With primary TR, or secondary TR being in an advanced stage, patients may experience fatigue and decreased exercise tolerance because of decreased cardiac output. They may also experience the classic symptoms of "right-sided heart failure" from elevated right atrial pressures such as ascites, congestive hepatopathy, peripheral oedema and dyspepsia.<sup>1-28</sup> Clinically, and in the literature, tricuspid valve disorders receive less attention as compared to the primary left-sided disease. It is frequently labelled as "the forgotten valve" since surgical correction is still often ignored. Appropriate treatment of the tricuspid valve



disease, even when secondary to left heart diseases, may improve long-term functional outcome.



**Figure.5. normal right sided blood flow pattern inset: tricuspid regurgitation<sup>12</sup>**



**Figure.6. Two dimensional echo-cardiographic view of tricuspid regurgitation in both inspiratory & expiratory phase**

### Echocardiographic Examinations

Echocardiography is one of the most frequently used techniques for diagnosing cardiovascular diseases since real-time two-dimensional (2D) echocardiography provides high-resolution images of cardiac structures and their movement so that detailed anatomic and functional information about the heart can be obtained. Comprehensive 2D echocardiographic exams were performed with a commercially available system (e.g. Philips Medical Systems, Eindhoven, IE33®) by Registered Diagnostic Cardiac Sonographers. There were two kinds of echocardiographic examinations that can be performed, transthoracic echocardiographic examinations (TTE) and transoesophageal echocardiographic examinations (TEE). TTE is completely non-invasive, a transducer is placed directly on the chest while emitting harmless ultrasound beams to create an image. The patient was asked to pull out his/her shirt before laying in the appropriate left lateral decubitus position (Figure 7 to 10). ECG patches and electrodes were placed on the patient's body to monitor the heart rate in a standard manner. For proper movement of the transducers tip and realizing a good image quality, a small amount of aquasonic ultrasound transmission gel was used. TEE on the other hand is a semi-invasive procedure because of a transoesophageal probe intubation that can be uncomfortable in unprepared patients. Mostly, a TEE was performed in addition to a TTE to evaluate the mitral valve, left atrium (LA), atrial septal defects, endocarditis, and its complications in more detail. The patient fasted for at least 4 hours before undergoing TEE, and a history of oesophageal pathology was evaluated before. Patients with dentures removed these. An

intravenous access for administration of contrast agent or medication was placed by qualified employees. Again, ECG patches and electrodes were placed on the patient's body to monitor the heart rate. Immediately before intubation of the transoesophageal probe, xylocaine spray (10%) was used to anesthetize the posterior pharynx locally. A short-acting sedative diprivan (1%), 1 to 10 mg, was mostly necessary to make the TEE examination more comfortable for the patient. This agent was used with caution because of potential respiratory suppression. No problems associated with this medication were noted during the investigations. A special TEE probe, with a transducer tip of 10 to 14 mm which can be maneuvered to various positions in the oesophagus and the stomach, was used. Additionally, the multiplane transducer could rotate 180 degrees. Altogether, this allowed us to visualize the heart and other structures in a detailed manner. Rotation of the transducer was accomplished by a finger pressure-sensitive switch at the proximal operator end. The examination began with the patient in the left lateral decubitus position. To protect the TEE scope, a bit guard and a latex free cover was used. At probe introduction, the imaging surface of the transducer faced the tongue, which directs the ultrasound beam anteriorly toward the heart when the probe is in the oesophagus. With guidance of the left index finger the transducer was advanced smoothly and slowly posteriorly toward the oesophagus. At that time, the patient was asked to swallow. The tip of the TEE was advanced into the oesophagus without force or significant resistance. From this point on the visualization started to evaluate the different heart structures. The used probe was cleaned afterwards with glutaraldehyde disinfectant. Mostly, patients needed a recovery time after the procedure. The intravenous catheter was also removed when it was not applicable anymore for other procedures afterwards. The patients were told not to drive for 12 hours if sedation was used. Typically, a TTE examination was initially performed by a echocardiographer/sonographer and required 20 to 30 minutes. Afterwards, these images were reviewed and supervised by a staff echo cardiologist. A TEE examination was performed by an echo cardiologist, assisted by a sonographer to sedate, and hold the patient in the appropriate position during the examination.

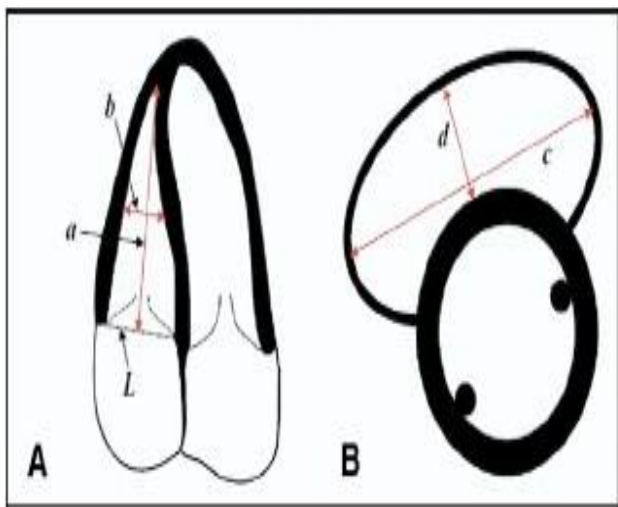
### This procedure took 30 to 45 minutes.

Echocardiography uses high-frequency ultrasound (2.0 to 7.5 MHz) to evaluate the structural, functional, and hemodynamic status of the cardiovascular system. A 2D TTE was performed in a standard manner from three standard transducer positions: the parasternal, apical, and subcostal windows. From each transducer position, multiple tomographic images of the heart relative to its long- and short-axis are obtained by manually rotating and angulating the transducer. The long axis view bisects the heart from the base to the apex. The short-axis view is perpendicular to the long-axis view. M-mode echocardiography complements 2D echocardiography by recording detailed motions of cardiac structures. M-mode is used for the measurement of dimensions and is essential for the display of subtle motion abnormalities of specific cardiac structures. The hemodynamic status is evaluated using the Doppler technique. Doppler echocardiography measures blood-flow velocities in the heart and great vessels and is based on the Doppler effect. The Doppler effect states that sound frequency increases as a sound source moves towards the observer and decreases as the source moves away. The most common uses of Doppler echocardiography are in the pulsed- and continuous-wave forms. In the pulsed-wave mode, a single ultrasound crystal sends and receives sound beams.

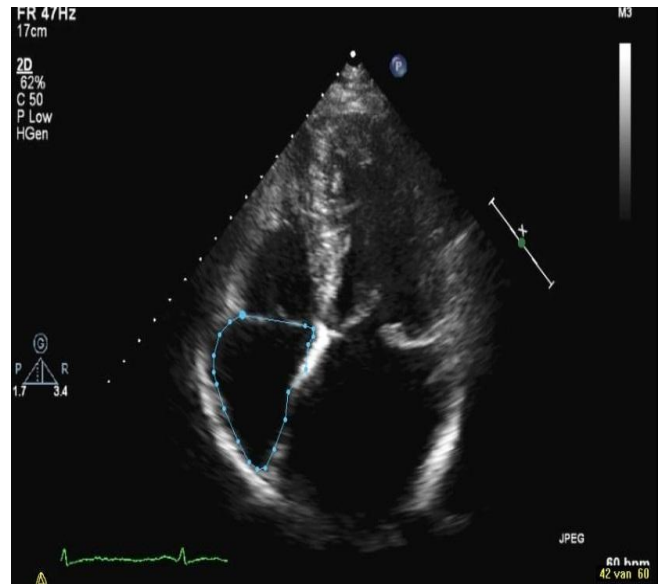
The ultrasound is reflected from moving red blood cells and is received by the same crystal. In the continuous-wave mode, the transducer has two crystals: one to send and the other to receive the reflected ultrasound waves continuously. Additionally, blood flow can also be visualized with the colour-flow imaging based on pulsed-wave Doppler principles. Blood flow directed toward the transducer is standard color-coded in shades of red. Blood flow directed away from the transducer is standard color-coded in shades of blue. Each colour has multiple shades, and the lighter shades within each primary colour are assigned to higher velocities. Optimization at the time of the examination was needed by using the optimal transducer, knob settings and transducer positions. Grey scales and gains were controlled manually to minimize background noise and to maximize the delineation of cardiac structures. The depth of the fields was also controlled to provide the optimal image size. Filter settings depend on the type of Doppler study and were selected automatically. These optimizations were important to make certain that the area of abnormal blood flow was not underestimated by a low gain setting or overestimated by a low filter setting, because severity of valvular regurgitation or shunt flow depends on the area of abnormal blood flow detected with colour-flow imaging. The optimal setting displayed the entire flow jet with minimal background noise.



**Figure.7.** Left lateral decubitus for standard echocardiographic position



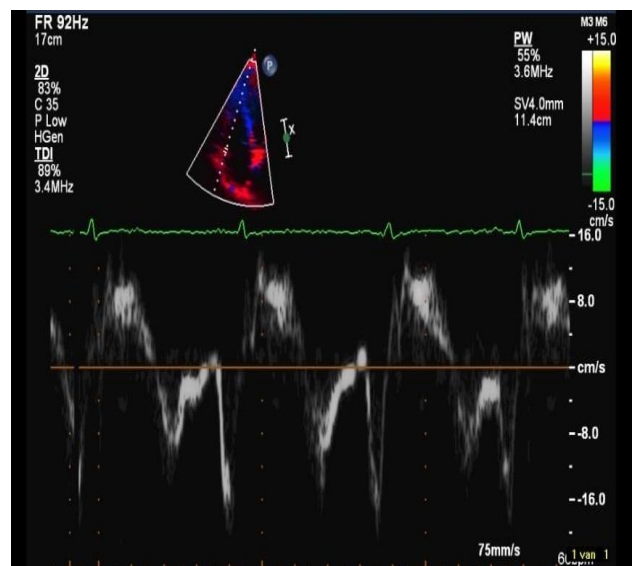
**Figure.8.** Long axis (a) & short axis (b) four chambers view (fig-8a) longest lateral distance (c) & Free wall midseptal (d)(fig-8b)



**Figure.8a.** Right atrial area in four chamber views

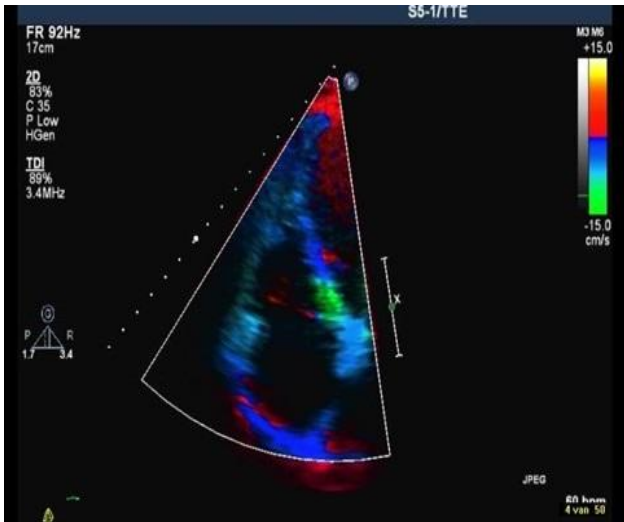


**Figure-8b.** Tricuspid annular plane systolic excursion



**Figure.9.** Tissue doppler imaging of right ventricular free wall

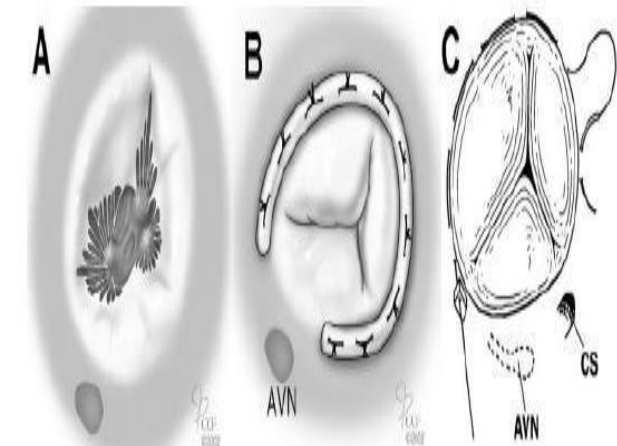




**Figure.10. Zoomed in photograph of framed area of figure-9**

**Surgical procedures**

All surgical procedures were performed through midline sternotomy (i.e. cutting the breast bone) and under cardiopulmonary bypass with antegrade blood cardioplegia (to stop the heart movements but to maintain circulations) and subsequently according to standard techniques. Standard Protocols as commonly performed are modified as required and if indicated. The restored leaflet coaptation was confirmed at the time of surgery by filling the RV/LV with disposable 20 c.c syringe fitted at the tip with three or four-inch polyvinyl chloride tube and filled with Normal Saline. Most diseased tricuspid valves can be easily repaired. Severe lesions are usually treated, but real or erroneously labelled “moderate” lesions are ignored. The problem often lies in the absence of a detailed preoperative search for tricuspid valve disease. Intraoperative decision-making is unreliable unless the lesion is severe. Preoperative transthoracic echocardiography specifically interrogating the tricuspid valve is an absolute requirement. Unfortunately, this is not always available to the surgeon, who should rely on intraoperative transoesophageal echocardiography. The main surgical approach to repair functional TR is tricuspid annuloplasty (TVP), which involves rigid and flexible annular bands that are used to reduce annular size and achieve leaflet coaptation. Another approach includes the De Vega non-ring annuloplasty, also called the partial purse-string suture technique, to reduce the anterior and posterior portions of the annulus but recurrent TR after surgery is more common, none of the devices & method provide sure success (Figure 11&16).



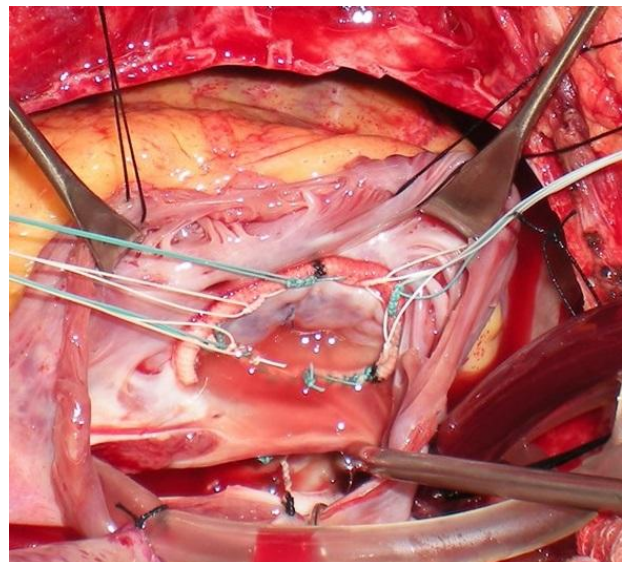
**Figure.11. Predominant surgical repair techniques for tricuspid regurgitation....cs: coronary sinus<sup>15</sup>**



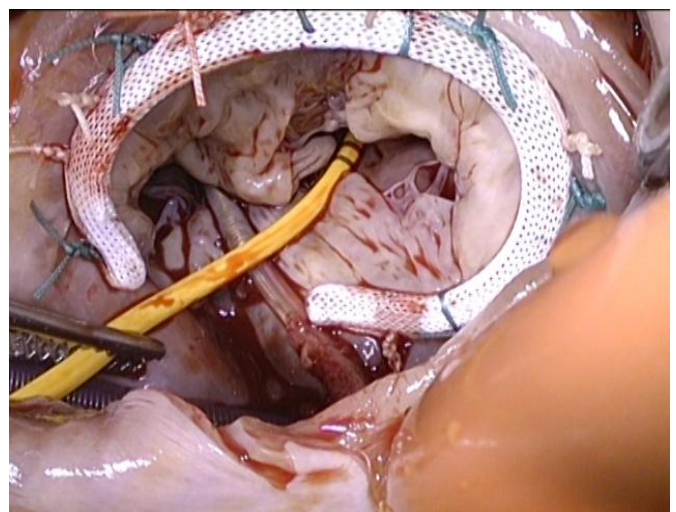
**Figure.12. Non-planar Tricuspid Annuloplasty Ring**



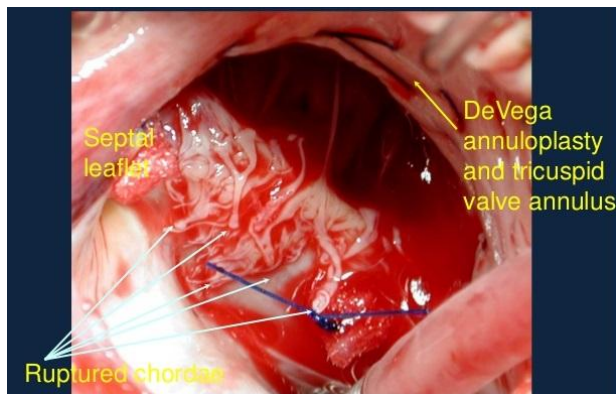
**Figure.13. Tricuspid Annuloplasty Ring with holder**



**Figure.14. Intra-operative picture after using Tricuspid Annuloplasty Ring to reduce Tricuspid Regurgitation**



**Figure.15. Checking of coaptation after tricuspid valve flexible ring annuloplasty**



**Figure.16. DeVega annuloplasty**

## II. DISCUSSION

Contrary to what might have been expected, concomitant tricuspid valve surgery did not lead to a reduction in RV size at short-term follow-up in the overall study population. The likely explanation for this finding is the fact that most patients in our study population underwent TVP because of TA dilatation rather than severe TR (65% vs. 35% respectively), also underscored by the fact that the average grade of TR severity was only 2 at baseline. Our findings indicate a significant correlation between the preoperative severity of TR and the subsequent change in end-diastolic RV size ( $r=0.5$ ,  $p=0.003$ ), and support previous findings by Kim et al. which showed that the extent of RV reverse remodelling post-TVP is directly proportional to the extent of RV volume overload prior to surgery<sup>28</sup>.

## III. CONCLUSIONS

As most of the day to day interventions occur, despite best effort by doctors the ultimate results are quite far cry as promised by theoretical explanations of biomedical engineering & mathematical formulas except few case reports in journal. Selection of patients may provide exceptionally good results however in subcontinents like ours where patients due to lower socio-economic conditions not only presents late but also cannot afford more precise high valued rings. Not only surgeons but also biomedical engineers need a great concern to bridge the gap.

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