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FACULTÉ DE MÉDECINE
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Year 2018

Thesis N° 176

The short and midterm outcomes of the De Vega annuloplasty of the tricuspid valve during the surgical management of rheumatic valvular heart disease

THESIS

PRESENTED AND PUBLICLY DEFENDED IN July 19th, 2018

BY

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KEYWORDS

Tricuspid valve – Tricuspid valve repair– De Vega annuloplasty

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سُبْحَانَكَ رَبِّيَ الرَّحْمَنُ الرَّحِيمُ



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أَنْعَمْتَ عَلَيَّ وَعَلَىٰ وَالِدَيَّ وَأَنْ أَعْمَلَ
صَالِحًا تَرْضَاهُ وَأَدْخِلْنِي بِرَحْمَتِكَ فِي
عِبَادِكَ الصَّالِحِينَ"



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DEDICATIONS



There are a number of people who helped shape my life for the better and to whom I am deeply indebted. Words can never suffice to properly give them their merit.



This humble work is affectionately dedicated to them...

To my loving father

You supported me unconditionally, and you continue to do so, in whatever path I took during my very long academic journey

You relentlessly encouraged me to strive for excellence and you taught me that the sky is the limit for a person's dream

Whatever I achieve or hope to accomplish in the future, I know none of it is possible without your love and endless support and I hope that someday I'll make you proud . . .

Because of all the previously cited, I put you first in my list of dedications. Plus, I kind of had to too knowing that you are going to be reading this manuscript cover to cover. I know because that's how dedicated you are to following my academic journey step by step

To my amazing mother

You are the best human being I know and the best mother I could have hoped for With your strong and gentle soul, you taught me to trust Allah, believe in hard work, and that so much can be accomplished with little

You have always been the rock of stability in my life. You supported me with your endless wisdom and found the right words to lift up my spirits

I can't thank you enough for putting up with my constant complaining, for being nothing but supportive of my choices and for having enough Faith for both of us

I dedicate this work to you, for your interest in it, as in all my ventures, was never less than mine

To my smart, mischievously creative sister

Continuous fighting is our thing apparently, but you know that I love you and I can't imagine my life without you. Thank you for your support and for speaking your mind without holding back when I am being stupid. I hope you get everything you wish for.

To my troublesome little brother

Thank you for always being there to do my errands. I am highly indebted to you for providing my usual sugar supply whenever stress took over. I love you. I hope you will grow up to be the decent man I know you can be

To my dear grandmothers, grandfather, aunties, uncles and cousins (maternal and paternal)

Thank you for your love, encouragement and prayers

To the memory of my paternal grandfather

May ALLAH rest his soul in peace and grant him the best place in paradise

To my sister and best friend Fatima Zahra and her cutie son Mohamed Anas (always Ahmed Anas for me)

Words cannot describe the hold you have on my heart. We share the most wonderful memories. You are my best friend in the whole world. Neither time nor distance is capable of changing that. Thank you for always being there for me. You deserve everything beautiful in this world. I love you.

To my sweet cousin Fatima Ezzahra and her adorable son Ghassan

You have always been the gentle soul in the family. Thank you for your endless support and countless prayers. May Allah grant all your wishes

To Fadwa, my best friend and the kindest person I know

Your big heart and generosity are both overwhelming and humbling. I cannot thank you enough for being by my side in some of the hardest times of my life, as I cannot properly express my gratitude for some of the best memories I have. I love you and I will always cherish our friendship.

To my brother and friend Yassine J

You are still the same helpful and decent person I met twelve years ago. I wish you happiness and success.

To all my friends Fatima B, Fatima Ezzahra T, Meryem B, Naila L, Fatima Ezzahra El M, Chihab B, Layla B, Aissam G, Sara I, Yassine Ch, Hajar J, Sanaa A, Rajaa B, Charaf Z, the two amazing Imane B and Imane B, Hassan O, Wassima B, Aassim B ...

I know I am not the most sensitive person in the world, but I do love you all. Thank you for both the memories and the headaches

To all my internship colleagues

Thank you for the memorable two years.

To Mr Aouss

I am indebted to you for helping me improve my English and for reincarnating my love for reading and books.

To all my other family members, big and small, to my friends and colleagues who I inadvertently failed to mention

To all my teachers in primary, secondary and high school

To all my teachers in medical school of Marrakech

To all those who touched my life, in anyway



ACKNOWLEDGMENTS



***To our master and jury president Professor EL HATTAOUI Mustapha
Professor of cardiology***

You granted us a great honor by agreeing to preside over the jury of our thesis. I admire the simplicity and the ease of your approach and the extent of your knowledge. Please accept the expression of my deep gratitude and respect.

This work is an opportunity to our consideration and deep admiration for all of your scientific and human qualities.

***To our master and thesis director Professor BOUMZEBRA Drissi
Professor of cardiovascular surgery***

You have done me a great honor by agreeing to entrust me with this work. I have nothing but high esteem and admiration for your competence, your seriousness, your dynamism and your kindness. I would always be grateful for your availability and your patience during this work preparation despite your busy schedule and professional obligations. Mere words cannot express my deep gratitude and admiration. Please accept my sincere acknowledgments and the expression of my great respect.

***To our master and thesis judge Professor EL HAOUATI Rachid
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Thank you for honoring us with your presence and your interest in our thesis topic. Thank you for your valuable participation in the development of this work. Allow me to express my admiration for your human and professional qualities. Please accept the expression of my high esteem, consideration and deep respect.

***To our master and thesis judge Professor EL KARIMI Saloua
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It is a great honor for us that you have agreed to be a member of this honorable jury. Your professional skills and your human qualities have been always an example to me. Please find here the expression of my respect and admiration.

***To our master and thesis judge Professor BEN DRISS Laila
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You have honored us with great sympathy when accepting to sit among our thesis jury. Please find here the expression of my profound respect and acknowledgments.

To all the cardiovascular surgery department team

To Professor ZOUIZRA Zahira

I cannot express in words how thankful I am for your support, your guidance and your kindness. This work couldn't have possibly been accomplished without your help.

To Siham, Amal, Sanaa, Loubna and Khadija

For making my time in the department's archives as smooth as can be. For always being there when needed without ever complaining despite my constant annoyance.

Dr El Mardouli, Dr El Alaoui, Dr Issaka and Dr Kane

Thank you for your kindness and support. I owe you a lot for your understanding and help in completing this work

The amazing nursing team: Sara Nabil, Hanane Hajji, Fatimzehra Larhbira, Naima Ihend, Fatimzehra Samouni, Laila Bentajri, Touria Berjoul, Hamza Znati, Ayoub El Ouadi, Mohamed Choukri, Oussama Rouhy, Hidaya El Adrani, Mohamed El Alami, Mohamed Choukrat, Houcine Hamni, Khadija Ismaoune, Hanane Outama, Hayat Tadili, Meryem El Idrissi, Youness El Aouzi, Abdellatif Lagssir, Mohamed Belkayed, Karim Elbyad

Your hard work and dedication are remarkable. Thank you for making working with you so enjoyable

To Dr El Moktadir, Dr Boukaidi, Dr Fahd, Dr Gael

To all the cardiovascular medicine department team

Thank you for your undeniable efforts and contribution

And to all those who, in a way or another, contributed to the elaboration of this work. Find here the expression of my endless gratitude.



ABBREVIATIONS



Abbreviations list

AF	: Atrial fibrillation
AV	: Aortic valve
AVR	: Aortic valve replacement
CPB	: Cardiopulmonary bypass
DVR	: Double valve replacement
ICU	: Intensive care unit
LV	: Left ventricle
LVEF	: Left ventricle ejection fraction
MV	: Mitral valve
MVR	: Mitral valve replacement
NYHA	: New York Heart Association
PAH	: Pulmonary artery hypertension
PASP	: Pulmonary artery systolic pressure
RV	: Right ventricle
TR	: Tricuspid regurgitation
TV	: Tricuspid valve



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INTRODUCTION

Right-sided cardiac valvular disease has traditionally been considered less clinically important than mitral or aortic valve pathology. The tricuspid valve was relatively «forgotten» over the years, even though severe tricuspid regurgitation can cause relevant functional impairment. Lately, the once “forgotten valve” has garnered increasing attention (1).

With the advent of surgical options for rheumatic heart disease the general life expectancy of the patients has increased. This has resulted in increased diagnosis of tricuspid regurgitation (2).

There is no debate regarding the ‘malignant nature’ of this entity as is evidenced by the poor long-term outcomes associated with surgical management of left-sided heart disease without addressing the tricuspid valve.

Pathologic tricuspid regurgitation is divided into primary (organic) and secondary (functional). The most common mechanism is functional (80% to 90% of tricuspid regurgitation) caused by annular dilation, secondary to left-sided valvular disease and pulmonary hypertension (3-6). Even though structural pathology of the valve is not included in the definition of functional tricuspid regurgitation, leaflet tethering involvement in the pathogenesis of functional tricuspid regurgitation has been recently cited (7).

Outcome of isolated tricuspid valve surgery is poor because right ventricular dysfunction has already established. Therefore, tricuspid valve repair is often concomitant to left-heart valve surgery(1,7).

Recent years have seen a surge in surgery for functional tricuspid regurgitation and numerous techniques are available to correct tricuspid regurgitation. The decision whether or not to address the tricuspid valve at the time of primary surgery had been a debatable topic for

some time both in terms of timing, indication, surgical techniques and results. Whereas the short-term outcome of surgical therapy is satisfactory, the majority of surgical techniques are limited in the mid- and long term by unacceptably high rates of residual and/or recurrent TR (8-10).

There is no discussion whether to treat the tricuspid valve in severe TR, but guidelines provided less firm recommendations for patients with moderate tricuspid regurgitation (10).

Another controversy is the technique of repair. In 1970s, Dr. Norberto De Vega introduced into clinical practice his tricuspid valve suture annuloplasty technique. Because of its simplicity and efficacy, the procedure became widespread (11).

The De Vega annuloplasty has been modified many times and widely used with accepted results by many surgeons. It is a simple and moreover economic procedure. The longevity of the outcome of this technique highly questioned the risk off its failure with subsequent development of significant tricuspid regurgitation requiring a redo surgery. This has led many surgeons to adopt the use of annuloplasty rings (12,13).

While transcatheter therapies for aortic, mitral and pulmonary valve disease are well established, interventional treatment of tricuspid valve disease is still in its early stages of development. The implementation of less invasive therapies in this field is therefore of major clinical interest. Percutaneous technologies might become an alternative option to treat selected tricuspid regurgitation patients with high surgical risk (14).

However, in a developing country such as ours, giving up an efficient and low-cost method like the De Vega annuloplasty for a method with an additional cost is not a luxury. The De Vega annuloplasty is still a gold standard technique for tricuspid valve repair.

Up-to-date ESC/EACTS guidelines for the management of valvular heart disease have recently been published in August 2017. The fact that once again new guidelines were published shows that this is interesting and timely topic.

Rheumatic fever is still a major health problem in our country with a growing number of young patients with rheumatic heart disease to manage. This unfortunate situation makes our health professionals (cardiologists and cardiac surgeons particularly) better placed to make guidelines based on results from rigorous randomized controlled trials on large cohorts.

PATIENTS

AND

METHODS

I. Study design:

1. Hypothesis:

The De Vega annuloplasty is still a relevant tricuspid valve repair method with satisfactory results.

2. Aims:

- Assessment of the short and mid-term outcomes of the De Vega annuloplasty in patients undergoing valve surgery in the context of rheumatic heart disease.
- Assessment of risk factor of failure of the De Vega annuloplasty

3. Study type:

A retrospective, single center, observational study

4. Setting:

Cardiovascular surgery department, Mohammed VI teaching Hospital, a tertiary referral center.

The preoperative echocardiographic assessment and postoperative follow-up was mainly realized by the cardiovascular medicine department of Mohammed VI teaching Hospital.

II. Methods:

We retrospectively analyzed the perioperative and follow-up data of patients who underwent surgery for rheumatic heart disease with a De Vega annuloplasty in our institute from February 2007 to June 2017.

1. Inclusion criteria:

- Patients with tricuspid regurgitation who underwent concomitant De Vega annuloplasty and another valve repair or replacement for rheumatic heart disease
- Patients with organic and functional tricuspid regurgitation were included as long as it was treated with the De Vega annuloplasty
- Patients who underwent other repair procedures on the tricuspid valve, besides the De Vega annuloplasty, were also included.
- Patients who underwent additional concomitant cardiac surgical procedures were also included.

2. Exclusion criteria:

- Patients who underwent the De Vega annuloplasty for non-rheumatic tricuspid regurgitation
- Patients with incomplete or missing medical records

3. Subgroups:

Because of the high diversity in our study group, we identified some subgroups:

- Patients who underwent double valvular procedures : mitral and tricuspid
- Patients who underwent triple valvular procedures : mitral, aortic and tricuspid
- Patients who were put postoperatively on oral Sildenafil

4. Data collection and analyze:

4.1. Data collection

- The preoperative data (demographic, history, clinical examination and paraclinical exams) was collected through review of medical records in the cardiovascular surgery department archives.
- Bidimensional and color doppler echocardiography were used to assess tricuspid valve disease, other valvular abnormalities and their impact on cardiac cavities.
- The outcomes were analyzed by evaluating data from outpatient follow-up visits. In addition, telephonic follow-up was conducted in order to account for follow-up data

4.2. Data analysis:

- The data were recorded in a questionnaire sheet
- Descriptive statistics were the primary focus of this analysis. Numeric data were presented by the following descriptive statistics as most appropriate: mean, median, standard deviation, range. Categorical data were presented by frequency tables: count and percentage.
- Descriptive data were analyzed using Microsoft Excel 2016.
- SPSS version 21 was used for multivariable analysis. A p value <0.05 was considered as statistically significant.



RESULTS

I. Synthetic/descriptive study:

A total of 595 patients underwent concomitant De Vega annuloplasty and another cardiac procedure for rheumatic valvular heart disease from January 2007 to June 2017.

Only 356 patients were included in our case series. The remaining 239 were excluded due to missing or incomplete medical records.

1. Demographic data:

1.1. Age:

The mean age in our case series was 40.6 with a range from 10 years-old to 73 years-old and a standard deviation of 12.14.

The age of 73,6 % of our patients ranged from 20 to 50 years-old.

The detailed distribution of our patients by their age is presented in table I.

Table I: Age distribution of our patients

AGE GROUP IN YEARS	FREQUENCY	PERCENTAGE
< 20	14	3.90%
20 – 30	56	15.70%
30 – 40	96	27%
40 – 50	110	30.90%
50 – 60	53	14.90%
> 60	27	7.60%
TOTAL	356	100.00%

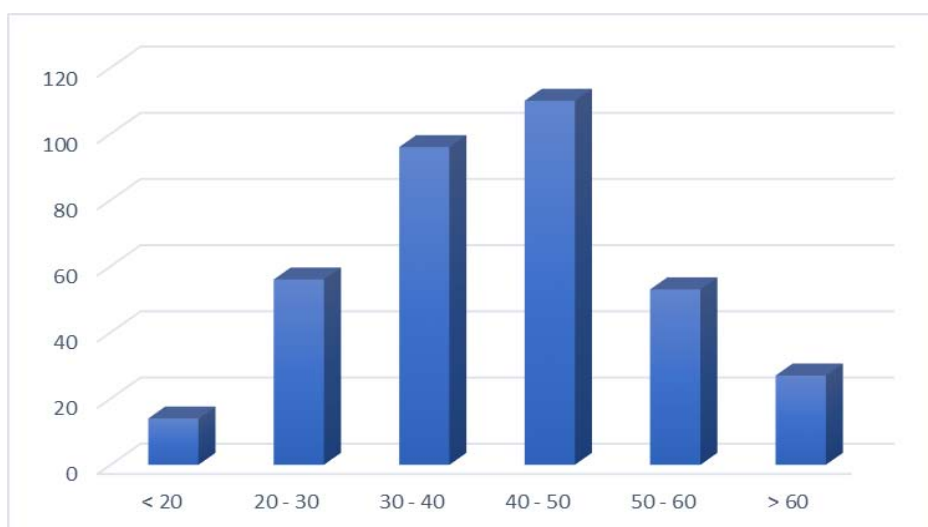


Figure 1: Age distribution of our patients

1.2. Sex:

Out of the total of 356 patients in our study, 66.3% were female and 33.7% were male (figure 3).

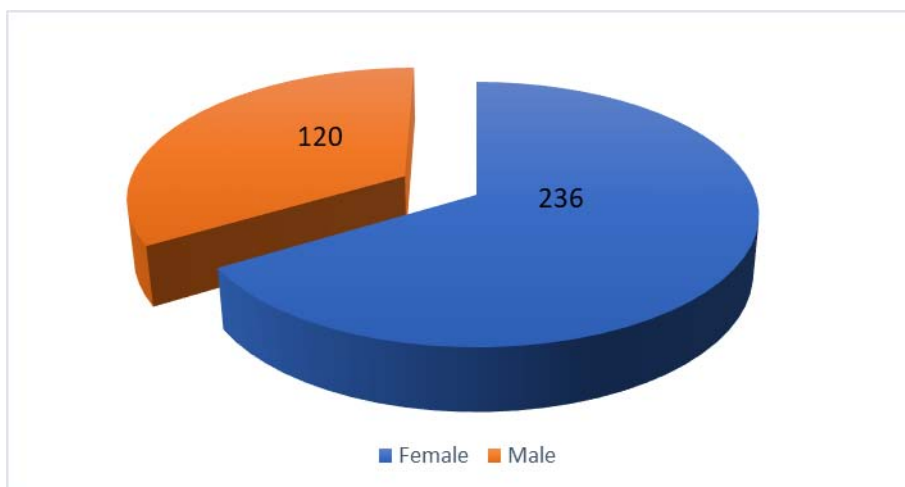


Figure 2: Sex distribution of our patients

Table II: Age and sex distribution of our patients

	MEAN AGE	MINIMUM	MAXIMUM
FEMALES	41	12	73
MALES	39	10	68

1.3. Medical history:

A history of recurrent tonsillitis was present in 152 (42.7%) patients and 116 (32.6%) were previously treated for acute rheumatic fever. A total of 63 (17.7%) of our patients had at least one previous episode of heart failure.

The extent of our patients' medical history is presented in Table 3.

Table III: Medical history in our case series

	NUMBER OF PATIENTS	PERCENTAGE
RECURRENT TONSILLITIS	152	42.7%
ACUTE RHEUMATIC FEVER	116	32.6%
HEART FAILURE	63	17.7%
ISCHEMIC STROKE	46	12.9%
CHRONIC SMOKING	36	10.1%
INFECTIVE ENDOCARDITIS	17	4.8%
HIGH BLOOD PRESSURE	14	3.9%
DIABETES MELLITUS	13	3.6%
ACUTE LIMB ISCHEMIA	6	1.7%
DYSLIPIDEMIA	4	1.1%
CORONARY HEART DISEASE	2	0.6%

1.4. Surgical history:

A history of mitral valve surgery was present in 24 (6.7%) patients (Table 4). None of our patient had a history of tricuspid valve surgery.

Table IV: History of cardiac surgery in our case series

SURGICAL PROCEDURE	NUMBER OF PATIENTS	PERCENTAGE
CLOSED MITRAL COMMISSUROTOMY	16	4.5%
OPEN MITRAL COMMISSUROTOMY	5	1.4%
MITRAL VALVE ANNULOPLASTY	3	0.8%
TOTAL	24	6.7%

2. Clinical data:

2.1. NYHA status:

The majority of our patients (82.9%) were in New York Heart Association (NYHA) functional classes III and IV. These results reflect the state of advanced heart failure in which our patients are usually operated on. Only 17.1% of our patients were in NYHA functional class II and none of them was in class I (figure 4).

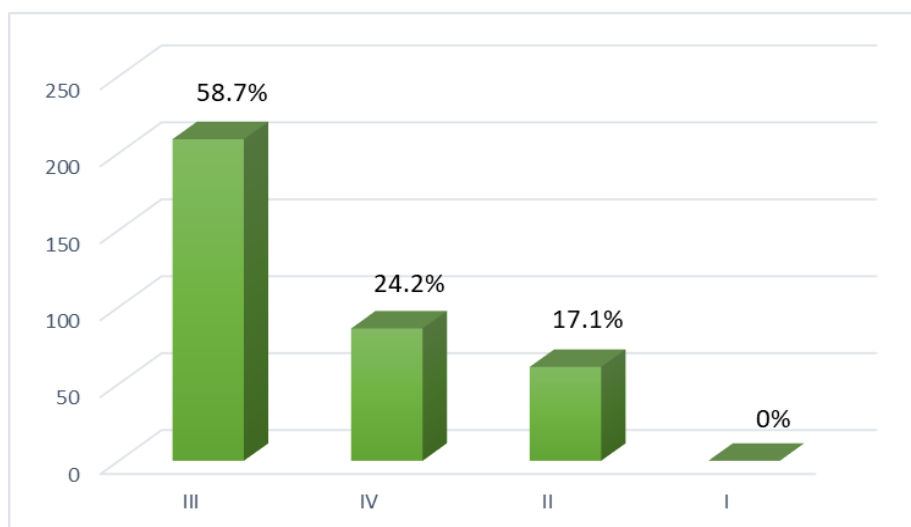


Figure 3: Distribution of our patients by their NYHA status

2.2. Congestive heart failure signs:

Seventy-seven (21.6%) patients presented with congestive heart failure signs, ranging from jugular venous distension (Kussmaul sign) to edema, ascites and orthopnea (Table 5).

Table V: Distribution of heart failure signs in our patients

CONGESTIVE HEART FAILURE SIGNS	NUMBER OF PATIENTS	PERCENTAGE
JUGULAR VEIN DISTENSION	77	21.60%
HEPATOJUGULAR REFLUX	62	17.40%
EDEMA	45	12.60%
RIGHT UPPER QUADRANT PAIN	31	8.70%
HEPATOMEGALY	17	4.70%
ASCITES	12	3.40%
ORTHOPNEA	12	3.40%

2.3. Heart murmurs:

A diastolic murmur of mitral stenosis was the most commonly found heart murmur. It was present in 67.4% of patients.

Cardiac auscultation found a systolic murmur of mitral regurgitation in 45.2%, a diastolic murmur of aortic regurgitation in 22.2% and a systolic murmur of aortic stenosis in 20.2%.

A systolic murmur of tricuspid regurgitation was present in only 29.2% of our patients and only 1.4% had a diastolic murmur of tricuspid stenosis.

Table VI: Distribution of cardiac auscultation findings in our case series

HEART MURMUR	NUMBER OF PATIENTS	PERCENTAGE
MITRAL STENOSIS	240	67.4%
MITRAL REGURGITATION	161	45.2%
AORTIC REGURGITATION	79	22.2%
AORTIC STENOSIS	72	20.2%
TRICUSPID REGURGITATION	104	29.2%
TRICUSPID STENOSIS	5	1.4%

2.4. Other clinical signs:

Other signs found in our case series included heart palpitations, angina, syncope and irregular heart rhythm (Table 7).

Table VII: Distribution of other clinical signs in our case series

OTHER SIGNS	NUMBER OF PATIENTS	PERCENTAGE
HEART PALPITATIONS	192	53.9%
ANGINA	22	6.1%
SYNCOPE	12	3.3%
IRREGULAR HEART RHYTHM	265	74.4%

2.5. Preoperative medication:

The majority of our patients (92.1%) were on a preoperative medical therapy (Table 8).

Table VIII: Distribution of our patients according to preoperative medication

PREOPERATIVE MEDICATION	NUMBER OF PATIENTS	PERCENTAGE
DIURETICS	295	82.8%
ANTICOAGULANT	271	76.1%
BETA BLOCKERS	67	18.8%
ACE INHIBITOR	56	15.7%
AMIODARONE	15	4.2%
TOTAL	328	92.1%

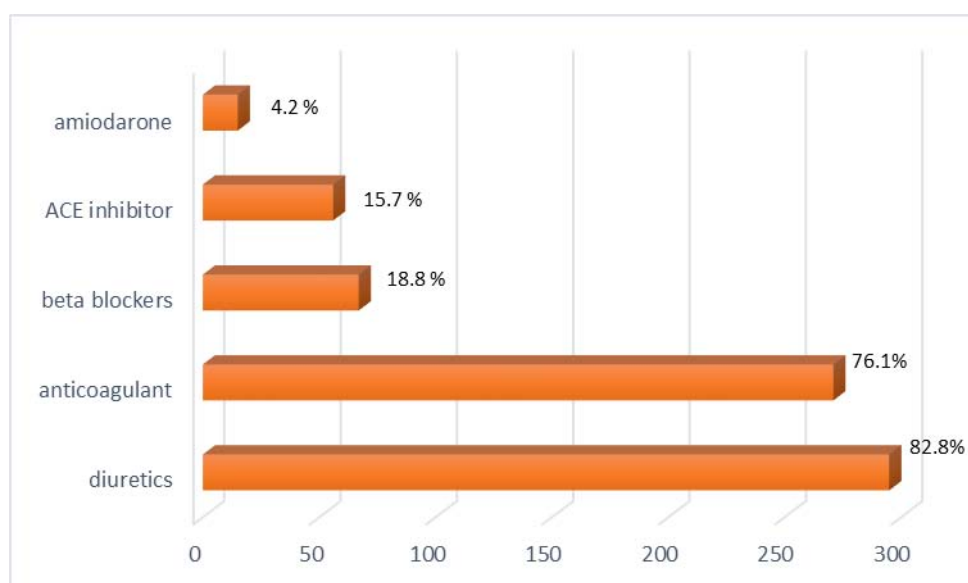


Figure 4: Preoperative medication in our case series

3. Chest X-ray:

Cardiomegaly was present in 312 patients (87.6%). The distribution of our patients according to their preoperative Cardiothoracic Index is presented in table 9.

Table IX: Distribution of patients according to cardiothoracic index

CARDIOTHORACIC INDEX	NUMBER OF PATIENTS	PERCENTAGE
<0.5	44	12.4%
0.5 - 0.7	250	70.2%
>0.7	62	17.4%
TOTAL	356	100.0%

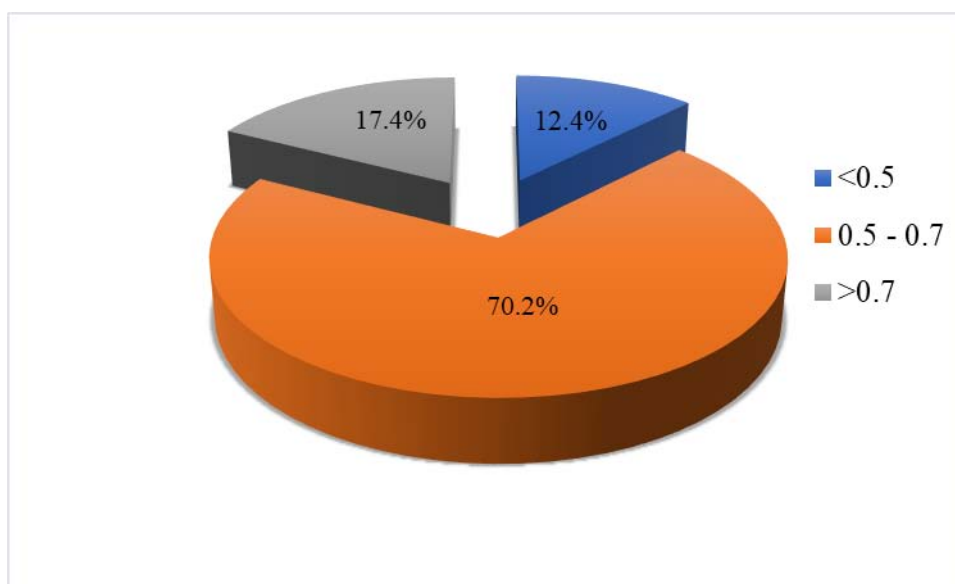


Figure 5: Cardiothoracic index in our case series

4. Electrocardiogram:

4.1. Rhythm abnormalities:

In our case series, 268 (75.3%) patients had atrial fibrillation. Only 75 (21.1%) of our patients had a sinus rhythm (Table 10).

Table X: Distribution of rhythm abnormalities in our patients

RHYTHM ABNORMALITY	NUMBER OF PATIENTS	PERCENTAGE
ATRIAL FIBRILLATION	268	75.3%
ATRIAL FLUTTER	8	2.2%
SUPRAVENTRICULAR EXTRASYSTOLES	5	1.4%

4.2. Conduction abnormalities:

The different conduction abnormalities found in our patients are presented in table 11.

Table XI: Distribution of conduction abnormalities in our patients

CONDUCTION ABNORMALITY	NUMBER OF PATIENTS	PERCENTAGE
RIGHT BUNDLE BRANCH BLOCK	25	7%
LEFT BUNDLE BRANCH BLOCK	13	3.6%
ATRIOVENTRICULAR BLOCK	0	0%

5. Echocardiographic data:

5.1. Tricuspid valve:

a. Leaflets:

The echocardiographic study of tricuspid valve anatomy showed normal leaflets in 268 (75.3%) patients and altered or thickened leaflets with suggesting organic lesions in 88 (24.7%) patients.

b. Tricuspid annulus:

The tricuspid annular diameter was measured in 316 patients. The mean was 37.7 mm +/- 6.8 mm with a minimal diameter of 23 and a maximal diameter of 58 mm (figure 9).

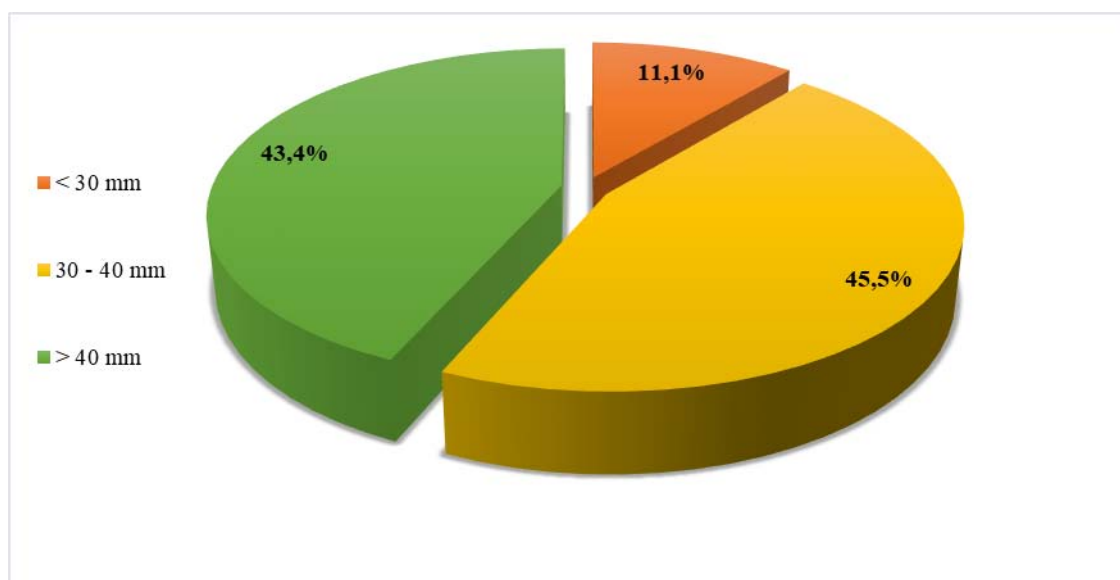


Figure 6: Distribution of patients by tricuspid annular diameter

c. Tricuspid regurgitation:

Preoperative tricuspid regurgitation was found in 343 (96.3%) of our patients. The remaining 13 (3.7%) patients had no preoperative regurgitation and the decision of tricuspid repair was taken peroperatively because of the enlarged tricuspid annulus. This situation can be

explained by the long delay between the echocardiographic assessment and surgery, giving time for tricuspid regurgitation to develop.

A grade 3 tricuspid regurgitation or higher was found in 226 (63.4%) patients.

Table XII: Distribution of our patients by the grade of tricuspid regurgitation

TR GRADE	NUMBER OF PATIENTS	PERCENTAGE
0 (NONE)	13	3.7%
1+ (MILD)	28	7.9%
2+ (MODERATE)	89	25.0%
3+ (MODERATE TO SEVERE)	150	42.1%
4+ (SEVERE)	76	21.3%
TOTAL	356	100.0%

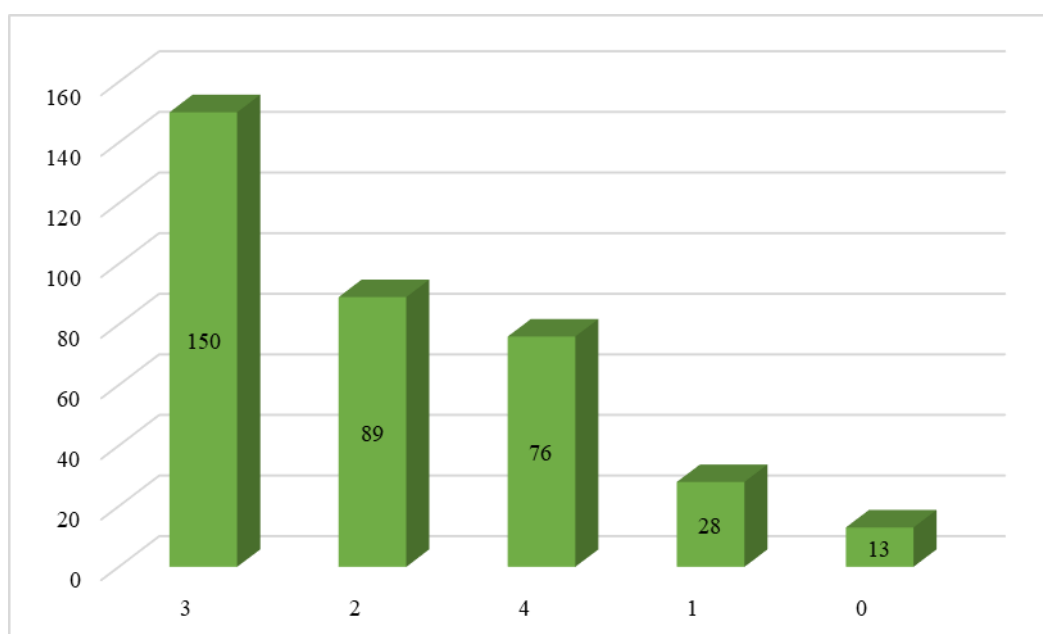


Figure 7: Distribution of our patients by the grade of tricuspid regurgitation

d. Tricuspid stenosis:

Nineteen patients had associated tricuspid stenosis which was mild in 8 cases, moderate in 6 and severe in 5.

e. The mechanism of the tricuspid regurgitation:

We identified three mechanisms of tricuspid regurgitation in our case series as organic, functional and mixed (dilated tricuspid annulus with altered leaflets). The repartition of our patients according to these mechanisms is detailed in table 13.

Table XIII: Tricuspid regurgitation mechanisms in our case series

TR MECHANISM	NUMBER OF PATIENTS	PERCENTAGE
FUNCTIONAL	255	74.3%
ORGANIC	17	5.0%
MIXED	71	20.7%
TOTAL	343	100.00%

5.2. Mitral valve:

All patients included in our case series had a pathological mitral valve in the echocardiographic study.

The majority of patients (53.1%) had mitral valve disease with an association of different degrees of mitral stenosis and regurgitation.

Isolated mitral stenosis interested 100 (28.1%) patients.

Pure mitral regurgitation was found in 67 (18.8%) patients.

Table XIV: Mitral valve involvement in our case series

MITRAL ABNORMALITY	NUMBER OF PATIENTS	PERCENTAGE
NONE	0	0%
MITRAL DISEASE	189	53.1%
PURE MITRAL STENOSIS	100	28.1%
PURE MITRAL REGURGITATION	67	18.8%
TOTAL	356	100%

a. Mitral stenosis:

Mitral stenosis was found in 289 (81.2%) of our patients. The degree of stenosis was graded as mild, moderate or severe. The distribution of our patient by the grade of mitral stenosis is detailed in table 14.

Table XV: Mitral stenosis grading in our patients

GRADE OF MITRAL STENOSIS	NUMBER OF PATIENTS	PERCENTAGE
NONE	67	18.8%
MILD	19	5.3%
MODERATE	126	35.4%
SEVERE	144	40.5%
TOTAL	356	100.0%

b. Mitral regurgitation:

Among the patients included in our case series, 256 had mitral regurgitation. The preoperative assessment of mitral regurgitation was graded from 1 to 4 (from mild to severe).

Table XVI: Mitral regurgitation grading in our patients

GRADE OF MITRAL REGURGITATION	NUMBER OF PATIENTS	PERCENTAGE
NONE	100	28.1%
1+	46	12.9%
2+	96	27.0%
3+	10	2.8%
4+	104	29.2%
TOTAL	356	100.0%

5.3. Aortic valve:

An involvement of the aortic valve was identified in 217 (60.9%) of our patients.

Pure aortic regurgitation was predominant with 132 (37.1%) patients.

Isolated aortic stenosis was found in 7 patients (1.9%) only.

The remaining 78 (21.9%) patients had an aortic valve disease with coexistent aortic regurgitation and stenosis.

Table XVII: Aortic valve involvement in our case series

AORTIC VALVE ABNORMALITY	NUMBER OF PATIENTS	PERCENTAGE
NONE	139	39.1%
ISOLATED AORTIC REGURGITATION	132	37.1%
AORTIC DISEASE	78	21.9%
ISOLATED AORTIC STENOSIS	7	1.9%
TOTAL	356	100.0%

a. Aortic stenosis:

Aortic stenosis was identified in 85 (23.9%) of patients. The distribution of patients by degree of stenosis is detailed in table 16.

Table XVIII: Aortic stenosis grading in our patients

GRADE OF AORTIC STENOSIS	NUMBER OF PATIENTS	PERCENTAGE
NONE	271	76.1%
MILD	22	6.2%
MODERATE	30	8.4%
SEVERE	33	9.3%
TOTAL	356	100.0%

b. Aortic regurgitation:

A total of 210 (59%) patients had aortic regurgitation.

Table XIX: Aortic regurgitation grading in our patients

GRADE OF AORTIC REGURGITATION	NUMBER OF PATIENTS	PERCENTAGE
0	146	41%
1+	58	16.3%
2+	92	25.9%
3+	21	5.9%
4+	39	10.9%
TOTAL	356	100.0%

5.4. Right ventricle:

a. **Right ventricular size:**

Right ventricular dilatation was present in 251 (70.5%) of our patients.

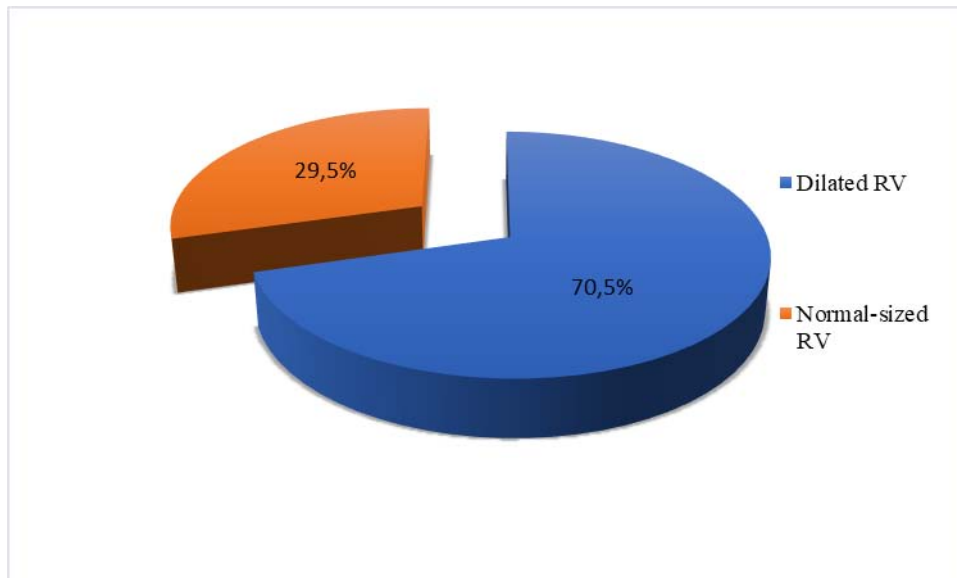


Figure 8: Right ventricular size in our case series

b. **Right ventricular systolic function:**

Right ventricular systolic dysfunction was present in 107 (30%) of our patients.

5.5. Left ventricle:

a. **Left ventricular size:**

Left ventricular dilatation was present in 58 (16.3%) of our patients.

b. **Left ventricular systolic function:**

The mean ejection fraction of the right ventricle was 56.8% +/- 9.1% with a maximum of 78% and a minimum of 30%. The majority of our patients (65.7%) had a normal left ventricular systolic function. None of our patients was in severe dysfunction of the left ventricle.

Table XX: Distribution of our patients by degree of LV dysfunction

LV SYSTOLIC EJECTION FRACTION	NUMBER OF PATIENTS	PERCENTAGE
≥ 55%	234	65.7%
45% – 54% (MILD LV DYSFUNCTION)	89	25.0%
30 – 44% (MODERATE LV DYSFUNCTION)	33	9.3%
< 30% (SEVERE LV DYSFUNCTION)	0	0.0%
TOTAL	356	100.0%

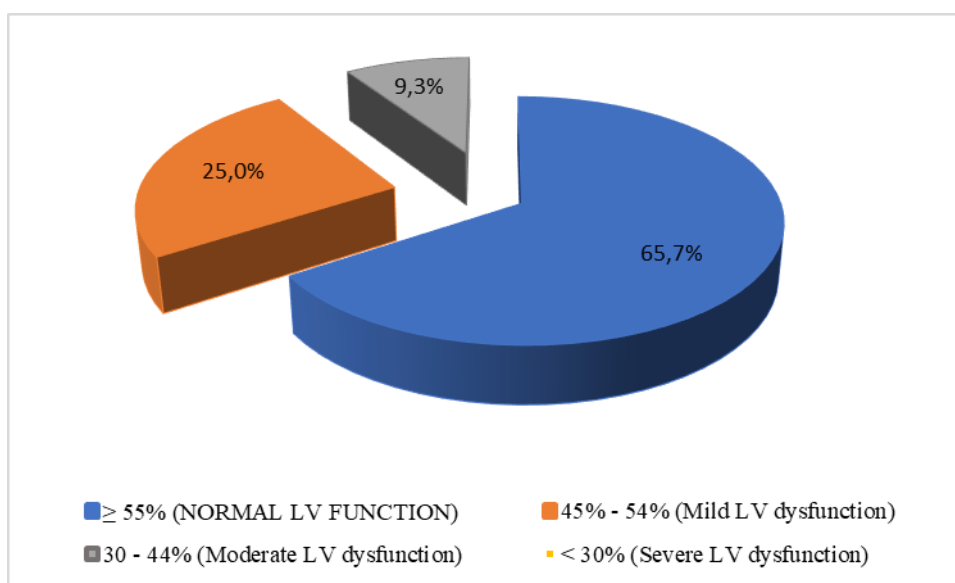


Figure 9: Left ventricular systolic function in our case series

5.6. Pulmonary artery systolic pressure:

The mean pulmonary artery systolic pressure was 61.7 +/- 23.9 mmHg with a maximum of 146 mmHg and a minimum of 20 mmHg.

Pulmonary hypertension was found in 347 (97.5%) patients.

Severe pulmonary hypertension was present in 203 (57%) of our patients.

A normal pulmonary artery systolic pressure was found in only 9 (2.5%) patients.

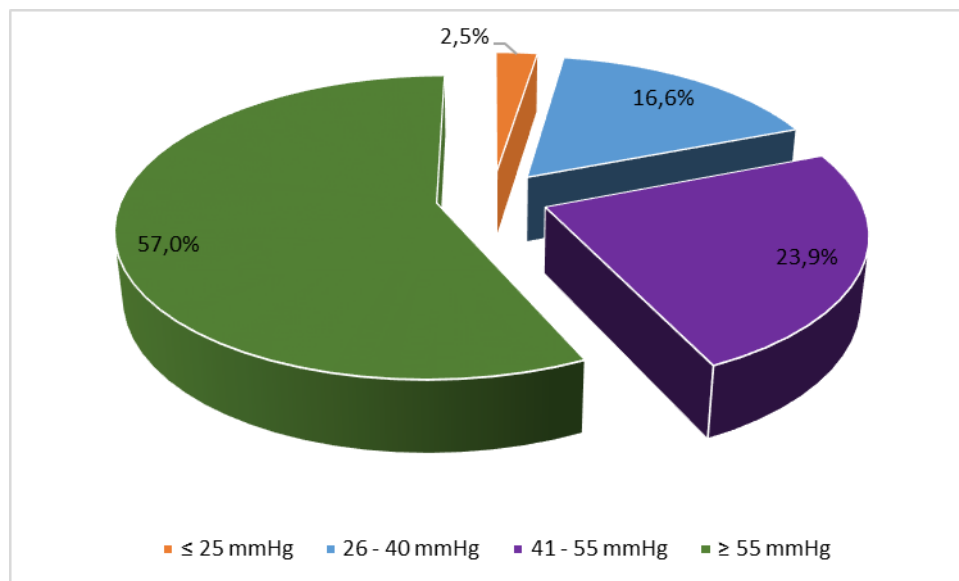


Figure 10: Pulmonary artery systolic pressure in our case series

5.7. Left atrium:

The left atrium was dilated in 193 (54.2%) of patients. An atrial thrombus was found in 23 (6.4%) patients.

5.8. Etiology of valvular disease:

The presumed etiology of valvular lesions was identified to be rheumatic in all our patients on echocardiographic study, confirmed later by surgical findings. Although, additional infectious endocarditis was present in 9 (2.5%) patients.

6. Perioperative data:

6.1. Surgical approach:

A median sternotomy was the surgical approach in the majority of our patients (352 which accounts for 98.9% of our patients). Only four surgeries were done mini invasively.

6.2. Cardiopulmonary bypass:

All the surgeries in our case series were done on cardiopulmonary bypass.



Figure 11: Cardiopulmonary bypass machine/ Cardiovascular Surgery Department – University Hospital Mohammed VI, Marrakech

Blood cardioplegia was used in 330 (92.7%) of patients while crystalloid cardioplegia was used in only 26 (7.3%) of patients. Crystalloid cardioplegia was used only in the beginning of our experience (before 2010). The surgeries were carried out in slight hypothermia in the majority of cases (88.2%).

The mean cardiopulmonary bypass time was 130 min \pm 50.4 min with extremes ranging from a minimum of 42 min to a maximum of 412 min.

The mean aortic cross-clamp time was 92.7 min \pm 38.4 min with a maximum of 28 min and a maximum of 300 min.

Weaning from cardiopulmonary bypass was possible without inotropic support in 58 (16.3%) patients. In the rest of cases, an inotropic (or more) drug was introduced to help coming off the bypass as detailed in table 21.

Table XXI: Use of inotropic agents when coming off cardiopulmonary bypass in our patients

INOTROPIC DRUGS	NUMBER OF PATIENTS	PERCENTAGE
DOBUTAMINE	298	83.7%
DOBUTAMINE + NOREPINEPHRINE	138	38.8%
DOBUTAMINE + NOREPINEPHRINE + EPINEPHRINE	29	8.1%

6.3. Surgical exploration:

a. Tricuspid valve:

The tricuspid annulus was dilated in all patients.

An associated organic lesion of the tricuspid valve was found in 102 (28.6%) patients. Leaflets thickening was found in all these patients. The other associated lesions were commissural fusion or abnormal chordae tendinea.

Table XXII: Profile of peroperative tricuspid valve organic lesions found in our patients

ORGANIC LESION	NUMBER OF PATIENTS	PERCENTAGE
LEAFLETS THICKENING	102	28.6%
COMMISSURAL FUSION	26	7.3%
ABNORMAL CHORDAE	13	3.6%

b. Right heart geometry:

Right ventricular dilatation was reported in 161 (45.2%) patients. Right atrial dilatation was reported in 155 (43.5%) patients.

c. Pericardial adhesions:

Pericardial adhesions were found in only 17 (4.7%) cases.

6.4. Surgical procedures:

a. Tricuspid valve:

➤ The De Vega annuloplasty:

De Vega annuloplasty was performed in all patients.

▪ Technique:

In our department, we used the classic technique of De Vega annuloplasty in the beginning of our experience, and then a modified technique was introduced.

In the modified technique, the suture is placed over a pledget from the anterior commissure to the posterior commissure using a 2.0 Ethibond (Contrary to the segmental technique, a single suture thread is used). The suture is doubled and the tread tips are met in the center of the annulus and a pledget is placed. This technique permits a more symmetrical reduction of the tricuspid annulus.

The tightening is controlled over two fingers of the surgeon.

The result is controlled with a saline test.

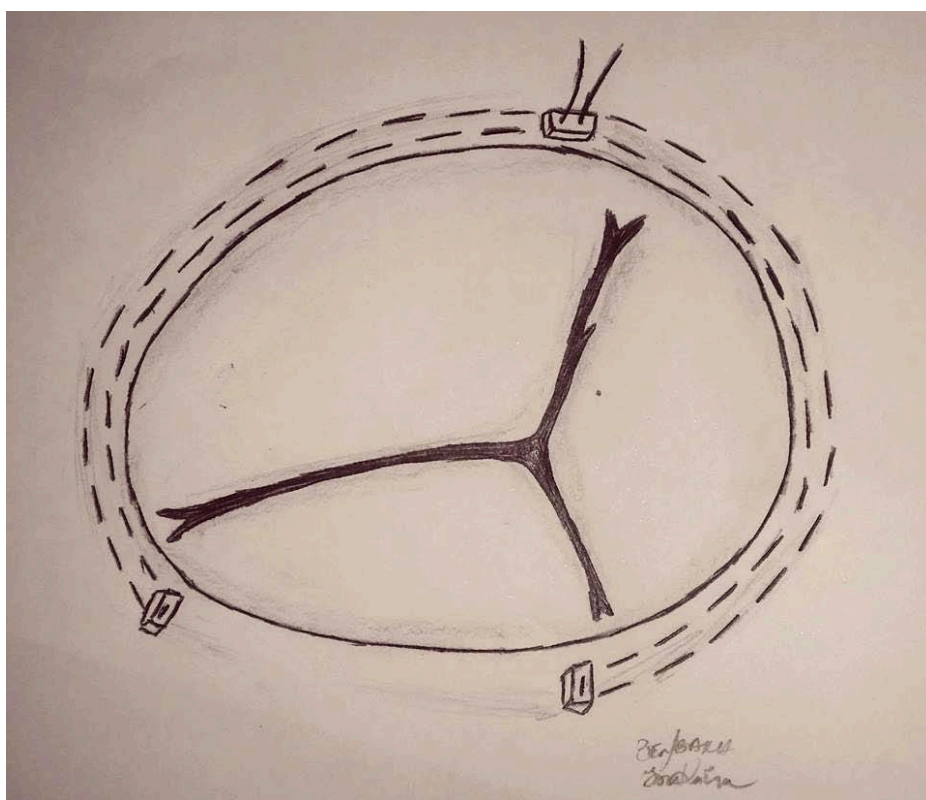


Figure 12: Schematic representation of our modified De Vega annuloplasty technique used in our department

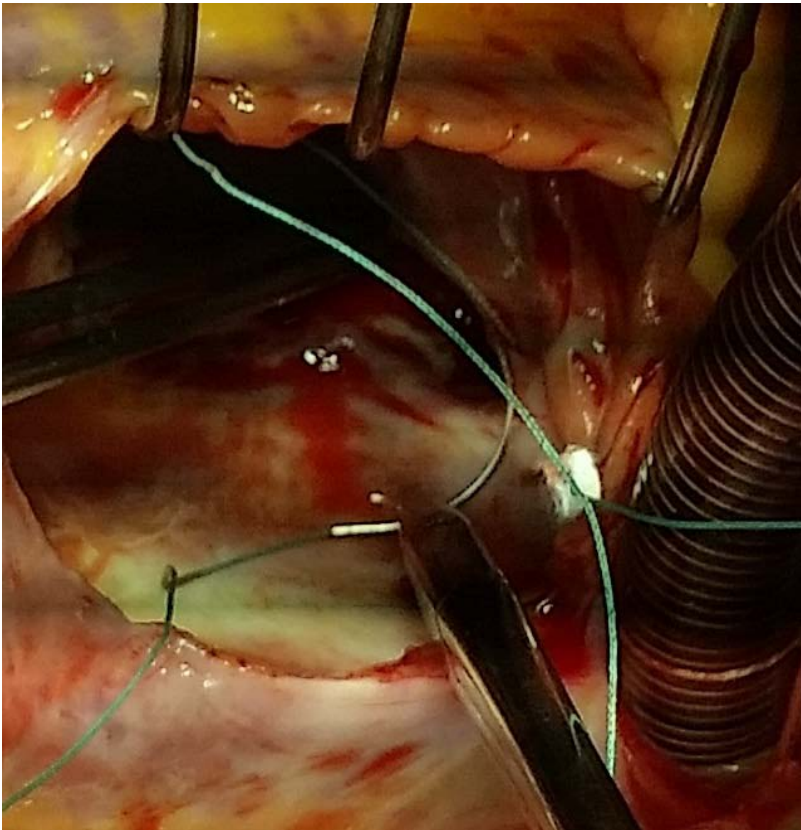


Figure 13: Placement of the first pledget

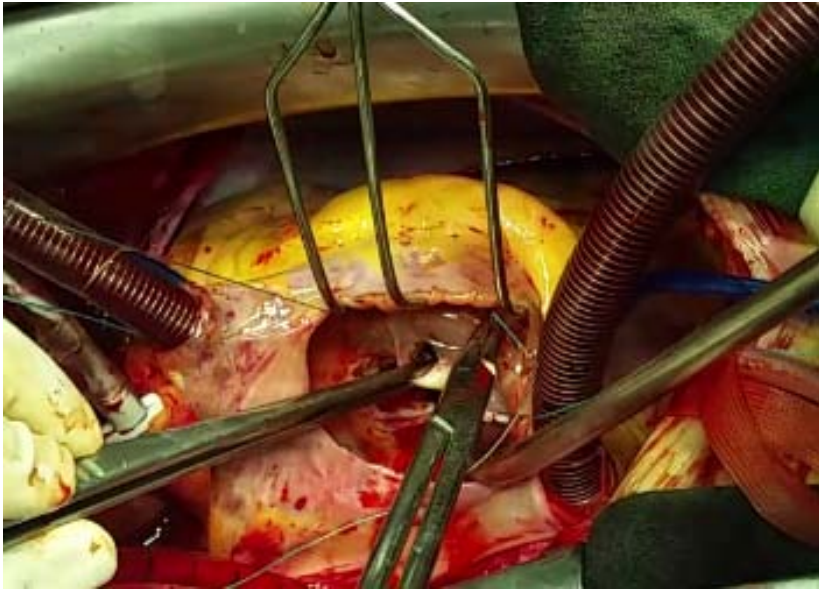


Figure 14: Placement of the first line of sutures

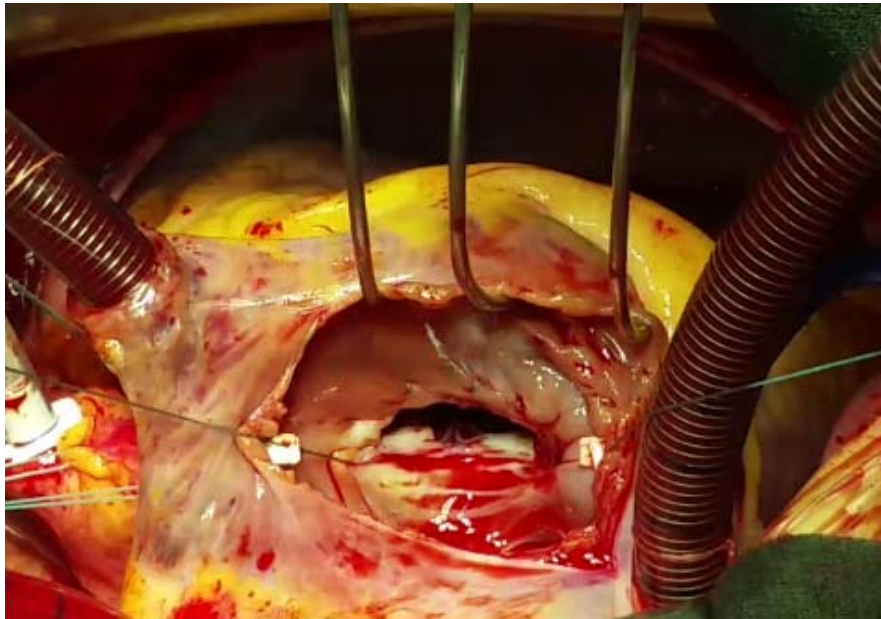


Figure 15: Placement of the second pledget

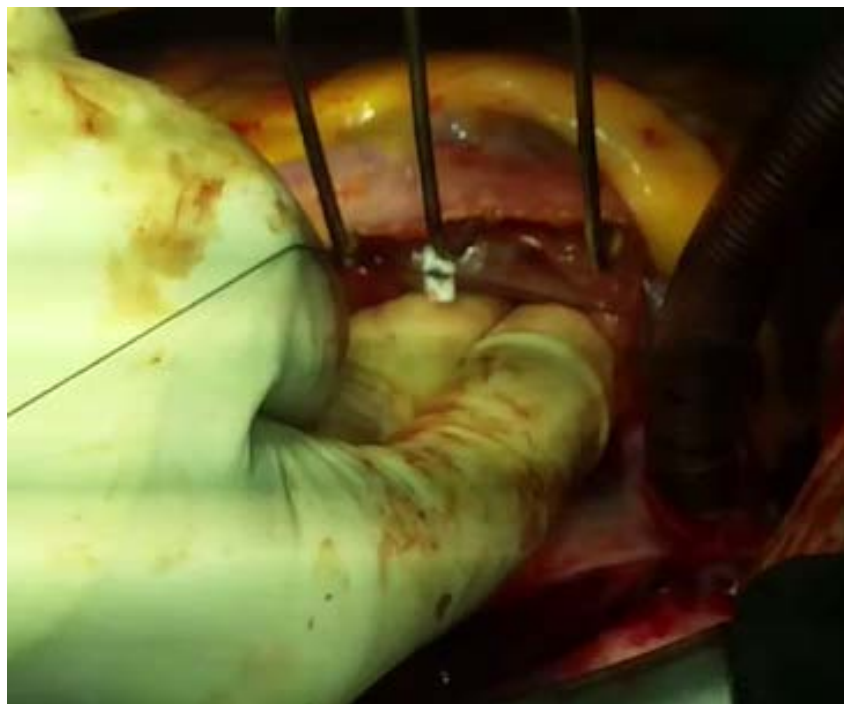


Figure 16: Placement of the third pledget where the sutures are met and tightened over two fingers

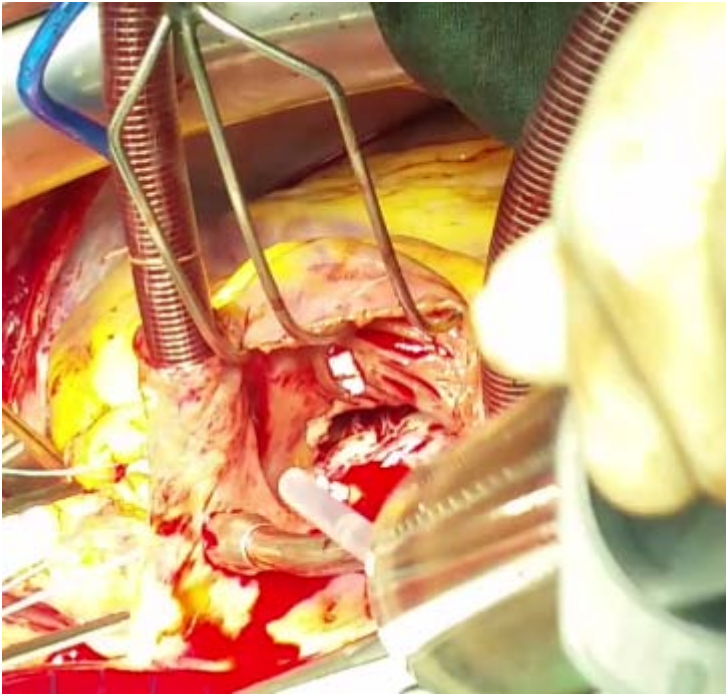


Figure 17: saline test

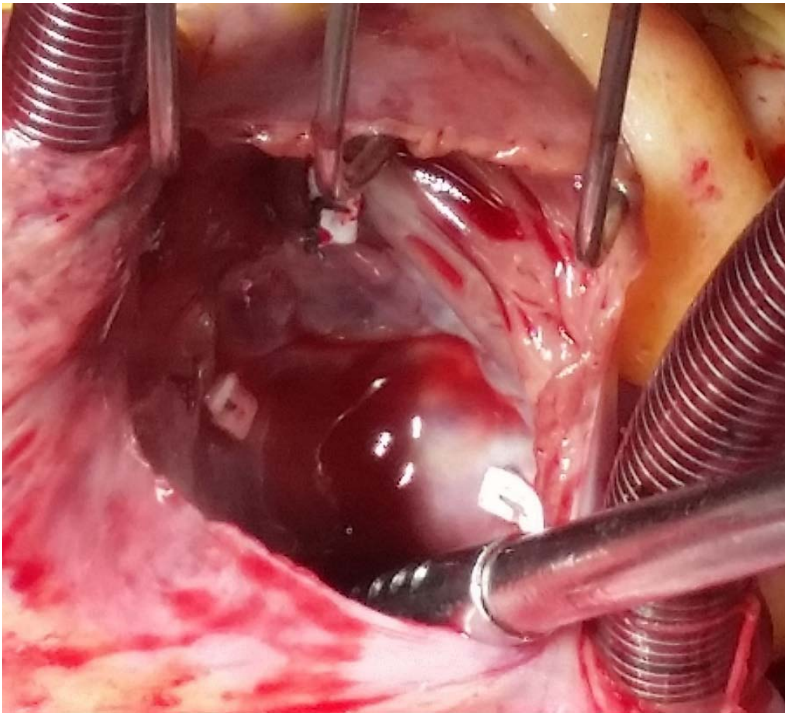


Figure 18: Leaflet coaptation after saline test

➤ **Concomitant procedures:**

When the saline test is not satisfactory, an additional tricuspid repair is performed. This was necessary in 61 (17.1%) patients.

Eliminating leaflet tissue excess (secondary to leaflet tethering) is one of the most frequently performed procedures in our institution. Unfortunately, we don't have the exact number of patients that needed this procedure in our case series as it was rarely mentioned on surgical reports.

Table XXIII: Distribution of concomitant tricuspid valve procedures

TRICUSPID PROCEDURE	NUMBER OF PATIENTS	PERCENTAGE
COMMISSUROTOMY	47	13.2%
KAY ANNULOPLASTY	14	3.9%
CHORDAE SHORTENING BY PLICATION	4	1.1%

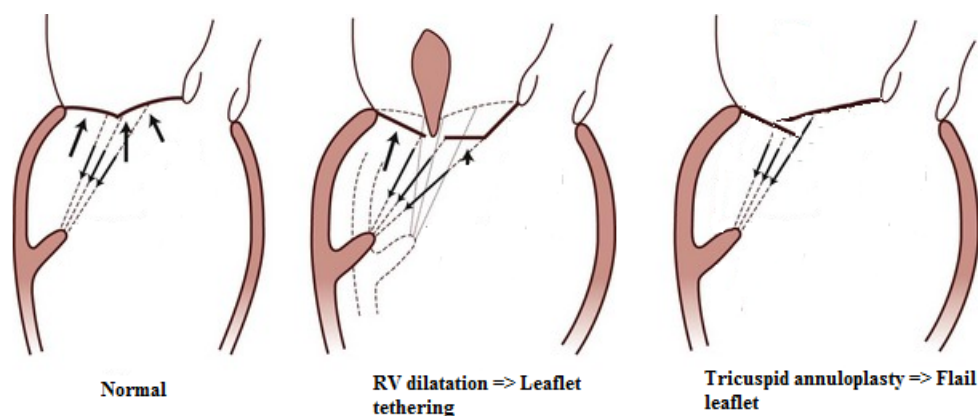


Figure 19: Schematic representation of leaflet tissue excess (flail leaflet)

➤ **Intraoperative assessment of tricuspid repair result:**

A saline injection leak test was used to assess leaflet coaptation after tricuspid valve repair. The test was satisfactory in all patients.

The saline test is considered satisfactory when the right ventricle remains distended for a few seconds after filling it with only a small central leak of the tricuspid valve and with a clear coaptation of the leaflets.

A residual central leak after tricuspid valve repair was noted in 31 (8.7%) patients.

We usually respect some degree of tricuspid regurgitation, especially in patient with severe pulmonary hypertension with right ventricular dysfunction, to relieve the dysfunctional ventricle.

b. Other valve procedures:

Major valve procedures in our case series interested left sided valves. An isolated mitral valve replacement was the most common procedure interesting 246 (69.1%) patients. There was 94 (26.4%) double valve replacements (mitral and aortic valves), 4 aortic valve replacements associated with a mitral valve repair, 6 mitral valve replacements with aortic valve repair and 6 isolated mitral repairs.

Table XXIV: Distribution of concomitant left-heart procedures

LEFT-HEART VALVE PROCEDURE	NUMBER OF PATIENTS	PERCENTAGE
MITRAL VALVE SURGERY	252	70.8%
AORTIC VALVE SURGERY	0	0%
COMBINED MITRAL AND AORTIC SURGERIES	104	29.2%

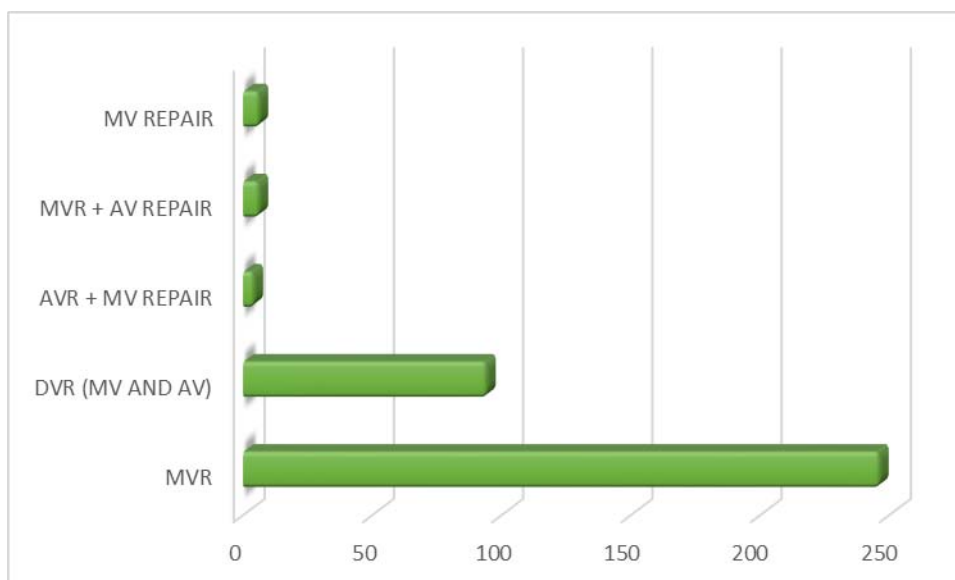


Figure 20: Distribution of left-heart valve procedures in our case series

There was a total of 444 valve replacements in our case series. A mechanical prosthesis was used in 440 patients. A bioprosthesis was used in only 4 patients.

Table XXV: Types of mechanical valve used in our case series

TYPE OF MECHANICAL PROSTHESIS	NUMBER	PERCENTAGE
ONYX	211	47.90%
SORIN	138	31.40%
SJM	71	16.10%
ATS	20	4.60%
TOTAL	440	100.00%

Mitral valve repair procedures included:

- Ring annuloplasty: 6 patients
- Kay annuloplasty: 4 patients
- Sub commissural annuloplasty: 1 patient
- Chordae shortening: 1 patient
- Surgical excision of a vegetation: 1 patient

Aortic valve repair procedures included:

- Sub commissural annuloplasty: 6
- Surgical excision of a vegetation: 2
- Leaflet plication: 1
- Chordae Shortening: 1

7. Critical care data:

7.1. Length of stay in the ICU:

The mean ICU stay duration in our case series was 4.2 ± 2.4 days with a maximum of 28 days and a minimum of 3 days.

7.2. Duration of intubation:

The average duration of respiratory assistance in our case series was 7.6 ± 15 hours with a maximum of 288 (accounting for 12 days). Only 13 patients (3.6%) were extubated after 24 hours.

Reintubation was necessary in 14 (3.9%) patients.

Table XXVI: Estimated causes for reintubation in our case series

CAUSE OF REINTUBATION	NUMBER OF PATIENTS	PERCENTAGE
ACUTE RESPIRATORY FAILURE	7	2%
NEUROLOGICAL IMPAIRMENT	4	1.1%
MULTIORGAN FAILURE	3	0.8%

7.3. Postoperative medication:

a. Antibiotic prophylaxis:

All patients were put postoperatively on an antibiotic prophylaxis using a first-generation cephalosporin (Cefalotin). The antibiotic is stopped after removing the chest drainage.

b. Anticoagulation:

Postoperative anticoagulation was prescribed in all patients using Heparin Sodium and Warfarin. The later was starter in the second postoperative day.

c. Inotropic agents:

Inotropic support was necessary in 304 (85.4%) patients.

Dobutamine was used in all these patients with a minimum duration of 3 hours and a maximum duration of 288 hours (12 days).

d. Antiarrhythmic agents:

The use of an antiarrhythmic drug during the ICU course was necessary in 170 (47.7%) of patients.

Table XXVII: Types of antiarrhythmic drugs used in our case series

ANTIARRHYTHMIC AGENT	NUMBER OF PATIENTS	PERCENTAGE
AMIODARONE	87	24.4%
DIGOXIN	40	11.2%
AMIODARONE + DIGOXIN	19	5.3%
LIDOCAINE	16	4.5%
BISOPROLOL	8	2.3%
TOTAL	170	47.7%

e. Other medications:

Diuretics (Furosemide) were used in all patients in immediate postoperative course.

Sildenafil was used in 61 (17.1%) of our patients.

8. Immediate and short-term surgical results: (<3 months)

8.1. Mortality:

Early postoperative death occurred in 4.8% (n=17) of patients in our case series.

Table XXVIII: Causes of early postoperative deaths in our case series

CAUSE OF DEATH	NUMBER OF PATIENTS
LOW CARDIAC OUTPUT SYNDROME	6
CARDIAC TAMPONADE	3
MULTIPLE ORGAN FAILURE	3
SEPSIS	3
REFRACTORY HYPOVOLEMIC SHOCK	1
UNEXPLAINED CARDIAC ARREST	1

8.2. Complications:

During the ICU stay, 23.3% (n=79) of surviving patients presented various complications.

a. Cardiac complications:

a.1. Arrhythmias:

Table XXIX: Postoperative arrhythmias in our case series

ARRHYTHMIA	NUMBER OF PATIENTS	PERCENTAGE
ATRIAL FIBRILLATION	198	58.4%
ATRIAL TACHYCARDIA	62	17.4%
SINUS TACHYCARDIA	47	13.8%
SINUS BRADYCARDIA	8	2.3%
VENTRICULAR FIBRILLATION	12	3.5%
EXTRASYSTOLES	9	2.7%

a.2. Conduction disturbances:

- Postoperative atrioventricular block occurred in 8 (2.3%) of our patients. It was transient in 5 patients and permanent in 3 patients who consequently underwent pacemaker implantation.
- A bundle branch block was observed in 3 (0.9%) patients.

a.3. Other cardiac complications:

- Hemopericardium: 18 (5.3%) patients.
- Right sided heart failure: 24 (7.1%) patients.
- Post pericardiotomy syndrome: 3 (0.9%) patients.

b. Hemorrhagic complications:

- Postoperative hemorrhage occurred in 58 (17.1%) patients.
- The management of the hemorrhage was:
 - Medical in 49 (84.5%) patients using hemostatic agents and transfusion.
 - Surgical in 9 (15.5%) patients in which medical measures were not sufficient and reoperation was necessary.

c. Pulmonary complications:

Pulmonary complications occurred in 48 (14.1%) patients.

Table XXX: Postoperative pulmonary complications

PULMONARY COMPLICATION	NUMBER OF PATIENTS	PERCENTAGE
PNEUMONIA	24	7.1%
PLEURAL EFFUSION	13	3.8%
ACUTE PULMONARY EDEMA	16	4.7%

d. Neurological complications:

- Postoperative ischemic stroke occurred in 2 patients. The stroke was transient in both patient and they both had a full recovery.
- A transient delirium during the ICU stay was observed in 5 patients.

e. Infectious complications:

A total of 53 (15.3%) patients had infectious complications. Other than pneumonia, the other sites of infection were the surgical site and in a sepsis in 1 patient. We recorded no case of infectious endocarditis in our study.

Table XXXI: Postoperative infectious complications

INFECTION SITE	NUMBER OF PATIENTS	PERCENTAGE
PNEUMONIA	24	7.1%
SURGICAL-SITE INFECTION	28	8.3%
- SUPERFICIAL	23	6.8%
- DEEP	5	1.5%
SEPSIS	1	0.3%
INFECTIOUS ENDOCARDITIS	0	0.0%

f. Infectious complications:

- Postoperative acute renal failure occurred in 4 patients. Dialysis was necessary in 1 patient. Renal failure was reversible in all cases.
- Postoperative vocal cord paralysis occurred in 1 patient.

8.3. Clinical results:

We studied clinical evolution of our surviving patients (n=339) after surgery by evaluating their NYHA status and heart failure signs.

a. NYHA status:

In the first month after surgery, 77.6% of patients were class I or II of the NYHA functional classification.

Table XXXII: Immediate postoperative NYHA status

NYHA STATUS	NUMBER OF PATIENTS	PERCENTAGE
I	39	11.5%
II	224	66.1%
III	65	19.2%
IV	11	3.2%
TOTAL	339	100.0%

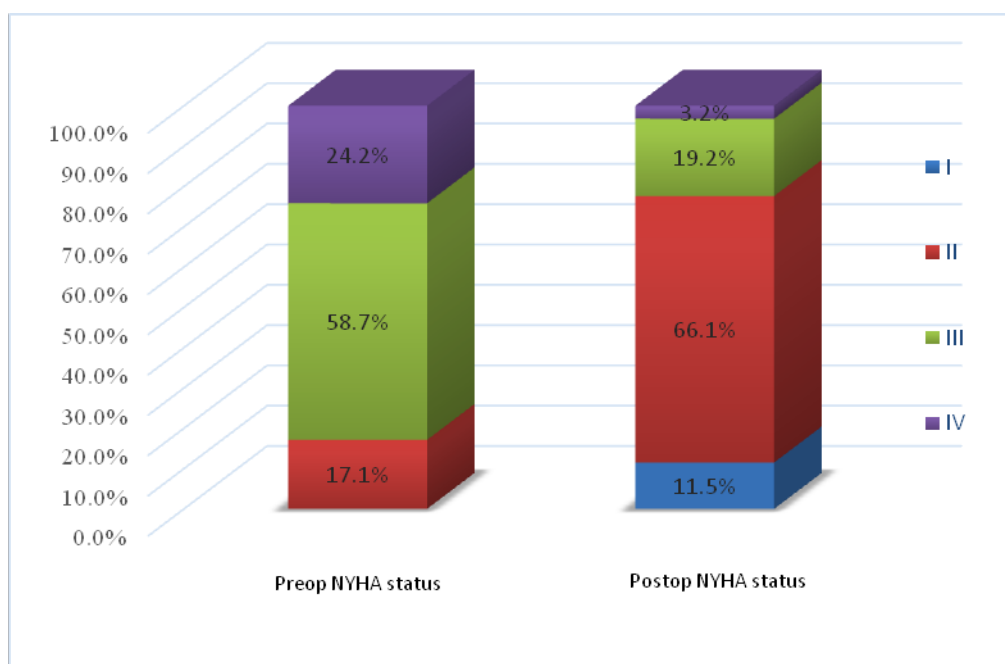


Figure 21: Evolution of NYHA status in the immediate postoperative course

b. Heart failure signs:

Out of the 339 surviving patients, 30 (8.8%) had postoperative congestive heart failure. Thirteen patients did not have heart failure signs before surgery and only developed them in the first postoperative month.

8.4. Echocardiographic results:

All surviving patients had an echocardiographic assessment during the first postoperative month. The average time delay after surgery was 7.2 ± 3.4 days with a minimum of 3 days and a maximum of 20 days.

a. De Vega annuloplasty results:

Postoperative tricuspid regurgitation was assessed in only 242.

The majority of patients (92.1%) had a class 2 or lower degree of tricuspid regurgitation (Moderate, mild or none). The remaining 7.9% of patients had a class 3 or 4 tricuspid regurgitation (moderate to severe or severe).

Tricuspid stenosis was present in 8 (2.3%) patients. It was mild in 5 and moderate in 3 patients.

b. Other echocardiographic data:

Table XXXIII: Comparison between preoperative and postoperative results

	PREOPERATIVE DATA	POSTOPERATIVE DATA
MEAN LVEF	56.8%	55.3%
MEAN PASP	61.7 mmHg	44.9 mmHg
RV DILATATION	70.5%	32.7%
RV DYSFUNCTION	30.0%	35.0%

9. Mid-term surgical results: (6 months to 2 years)

9.1. Follow-up:

The mean follow-up duration was 31 months with a minimum of 6 months and a maximum of 78 months.

The follow-up was insured with phone calls and outpatient consultations in a total of 273 patients while we lost track of 43 patients and late mortality occurred in 23 patients.

In the following segments, we only expose the results of the first 2 years following tricuspid repair.

9.2. Mortality:

During the follow-up, we noted 23 deaths, accounting for 6.7% of our cohort patients.

The causes of death are exposed in table 30. The cause was undetermined in 18 patients, but we know that it is most likely to be Warfarin related accidents.

Table XXXIV: Causes of death in mid-term follow-up

CAUSE OF DEATH	NUMBER OF PATIENTS
VITAMIN K ANTAGONISTS-RELATED HEMORRHAGE	3
INFECTIOUS ENDOCARDITIS	2
UNDETERMINED CAUSE OF DEATH	18

9.3. Devega annuloplasty mid-term results:

a. Clinical outcomes

The majority of our patients were asymptomatic in mid-term follow up with a total of 228 (83.5%) of patients having normal effort tolerance.

The remaining 58 patients had clinical signs as follows:

- Dyspnea:
 - o NYHA II: 12
 - o NYHA III: 18
 - o NYHA IV: 15
- Right-sided heart failure signs were found in 34 patients ranging from jugular vein distension to recurrent ascites.

b. Echocardiographic data:

b.1. Tricuspid regurgitation

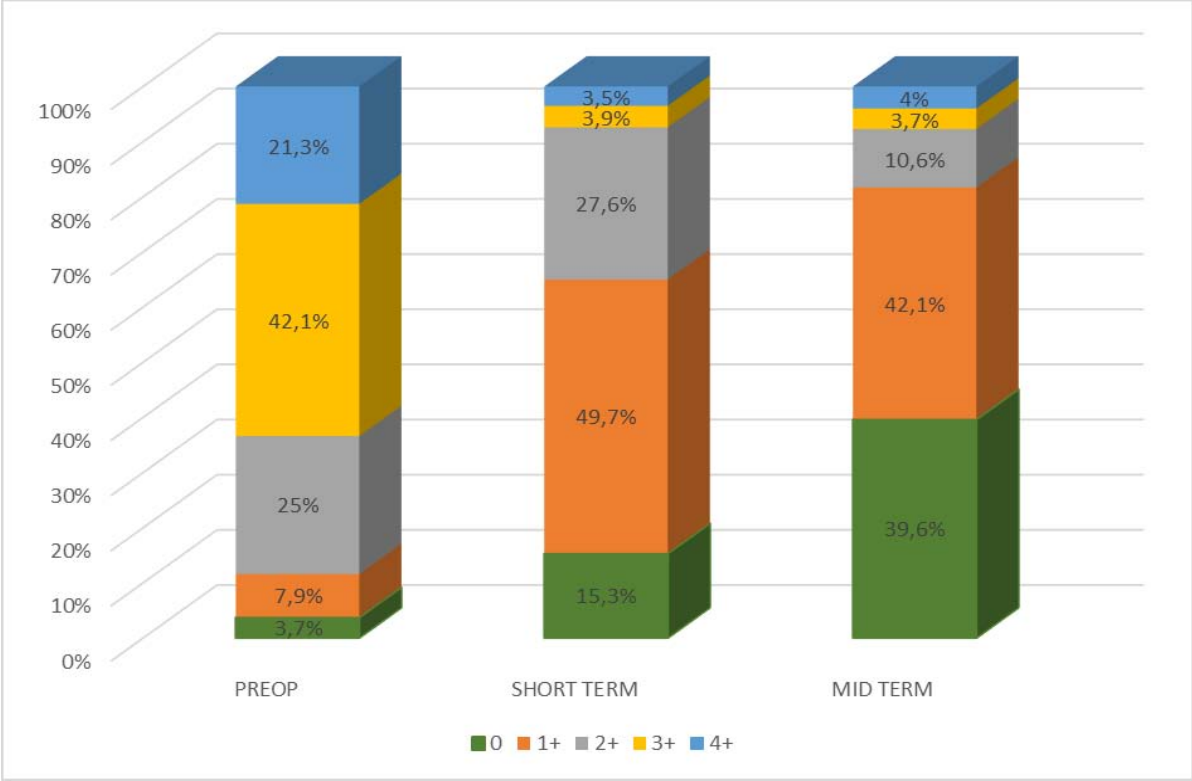


Figure 22: Postoperative tricuspid regurgitation evolution

Table XXXV: Postoperative evolution of each grade of tricuspid regurgitation

Preoperative tricuspid regurgitation	Postoperative (Mid-term results)		
None or mild (0, 1+): 41 patients	0, 1+	25	61%
	2+	1	2.4%
	3+	1	2.4%
	4+	0	0%
	Unknown	14	34.2%
Moderate (2+): 89 patients	0, 1+	73	82%
	2+	4	4.5%
	3+	1	1.1%
	4+	0	0%
	Unknown	11	12.4%
Moderate to severe (3+): 150 patients	0, 1+	103	68.7%
	2+	11	7.3%
	3+	1	0.7%
	4+	3	2%
	Unknown	32	21.3%
Severe (4+): 76 patients	0, 1+	26	34.2%
	2+	12	15.7%
	3+	6	7.9%
	4+	8	10.6%
	Unknown	24	31.6%

b.2. Tricuspid valve stenosis:

In mid-term follow-up, tricuspid valve stenosis was found in 5 patients (1.4%). It was mild in 4 patients and moderate in 1 patient. All 5 patients had tricuspid stenosis in short-term follow up. The 3 remaining patients were lost in mid-term follow-up.

b.3. Pulmonary arterial hypertension:

The mean postoperative pulmonary artery systolic pressure in mid-term follow-up is 38.5 mmHg.

The short and midterm outcomes of the De Vega annuloplasty of the tricuspid valve during the surgical management of rheumatic valvular heart disease

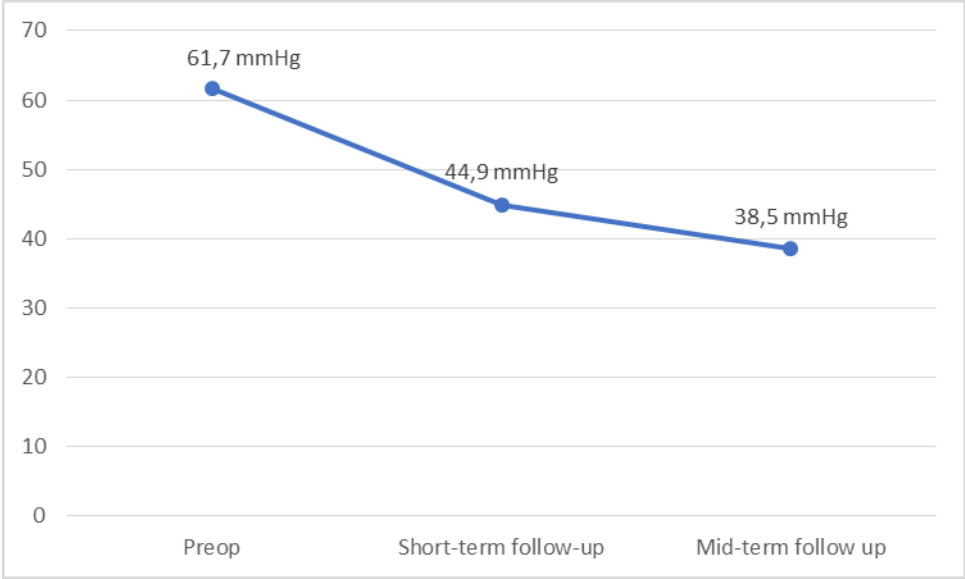


Figure 23: Evolution of mean PASP in our case series

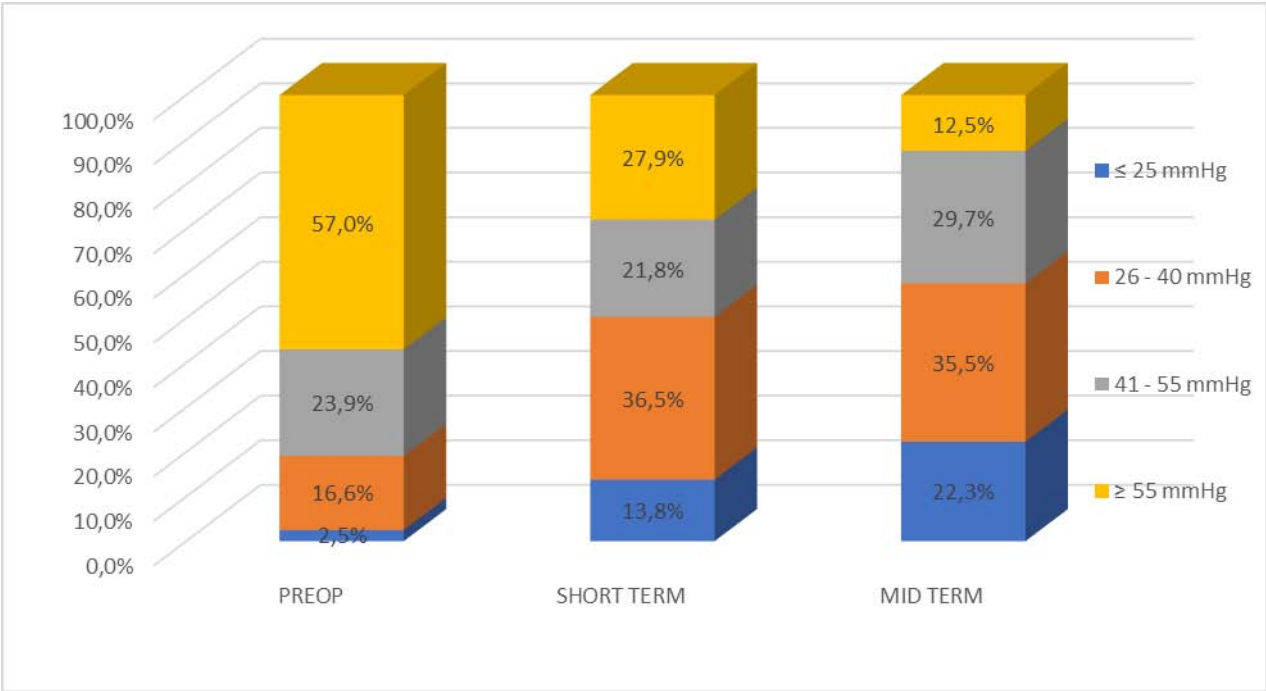


Figure 24: Postoperative evolution of pulmonary artery systolic pressure

❖ Impact of Sildenafil use on PASP evolution in our case series:

In our study, 61 patients were put on sildenafil in the post-operative course. We have no data on the treatment duration.

The indications of Sildenafil use in our study were:

- Severe preoperative pulmonary hypertension
- Postoperative persistence of right-sided heart failure
- Severe postoperative pulmonary hypertension

The mid-term follow-up data are available for 53 patients. We found no statistically significant difference in the evolution of PASP with or without Sildenafil use ($p=0.7$).

Table XXXVI: Evolution of the mean PASP in the Sildenafil group versus the rest of the cohort

MEAN PASP	SILDENAFIL	NO SILDENAFIL
PREOPERATIVE	72.6 mmHg	61.7 mmHg
SHORT-TERM FOLLOW-UP	49.3 mmHg	44.9 mmHg
MID-TERM FOLLOW-UP	42.3 mmHg	38.5mmHg

b.4. Left ventricular systolic function:

The mean postoperative LVEF in mid-term follow-up is 58.4%.

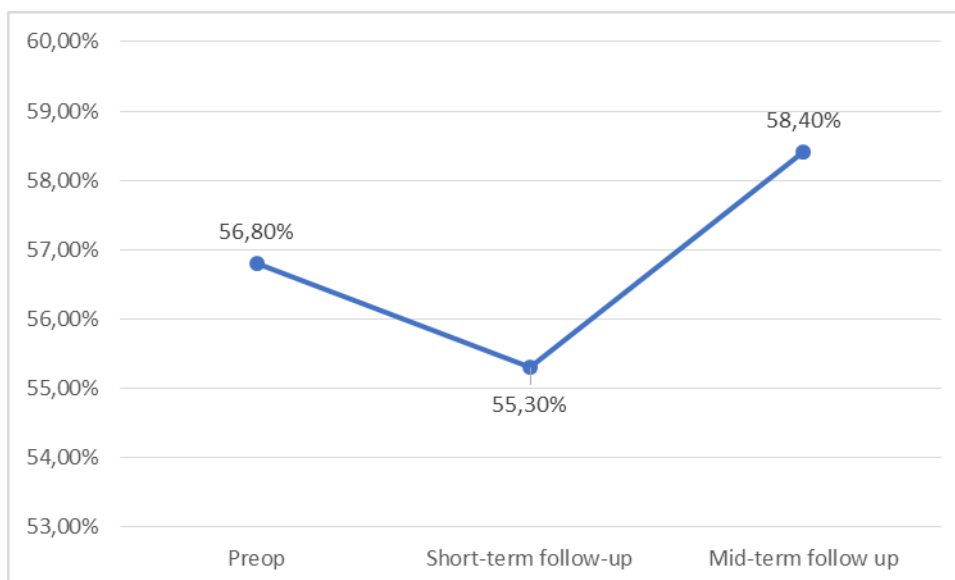


Figure 25: Evolution of mean LVEF in our case series

II. Analytic study:

1. Mortality:

We had a total of 40 (11.2%) deceased patients during the 2 years period of follow up:

- 17 (4.8%) occurred in the first month.
- 23 (6.4%) occurred later on.

The clinical characteristics of this group are displayed bellow.

1.1. Preoperative data:

Table XXXVII: Preoperative data in deceased patients versus the total of the cohort

	Deceased group	Cohort data
Mean age	50.4 years	40.6 years
Female predominance	72.5%	66.3%
Heart failure signs	47.5%	21.6%
NYHA status:		
- I	0%	0%
- II	5%	17.1%
- III	50%	24.2%
- IV	45%	58.7%
Atrial fibrillation	75%	75.3%
Dilated right ventricle	70%	70.5%
Right ventricle dysfunction	55%	30%
Tricuspid regurgitation grade:		
- 0, 1+	10%	11.6%
- 2+	12.5%	25%
- 3+	60%	42.1%
- 4+	17.5%	21.3%
Mean PASP	65.5 mmHg	61.7 mmHg
Mean LVEF	56.6%	56.8%

1.2. Surgical data:

Table XXXVIII: Surgical data in deceased patients versus the total of the cohort

	Deceased group	Cohort data
Aortic cross clamp time	98.7 min	92.7 min
CPB time	149.6 min	130 min
Left-sided valvular procedures:		
- MV and AV replacement	72.5%	26.4%
- MV replacement	22.5%	39.1%
- MV replacement + AV repair	5%	1.9%

1.3. Postoperative data:

Only 27 out of the 40 deceased patients had a postoperative echocardiography done during the ICU stay.

The mean LVEF was 43.6% with 4 patients having a severe left ventricle dysfunction.

The mean immediate postoperative PASP in these patients was 61 mmHg.

Only 10 patients had postoperative tricuspid valve regurgitation quantification:

- Severe: 2 patients
- Moderate to severe: 1 patient
- Moderate: 4 patients
- Mild: 3 patients

2. Factors of failure of De Vega tricuspid repair:

With the use of the IBM SPSS Statistics 21 program, we analyzed risk factors for the De Vega annuloplasty failure. A p value <0.05 was considered as statistically significant.

We defined as failure a postoperative tricuspid regurgitation grade 3+ or 4+. In total, we had 23 cases (7.3%) of De Vega annuloplasty failure.

Table XXXIX: Different potential De Vega annuloplasty failure risk factors tested in our study

	3+ and 4+ postop TR group data	Cohort data	P value
Mean age	47.9 years	40.6 years	0.7
Female predominance	67.5%	66.3%	0.1
Preoperative heart failure signs	65.2%	21.6%	0.04
Preoperative NYHA status III or IV	86.9%	82.9%	0.07
Preoperative atrial fibrillation	91.3%	75.3%	0.3
Preoperative dilated right ventricle	78.3%	70.5%	0.03
Preoperative right ventricle dysfunction	82.6%	30%	0.01
Preoperative TR grade 4+	69.6 %	21.3%	0.03
Preoperative TR grade 3+	26 %	42.1 %	0.7
TV annulus diameter > 40 mm	91.3%	43.4%	0.01
Organic lesions of the TV	65.2%	25.7%	0.1
Severe preoperative PAH	91.3%	57%	0.002
Mean preoperative LVEF	54.3%	56.8%	0.07
Isolated mitral valve surgery	56.5%	70.8%	0.7
Combined MV and AV surgery	43.5%	29.2%	
Severe postoperative PASP	69.6%	12.5%	0.004
Postoperative mean LVEF	55.6%	58.4%	0.3

The predictive factors of failure in our study were:

- Preoperative heart failure signs (p=0.04)
- Preoperative dilated right ventricle (p=0.03)
- Preoperative right ventricular dysfunction (p=0.01)
- Preoperative TR grade 4+ (p=0.03)
- Preoperative TV annulus diameter > 40 mm (p=0.01)
- Severe preoperative pulmonary artery hypertension (0.002)
- Severe postoperative pulmonary hypertension (0.004)

3. Data comparison by left-heart valvular procedures:

We identified two sub-groups in our study:

- **Group 1:** Patients who underwent tricuspid repair + isolated mitral valve surgery

- **Group 2:** Patients who underwent tricuspid repair + combined mitral valve and aortic valve surgery

Table XL: Data comparison between group 1 and 2

	Group 1 (70.8%)	Group 2 (29.2%)
Mean age	39.6 years	43.7 years
Female predominance	65.8%	68.1%
Preoperative heart failure signs	26.2%	18.5%
Preoperative NYHA		
- I	0%	0%
- II	13.6%	19.2%
- III	66.7%	49.3%
- IV	19.6%	31.5%
Preoperative atrial fibrillation	75.9%	74.6%
Preoperative dilated right ventricle	72.2%	69.8%
Preoperative right ventricle dysfunction	28.2%	34.4%
Preoperative TR grading:		
- 0, 1+	6.6%	15.2%
- 2+	29.8%	24.6%
- 3+	41.9%	43.8%
- 4+	21.7%	16.4%
Mean TV annulus diameter	37.6 mm	39.9 mm
Organic lesions of the TV	24.2%	27.4%
Mean preoperative PASP	60.7 mmHg	61.9 mmHg
Mean preoperative LVEF	57.3%	55.9%
Postoperative NYHA		
- I	83.8%	80.8%
- II	6.6%	4.1%
- III	4.6%	8.2%
- IV	5%	6.8%
Postoperative TR grading:		
- 0, 1+	82.8%	79.6%
- 2+	9.6%	10.9%
- 3+	5.1%	2.7%
- 4+	2.5%	6.8%
Postoperative dilated right ventricle	21.2%	26%
Postoperative right ventricle dysfunction	19.2%	23.2%
Mean postoperative PAH	37.5 mmHg	41.5 mmHg
Mean postoperative LVEF	58.5%	57.9%



DISCUSSION

Functional TR was a forgotten entity for so long. When existing with concomitant left sided lesions such mitral or aortic valve disease, it is associated with high mortality and increased risk of adverse events (15).

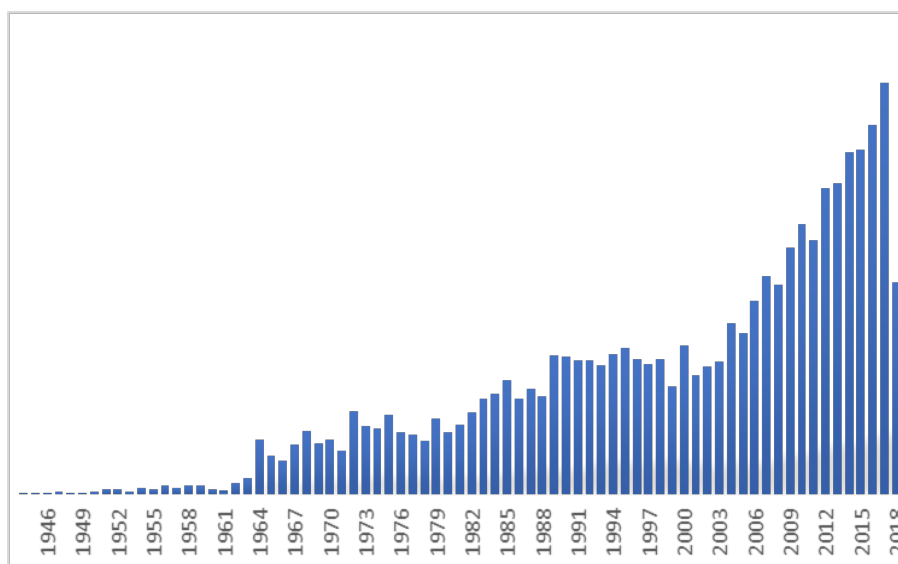


Figure 26: Published articles on "tricuspid valve regurgitation" throughout the years – PubMed

In patient with secondary TR concomitant with mitral or aortic valve(s) lesion(s), surgical treatment of left-sided valvular disease without addressing the tricuspid valve may improve mild TR (16,17). However, uncorrected moderate and severe TR may persist or even worsen after mitral valve surgery leading to progressive heart failure and death. In addition, reoperation for residual TR carries significant risks and may is associated with poor outcomes and (18,19).

Our study reports the short- and midterm results of 356 patients who underwent a De Vega tricuspid annuloplasty during the surgical treatment of left heart valvular disease. It was conducted in order to evaluate the effectiveness of this technique and to analyze the effects of preoperative, operative and postoperative parameters on outcomes. The limits of our study are:

- Our study is a retrospective study with all of the inherent limitations; some data were not mentioned on medical records or were lost in handwritten archives rendering many files unusable in our study.

- The absence of a group of patients operated on with another tricuspid valve annuloplasty technique for comparison.
- Furthermore, the data may be subject to selection bias. Symptomatic patients might have been more likely to receive the follow-up echocardiography than asymptomatic patients.
- During study period, preoperative diagnostic evaluation, surgical technique, anesthesiologic management and intensive care changed considerably and it is difficult to take into consideration all the factors that might have influenced the results. Two different De Vega annuloplasty techniques were used in our study.

I. Demographic, clinical and paraclinical data:

1. Age:

The average age of our patients was 40.6 years \pm 12.14 and 73.6% of our patients were aged between 20 and 50 years old.

Our study population is younger compared to western studies. This result is comparable to studies from other developing countries. At the national level, the average age of our patients is close to that found patients in the Kachchour series with an average of 39.8 years (20).

This result is explained by the etiology of tricuspid regurgitation in our study, being rheumatic valvular disease.

Thus, tricuspid regurgitation occurs especially in the young active adult in developing and emerging countries in contrast to developed countries where it occurs in an older population. This predominance has a great impact on socio-economic development of our country.

The distribution of patients by gender in our series found a clear female predominance with 66.3% of female patients. The same result was also reported by the majority of published studies.

This female predominance can be attributed to the preponderance of rheumatic valvular disease in women (21).

Table XLI: Age and sex of patients with tricuspid regurgitation (Literature review)

SERIES	COUNTRY	YEAR OF PUBLICATION	NUMBER OF CASES	AVERAGE AGE	FEMALES %
ANNOUKHAILI (22)	MOROCCO (Casablanca)	2007	54	29	60%
KACHCHOUR (20)	MOROCCO (FEZ)	2016	120	39.8	69.2%
ALNAWASEH (23)	JORDAN	2017	320	48	54.7%
CHARAFEDDINE (24)	TUNISIA	2017	91	43	78.1%
KHALLAF (25)	EGYPT	2016	80	33	63%
SUNG HO (26)	USA	2016	415	71	62%
ABDUL MALIK (27)	PAKISTAN	2012	160	21	55.6%
CISS AHMADOU (28)	SENEGAL	2017	58	26	58%
OUR CASE SERIES	MOROCCO (Marrakech)	2018	356	40.6	66.3%

2. Medical and surgical history:

We noted the presence of a history of recurrent tonsillitis in 42.7% of our patients and rheumatic fever in 32.6% of our patients. This has been reported in different series (29,30) that considered it as an indication of the rheumatic context of patients, being mostly from underdeveloped countries, without establishing a direct causality relationship.

Two patients had a history of myocardial infarction, 46 patients had during the course of their valvular disease an ischemic stroke, and 17 patients had a history of infective endocarditis

The association with other chronic diseases was observed in some patients of our series: 3.9% of patients had high blood pressure and 3.6% have diabetes mellitus. In the Shinn et al series, which included older patients, there was higher percentages of chronic conditions with 62% of hypertension and 17% of diabetes (31).

Only 6.7% of patients underwent a previous cardiac surgery. No patient had previous surgical procedure on the tricuspid valve. This rate is lower than that reported in the Bernal (32) study (26%) and higher than that of Han (0%) (33).

McCarthy (34) reported in his series that 51% of patients had prior valvular surgery on the mitral, aortic or tricuspid valve.

In the Colombo series (30) of 50 cases, 64% of patients had undergone previous surgical treatment: 52% had an open mitral commissurotomy and 16% a closed mitral commissurotomy. While in our series, only 1.4% of patients had previous open mitral commissurotomy and 4.5% had closed mitral commissurotomy.

Closed mitral commissurotomy was always privileged when possible: it is attempted first because it is fast, less dangerous, with satisfactory results, in addition to better accessibility compared to open mitral commissurotomy. However, it has many limitations (thrombosis of the right atrium, major damage to the subvalvular apparatus). We noticed in our study that the closed mitral commissurotomy was less and less practiced in recent years of our study.

The low number of patients previously operated in our series compared to those of Bernal (32), Colombo (30) and McCarthy (34) can be explained by the delay of the surgical management in our patients. Our case series consists of patients treated at least for a double valvular disease, that is to say at an advanced stage of valvular rheumatic disease.

3. Etiology of tricuspid regurgitation:

The majority of our patients had tricuspid regurgitation associated with mitral stenosis (67.7%). Only 1.4% of patients had additional tricuspid stenosis.

According to literature, functional tricuspid regurgitation is the most common. In our series, the mechanism of regurgitation was functional by isolated annular dilation in 74.3% (10).

The etiology of valvular disease was presumed to be rheumatic in all our patients.

The incidence of rheumatic fever has declined in industrialized countries since the 1950s, but in developing countries, it remains an endemic disease with annual incidences ranging from 100 to 200 per 100,000 school-aged children (35).

Rheumatic heart disease is still the major cause of valvular disease in our country and in other developing countries. It represents a real health problem. In developed countries, it became rare since the 1970s.

The prevalence of rheumatic heart disease in Morocco is 0.3 to 1%. It is responsible for 10% of medical hospitalizations and more than 50% in cardiology departments. It is a notifiable disease because of its cardiac complication, its economic impact at the individual and community levels (36).

The prevention of rheumatic fever is a necessity in our context. It is the only way to reduce morbidity and mortality of rheumatic heart disease. Rheumatic fever has been a disease under surveillance since 1986 in Morocco. And since, the epidemiological trend is clearly increasing. In 1995, the Health Department launched the rheumatic fever control program in the same way as its declaration (referred to as 10.02.95) (37–39).

The aims of the program were to treat 40–50% of acute tonsillitis and reduce the incidence of rheumatic fever and the prevalence of rheumatic heart disease (39).

The main components of this program are:

- **Primary prevention:** systematically treat all acute tonsillitis with penicillin. In case of allergy to penicillin, macrolides can be used.
- **Secondary prevention:** aims to prevent relapses that are secondary to streptococcal reinfection; it is aimed at subjects with a history of rheumatic fever and / or with rheumatic heart valve disease. It consists of an injection of Benzathine benzylpenicillin every 3 weeks. Erythromycin remains the alternative in case of allergy to penicillin. Treatment for secondary prevention should be maintained until the age of 45 in case of carditis and at least for 5 years in the absence of it.
- **Health education:** must be generalized but must focus on the children affected and their parents. The school environment is also a very important target, it constitutes a population with a high risk for the rheumatic fever.

- **Continuous training:** should gradually cover doctors and paramedical staff, particularly those who work in level I health care centers and in school medical facilities.
- **Epidemiological surveillance:** all cases of rheumatic fever should be monitored by the Department of Infrastructure and Prefectural/Provincial Ambulatory Actions (SIAAP) every trimester.

Despite this program, as our study shows, rheumatic heart disease is still a heavy burden as much by physical impotence as by the mortality it generates. An evaluation of the program activities is necessary to actually guide its actions.



Figure 27: Some examples of the tools provided by the national rheumatic fever control program (36)

Rheumatic heart disease is progressive, and heart valve surgery used to repair or replace diseased heart valves is a palliative therapy. Progression of rheumatic heart disease together with valve-related complications of cardiac prostheses prevents heart valve surgery from being considered curative (40).

4. NYHA status:

The majority of patients presented with NYHA status III in 58.7% of cases and status IV in 24.2% of cases. This result reflects the advanced stage of valvular heart disease.

Similarly, in the majority of international published series, patients had advanced valvular disease.

Table XLII: NYHA classification in patients with tricuspid regurgitation

	I	II	III	IV
GUGLIERMO (41) (Italy)	0%	20%	55%	5%
ABDELGAWAD (42) (Egypt)	0%	15%	65%	20%
KUNWA (43) (Czech)	4.6%	23.3%	53.5%	18.6%
OUR CASE SERIES	0%	17.1%	58.7%	24.2%

The comparison of the preoperative functional status and the degree of tricuspid regurgitation of patients in our study and other series (23,27,28) showed an overlap in terms of results, which confirms the correlation between tricuspid regurgitation degree and stage of dyspnea.

5. Congestive heart failure signs:

In the majority of studies, the presence of peripheral signs of heart failure was not addressed and only NYHA status was used to assess heart failure. Unfortunately, this made the comparison to a western study impossible.

In our case series, 21.6 % of our patients had signs of right heart failure meaning that almost one quarter of our patients were in late stage valvular heart disease. The impact of this data on the general results of valvular surgery is undeniable.

Table XLIII: Peripheral signs of heart failure

SERIES	COUNTRY	YEAR OF PUBLICATION	% OF PATIENTS
KACHCHOUR (20)	MOROCCO (FEZ)	2016	32.5%
ZAHIRI (44)	TUNISIA	2016	50%
OUR CASE SERIES	MOROCCO (Marrakech)	2018	21.6%

6. Chest X-ray:

We found cardiomegaly in 87.6% of our patient. This percentage was higher in other case series. This result is another sign of advanced valvular disease in our series.

Table XLIV: Cardiomegaly in patients with tricuspid regurgitation

SERIES	COUNTRY	YEAR OF PUBLICATION	% OF PATIENTS
KACHCHOUR (20)	MOROCCO (FEZ)	2016	55%
CISS AHMADOU (28)	SENEGAL	2017	67%
OUR CASE SERIES	MOROCCO (Marrakech)	2018	87.6%

7. Electrocardiogram:

In our series, we had 268 cases of atrial fibrillation (75.3%), a close rate to the majority of other series.

This is explained by the impact of left valvular lesions on the left atrium, which is usually dilated.

Table XLV: AF in patients with tricuspid regurgitation

SERIES	COUNTRY	YEAR OF PUBLICATION	% PREOPERATIVE AF
KACHCHOUR (20)	MOROCCO (FEZ)	2016	35%
ANNOUKHAILI (22)	MOROCCO (Casablanca)	2007	75%
ZAHIRI (44)	TUNISIA	2016	84.4%
HIROKI (45)	JAPAN	2017	79.5%
SUNG HO (26)	USA	2016	60%
OUR CASE SERIES	MOROCCO (Marrakech)	2018	75.3%

8. Echocardiography:

Two-dimensional echocardiography and pulsed Doppler allows an objective, reproducible evaluation and non-invasive diagnosis of valvular disease (10).

All series agree that transthoracic echocardiography is the key examination for the diagnosis of tricuspid regurgitation, the evaluation of its severity, the determination of its etiology and the orientation of surgical indications.

In our series, two-dimensional echocardiography was used to assess the valvular and subvalvular lesions. The color doppler visualize the regurgitant jet directly and calculates its velocity.

8.1. Tricuspid regurgitation grading:

Tricuspid regurgitation was moderate to severe in 63.4 % of our patients. This rate is close to other studies.

Table XLVI: Percentage of patients with preoperative grade 3+ and 4+ tricuspid regurgitation

SERIES	COUNTRY	YEAR OF PUBLICATION	% OF PATIENTS WITH 3+ AND 4+ TR
KACHCHOUR (20)	MOROCCO (FEZ)	2016	53.8%
ANNOUKHAILI (22)	MOROCCO (Casablanca)	2007	74%
CHARAFEDDINE (24)	TUNISIA	2017	83.5%
SUNG HO (26)	USA	2016	62%
OUR CASE SERIES	MOROCCO (Marrakech)	2018	63.4%

8.2. Tricuspid annulus:

Like it is reported in the literature, a predominance of functional TR was found in our patients with a rate of 74.3%. We also noted, on the echocardiographic study, that 45.5% had a tricuspid annulus diameter > 40mm.

8.3. Pulmonary arterial systolic pressure:

The average PASP in our series finds was 61.7 mmHg. This value is close to those reported in other studies conducted in developing countries. However, the value was much lower in developing countries.

Table XLVII: Average PASP in patients with tricuspid regurgitation

SERIES	COUNTRY	YEAR OF PUBLICATION	AVERAGE PASP
KACHCHOUR (20)	MOROCCO (FEZ)	2016	63.69 mmHg
ANNOUKHAILI (22)	MOROCCO (Casablanca)	2007	71 mmHg
CISS AHMADOU (28)	SENEGAL	2017	73 mmHg
HIROKI (45)	JAPAN	2017	49 mmHg
SUNG HO (26)	USA	2016	52 mmHg
OUR CASE SERIES	MOROCCO (Marrakech)	2018	61.7 mmHg

8.4. Left ventricle function:

In our series the mean ejection fraction of the left ventricle (LVEF) was 56.8%. This value is very close to those found in other studies.

Table XLVIII: Average LVEF in patients with tricuspid regurgitation

SERIES	COUNTRY	YEAR OF PUBLICATION	AVERAGE LVEF
KACHCHOUR (20)	MOROCCO (FEZ)	2016	59%
CHARAFEDDINE (24)	TUNISIA	2017	57.3%
CISS AHMADOU (28)	SENEGAL	2017	63%
HIROKI (45)	JAPAN	2017	33.4%
SUNG HO (26)	USA	2016	60%
OUR CASE SERIES	MOROCCO (Marrakech)	2018	56.3%

8.5. Operative indications:

Echocardiography gives the surgeon a complete assessment of valvular disease. Therefore, it can be capable of guiding surgical indications and operative choices: functional TR cannot be ignored because its postoperative persistence can compromise the long-term results of left heart valvular surgery. No formal argument allows to distinguish with certainty patients in which TR will regress from those in which it will persist.

Some clinical and echocardiographic criteria can help choose between surgical abstention and annuloplasty:

- In cases with significant pulmonary hypertension, cardiomegaly, and preoperative significant TR, an annuloplasty is indicated. Some authors have identified echocardiographic data as indicators of the need for tricuspid valve surgery (46). In a study published by Chopra (46), a diameter of the annulus greater than 21 mm / m² in diastole, and 16 mm / m² in systole, a regurgitant jet surface / right atrial area ratio greater than 34%, and a systolic shortening of the tricuspid annulus less than

25% should lead to surgical correction of TR. In the study of Colombo (34), the measurement of this degree of shortening of the tricuspid annulus preoperatively is a predictor of the incidence of late poor outcome; It is 57.1% when this degree of shortening of the tricuspid ring is less than 25%, and it is 0% when it is greater than 25%.

- Organic lesions of the tricuspid valve and significant right ventricle dysfunction must be corrected.

In most cases, surgical management of tricuspid disease is possible by repairing the native valve. In cases with extreme valvular alterations, the only choice is tricuspid valve replacement.

II. Intraoperative data:

1. Cardiopulmonary bypass:

All our interventions took place on cardiopulmonary bypass, on aortic cross clamp.

The average CPB time in our patients was 130 ± 50.4 min and the mean aortic clamp time was 92.7 ± 38.4 min.

These durations highly differ between studies. This is explained by the difference between the number and nature of different valvular interventions conducted on the study patients.

Table XLIX: CPB data in various studies

SERIES	COUNTRY	YEAR OF PUBLICATION	AVERAGE CPB TIME	AVERAGE CLAMP TIME
KACHCHOUR (20)	MOROCCO (FEZ)	2016	95 min	67 min
HIROKI (45)	JAPAN	2017	176 min	117 min
SUNG HO (26)	USA	2016	82 min	48 min
OUR CASE SERIES	MOROCCO (Marrakech)	2018	130 min	92 min

The use of inotropic agents was necessary for CPB weaning in 298 patients (83.7%) in our series. Lower rates of use of inotropic agents have been published in other studies (20).

2. Surgical details:

Surgeons agree that surgical indications for tricuspid regurgitation are difficult to establish because studies are rare, not randomized and spread over many years with small series of heterogeneous patients. In fact, the majority of these studies include organic and functional TR, early and late TR, isolated interventions on the tricuspid valve and associated interventions on mitral and / or aortic valves with very different surgical techniques.

All of these elements lead to controversial attitudes towards the need or not of a tricuspid valve intervention, its optimal timing and type. It is why the level of evidence of these recommendations is still level C (results from expert consensus, records or retrospective studies with small cohorts of patients).

2.1. Left sided valve surgical procedure:

104 patients (29.2%) in our series had triple valvular surgery and 252 patients (70.8%) had mitral and tricuspid surgery.

Table L: Left sided valvular surgery procedure concomitant to tricuspid valve repair

SERIES	COUNTRY	YEAR OF PUBLICATION	MITRAL VALVE SURGERY	AORTIC VALVE SURGERY	COMBINED SURGERY
KACHCHOUR (20)	MOROCCO (FEZ)	2016	64.5%	0%	35.5%
ALNAWAISEH (23)	JORDAN	2018	78%	15%	7%
CHARAFEDDINE (47)	TUNISIA	2017	64.8%	3.3%	28.6%
KHALLAF (25)	EGYPT	2016	100%	0%	0%
HIROKI (45)	JAPAN	2017	88.5%	0%	11.5%
SUNG HO (26)	USA	2016	100%	0%	0%
OUR CASE SERIES	MOROCCO (Marrakech)	2018	70.8%	0%	29.2%

There is a large contrast between the rates reported in other studies. Mitral valve surgery associated with tricuspid repair is the most reported. This can be explained by the fact that mitral valve disease is the major cause of functional tricuspid regurgitation.

Requiring triple valve procedure is another indicator of advanced valvular disease.

a. Tricuspid valve surgical procedure:

Operative procedures on the tricuspid valve were conservative in all patients.

The De Vega tricuspid annuloplasty was done in all patients. Concomitant procedures on the tricuspid valve were necessary in 17.1%.

Table LI: Tricuspid valve repair procedures

SERIES	COUNTRY	YEAR / N° OF PATIENTS	DE VEGA ANNULOPLASTY	RING ANNULOPLASTY	OTHER PROCEDURES
KACHCHOUR (20)	MOROCCO (FEZ)	2016 120 patients	63.3%	29.2%	7.5% (Kay annuloplasty)
ANNOUKHAILI (22)	MOROCCO (Casablanca)	2007 54 patients	100%	0%	0%
ALNAWASEH (23)	JORDAN	2018 320 patients	43.7%	56.3%	0%
CHARAFEDDINE (47)	TUNISIA	2017 91 patients	50.5%	49.5%	0%
KHALLAF (25)	EGYPT	2016 80 patients	77.5%	22.5%	0%
CISS AHMADOU (28)	SENEGAL	2017 58 patients	100%	0%	0%
HIROKI (45)	JAPAN	2017 684 patients	45.6%	54.4%	0%
SUNG HO (26)	USA	2016 415 patients	46.7%	53.3%	0%
OUR CASE SERIES	MOROCCO (Marrakech)	2018 356 patients	100%	0%	17.1%

III. Complications:

In our series, we noted 79 cases of postoperative (23.3%) complications. This rate is lower than the one reported in the study series of Gabriel et al (28) being 52% while it was higher than the one reported in the study conducted in the university hospital of Fez being 15% (20).

IV. Tricuspid valve repair result:

The association of tricuspid regurgitation to left side heart valve diseases is common but surgical indications during left side heart surgery is still controversial and concomitant tricuspid valve annuloplasty ranges from more than 60% to 6% depending on each single institution strategy (41).

Tricuspid valve surgery is recommended for symptomatic patients with the signs of right heart failure and severe tricuspid valve regurgitation, but there is growing evidence that even patients presenting with annular dilatation without significant regurgitation benefit from valve repair (48). Uncorrected trivial and moderate TR may persist or even worsen after mitral valve surgery, leading to progressive heart failure and death (49).

Outcome assessment of tricuspid valve repair is influenced by the competing risk of left heart valve-related death and complications such as thromboembolism, bleeding or reoperation. The mortality and morbidity associated with tricuspid valve annuloplasty may be overestimated. The majority of studies agree that concomitant tricuspid repair and left sided valvular surgery has more benefits than risk (7,7,49).

Published studies agree that the benefit of the conjunction of tricuspid repair to left-heart valvular surgery is superior to its surgical risk.

Functional TV regurgitation is primarily treated by annuloplasty. Valve replacement is rarely necessary and is associated with a worse outcome (50,51). However, there is an ongoing debate on whether the TV should be repaired using either a suture-based or a prosthetic ring annuloplasty (2,25).

1. De Vega annuloplasty results:

The reported benefits of the De Vega annuloplasty include (52–54):

- It is a secure and reproducible technique that requires very short additional operative time (10 or 15 additional minutes of operating times).
- It preserves the 3D shape of the tricuspid valve annulus and its dynamics during systole and diastole, contrary to ring annuloplasty that fixes the annulus, thus preventing its natural dynamics during the cardiac cycle. Even the flexible ring, which was believed to permit the dynamic modifications of the annulus shape, still prevents dilatation of the tricuspid annulus during diastole. Besides, it becomes progressively stiff with time as a consequence of endothelial ingrowth, eventually even calcification (55,56).
- Apart from the suture material (and of pledgets), there is an absence of prosthetic material, which is an important consideration.
- The fibrosis around the sutures (and the pledgets) tends to transform it into a kind of band without altering the annulus flexibility.
- Much cheaper than any commercially available devices and this was a main consideration when De Vega advised the technique.

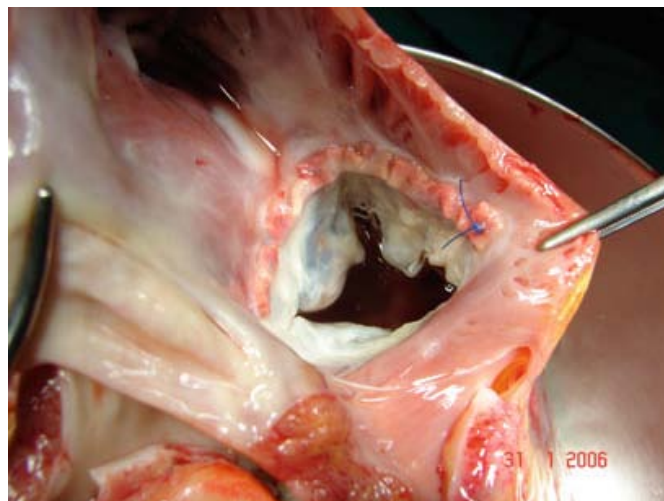


Figure 28: Photograph of a heart retrieved during heart transplantation showing a well preserved modified De Vega annuloplasty (54)

Like it is reported in other studies, we noted an improvement of the NYHA status in the majority of our patients. In the midterm follow up, 83.5% of our patients were asymptomatic.

CISS AHMADOU (28) reported a rate of 89% of asymptomatic patients in long term follow up of 58 patients who underwent De Vega annuloplasty.

Less favorable clinical outcomes were reported in studies published in early development of tricuspid valve repair. Modification of the De Vega annuloplasty method had a clear impact on the technique's results (57-61).

1.1. Echocardiographic outcomes:

Table II: Echocardiographic results after De Vega annuloplasty

SERIES	COUNTRY	YEAR / N° OF PATIENTS	3+ or 4+ TR	PASP mmHg	LVEF
KACHCHOUR (20)	MOROCCO (FEZ)	2016 120 patients	12.5%	39.78	55.69%
ANNOUKHAILI (22)	MOROCCO (Casablanca)	2007 54 patients	8%	45	58%
CISS AHMADOU (28)	SENEGAL	2017 58 patients	5.26%	42	-
HIROKI (45)	JAPAN	2017 684 patients	4.8%	38	30.9%
SUNG HO (26)	USA	2016 415 patients	3%	-	-
OUR CASE SERIES	MOROCCO (Marrakech)	2018 356 patients	7.3%	38.5	58.3%

1.2. Mortality:

Hospital mortality varies significantly from one series to another, ranging from 0.6% to 37.1% (16). In our series this rate was 11.2%. The review of literature shows that this mortality rate is largely influenced, on one hand, by the demographic characteristics of the included patients, cardiovascular status and, on the other hand, by the nature of the tricuspid

intervention. Tricuspid surgery is considered to be a reflection of neglected left valvular disease usually with severe ventricular dysfunction (62).

All authors highlight the difficulty of assessing the operative mortality of tricuspid interventions since the surgery is double or triple: mitral, tricuspid and/or aortic.

1.3. Reoperation:

Despite a rate of 7.3% severe tricuspid regurgitation during follow up, none of our patients was reoperated on. Due to poor outcomes and high mortality risk in redo surgery for persistent tricuspid valve regurgitation, our patients were managed medically. In addition, the mean follow-up period in our study is relatively short.

In published studies discussing long-term results of tricuspid valve repair, a rate of reoperation ranging from 2% to 30% at 10 years was reported (62). A great number of factors influence these results:

- Reoperation is associated with a high mortality rate making it a last resort in the majority of centers (63).
- Preoperative patient's clinical status influence and the degree of left heart disease and its impact on ventricular function.
- Reoperations for left-heart valvular dysfunction are not always excluded.

2. Comparison between De Vega annuloplasty and ring annuloplasty:

There is a considerable controversy among surgeons regarding the optimal treatment of functional tricuspid valve regurgitation.

Patients who require TV surgery are a very heterogeneous group. Therefore, the comparison of surgical results between different methods is difficult because it theoretically requires two comparable study groups.

The De Vega suture annuloplasty was first proposed as an easy and fast, cost effective method for tricuspid valve repair, which preserves the flexibility of the annulus and avoids prosthetic material, with good short term outcomes (62,64).

Some studies suggest that late results of TV repair with ring annuloplasty are superior to repair with suture annuloplasty. In the study reported by McCarthy (34), the surgical outcomes of TV annuloplasty using 4 techniques were compared in patients undergoing operation between 1990 and 1999. Severity of residual regurgitation was similar in early follow up, but over time residual TR rose more rapidly in patients with De Vega repair compared with ring methods. In another report by Guenther (62),¹³ 10-year survival after TV repair with either a ring annuloplasty or De Vega annuloplasty were 46% \pm 7%, and 39% \pm 3% , and freedom from TV reoperation after TV repair with De Vega suture annuloplasty was 88% compared to 98% after a ring annuloplasty. The authors concluded that a ring annuloplasty was associated with improved survival and lower rate of reoperation than a suture annuloplasty.

The problem with the longevity of the outcome of this technique highly questioned the risk off its failure with subsequent development of significant tricuspid regurgitation requiring a redo surgery (34,53,65). This has led many surgeons to adopt the use of annuloplasty rings. Although, for a technique with numerous advantages, we believe that it was given up on the De Vega annuloplasty technique too quickly.

Fortunately, other studies differ from these previous reports and defended the De Vega technique. Shinn (26) reported that results of patient survival were similar between ring and De Vega annuloplasty for the overall cohort as well as propensity-matched patients. Further, there was no difference in patient's outcome with suture or ring annuloplasty in regard to late functional status, progression of late TR, or TV reoperation.

There are several possible explanations for discordance. Era of operation may have influenced the results. In both of the previously mentioned investigations, ring annuloplasty was used in the later periods of the study. Further, the methods used for suture annuloplasty and the degree of annular reduction were highly variable. The experience of the surgical team is a major

influential factor too. Ring annuloplasty has a standard technique compared to De Vega annuloplasty.

Other investigations have shown no important differences in clinical outcomes of De Vega annuloplasty versus ring annuloplasty (25,66,67). In a study of 299 patients with multivalvular rheumatic heart disease, Sarralde (35) reported better late survival of patients undergoing De Vega repair compared with those having ring annuloplasty for TV repair. In a subgroup meta-analysis with only randomized studies, there was no difference in early mortality (40).

All of the afore mentioned studies should be interpreted cautiously because of wide variation in surgeons' attitudes toward patient selection and choice of procedure. TV repair with ring annuloplasty is popular now, and selection of this technique may be influenced by surgeons' experience with use of rings for MV repair.

There is another plausible justification for these differences in reported results of the De Vega annuloplasty. While the technique of implantation of a prosthetic ring is very standardized, the performance of the De Vega annuloplasty is not, depending on small technical details and experience that can transform the outcome (68).

For example, and contrary to what may be understood from most technical drawings, and from the initial description by De Vega, we have learnt that the annuloplasty must be extended to the infero-posterior and supero-posterior parts of the septal portion of the tricuspid annulus, exactly in the same extent as the ring does. This conclusion had been described by Calafiore and colleagues (69).

While the classic De Vega annuloplasty method consisted of a single polypropylene suture line, numerous modifications to the De Vega annuloplasty technique were adopted in our institution:

- The annuloplasty is executed first, before mitral valve replacement, for two reasons:
 - To avoid the distortion of the tricuspid annulus by the metallic prosthesis.
 - To permit the best repair possible while the surgeon is in full concentration

- Other repair techniques are used in conjunction to the annuloplasty as much as needed. For example, we noticed that in case of major annular dilation and leaflet tethering, leaflet flail and prolapse made the coaptation non-satisfactory. The results of the annuloplasty controlled with the saline test were better after reducing the excessive tissue with a pledgeted suture. Moreover, our patients have more organic involvement than reported in other studies (25.7% of our cohort subjects), explaining the need of a more extensive repair.
- The infero-posterior and supero-posterior parts of the septal portion of the tricuspid annulus are always included.
- Ethibond sutures are used. It confers a superior inflammatory reaction with a rapid stabilization in 3 to 4 weeks (70,71).

3. Predictive factors of tricuspid valve repair failure:

Predictors of failure of tricuspid repair in our case series were similar to other published studies:

- Preoperative heart failure signs
- Preoperative dilated right ventricle
- Preoperative right ventricular dysfunction
- Preoperative TR grade 4+
- Preoperative TV annulus diameter > 40 mm
- Severe preoperative pulmonary artery hypertension
- Severe postoperative pulmonary hypertension

These predictive factors are not exclusive to the De Vega repair.

Additional risk factors identified in other studies include:

- Atrial fibrillation
- Left ventricle dysfunction
- Leaflet tethering area

All these factors are linked and have in common reflecting an advanced stage of valvular disease.

3.1. Pulmonary hypertension and tricuspid repair:

This study demonstrated that the preoperative and postoperative severity of pulmonary hypertension did influence mid-term outcome of tricuspid annuloplasty, which is consistent with the results of previous studies of TV surgery (34,72,73).

Pulmonary hypertension can affect tricuspid repair's outcome in two ways:

- Increasing RV pressure that results in dilatation and tricuspid regurgitation. The later increases the RV overload and worsens the degree of regurgitation. This closed circle is unbreakable by tricuspid repair unless pulmonary artery pressure is reduced (74).
- The most interesting mechanism was studied by Anup D. Pant and colleagues (75) who, using an experimental setup, measured the alteration of porcine tricuspid leaflets microstructure in response to pressure increase. He concluded that tricuspid leaflets extracellular matrix alterations induced thickening of the valve in response to pulmonary hypertension and can play a prominent role in the onset and progression of tricuspid valve disease. This could directly impact tricuspid valve repair outcomes in patients with severe pulmonary hypertension.

The use of approaches alleviating pulmonary hypertension could potentially be a part of the effective management strategies for patients after TV annuloplasty.

We didn't find any previous published papers studying the impact of medical treatment of pulmonary hypertension on the outcome of tricuspid repair. However, in a recent multicenter, double blind clinical trial, studying the use of Sildenafil for improving outcomes in patients with corrected valvular heart disease and persistent pulmonary hypertension was published. It discourages the use of Sildenafil for treating pulmonary hypertension after successful correction of valvular disease as it is associated to unfavorable clinical outcomes as compared to placebo

(76). Although the mechanisms leading to worse outcomes of patients taking Sildenafil in this study are necessarily speculative, a chronic increase in pulmonary capillary pressure is the most plausible explanation.

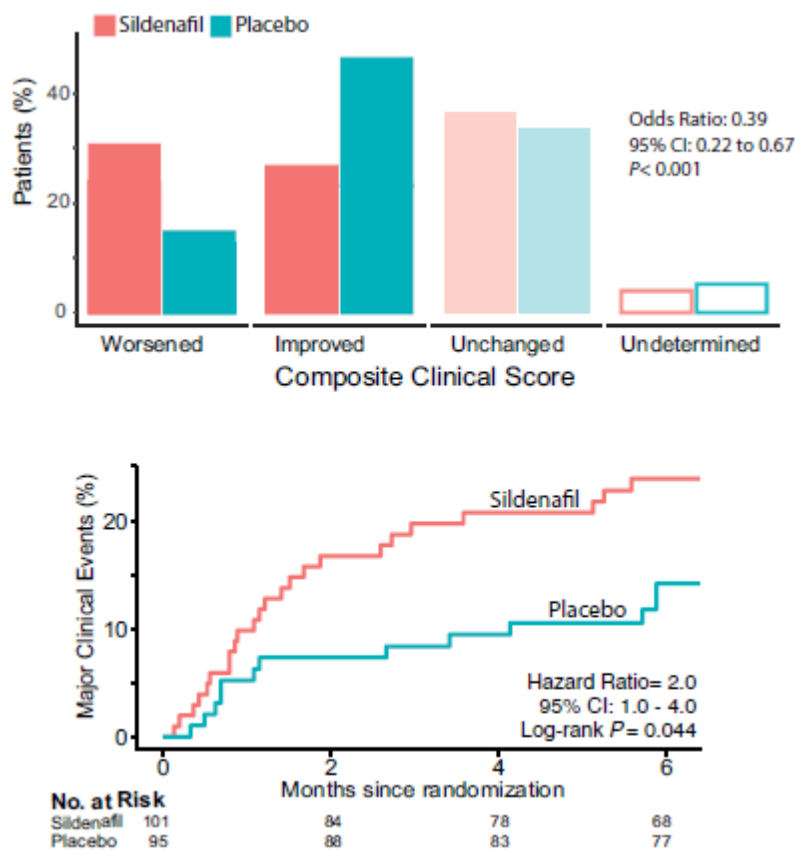


Figure 29: Sildenafil is associated to impaired clinical outcomes, as demonstrated by a higher proportion of patients who worsen their clinical status (upper panel) mainly due to a higher risk of readmission due to heart failure in the following six months (lower panel). (76)

3.2. Other factors impacting tricuspid repair outcome:

Preoperative right ventricular dysfunction is associated with poor outcome after tricuspid repair. While the later improves RV function in some patients, in other cases the dysfunction is so severe that it alters the dynamics of the tricuspid valve causing residual tricuspid regurgitation (77).

Preoperative dilated RV is causes papillary muscles displacement and leaflet tethering rendering tricuspid annuloplasty insufficient. Goro Matsumiya and colleagues recently proposed a surgical method of RV papillary muscles approximation to improve tricuspid valve repair (78).

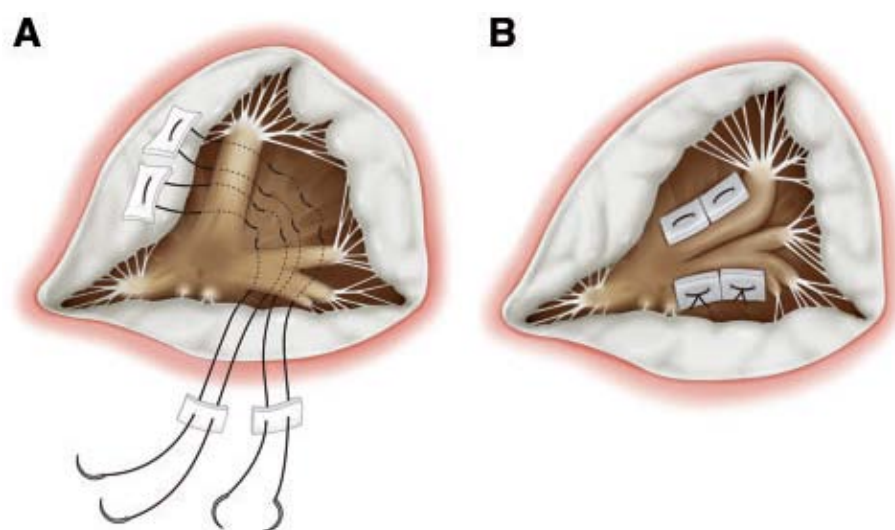


Figure 30: Schema of right ventricular papillary muscle approximation as proposed by Goro Matsumiya and colleagues(78) (A) semi circumferentially placed horizontal mattress sutures (B) the position of papillary muscles after the approximation.

Atrial fibrillation reflects atrial enlargement and high-pressure secondary to pulmonary hypertension, causing tricuspid annular dilatation (79).

3.3. Technical considerations when repairing the tricuspid valve:

The fact that surgeons generally leave the tricuspid repair until the end of the surgical procedure, after left-sided valve repair or even after restarting the heart, suggests that they consider it as a less complex repair that does not require much time. This may be the result of lack of understanding of tricuspid regurgitation mechanisms.

Treating the tricuspid valve as a “forgotten valve” or a “second class valve” subjects it to a higher risk of repair failure.

In our experience, some technical considerations must be respected when repairing the tricuspid valve in general, and when doing a De Vega repair in particular:

- The priority in operative time should be given to the tricuspid valve as:
 - o The repair or replacement of left-sided valves are more standardized.
 - o Additional procedures are often required to optimize tricuspid valve needing more time and concentration.

- A tension should be equally distributed on the antero-posterior part of the annulus. To achieve this, a certain tension should be constantly applied on the end of the suture thread to insure a symmetrical reduction of the annulus.
- The De Vega suture should include the antero-and the postero-septal commissures. Contrarily to what was once believed, the septal segment of the annulus distends too. They should not be considered as a part of the septal annulus.

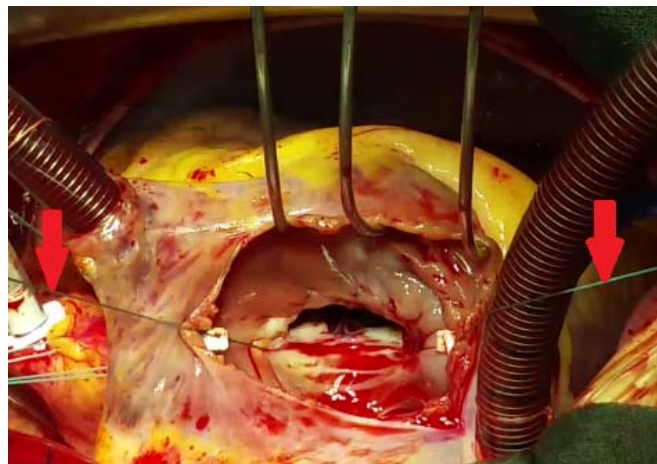


Figure 31: Peroperative view of the De Vega illustrating the continuous tension applied on the thread's ends

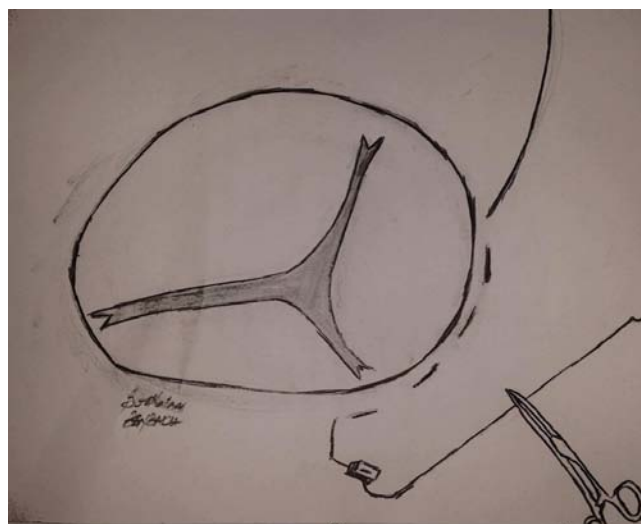


Figure 32: Continuous tension should be applied on the thread's first end by placing a clamp

- The preference should be given to Braided & Coated Polyester Suture when doing a De Vega repair for as it gives a better inflammatory response, a better consequent fibrosis and it is less allergenic. This is a very important concept as demonstrated by many studies.
- The stitches should be deep to prevent the guitar string effect. Yung-Tsai Lee and colleagues (80) demonstrated that Stitch depth within 10 mm when doing a De Vega repair will not injure the right coronary artery or aortic annulus.

The continuous follow-up of our patients is necessary to confirm the impact of the modification of the De Vega annuloplasty on long-term tricuspid repair outcome.



ANNEX

ANNEX 1

I. HISTORY OF HEART VALVE SURGERY:

“One does not completely understand a science completely as long as one did not know the history” Auguste Comte (52).

1. Before the advent of cardiopulmonary bypass:

The history of heart valve surgery runs parallel to the development of heart surgery. The interest in cardiac anatomy, physiology and pathology started since early Greek times. In fact, the role of Hippocrates and Galen in the early development of cardiology is underappreciated (52). And since, a lot of great scientists collaborated to move this knowledge. In the middle ages, the Islamic civilization carried on this path (81). Ibn Sina (Avicenna) gave a logically presented description of heart diseases and was the first to discuss the effects of many cardiac drugs. His book, the “Canon of medicine”, remained the standard textbook in both the east and the west until the 16th century .

In the 15th century, Leonardo Da Vinci’s work highlighted valve function, hemodynamic phases of the cardiac cycle and other anatomic and physiological features of the heart (82).

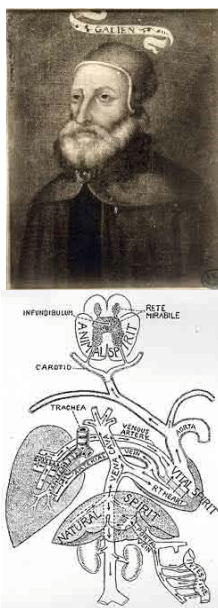


Figure 33: Galen
131 -201



Figure 34: Ibn Sina
980 - 1037

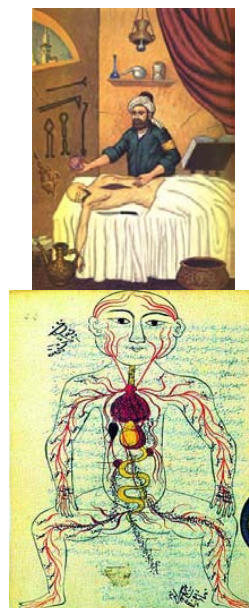


Figure 35: Ibn Nafees
1210 - 1288



Figure 36: Leonardo Da Vinci
1452- 1519

By the 19th and 20th century, great cardiologists had well described heart diseases, including congenital and valvular diseases: anatomical lesions, their symptoms, signs collected by clinical examination and auscultation, and their evolution. All these findings were finally controlled at autopsy (83).

However, cardiac surgery is only almost a decade old. Even to the surgeons of the end of the 19th century, who, thanks to the twin discoveries of anesthesia and antiseptic surgery, were beginning to operate on all the other major organs of the body, heart surgery seemed to remain an impossible dream (84). For so long, the heart was considered by the surgical community to be the “no-go” area. In 1883, Theodor Billroth, a professor of surgery in Vienna and himself a pioneer of modern surgery, wrote: “The surgeon who would attempt to suture a wound of the heart should lose the respect of his colleagues”. And in 1893, Stephen Paget, an illustrious English surgeon, wrote: “surgery of the heart has probably reached the limits set by nature to all surgery. No method, no new discovery can overcome the natural difficulties that attend a wound of the heart” (52).

Ironically, a few months later, Ludwig Rehn, a surgeon at Frankfurt, reported the first successful wound suture of the right ventricle. Ten years later, Rehn reported 124 recorded cases of operations on stab wounds of the heart with a recovery rate of 40%, establishing the possibility of successful surgery on the heart (85).

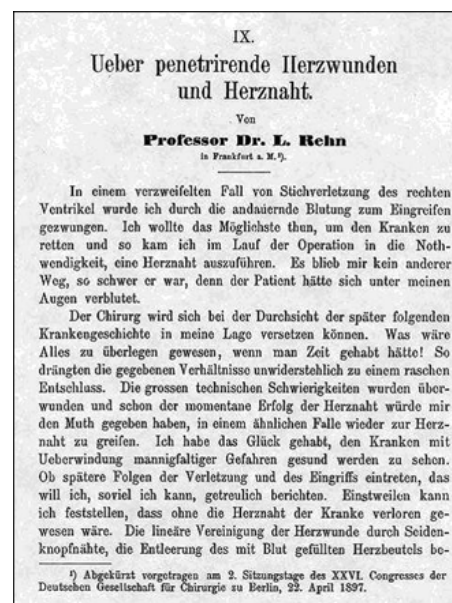


Figure 37: Ludwig Rehn (1849 – 1930) and his original published paper on heart wounds surgery

At the turn of the 19th century, rheumatic fever was an epidemic disease –still is in our context– and cardiologists were confronted with the ineffectiveness of medical treatment in advanced rheumatic valve disease, especially mitral stenosis (86). Sir Lauder Brunton, a British physician, was the first to suggest, in an article published in the Lancet in 1902, that it might be possible to treat mitral stenosis surgically (84,86).

The concept was first tried on animals. In 1923, Elliot Carr Cutler performed the first successful surgery of mitral stenosis on a 12-year-old girl using a trans-ventricularly introduced tenotom. The next five surgeries were not successful and the patients died from surgically induced mitral incompetence (86).



Figure 38: Elliott Carr Cutler, MD

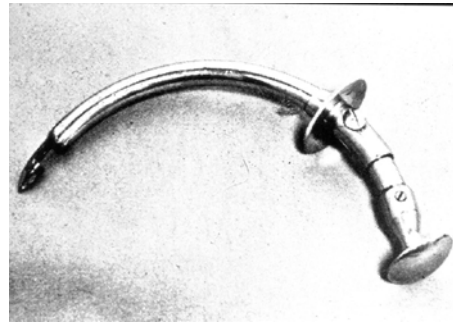


Figure 39: Cardiovalvulotome by Elliott Carr Cutler



Figure 40: First patient to have successful valve surgery, 4 days after the operation

In 1925, Sir Henry Session Souttar proposed a different method and performed the first digital commissurolysis of a mitral stenosis by introducing a forefinger into the left atrium to loosen the fused commissures (84,86).



Figure 41: Henry Session Souttar, MD

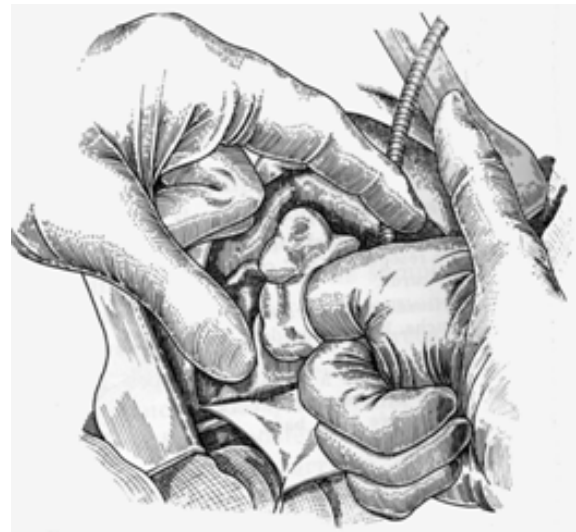


Figure 42: Digital commissurolysis

2. After the advent of cardiopulmonary bypass:

Jon Gibbon's advent of cardiopulmonary bypass in 1953, along with hypothermic cardiac arrest and anticoagulation, opened the possibility of open heart surgery under direct visual

control (84). The technique was first applied to the repair of congenital heart disease. Heart valve surgery developed in a second phase (52,83,84).



Figure 43: Jon Gibbon and the first cardiopulmonary bypass machine (52)

The first artificial valve implantations were performed in 1960. Nina Braunwald, the first female cardio-thoracic surgeon (87,88), and Andrew Morrow implanted a polyurethane heart valve of their own design into the mitral orifice. Although, due to only short-term survival of their patient, the priority was attributed to Albert Starr who implanted a ball cage valve into the mitral position with a long-term survival (83,84,87). Mechanical heart valve replacements in other positions were soon tried.



Figure 44: Nina Braunwald in 1963, the year she was certified by the American Board of Thoracic Surgery (87)



Figure 45: Nina Braunwald (far left) and Andrew G. Morrow (far right) performing surgery (87)



Figure 46: The Braunwald-Morrow mitral valve (87)



Figure 47: Albert Starr (52)



Figure 48: Jean Paul Binet (52)

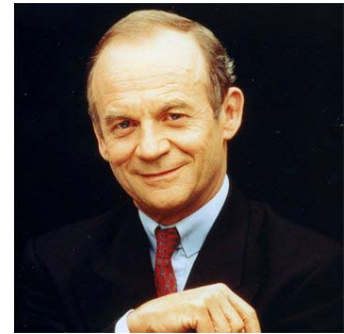


Figure 49: Alain Carpentier (52)

Soon after the first mechanical valve implantation, the era of biological valves started thanks to the work of Jean Paul Binet and then Alain Carpentier. The later carried a tremendous campaign to promote valve-sparing procedures.

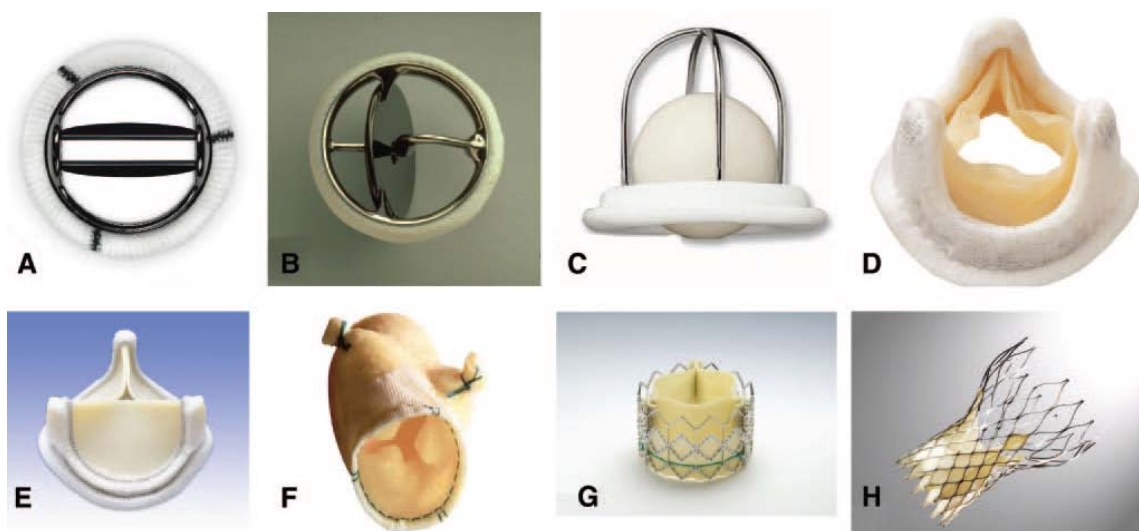


Figure 50: Different types of prosthetic valves (89).

A, Bileaflet mechanical valve (St Jude); B, monoleaflet mechanical valve (Medtronic Hall); C, caged ball valve (Starr-Edwards); D, stented porcine bioprosthesis (Medtronic Mosaic); E, stented pericardial bioprosthesis (Carpentier-Edwards Magna); F, stentless porcine bioprosthesis (Medtronic Freestyle); G, percutaneous bioprosthesis expanded over a balloon (Edwards Sapien); H, self-expandable percutaneous bioprosthesis (CoreValve)

Valvuloplasty is a technique of recent appearance and it owes a major part of its development to the pioneer work of Alain Carpentier. Since 1970, his techniques of valvuloplasty, particularly mitral annuloplasty, have been gradually adopted by all surgical teams. His techniques are regrouped in varied published books.

3. What about the tricuspid valve?

Unfortunately, in the era of developing valve surgery, the tricuspid valve has been considered “A second class valve” and has not received as much attention as the mitral and aortic valve. It has been referred to as “the forgotten valve” (52).

Tricuspid regurgitation (TR) was first described by T.W. King in 1837, who showed that distension of the right ventricle (RV) with water induced considerable tricuspid valve reflux and concluded that “The tricuspid valve is designed to be(come) incompetent” (52).

In 1967, Grodin published an article on the Journal of Cardiothoracic Surgery pointing out the challenge in the decision making and the treatment of the tricuspid valve (90).

Nowadays, it is believed that this valve was poorly understood in the past and was left undertreated. A special consideration is attributed to the tricuspid valve now that its impact in short and long-term prognosis of valvular patients has been proven (52).

The replacement of the tricuspid valve was tried since the advent of biological valves. It is now reserved to cases when the valve is unrepairable because of its high morbidity and mortality. In the other hand, several surgical procedures have been described for the repair of the tricuspid valve.

Kay was the first to apply a reconstructive surgery technique to the tricuspid regurgitation. The Kay annuloplasty consisted of creating a bicuspid valve by excluding the posterior leaflet. However, this technique does not repair the anterior position of the annulus (91).

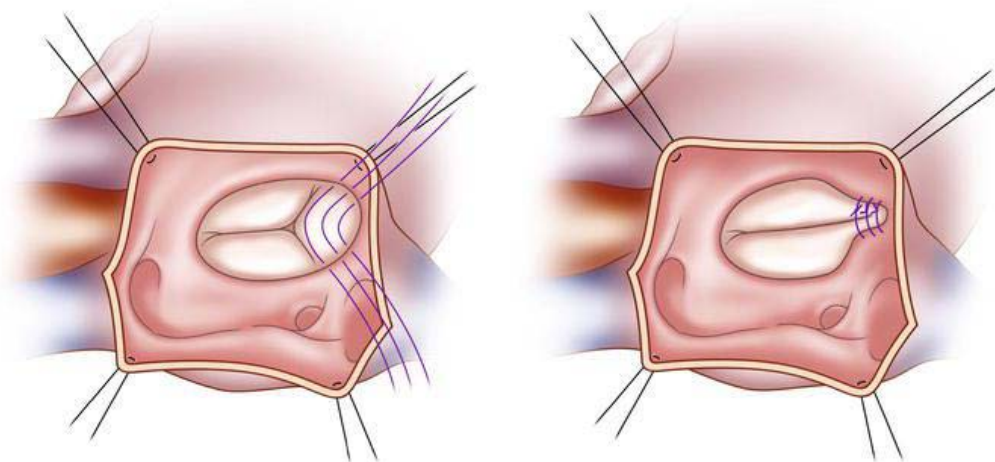


Figure 51: Kay tricuspid valve annuloplasty technique (52)

The De Vega annuloplasty or suture annuloplasty, the subject of our study, was introduced in 1972 by Noberto G. Vega (92). Since then, it has been used extensively with satisfactory long-term results. It was referred to as “the magic stitch” (52). It consists of two semi-circular sutures along the anterior and lateral positions of the tricuspid valve, tied on both sides on pledgets over a 28 or 30 mm dilatator to avoid the purse string effect. This technique has been modified by various surgeons to enhance its effectiveness.

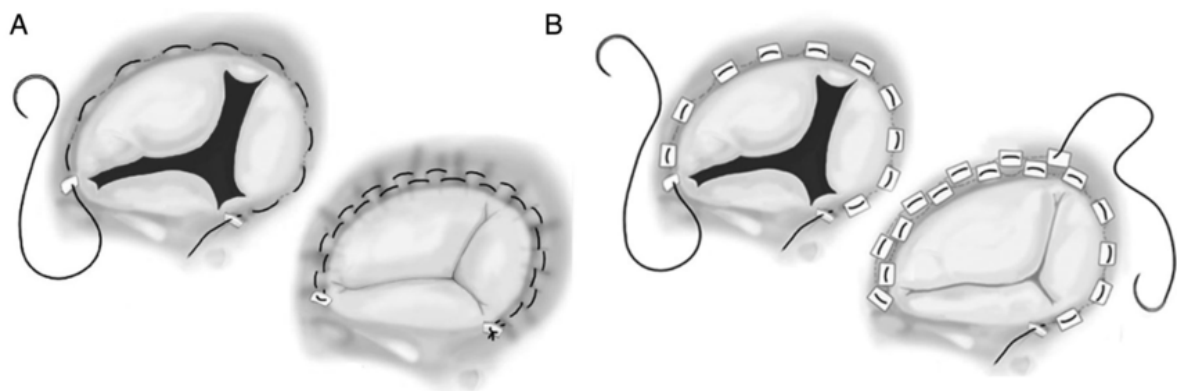


Figure 52: Suture annuloplasty (86): De Vega (A) and modified De Vega annuloplasty (B)

In the same period of the De Vega annuloplasty, exactly in 1974, Carpentier presented the semi-rigid prosthetic ring annuloplasty. He based its design on anatomical and pathological studies (93).



Figure 53: Carpentier-Edwards tricuspid ring (54)

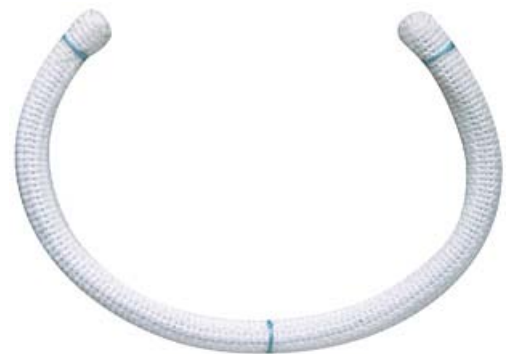


Figure 54: Duran's flexible tricuspid ring (54)

In 1975, Duran introduced a flexible prosthetic ring to reconstruct the native annulus geometry (94).

In recent years other techniques have been proposed: the edge-to-edge “Clover” technique, performed by suturing the free margins of the tricuspid leaflets in conjunction with ring annuloplasty (54).

The innovation continues to this day with transcatheter repair and replacement of the tricuspid valve. These techniques are still under trial but they offer promising solutions for critical patients (95–98).

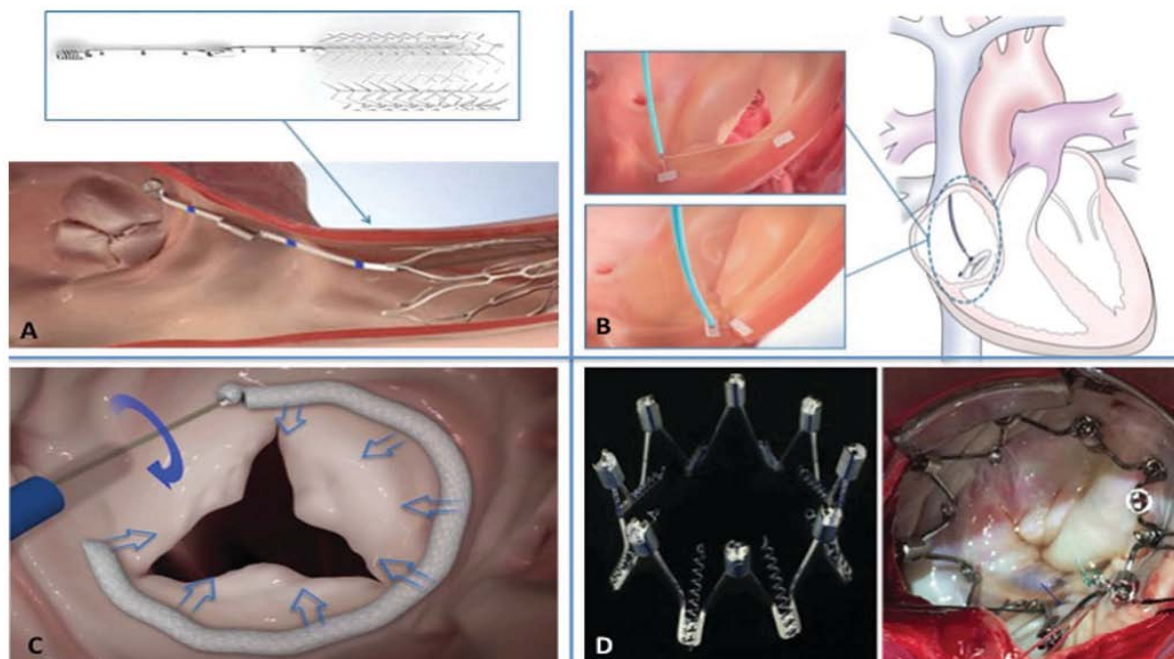


Figure 55: Transcatheter Tricuspid valve repair devices (1)

(A) TriCinch™; (B) Trialign™; (C) Cardioband; (D) Milipede

II. SURGICAL ANATOMY OF THE TRICUSPID VALVE:

The tricuspid valve is part of a complex functional system that also includes the right atrium, the right ventricle and the pulmonary circulation.

With the tricuspid valve exposed, the surgeon observes sequentially the right atrial cavity, the atrio-valvular junction, the tricuspid leaflets, and the subvalvular apparatus.

1. Right atrium:

The right atrium consists of a curved posterior groove continuous with the superior and inferior vena cava, a flat interatrial septum, a trabeculated dome, and the tricuspid valve.

The posterior groove is a smooth wall separated from the trabeculated dome by a ridge of muscle, the crista terminalis, that extends from the superior vena cava to the inferior vena cava.

The interatrial septum has a central shallow depression, the fossa ovalis, and the orifice of the coronary sinus guarded by a valvelike fold, the thebesian valve.

The short and midterm outcomes of the De Vega annuloplasty of the tricuspid valve during the surgical management of rheumatic valvular heart disease

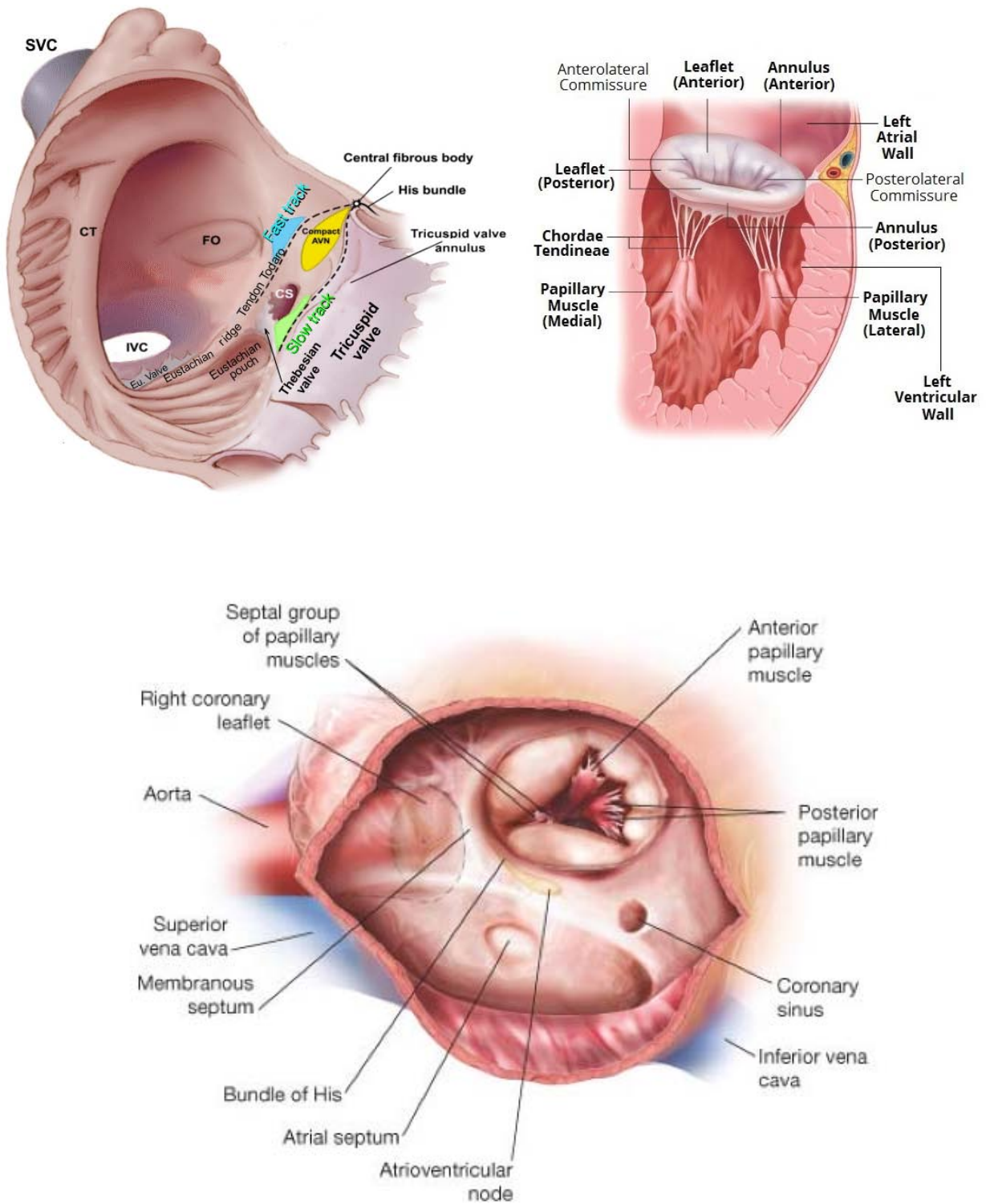


Figure 56: Tricuspid valve anatomy

The Eustachian valve, guards the anterior extremity of the inferior vena cava.

The tendon of Todaro is a fibrous structure formed by an extension of both the Eustachian and thebesian valves. This tendon in conjunction with the orifice of the coronary sinus and the base of the septal leaflet of the tricuspid valve delineate the triangle of Koch. This triangle helps the surgeon locate two important components of the conduction system:

- ❖ The atrioventricular node, at the base of the triangle, and
- ❖ The bundle of His, which extends towards the apex of the triangle. The bundle of His crosses the septal segment of the tricuspid annulus approximately 5 mm from the anteroseptal commissure.

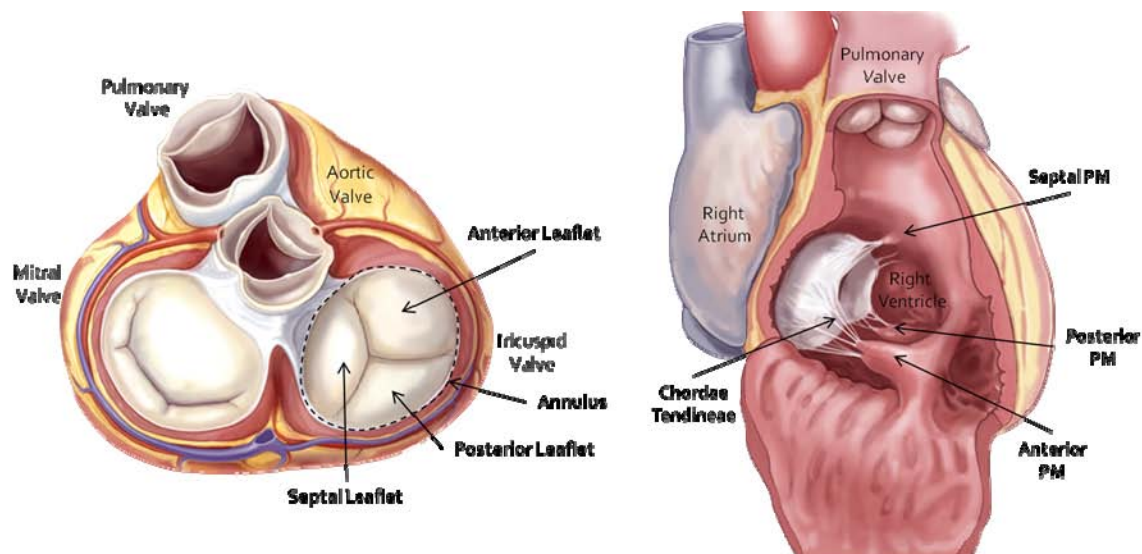


Figure 57: Right Heart Anatomy, showing location of tricuspid valve, annulus, leaflets, chordae tendineae and papillary muscles (99) (Modified from Carpentier's Reconstructive Valve Surgery)

2. The atrio-valvular junction:

The junction between the right atrium and the tricuspid valve is usually well-delineated by the change in color and structure at the junction. While the atrium is pale pink and trabeculated, the valve tissue is yellowish, smooth, and regular.

In pathological valves, the atrio-valvular junction can be masked by endothelial thickening or jet lesions. In these circumstances, the atrio-ventricular junction can be identified by mobilizing the leaflets upward and downward. This mobilization helps visualize the hinge of the leaflets, which in turn allows location of the annulus fibrosus, which attaches three leaflets – anterior, posterior, and septal– separated by three commissures –anteroposterior, posteroseptal, and anteroseptal.

3. The tricuspid annulus

The annulus fibrosus is not visible from an atrial view because it is deeper and 2 mm external to the hinge.

This characteristic has important surgical implications: sutures passed only through the hinge would compromise the motion of the leaflets and would not reach the resistant fibrous body of the annulus; on the contrary, sutures placed 2 mm external to the hinge and orientated towards the ventricle reach the fibrous body of the annulus and preserve the free motion of the leaflets.

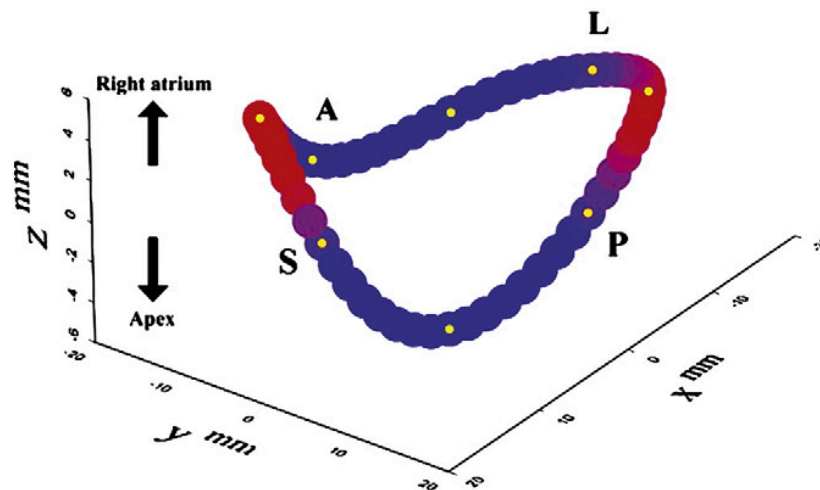


Figure 58: 3D shape of the tricuspid annulus (100)

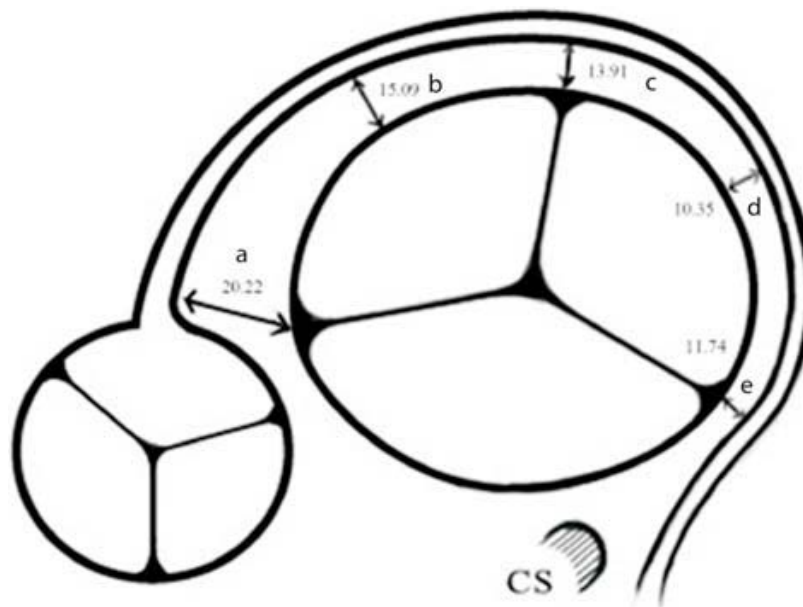


Figure 59: Anatomical relationship between tricuspid annulus, right coronary artery, coronary sinus and aortic annulus (a surgeon's view) (80)

Studies have shown it to be a dynamic structure which contracts during systole and expands during diastole. The tricuspid valve annulus area significantly increases from early to late systole in healthy subjects and experiences differences ranging from 28% to 48% from its minimum to maximum area throughout the cardiac cycle.

The annulus changes its shape as well. The annulus of the tricuspid valve has a 3-dimensional saddle shape, with the annulus becoming more planar during diastole. This saddle shape, or hyperbolic parabola, has been shown to significantly reduce peak leaflet stress in the mitral valve. Rigid structure such as stented prostheses or rigid annuloplasty rings, destroy this configuration and probably negatively impact the function of the right ventricle.

4. The tricuspid valve Leaflets:

The tricuspid valve consists of three leaflets named for their base attachment (septal, anterior, and posterior).

The anterior and posterior leaflets originate from the free wall while the septal leaflet originates from the intraventricular septum.

The leaflets are separated by three clefts or commissures—the anteroseptal, anteromedial and posteroseptal. These clefts do not reach the annulus but delineate small “commissural leaflets”. This is an important surgical point because in cases of fused commissures, their incision should not extend all the way to the annulus, which destroys the commissural leaflets.

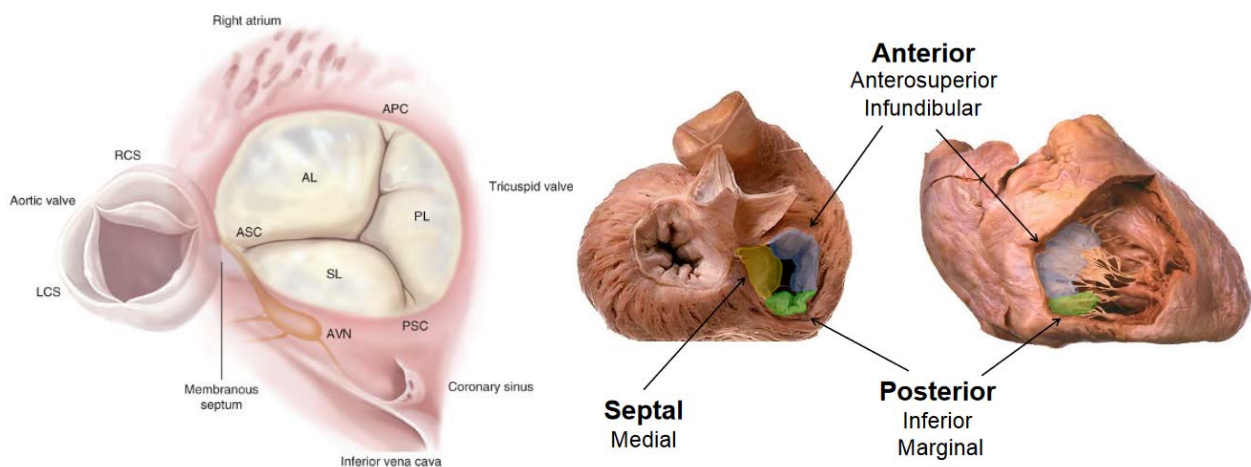


Figure 60: Tricuspid valve leaflets and commissures position (86)

5. The tricuspid subvalvular apparatus: The Chordae Tendineae and Papillary Muscles:

The tricuspid subvalvular apparatus consists of anterior, posterior and septal papillary muscles, and their true chordae tendineae. It is similar to the mitral valve but has greater variability.

Each leaflet has chordal attachments to one or more papillary muscles. The anterior papillary muscle, the most prominent, provides chordae to the anterior and posterior leaflets, and the medial papillary muscle provides chordae to the posterior and septal leaflets. The posterior papillary muscle is smaller and is missing in 20% of healthy subjects. The septal wall may give chordae directly to the anterior and septal leaflets, without a specific septal papillary muscle. These multiple chordal attachments are important mediators of tricuspid regurgitation, as they impair proper leaflet coaptation in the setting of RV dysfunction and adverse remodeling.

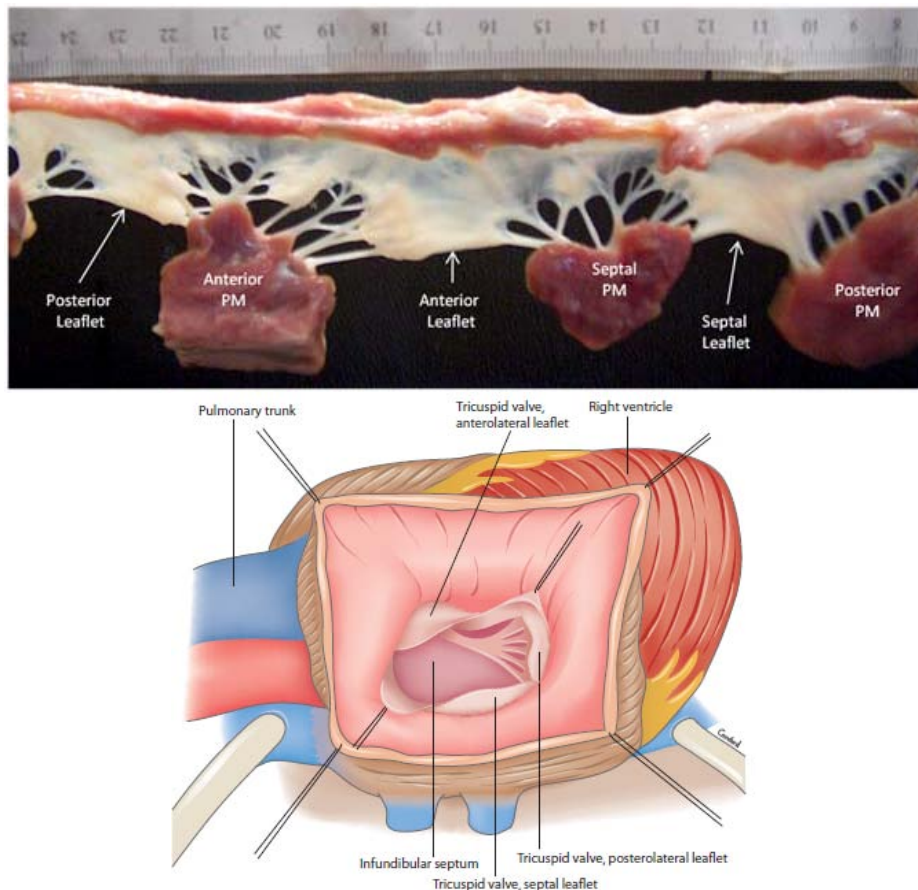


Figure 61: Explanted porcine valve, showing leaflet and PM locations relative to another Exposure of the inferior papillary muscle, schematic drawing (86)

III. EMBRYOLOGY OF THE TRICUSPID VALVE:

The heart is the first organ to function during embryonic development. It forms initially as a primitive tube composed of a myocardial cell layer surrounding an endocardial endothelial cell layer.

Formation of the atrioventricular valves begins during the fifth embryonic week

as swellings form the atrioventricular canal. In response to local signaling from the underlying myocardium, the cardiac jelly between the myocardium and endocardium accumulates hydrated hyaluronic acid, proteoglycans, and other substances forming block-like endocardial cushions. This is the first indication of valve development during vertebrate embryogenesis.

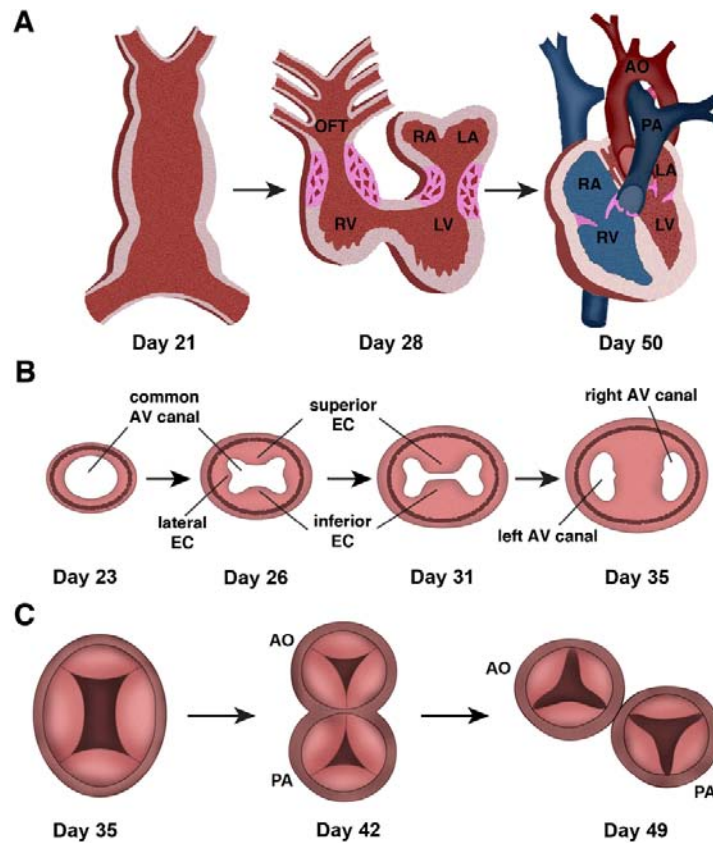


Figure 62: Cardiac valve development (101)

The two principal cushions, the superior and inferior cushions, form first, followed in a few days by smaller left and right lateral cushions. At this point, the endocardial cushions function efficiently to prevent retrograde flow of blood.

While cushions are developing, the atrio-ventricular canal and its accompanying portions are gradually moving toward the right, so that the tricuspid orifice first straddles the developing ventricular septum and then opens completely into the right ventricle.

At the completion of septation of the atria and ventricles, the greater part of the atrioventricular valve leaflet tissue has not begun to develop.

At about the time of formation of the septum primum, the primitive canal is fashioned into mitral and tricuspid orifices, with responding valves, by the fusion of the endocardial cushions.

The endocardial cushions, the dextrodorsal bulbar cushion and the connections of the endocardial cushions with the muscular trabeculae of the ventricular myocardium fashion the formation of the atrio-ventriculo-valvular apparatus.

Not only is valve maturation molecularly compartmentalized, it also bears remarkable similarity to skeletal development. The adult trilaminar arrangement of the valve leaflets develops postnatally. Collagen fibers become densely packed on the ventricular side of the leaflet forming the fibrosa which strengthens the leaflet and resists stretching. The atrial surface of the leaflets contain numerous elastic fibers. The central layer or spongiosa is rich in glycosaminoglycans and is thought to absorb energy associated with closure.

IV. TRICUSPID VALVE REGURGITATION:

The tricuspid valve was often ignored by cardiologists and surgeons because of its unique characteristics:

- 1- With the exception of infective endocarditis, it is rarely affected in isolation. Most often, the prominent impact of other diseased valves minimizes its importance.
- 2- Located at the entrance of the heart, its symptomatology is primarily extracardiac and is often silent.
- 3- Its behavior is closely related to the function of the right ventricle. In most cases, tricuspid regurgitation is secondary to right ventricular failure.
- 4- It follows the dictates of the mitral valve. Resolution of the mitral problem is often followed by improvement in the degree of tricuspid regurgitation.
- 5- Because it works in a low-pressure system, it is difficult to evaluate its preoperative importance and assess the value of different surgical techniques.

1. Definition:

Tricuspid regurgitation is defined by the presence of a retrograde flow from the right ventricle to the right atrium during systole.

Trivial amounts of tricuspid regurgitation are seen in the majority of healthy adults. As in all regurgitant valve lesions, the severity is most commonly graded on a semiquantitative scale. This scale begins at 0 (none), followed by 'trivial' (physiologic), 1+ (mild), 2+ (moderate), 3+ (moderate-severe), and 4+ (severe).

The tricuspid valve function depends on interactions between the tricuspid annulus, valvular leaflets, papillary muscles, chords, the right atrium and the right ventricle. Any congenital or acquired abnormality affecting one of these structures disturbs the valve's function and leads to tricuspid regurgitation.

Functional regurgitation is a lesion produced by a disturbance of the efficient coordination of all the elements of the valve complex such as caused by annular dilatation, elongation of the valve cords, perforation of the leaflet.

Tricuspid valve regurgitation is generally classified as primary or secondary (functional). The latter results from left heart disease. Clinically, secondary, or functional tricuspid regurgitation is the most common condition.

2. Etiology of tricuspid regurgitation:

Tricuspid valve diseases are primarily divided into two etiological groups: functional and organic.

2.1. Functional tricuspid regurgitation:

Functional tricuspid regurgitation is a complex valvular lesion in which the tricuspid valve leaks during systole in the presence of structurally normal leaflets and chordae. Functional tricuspid regurgitation is considered more of a “ventricular” disease (100). It is by far the most frequent cause of tricuspid valve dysfunction.

This condition is commonly observed in patients with mitral valve disease.

It can also be observed in a variety of diseases producing pulmonary hypertension and/or right ventricle dysfunction.

The most important characteristic of functional tricuspid regurgitation is its variability in a given patient, depending upon cardiac output, ventricular contractility, blood volume and medical treatment.

This variability makes difficult the assessment of the severity of tricuspid regurgitation and therefore the decision of repair.

2.2. Organic tricuspid regurgitation:

Organic tricuspid regurgitation mainly results from primary involvement of the tricuspid valve. Rheumatic valvular disease is prevalent in developing countries whereas degenerative valvular disease is common in western countries.

Organic tricuspid regurgitation may also result from primary myocardial diseases such as myocardial infarction and dilated cardiomyopathy.

Table LIII: Characteristics of functional and organic tricuspid regurgitation

Functional TR	Organic TR
<ul style="list-style-type: none">▪ No organic lesion▪ Potentially reversible TR	<ul style="list-style-type: none">▪ Valvular or myocardial lesions▪ Non-reversible TR

3. Functional classification of tricuspid valve regurgitation:

Based on leaflet motion, three types of valve dysfunction are described depending on whether leaflet motion is normal (type I), excessive (type II), or restricted (type III).

3.1. Type I: Tricuspid valve regurgitation with normal leaflet motion

The course of leaflets has normal amplitude and the regurgitation usually results from annular dilatation. Another cause is leaflet perforation.

3.2. **Type II: Tricuspid valve regurgitation with excess leaflet motion (leaflet prolapse)**

The motion of one or more leaflets is increased with the free edge of the leaflet overriding the plane of the orifice during systole. Leaflet prolapse may be a result either of chordae rupture or elongation or of papillary muscle rupture or elongation. The most common cause of type II regurgitation are bacterial endocarditis and trauma.

3.3. **Type III: restricted leaflet motion**

Leaflet motion is limited either during diastole (type IIIa) or during systole (type IIIb)

- **Type IIIa:** typically seen in rheumatic valve disease. Leaflet motion is restricted due to commissure fusion, chordae thickening and fusion.
- **Type IIIb:** displays a leaflet tethering usually attributable to ventricular dilatation and papillary muscle displacement.

4. **Pathophysiology of functional tricuspid regurgitation:**

Functional tricuspid regurgitation occurs due to loss of leaflet coaptation as a result of two pathologies: dilatation of the tricuspid annulus and/or leaflet tethering, both of which occur due to right ventricular remodeling and dilatation as a result of raised pulmonary artery pressures from left sided heart valve disease.

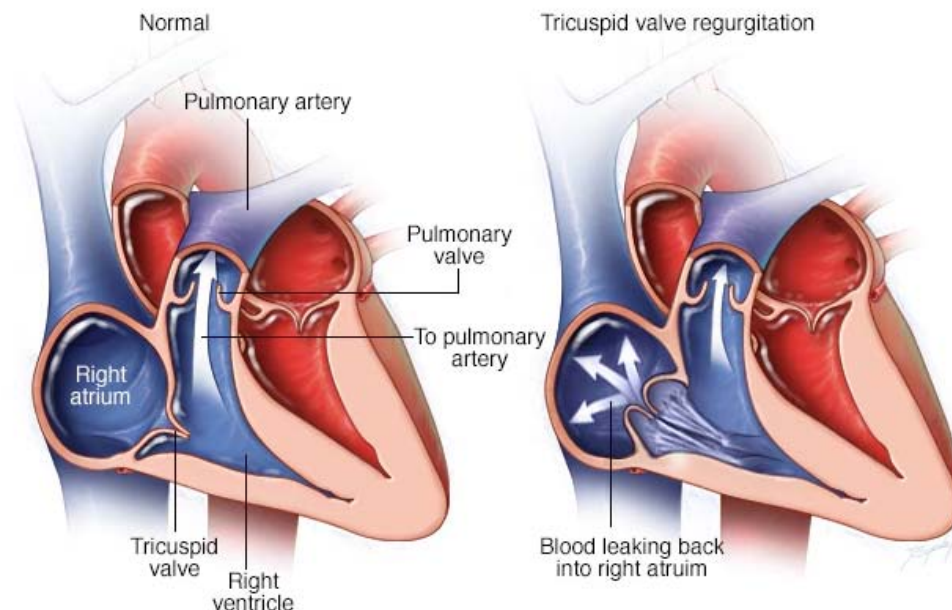


Figure 63: Tricuspid regurgitation (65)

These two pathologies may co-exist, with one pathology leading to the other, or they may be distinct. Tricuspid annular dilatation can also be caused by long standing atrial fibrillation.

It is referred to as functional as the leaflets are normal in morphology but their function is impaired due to dilatation of the tricuspid annulus which pulls them apart, or right ventricular dilatation, elongation and altered geometry which displaces the papillary muscles laterally and apically, tethering the leaflets and preventing adequate leaflet coaptation.

4.1. Annular dilatation:

Most of the focus has been directed at investigating how the annulus dilates in the diseased condition. It is believed to be a better indicator of TV pathology than TR since patients with no TR initially and a dilated annulus developed TR.

Only the anterior and posterior segments of the annulus can dilate since they are the only segments on the free wall.

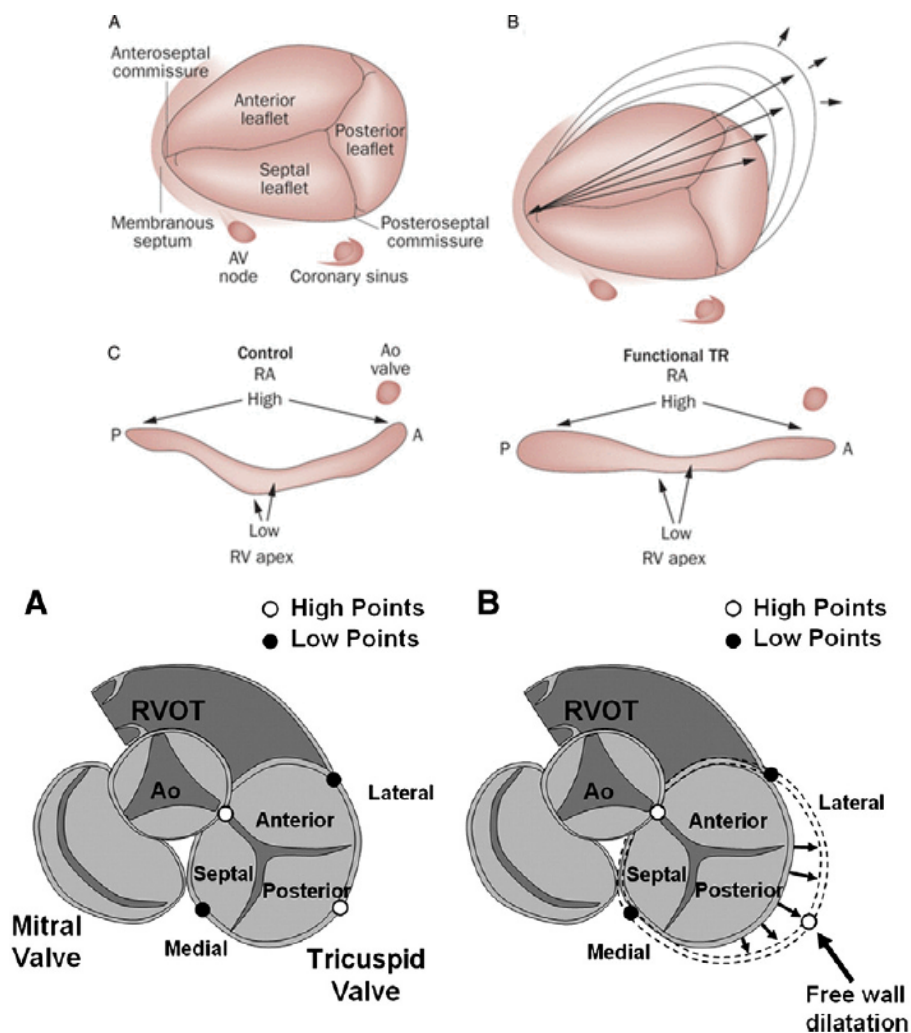


Figure 64: Normal TV (A) versus pathological TV (B) with annular dilatation
Annular dilatation occurs mainly along the anterior and posterior portions of the tricuspid annulus (100)

It is not just the increase in annulus size which is believed to cause TR, but the loss of the dynamic function of the annulus. The loss of systolic annular contraction and the 3D geometry in TR patients, with the annulus becoming planar, may be an important pathogenesis.

Although it is known that TR occurs with annular dilatation it is not known at which amount of dilatation TR begins.

4.2. Papillary muscles displacement and Leaflet Tethering:

Another effect of right ventricular dilation is papillary muscle displacement which leads to leaflet tethering and possibly leaflet prolapse depending on the type of dilatation.

Restoration of the annulus to its original size is not always enough to eliminate TR completely.

It may be a combination of these factors: annular dilatation and PM displacement in conjunction with nonspecific designed devices which do not adequately correct TR. It is our incomplete understanding of the normal and diseased anatomy of the valvular and subvalvular components of the tricuspid valve which do not allow us to completely repair the valve and remove all current and possibility for recurrent TR.

5. Diagnosis of functional tricuspid regurgitation:

5.1. Clinical diagnosis:

TR is most often diagnosed at the time of echocardiographic evaluation of left-side heart disease or evaluation of right heart failure.

TR is relatively well tolerated and patients may remain asymptomatic even with TR of moderate or severe degree.

When symptoms appear, patients may complain of asthenia, fatigue or decreased exercise tolerance as a result of lower cardiac output. They may also present with signs of elevated right atrial pressure, such as peripheral edema and abdominal fullness, congestive hepatomegaly and ascites.

Physical examination findings include jugular venous distension with a prominent v-wave, hepatomegaly and, rarely, a soft systolic regurgitant murmur can be heard that increases during inspiration owing to the increased venous return. Atrial fibrillation as a result of right atrial enlargement is common.

The holosystolic murmur typical of TR, when present, should prompt echocardiographic assessment. The respiratory variation of the murmur (Rivero-Carvalho's sign) occurs because inspiration increases annular diameter and leads to valvular tenting. As a result, the regurgitant volume more than doubles on average during inspiration. This leads to two clinical implications:

1. As TR varies over the respiratory cycle, multiple measurements during inspiration and expiration should be performed.
2. Clinical optimization of loading conditions may significantly decrease TR severity.

5.2. Imaging of tricuspid regurgitation:

a. Chest radiography:

Chest radiography is of limited utility. We can note:

- Marked cardiomegaly associated with prominent right-heart borders
- Evidence of elevated right atrial pressure may include distention of the azygous vein and pleural effusions
- Ascites with diaphragmatic elevation may be present
- Pulmonary arterial hypertension is common

There are no specific findings which suggest a diagnosis of TV disease.

b. Transthoracic echocardiography:

Two-dimensional echocardiography combined with spectral and color flow Doppler is now the main modality for detection and quantitation of TR.

“Physiological” FTR is associated with normal valve leaflet morphology and normal RV and atrial size. The color jet is localized in a small region adjacent to valve closure (usually < 1 cm), is thin, central, and often is limited to early-systole. However, the precise threshold at which FTR transitions from physiological to abnormal remains uncertain.

When TR is detected by color Doppler and may be abnormal or ‘pathological’ (moderate or more severe), comprehensive description of leaflet morphology and of the pathophysiological mechanisms underlying TR is warranted.

Color flow Doppler and spectral Doppler are highly sensitive for TR detection and generally provide good semi-quantitative assessment of its severity. Tricuspid regurgitation detection by color flow imaging uses parasternal (tricuspid inflow view and short-axis view at great vessels level), apical, or subcostal (four chamber view) approaches. Regurgitant jet area correlates roughly with TR severity.

In clinical practice, visual estimate rather than actual planimetry is the basis of TR severity assessment, which may participate to the vagueness of the qualitative approach.

Detection of eccentric jets adhering, swirling, and reaching the posterior wall of the right atrium suggests organic rather than functional TR and thereby a rather voluminous regurgitation.

Doppler evaluation of TR, despite its simplicity, is a source of errors, is limited by technical and hemodynamic factors and therefore it is not recommended to assess TR severity when it is more than mild. A more quantitative FTR assessment is provided by vena contracta width and proximal isovelocity surface area (PISA) measurements.

Vena contracta represents the cross-sectional area of the blood column as it leaves the regurgitant orifice; it thus reflects the regurgitant orifice area. Averaging measurements over at least 2—3 beats are recommended. Vena contracta width > 6.5 mm is usually associated to severe TR.

Proximal isovelocity surface area radius measurement is by itself a good indicator of severity of regurgitation, but complete application of the method is preferable to obtain physiological information on TR. The PISA radius is measured at midsystole using the first aliasing. Qualitatively, a TR PISA radius > 9 mm at a Nyquist limit of 28 cm/s has been associated with the presence of significant TR (corresponding to an effective regurgitant orifice area ≥ 40 mm² and a regurgitant volume ≥ 45 mL, the quantitative thresholds for severe TR), whereas a radius < 5 mm suggests mild regurgitation.

Finally, a comprehensive echocardiographic assessment of patients with FTR should include measurement of RV size and function.

Table LIV: Recommendations for the echocardiographic assessment of native valvular regurgitation: an executive summary from the European Association of Cardiovascular Imaging (Lancellotti et al) (102)

Parameters	Mild	Moderate	Severe
Grading the severity of TR			
Qualitative			
Tricuspid valve morphology	Normal/abnormal	Normal/abnormal	Abnormal/flail/large coaptation defect
Colour flow TR jet ^a	Small, central	Intermediate	Very large central jet or eccentric wall-impinging jet
CW signal of TR jet	Faint/parabolic	Dense/parabolic	Dense/triangular with early peaking (peak < 2 m/s in massive TR)
Semi-quantitative			
VC width (mm) ^a	Not defined	< 7	> 7
PISA radius (mm) ^b	≤ 5	6–9	> 9
Hepatic vein flow ^c	Systolic dominance	Systolic blunting	Systolic flow reversal
Tricuspid inflow	Normal	Normal	E-wave dominant (≥ 1 m/s) ^d
Quantitative			
EROA (mm ²)	Not defined	Not defined	≥ 40
R Vol (mL)	Not defined	Not defined	≥ 45
+ RA/RV/IVC dimension ^e			

CW: continuous wave; EROA, effective regurgitant orifice area; RA: right atrium; RV: right ventricle; R Vol, regurgitant volume; TR: tricuspid regurgitation; VC, vena contracta. a: At a Nyquist limit of 50–60 cm/s; b: Baseline Nyquist limit shift of 28 cm/s; c: Unless other reasons of systolic blunting (atrial fibrillation, elevated RA pressure); d: In the absence of other causes of elevated RA pressure. e: Unless for other reasons, the RA and RV size and IVC are usually normal in patients with mild TR. An end-systolic RV eccentricity index .2 is in favour of severe TR. In acute severe TR, the RV size is often normal. In chronic severe TR, the RV is classically dilated. Accepted cut-off values for non-significant right-sided chambers enlargement (measurements obtained from the apical four-chamber view): Mid-RV dimension ≤ 33 mm, RV end-diastolic area ≤ 28 cm², RV end-systolic area ≤ 16 cm², RV fractional area change .32%, and maximal 2D RA volume ≤ 33 mL/m².

Table LV: Echocardiographic parameters of severe tricuspid regurgitation (TR) (102)

Doppler derived parameters	Indirect parameters
<ul style="list-style-type: none"> ▶ Color Doppler regurgitant flow >30% of the area of the right atrium ▶ Dense/triangular flow with early systolic peak (maximum velocity <2 m/s in massive TR) ▶ Vena contracta width ≥ 7 mm ▶ PISA in tricuspid regurgitation <ul style="list-style-type: none"> - PISA* radius >9 mm at Nyquist limit of 28 cm/s - EROA* ≥ 40 mm² or regurgitant volume ≥ 45 mL ▶ Systolic flow reversal in hepatic vein flow ▶ Tricuspid inflow (E-wave) ≥ 1 m/s 	<ul style="list-style-type: none"> ▶ Abnormal/flail/large coaptation defect (tenting area >1 cm²) ▶ Inferior vena cava ≥ 21 mm with <50% collapse ▶ RV enlargement (RV eccentricity index >2) * <ul style="list-style-type: none"> *Obtained by dividing the longest right lateral distance by the distance connecting the ventricular septum and the RV free wall. ▶ PISA, proximal isovelocity surface area; EROA, effective regurgitant orifice area.

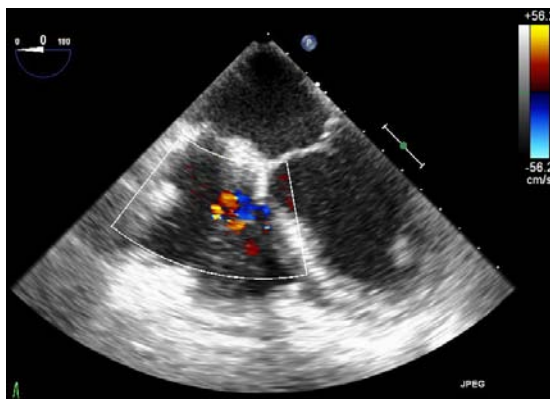


Figure 65: Trace tricuspid regurgitation on color flow Doppler (103)

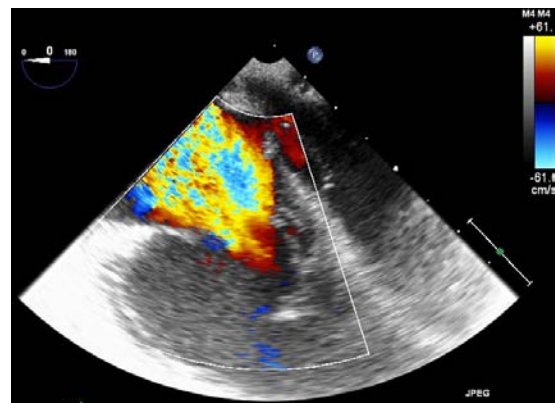


Figure 66: Severe tricuspid regurgitation on color flow Doppler (103)

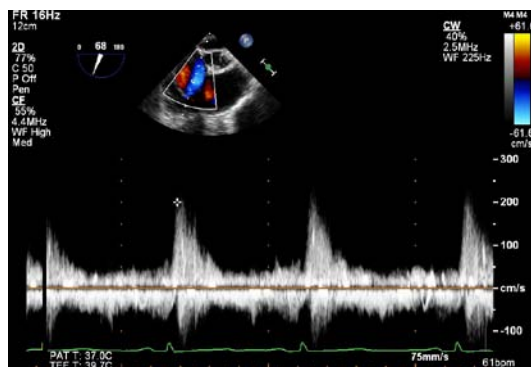


Figure 67: Mild tricuspid regurgitation on continuous wave Doppler (103)

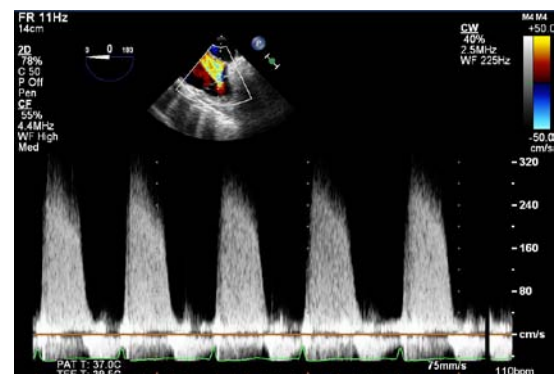


Figure 68: Severe tricuspid regurgitation on continuous wave Doppler (103)

c. Transesophageal echocardiography and 3D echocardiography:

Transesophageal echocardiography is only indicated when it is not possible to assess the etiology of TR by TTE because of poor echocardiographic window.

Additionally, transesophageal echocardiography is indicated in patients with left prosthetic valves and TR to ensure normal function before surgery of the tricuspid valve. It is also more useful intraoperatively, especially to assess the final result of repair.

In recent years, real-time 3D TTE has become a basic tool for the assessment of tricuspid valve pathology, since it permits simultaneous visualization of the three leaflets, the commissures and the anatomy of the annulus in a short-axis view.

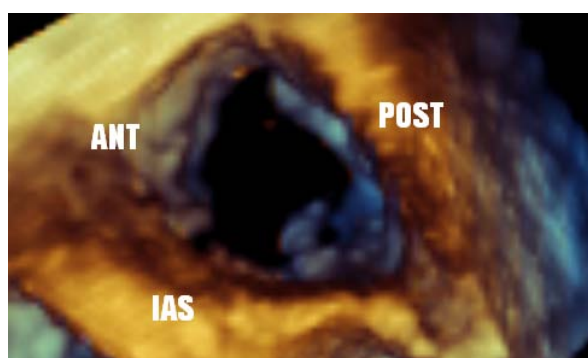


Figure 69: 3D imaging of the tricuspid valve (right atrial view).(103).

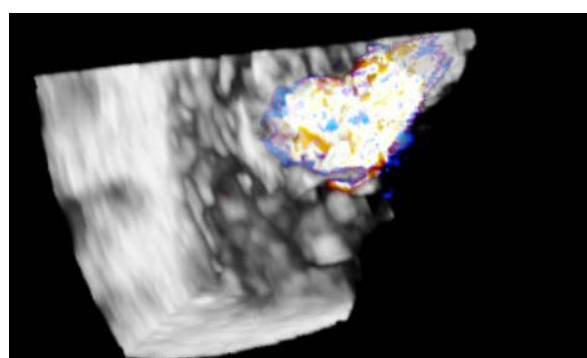


Figure 70: 3D color of large central tricuspid regurgitation jet.(103).

d. Cardiac catheterization:

Before the introduction of 2DE and Doppler echocardiography, cardiac catheterization was the main imaging modality to confirm the presence and severity of TR. The diagnosis of TR posed a greater challenge, as selective angiography into the RV would often distort the TV and catheter position in the RV would be difficult to maintain.

At present, diagnostic cardiac catheterization should rarely, if ever, be undertaken for the diagnosis or quantitation of TV disease alone.

e. Cardiac magnetic resonance:

In the absence of specific contraindications, when echocardiographic imaging is limited by a suboptimal acoustic window, cardiac magnetic resonance is the technique of choice to assess patients with significant TR.

Cardiac magnetic resonance is not limited by acoustic window and can image the whole heart in any plane providing excellent myocardial definition.

Cardiac magnetic resonance is currently the gold standard for the assessment of RV morphology and function, and accurate assessment of the RV is crucial to understand the underlying mechanisms and address management in patients with TR even if no threshold value of RV volume has been validated by its prognostic value with regard to TR.

It can also be used to assess the configuration of TA.

6. Management of tricuspid regurgitation:

6.1. Medical management:

For patients in whom tricuspid regurgitation is secondary to left-sided heart failure, treatment centers on adequate control of fluid overload and failure symptoms (eg, diuretic therapy). Patients should be instructed to reduce their intake of salt. Elevation of the head of the bed may improve symptoms of shortness of breath.

Digitalis, diuretics (including potassium-sparing agents), angiotensin-converting enzyme (ACE) inhibitors, and anticoagulants are all indicated in the care of these patients. Antiarrhythmics can be added to control atrial fibrillation.

6.2. Surgical management:

a. Indications and guidelines:

The timing of surgical intervention remains controversial, mostly due to the limited data available and their heterogeneous nature (see table of recommendations for indications for tricuspid valve surgery and Flowchart).

Surgery should be carried out sufficiently early to avoid irreversible RV dysfunction.

In severe primary tricuspid regurgitation, surgery is not only recommended in symptomatic patients but should also be considered in asymptomatic patients when progressive RV dilatation or decline of RV function is observed. Although these patients respond well to diuretic therapy, delaying surgery is likely to result in irreversible RV damage, organ failure and poor results of late surgical intervention.

In secondary tricuspid regurgitation, adding a tricuspid repair, if indicated, during left-sided surgery does not increase operative risk and has been demonstrated to provide reverse remodeling of the RV and improvement of functional status even in the absence of substantial tricuspid regurgitation when annulus dilatation is present. It should therefore be performed liberally.

Reoperation on the tricuspid valve in cases of persistent tricuspid regurgitation after mitral valve surgery carries a high risk, mostly due to the late referral and the consequently poor clinical condition of patients. To improve the prognosis of patients in this challenging scenario, the treatment of severe late tricuspid regurgitation following left-sided valve surgery should be considered earlier, even in asymptomatic patients, if there are signs of progressive RV dilatation or decline in RV function and in the absence of left-sided valve dysfunction, severe RV or LV dysfunction and severe pulmonary vascular disease/hypertension.

If possible, valve repair is preferable to valve replacement. Annuloplasty, is key to surgery for secondary tricuspid regurgitation. Valve replacement should be considered when the tricuspid valve leaflets are significantly tethered and the annulus is severely dilated. In the presence of trans-tricuspid pacemaker leads, the technique used should be adapted to the patient's condition and the surgeon's experience. Percutaneous repair techniques are in their infancy and must be further evaluated before any recommendations can be made.

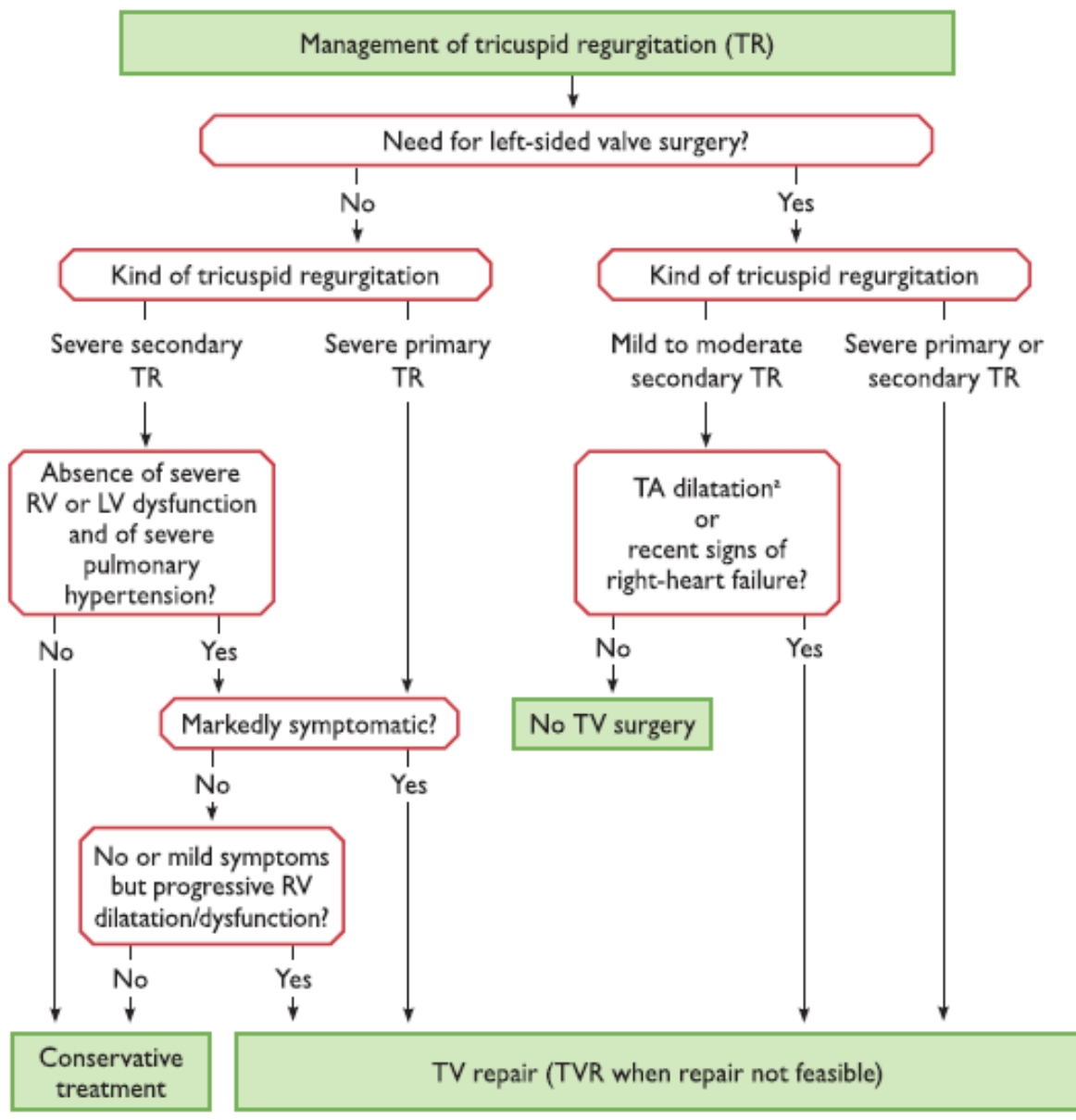


Figure 71: Indications for surgery in tricuspid regurgitation (104).
(2017 ESC/EACTS Guidelines for the management of valvular heart disease)

LV = left ventricular; RV = right ventricular; TA = tricuspid annulus; TR = tricuspid regurgitation; TV = tricuspid valve; TVR = tricuspid valve replacement. TA \geq 40mm or $>$ 21 mm/m².

Recommendations	Class ^a	Level ^b	Recommendations	Class ^a	Level ^b
Recommendations on primary tricuspid regurgitation			Recommendations on secondary tricuspid regurgitation		
Surgery is indicated in patients with severe primary tricuspid regurgitation undergoing left-sided valve surgery.	I	C	Surgery is indicated in patients with severe secondary tricuspid regurgitation undergoing left-sided valve surgery.	I	C
Surgery is indicated in symptomatic patients with severe isolated primary tricuspid regurgitation without severe RV dysfunction.	I	C	Surgery should be considered in patients with mild or moderate secondary tricuspid regurgitation with a dilated annulus (≥ 40 mm or > 21 mm/m ² by 2D echocardiography) undergoing left-sided valve surgery.	IIa	C
Surgery should be considered in patients with moderate primary tricuspid regurgitation undergoing left-sided valve surgery.	IIa	C	Surgery may be considered in patients undergoing left-sided valve surgery with mild or moderate secondary tricuspid regurgitation even in the absence of annular dilatation when previous recent right-heart failure has been documented.	IIb	C
Surgery should be considered in asymptomatic or mildly symptomatic patients with severe isolated primary tricuspid regurgitation and progressive RV dilatation or deterioration of RV function.	IIa	C	After previous left-sided surgery and in absence of recurrent left-sided valve dysfunction, surgery should be considered in patients with severe tricuspid regurgitation who are symptomatic or have progressive RV dilatation/dysfunction, in the absence of severe RV or LV dysfunction and severe pulmonary vascular disease/hypertension.	IIa	C

Figure 72: Indications for tricuspid valve regurgitation surgery (104)
(2017 ESC/EACTS Guidelines for the management of valvular heart disease)

2D = two-dimensional; LV = left ventricular; PMC = percutaneous mitral commissurotomy; RV = right ventricular.

a: Class of recommendation. b: Level of evidence. c: Percutaneous balloon valvuloplasty can be attempted as a first approach if tricuspid stenosis is isolated. d: Percutaneous balloon valvuloplasty can be attempted if PMC can be performed on the mitral valve.

b. Surgical operative techniques:

Only exceptionally will the TV need to be replaced as a first procedure, because the valve tolerates well a less-than-perfect repair (even in many cases of organic rheumatic disease, infective endocarditis, etc.). Hence, annuloplasty, when feasible, is the surgery of choice.

b.1. De Vega annuloplasty:

Dr. Norberto de Vega, described that “the tricuspid regurgitation is usually caused by a selective expansion of the part of the tricuspid annulus closely related to the free wall of the right ventricle (RV), area in which the anterior and posterior leaflets of the tricuspid apparatus are inserted. Hence, he devised the operation that carries his name and was then adopted by the majority of the surgeons.

Classified as a selective, adjustable and permanent annuloplasty, it consists of a double continuous suture plication of the annulus, commenced anchored to the right fibrous trigone, at

the level of the antero-septal commissure, and run along the annulus, ending at the level of the postero-septal commissure, both ends supported with Teflon pledgets.

The procedure is simple and reproducible and only implies one important technical consideration, which consists of avoiding the right coronary artery which runs close to the anterior portion of the annulus.

The classic procedure was often complicated by the 'guitar string syndrome' which resulted of the suture tearing from the tissues. To avoid this complication, in 1983 a simple modification of the De Vega technique was described which consists of the interposition of a small Teflon pledget in each bite in the annulus in both rows of the suture.

A similar principle was used in 1989 by Revuelta et al. who used interrupted sutures supported by the Teflon pledgets along the annulus and termed it segmental tricuspid annuloplasty.

Another modification was described in 2007 by Sarraj and Duarte, who used a type of adjustable De Vega which consists of dividing it into two parts and using tourniquets to sequentially adjust the length of each part, hence the tightening of the orifice until competence is achieved.

Many other modifications were suggested but all were based on the concept idealized by De Vega and should all be named as modifications of the initial procedure.

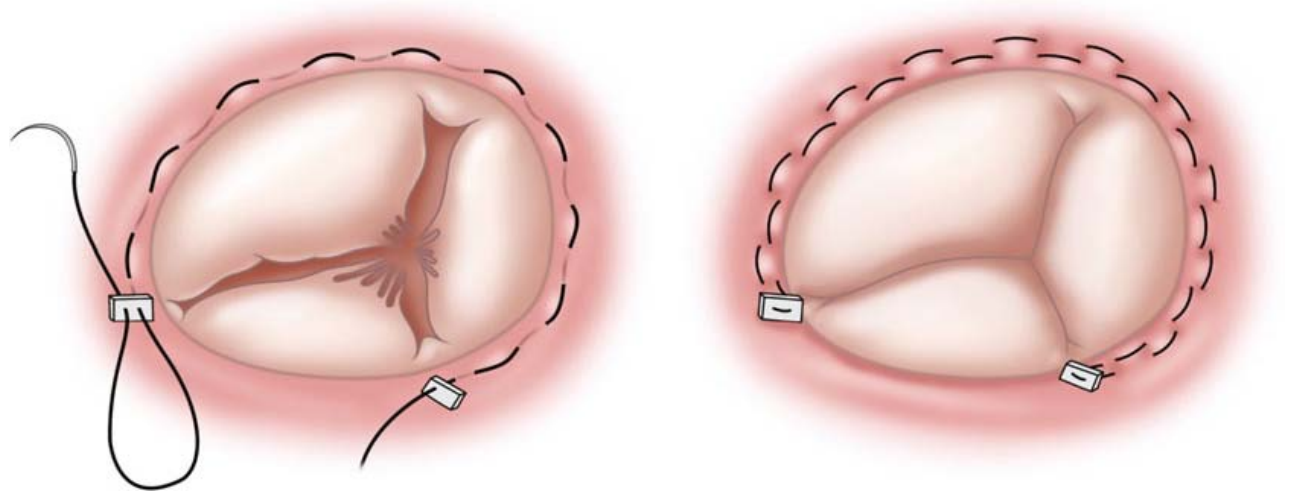


Figure 73: De Vega suture annuloplasty (54): It consists of a double continuous suture extending from the antero-septal to the postero-septal commissures.

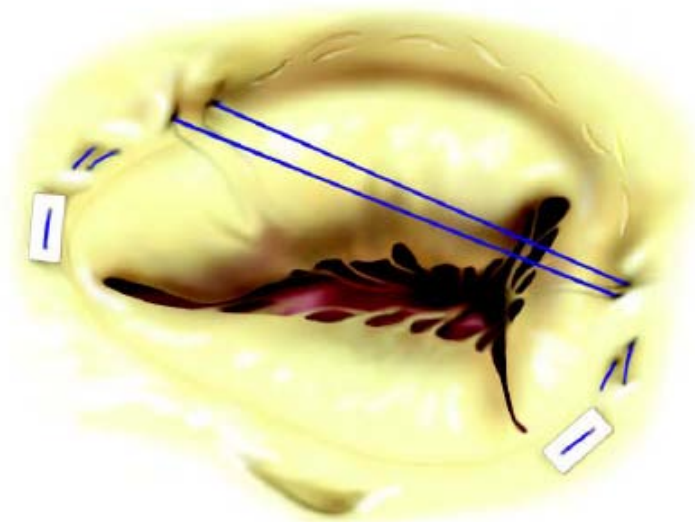


Figure 74: Failure of DeVega annuloplasty: cutting through of the stitches in a fragile tricuspid annulus (“guitar-string syndrome”)

A single row of suture (pledgeted at one end) is first placed along the annulus from the postero-septal commissure to the antero-septal commissure (where it is then passed through a pledget). The same suture is then placed along the annulus again in the opposite direction forming a second row of sutures, and passing through the initial pledget, and tied after adjusting to the required length

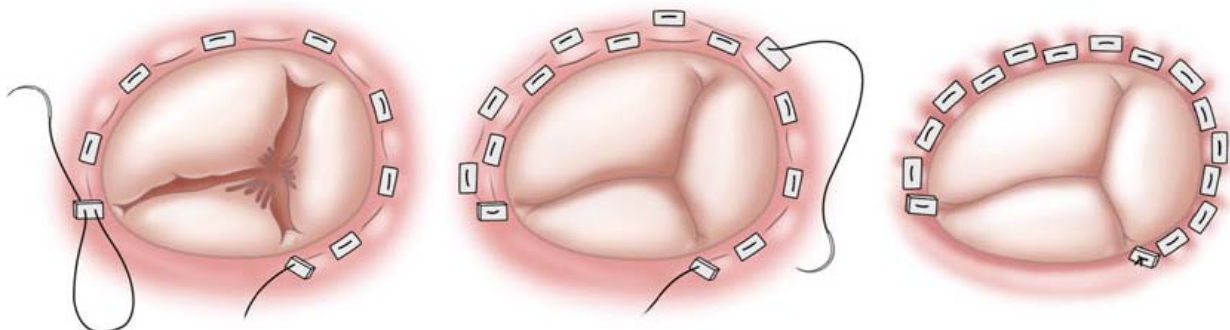


Figure 75: Modified De Vega annuloplasty described by Antunes and Girdwood (54).

It consists of the interposition of Teflon pledgets for each bite of the two-row suture.

- (a) A single row of suture (pledgeted at one end) is first placed along the annulus from the postero-septal commissure to the antero-septal commissure; each bite of the suture through the annulus passes through a pledget.
- (b) The same suture is then placed along the annulus again in the opposite direction forming a second row of sutures; each bite through the annulus is again passed through a pledget.
- (c) The final result after tying the suture having determined the appropriate size

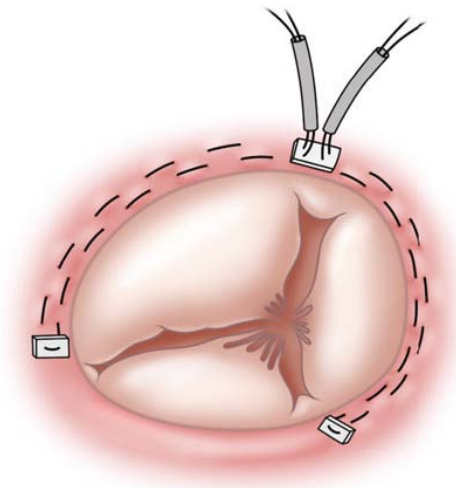


Figure 76: Adjustable annuloplasty as described by Saraj et al. (54)
(*Ann Thorac Surg.* 2007; 83: 698-9).

This modification of the De Vega annuloplasty consists of two double row sutures which can be tightened sequentially by tourniquets, thus selectively adjusting the size of the orifice.

b.2. Kay annuloplasty:

The first tricuspid annuloplasty was described in 1965 by Kay, as a modification of the mitral procedure he had described 2 years earlier. This pioneer used “three or four figure-of-eight sutures of 1-0 silk” to obliterate most, if not all, of the annulus of the septal leaflet to “decrease the size of the annulus from an initial four or five finger breadths to two and one-half finger breadths”, thus resulting in bicuspidization of the tricuspid valve. The results then described were very encouraging and the procedure was quickly adopted by the surgical community.

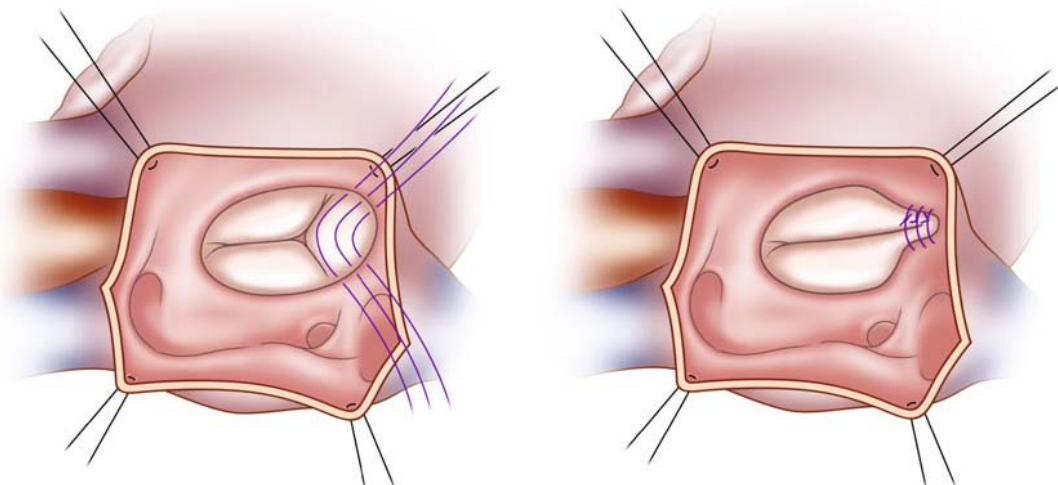


Figure 77: The Kay procedure (54) (the first tricuspid annuloplasty described)

b.3. Ring annuloplasty:

Several commercially available tricuspid annuloplasty rings are available with the newer rings shaped geometrically into a 3-dimensional configuration to match the normal geometry of the tricuspid annulus.

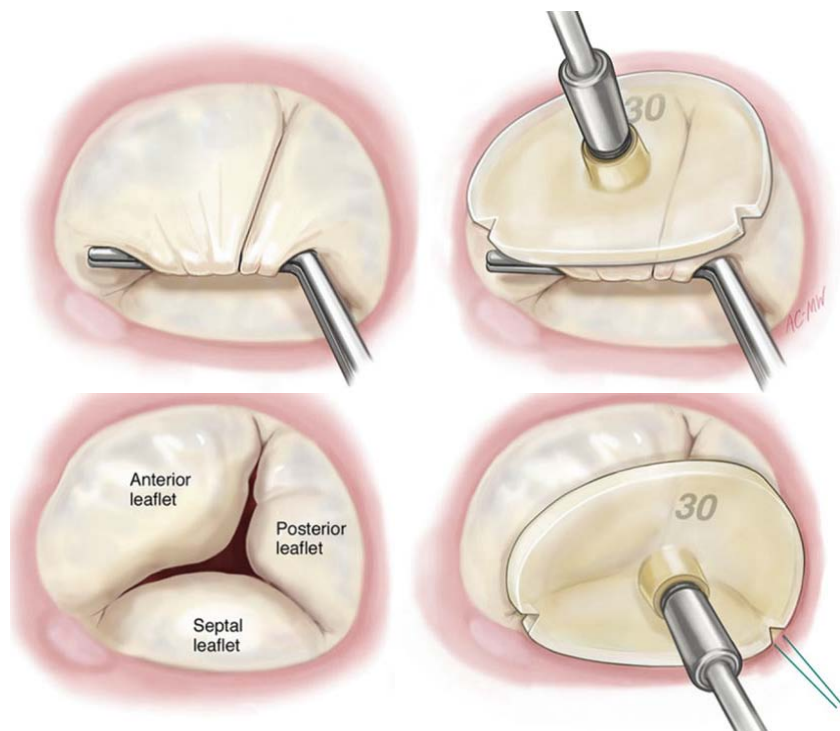
The tricuspid annulus can be sized by measuring its leaflet area by pulling on the anterior papillary muscle which supports both the anterior and posterior leaflets and using an obturator to determine this size.

In the presence of severe FTR, the annuloplasty ring can be undersized to increase leaflet coaptation. For example, if the annulus is sized at 34 mm, a 32 mm ring could be used instead.

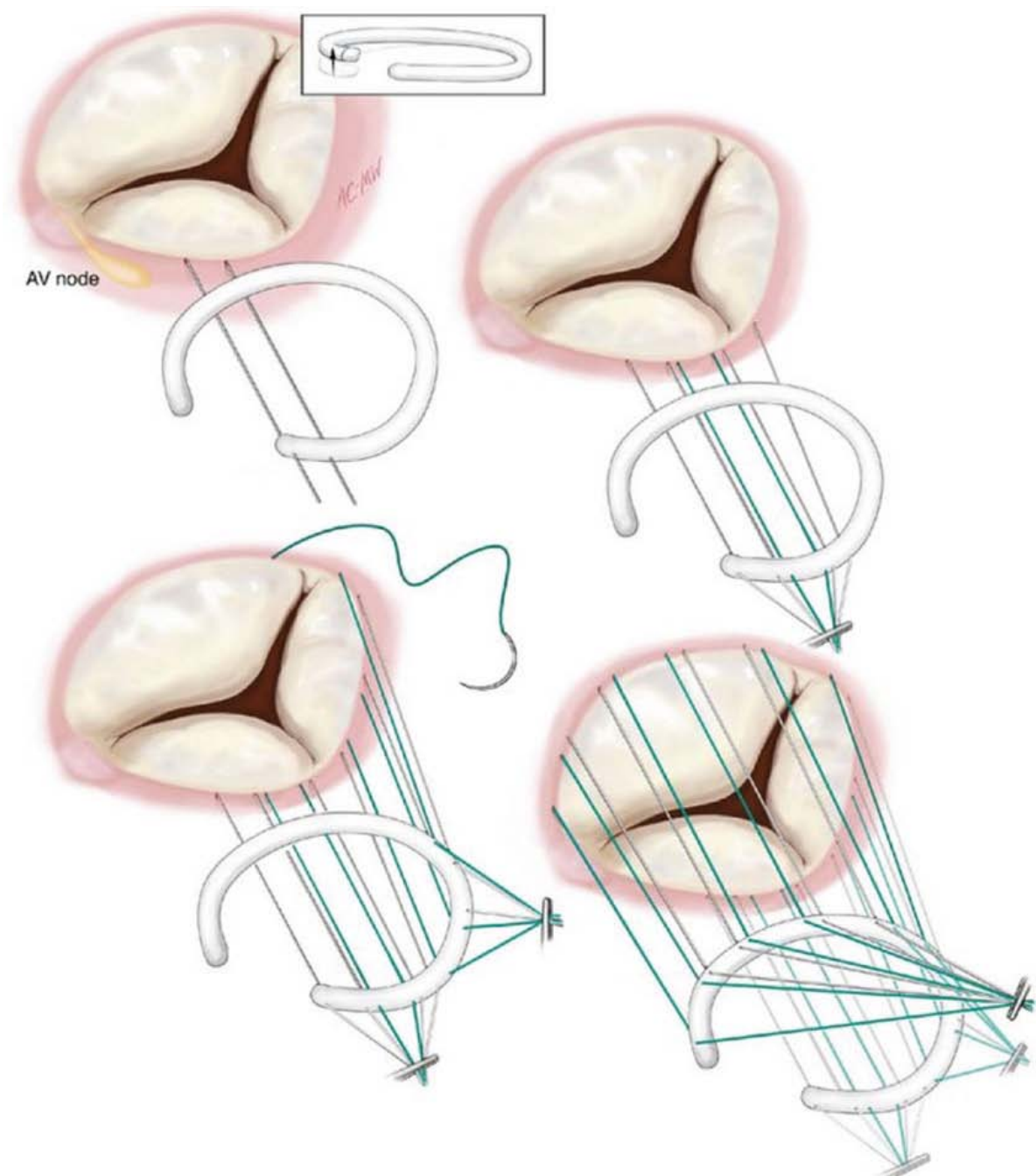
However, caution must be exercised particularly in elderly patients with weak tissue as the excess tension can cause annular dehiscence particularly at the septal annulus.

A series of 2/0 ethibond sutures or equivalent are placed around the tricuspid annulus except at the region of the conduction tissues at the septal annulus. The sutures are then in turn passed through the tricuspid annuloplasty ring.

Care must be taken during suture placement to ensure that the sutures are passed through the annuloplasty ring at their corresponding positions on the tricuspid annulus so as not to distort the tricuspid valve. Two important landmarks are the anteroposterior commissure and the posteroseptal commissures which should pass through the corresponding positions in the tricuspid annuloplasty ring. The annuloplasty ring is then lowered and the sutures tied. A water test is performed while occluding the pulmonary artery to confirm valve competency.



(a) Measuring the tricuspid annular size by pulling on the anterior papillary muscle;
b) Measuring the tricuspid annular size by determining the length of the septal annulus



- (a) 2/0 ethibond sutures or equivalent are placed around the tricuspid annulus. The tricuspid annuloplasty ring is designed such that no sutures are placed in the region of the conduction tissues.
- (b - d) Care is taken to ensure that sutures placed in the annulus are passed through the corresponding positions in the annuloplasty ring;

Figure 78: Ring tricuspid annuloplasty technique (54)

ANNEX 2

➤ The patient's information sheet:

Cardiovascular surgery department

SHEET N°:

Mohamed VI University Hospital Marrakech

PATIENT'S INFORMATION SHEET

I- Demographic data:

- Full name:
- Patient's index:
- Phone number:
- Date of surgery:
- Sex: Female
 Male
- Age: years old

II- Medical and surgical history:

- Recurrent tonsillitis: YES NO
- Acute rheumatic fever: YES NO
- Diabetes: YES NO
- Arterial hypertension: YES NO
- Dyslipidemia: YES NO
- Chronic smoking: YES NO
- Renal function impairment: YES NO
- COPD: YES NO
- Ischemic stroke: YES NO
- Lower limb ischemia: YES NO
- Myocardial infarction/Coronary heart disease: YES NO
- Infectious endocarditis: YES NO
- Hospitalization for heart failure: YES NO
- Preoperative medical treatment:
- Previous valvular surgery: YES NO
 - If yes, specify:
 - Valvular heart disease type:
 - Mitral
 - Aortic
 - Tricuspid

The short and midterm outcomes of the De Vega annuloplasty of the tricuspid valve during the surgical management of rheumatic valvular heart disease

- Surgical procedure(s):
 - Mitral valvuloplasty
 - MVR
 - AVR
 - DVR
 - CABG
 - Other/ specifics:
- The presumed etiology of valvular disease:
 - Rheumatic heart disease
 - Infectious endocarditis
 - Degenerative
 - Congenital
 - Other:

III- Clinical data:

- NYHA Status: I II III IV
- Heart murmurs: MV stenosis MV regurgitation AV stenosis AV regurgitation TV regurgitation
 - Other:
- Right heart failure signs: YES NO
 - If yes, specify:
- Irregular heart rhythm: YES NO
- Other clinical signs:

IV- Chest X-ray:

- Cardiothoracic ratio:
- Left ventricle dilatation:
- Left atrium dilatation:
- Right ventricle dilatation:
- Right atrium dilatation:
- Pulmonary hypertension signs:
- Pulmonary edema signs:

V- EKG:

- Atrial fibrillation:
- Sinus rhythm:
- AV block:
- Right bundle block:
- Left bundle block:

VI- Echocardiographic data:

The short and midterm outcomes of the De Vega annuloplasty of the tricuspid valve during the surgical management of rheumatic valvular heart disease

- Tricuspid valve anatomy:
 - Normal
 - Altered valvular leaflets Specify:.....
 - Fused commissures
 - Tricuspid annulus diameter:
- Tricuspid valve regurgitation: YES NO
 - If yes: specify
 - Type: Functional
 - Organic
 - Both
 - Grade: I II III IV
- Tricuspid valve stenosis: YES NO; => Gradient:
- Associated left-sided valvular disease: YES NO
 - If yes: specify
 - Type:
 - MR
 - MS
 - AoR
 - AoS
 - Etiology:
 - Rheumatic heart fever
 - Infectious endocarditis
 - Prosthetic valve malfunction
- Dilated right ventricle:
- Right ventricle dysfunction:
- Dilated right atrium:
- Dilated left ventricle:
- Dilated left atrium:
- PASP (mm Hg): mmHg
- LVEF: %
- Other mentionable abnormalities:

VII- Perioperative data:

- Surgical approach: Median sternotomy Mini-invasive
- Aortic cross-clamp time:
- CPB time
- Weaning from CPB/ Inotropic drugs:
- Tricuspid valve annulus:
- Organic abnormality of the TV: Specify:
- Dilated right ventricle :
- Concomitant procedure on the TV: Specify:
- Saline test of the TV:
- Associated procedures:

The short and midterm outcomes of the De Vega annuloplasty of the tricuspid valve during the surgical management of rheumatic valvular heart disease

- Mitral valve repair Specify:
- Aortic valve repair Specify:
- Mitral valve replacement Prosthesis type:
- Aortic valve replacement Prosthesis type:
- Double valve replacement Prosthesis type:
- CABG
- Thrombectomy
- Left atrial appendage exclusion
- Other:
- Other mentionable information:

VII- Postoperative data:

1- Critical care data and immediate results:

- Death: YES NO
 - If yes: specify the cause
 - Cardiac
 - Respiratory
 - Sepsis
 - Hemorrhage
 - Other:
- Length of stay in the ICU: days
- Duration of respiratory assistance: hours
- Postoperative medication:
 - Antibioprophylaxis:
 - Anticoagulation:
 - Inotropic drugs: Duration:
- Complications:
 - Cardiac: YES NO
 - Low cardiac output
 - Conduction disturbances: If AV block, specify:
 - Transient :
 - Permanent :
 - Arrhythmias:
 - Myocardial infarction
 - Tamponnade
 - Heart failure
 - Hemorrhage: YES NO

If yes, specify management method:

Transfusion

Reoperation

Other:

➤ Neurologic: YES NO

Stroke Transient or permanent?

Coma

Other:

➤ Infection YES NO

Pulmonary infection

Mediastinitis

Endocarditis

Sepsis

Isolated bacterial agent: YES NO

If yes:

➤ Respiratory failure: YES NO

➤ Liver failure: YES NO

➤ Kidney failure: YES NO

➤ Multiorgan failure: YES NO

▪ Clinical evaluation:

○ NYHA status:

○ Heart failure:

▪ Postoperative echocardiography: YES NO

➤ Time: days

➤ Results:

Pericardial effusion

Dilated right ventricle

Right ventricle dysfunction

TR Grade:

TS Grade:

LVEF:

PASP:

Other information:

ANNEX 3

Responses to some of the jury member's comments:

1. *More details are required about concomitant surgical procedures on the tricuspid valve beside the De Vega annuloplasty.*
 - As mentioned in page 31, the lack of information on the concomitant procedures is due to them being rarely mentioned on the surgical reports.
2. *Respected central tricuspid leak: is there any scientific evidence of its benefit?*
 - It's a common practice in most cardiac surgery centers but we could not find any studies confirming its benefit. On the contrary, the difficulty of controlling the degree of respected regurgitation makes it questionable because of the potential further development of a severe regurgitation. Further studies are required to sort this particular point out.
3. *What is the explanation for the long ICU stay period in this case series.*
 - In practice, the ICU stay period in our department is 2 to 3 days in a simple postoperative course (the patient is discharged after the removal of chest drainage). In our study, the mean ICU stay duration was 4.2 days. This result is mainly explained by the lack of regular hospitalization beds when discharge from the ICU is indicated more than it being secondary to a postoperative complication.
4. *More details are required on the short-term outcomes of the De Vega annuloplasty in this case series.*
 - The lack of details on the short-term outcomes is due to the absence of this information on the echocardiographic report. The echocardiographic assessment of tricuspid valve repair is not possible until the third postoperative month.
5. *More details are required about the subgroup of patients with failure of the De Vega annuloplasty.*
 - These details are available in page 47.
6. *The introduction should not contain diagrams.*
 - The diagram will be removed in the finale revision.

7. *The choice of subgroups would be more efficient if done by the etiological valvular lesion rather than type of surgical intervention.*
 - Most studies don't compare subgroups. We will make sure to compare by valvular lesion too in the rest of our study.

8. *The tricuspid regurgitation grading cited in the material and methods chapter is not the one adopted in the cardiology department.*
 - This grading will be removed in the finale revision. We will only preserve the one cited in page 18 which is the grading adopted in the cardiology department.

9. *How was the preoperative right ventricular function prior to surgery in this case series?*
 - Preoperative right ventricular function was addressed in page 22. However, the degree of ventricular dysfunction was not studied as it was rarely addressed in echocardiographic reports.

10. *What are the criteria of good or poor outcome after tricuspid regurgitation repair?*
 - Risk factors for poor surgical outcomes after tricuspid valve repair were addressed in the analytic part of our study in page 47.

11. *What is the percentage of patients with organic tricuspid lesions?*
 - There was typing mistake in the first version of the thesis manuscript that was corrected in the finale version. The percentage of patients having organic tricuspid valve lesions in our study is actually 24.7%.

12. *A tricuspid annular diameter > 40 mm was identified in our case series as a risk factor of poor outcome after tricuspid repair. Should we anticipate?*
 - This risk factor was mentioned in other studies too. However, further study is required to confirm this finding.

13. *This study needs a comparison group.*
 - A comparison group will definitely give our results a further statistical perspective. We mean to continue this study by including more patients. A comparison group where another tricuspid annuloplasty technique will be performed is another addition that is under discussion.

14. *Can patients with very dilated annuli also benefit from the De Vega repair?*
 - The extremely dilated tricuspid annulus is a challenge with all tricuspid valve repair methods. We believe that the De Vega annuloplasty is not ideal in the

case of very dilated annuli, but in the absence of recommendations, the decision making process in these cases is still unclear.

15. Why did you use only phone calls to collect follow-up data?

- We didn't only use phone calls. Actually, because of the large geographic area that our department covers, one of the major tools that enabled us to collect postoperative data was the Whatsapp application. Patients living far from Marrakech sent us photos of their echocardiographic reports and medical examination by their local doctors on Whatsapp.



CONCLUSION

This study examined the results of simultaneous tricuspid valve repair using the De Vega annuloplasty method for tricuspid regurgitation in 356 patients undergoing left-sided surgery for rheumatic valvular disease.

Tricuspid valve surgery in our study was associated with a significant improvement in the NYHA functional class and of tricuspid regurgitation grade. These results of tricuspid repair were satisfactory in our study.

The major predictive factor of tricuspid valve repair failure identified in our study was severe preoperative and postoperative pulmonary hypertension. Addressing this issue may rend the repair more effective.

The modifications of the classic De Vega technique adopted in our institution may be the answer to the previously reported shortcomings of the De Vega annuloplasty. A long-term follow-up is necessary to further support this conclusion.

We believe that giving-up so soon on the De Vega annuloplasty is unjustified as it is a low-cost method with undeniable benefits.



ABSTRACTS

Abstract

Tricuspid valve disease has been ignored for so long. It commonly consists of tricuspid valve regurgitation secondary to annular dilatation. Lately, it gained increasing attention after proving its impact on mortality and morbidity when treating left-sided valvular disease. The De Vega annuloplasty is one of the first methods proposed for treating this entity. This method was aggressively attacked after the advent of ring annuloplasty because of numerous reported shortcomings without enough further study.

Because we believe that the De Vega annuloplasty is still an efficient tricuspid valve repair method if proper considerations are respected, we conducted a retrospective study including 356 patients who underwent a modified De Vega repair during surgery for left-sided rheumatic valvular heart disease, in cardiovascular surgery department of the University Hospital Mohamed VI of Marrakech to properly analyze the method's results. Our cohort consisted of relatively young patients with a mean age of 40.6 years. The majority of our patients had clinical and echocardiographic signs of advanced valvular disease. Tricuspid valve regurgitation was present in all our patients. The functional etiology was preponderant interesting 74.3%. A significant percentage of our patients had organic lesions of the tricuspid valve (25.7%). An associated tricuspid valve stenosis was present in only 19 patients. In the two years follow-up period in our study, 83.5% of surviving patients were asymptomatic and 81.7% had only trivial or mild tricuspid regurgitation. The overall postoperative mortality in our case series is 11.2%. A persistent moderate to severe tricuspid regurgitation during midterm follow up was found in 7.3%. Severe preoperative and postoperative pulmonary hypertension was found to be the major risk factor of the De Vega annuloplasty failure in our case series.

These results were comparable to those reported in other studies with prosthetic tricuspid annuloplasty. Our choice of the De Vega annuloplasty is built on realistic perspective considering both results and cost of tricuspid valve repair. A long term follow-up is necessary to further support our findings.

Resumé

L'atteinte valvulaire tricuspide a été ignorée pendant longtemps. Elle est représentée généralement par une insuffisance de la valve tricuspide, le plus souvent secondaire à une dilatation de l'anneau. Dernièrement, il a attiré de plus en plus l'attention après avoir prouvé son impact sur la mortalité et la morbidité lors du traitement des valvuloplasties du cœur gauche. L'annuloplastie de De Vega est l'une des premières méthodes proposées pour traiter cette entité. Cette méthode a été agressivement attaquée après l'avènement de l'annuloplastie par anneau en raison de nombreux défauts reportés et insuffisamment étudiés.

Parce que nous croyons que l'annuloplastie De Vega est toujours une méthode efficace de réparation de la valve tricuspide si les considérations appropriées sont respectées, nous avons mené une étude rétrospective incluant 356 patients qui ont subi une réparation De Vega modifiée pendant la chirurgie d'une valvulopathie rhumatismale gauche, au service de chirurgie cardiovasculaire au CHU Mohamed VI de Marrakech, pour une meilleure analyse des résultats de cette méthode. Notre cohorte était composée de patients relativement jeunes avec un âge moyen de 40,6 ans. La majorité de nos patients présentaient des signes cliniques et échocardiographiques de maladie valvulaire avancée. Une insuffisance tricuspide était présente chez tous nos patients. L'étiologie fonctionnelle était prépondérante intéressante 74,3% des patients. Un pourcentage important de nos patients présentait des lésions organiques de la valve tricuspide (25,7%). Une sténose de la valve tricuspide associée était présente chez seulement 19 patients. Durant les 2 ans de suivi après la chirurgie, 83.5% de nos patients sont devenus cliniquement asymptomatiques et 81.7% avait seulement une insuffisance tricuspide minime. La mortalité globale dans notre série était de 11,2%. Une insuffisance tricuspide résiduelle moyenne ou sévère au cours du suivi au moyen-terme a été observée dans 7,3% des cas. L'hypertension artérielle pulmonaire sévère en pré et en postopératoire était identifié comme le facteur de risque majeur d'échec de la plastie tricuspide dans notre série.

Ces résultats étaient comparables à ceux rapportés dans d'autres études avec l'annuloplastie tricuspide prothétique. Le choix de la technique de De Vega dans notre service est basé sur une vision réaliste qui prend en considération à la fois les résultats et le coût de la plastie tricuspide. Un suivi à long terme est nécessaire pour renforcer nos résultats.

ملخص

لقد تم تجاهل مرض الصمام ثلاثي الشرف لفترة طويلة. يتلخص هذا المرض عادة في قلس الصمام ثلاثي الشرف الناتج في أغلب الحالات عن توسع في حلقة الصمام. في الآونة الأخيرة ، اكتسب هذا المرض اهتماما متزايدا بعد إثبات تأثيره على نسب الوفيات و خطر حدوث مضاعفات عند العلاج الجراحي لأمراض صمامات الجانب الأيسر من القلب. تعتبر عملية إصلاح الصمام ثلاثي الشرفات على طريقة "دي فيجا" واحدة من أولى الطرق الجراحية المقترحة لمعالجة قلس هذا الصمام. تعرضت هذه الطريقة لهجوم قوي بعد ظهور طريق زرع حلقة اصطناعية لإصلاح الصمام نظرا لبعض النواقص التي نسبت لطريقة "دي فيجا" من دون دراسة إضافية كافية و عميقة لحثيات هذه النواقص.

لأننا نؤمن بأن عملية إصلاح الصمام ثلاثي الشرفات على طريقة "دي فيجا" لا تزال طريقة فعالة إذا تم احترام الاعتبارات التقنية المناسبة ، أجرينا دراسة شملت 356 مريضاً خضعوا لهذا الإصلاح مع بعض التعديلات أثناء العلاج الجراحي لمرض صمامات الجانب الأيسر للقلب الروماتيزمي ، في قسم جراحة القلب و الشرايين بالمستشفى الجامعي محمد السادس بمراكش لتحليل نتائج الطريقة بشكل معمق. تألفت دراستنا من مرضى نسبيا في سن شاب مع متوسط عمر 40.6 سنة. كان لدى غالبية المرضى علامات سريرية و في الفحص بالصدى الصوتي تدل على حالة متقدمة من مرض صمامات القلب. كان قلس الصمام ثلاثي الشرفات موجوداً في جميع الحالات المدروسة. كانتالسبب الأساسي لهذا الخلل وظيفيا بنسبة 74.3 ٪ من مجموع المرضى. نسبة لا يستهان بها من مرضانا كان لديهم مرض عضوي أيضا لصمام ثلاثي الشرفات (25.7 ٪ من مجموع المرضى). تضيق الصمام ثلاثي الشرف كان متواجدا عند 19 مريضا فقط. بعد الجراحة، 83.7 ٪ من المرضى لم يعد لديهم أي أعراض سريرية للمرض و 81.7 ٪ لم يعد لديهم الا درجة ضئيلة من قلس الصمام ثلاثي الشرف. معدل الوفيات الإجمالي في سلسلة الحالات لدينا هو 11.2 ٪. بعد الجراحة تم العثور على درجة قلس متوسطة إلى

شديدة خلال فترة المتابعة في 7.3 % من المرضى. ارتفاع الضغط الرئوي قبل و بعد الجراحة كان من أهم أسباب فشل اصلاح الصمام في هذه الفئة من المرضى.

كانت هذه النتائج مماثلة لتلك التي تم نشرها في دراسات أخرى مع اصلاح الصمام ثلاثي الشرف بطريقة زرع حقة اصطناعية. اختيارنا لاصلاح الصمام ثلاثي الشرفات على طريقة "دي فيجا" مبني على مقارنة واقعية تخأذ بعين الاعتبار النتائج و التكلفة. المتابعة على المدى الطويل لمرضى هاته الدراسة ضرورية لدعم نتائجنا.



REFERENCES

1. **Van Praet KM et al.**
An overview of surgical treatment modalities and emerging transcatheter interventions in the management of tricuspid valve regurgitation. *Expert Review of Cardiovascular Therapy*. 2018 Feb;16(2):75–89.
2. **Khallaf AN, Saleh HZ, Elnaggar AM, Rasekh FS.**
Tricuspid valve repair by DeVega technique versus ring annuloplasty in patients with functional severe tricuspid regurge. *Journal of the Egyptian Society of Cardio–Thoracic Surgery*. 2016 Aug;24(2):131–4.
3. **Pozzoli A, Elisabetta L, Vicentini L, Alfieri O, De Bonis M.**
Surgical indication for functional tricuspid regurgitation at initial operation: judging from long term outcomes. *General Thoracic and Cardiovascular Surgery*. 2016 Sep;64(9):509–16.
4. **Sadeghpour A, Alizadehasl A, Azizi Z, Ghavidel AA.**
Tricuspid Regurgitation Dilemma: A Comparison Study between Surgical Versus Medical Management of Patients with Tricuspid Regurgitation. *Multidiscip Cardio Annal*. 2016 Oct;
5. **Yiu K–H, Chen Y, Liu J–H, Lin Q, Liu M, Wu M, et al.**
Burden and contributing factors associated with tricuspid regurgitation: a hospital–based study. *Hospital Practice*. 2017 Oct 20;45(5):209–14.
6. **Fender EA, Zack CJ, Nishimura RA.**
Isolated tricuspid regurgitation: outcomes and therapeutic interventions. *Heart*. 2018 May;104(10):798–806.
7. **Saitto G, Russo M, Nardi P, Gislao V, Scafuri A, Pellegrino A, et al.**
Tricuspid Valve Annuloplasty during Mitral Valve Surgery: A Risk or an Additional Benefit? *Ann Vasc Med Res*. 2016;3(1):1026.
8. **Muraru D, Surkova E, Badano LP.**
Revisit of Functional Tricuspid Regurgitation; Current Trends in the Diagnosis and Management. *Korean Circulation Journal*. 2016;46(4):443.
9. **Rodés–Cabau J, Taramasso M, O’Gara PT.**
Diagnosis and treatment of tricuspid valve disease: current and future perspectives. *The Lancet*. 2016 Nov;388(10058):2431–42.
10. **Huttin O, Voilliot D, Mandry D, Venner C, Juillièrè Y, Selton–Suty C.**
All you need to know about the tricuspid valve: Tricuspid valve imaging and tricuspid regurgitation analysis. *Archives of Cardiovascular Diseases*. 2016 Jan;109(1):67–80.

11. **Kunová M, Frána R, Toušek F, Mokráček A, Peší L.**
Tricuspid annuloplasty using De Vega modified technique – Short-term and medium-term results. *Cor et Vasa*. 2016 Aug;58(4):e379–83.
12. **Antunes MJ, Barlow JB.**
Management of tricuspid valve regurgitation. *Heart*. 2005 Dec 30;93(2):271–6.
13. **David TE, David CM, Manhiolt C.**
When is tricuspid valve annuloplasty necessary during mitral valve surgery? *The Journal of Thoracic and Cardiovascular Surgery*. 2015 Nov;150(5):1043–4.
14. **Yuh DD et al.**
Redefining tricuspid valve anatomy: Acknowledging the “forgotten valve.” *The Journal of Thoracic and Cardiovascular Surgery*. 2018 Apr;155(4):1520–1.
15. **Rodés–Cabau et al.**
Diagnosis and treatment of tricuspid valve disease: current and future perspectives. *Lancet* 2016; 388: 2431–42
16. **Sadeghpour A, Alizadehasl A, Azizi Z, Chavidel AA.**
Tricuspid Regurgitation Dilemma: A Comparison Study between Surgical Versus Medical Management of Patients with Tricuspid Regurgitation. *Multidiscip Cardio Annal*. 2016 October; 7(1):e9469.
17. **Mangieri A, Montalto C, Pagnesi M, Jabbour RJ, Rodés–Cabau J, Moat N, et al.**
Mechanism and Implications of the Tricuspid Regurgitation: From the Pathophysiology to the Current and Future Therapeutic Options. *Circulation: Cardiovascular Interventions*. 2017 Jul;10(7):e005043.
18. **Buzzatti N, De Bonis M, Moat N.**
Anatomy of the Tricuspid Valve, Pathophysiology of Functional Tricuspid Regurgitation, and Implications for Percutaneous Therapies. *Interventional Cardiology Clinics*. 2018 Jan;7(1):1–11.
19. **Kuh JH, Kim KH, Choi JB.**
Surgical Lessons from the Repair of Recurrent Tricuspid Regurgitation after DeVega Annuloplasty. *Texas Heart Institute Journal*. 2017 Feb;44(1):82–3.
20. **KACHCHOUR BOUTAINA.**
Thèse en médecine: RESULTATS DE LA CHIRURGIE DE L'INSUFFISANCE TRICUSPIDE (A propos de 120 cas).Fèz 2016.

21. **Imamura E, Ohteki H, Koyanagi H.**
An Improved de Vega Tricuspid Annuloplasty. *The Annals of Thoracic Surgery*. 1982 Dec;34(6):710-3.
22. **Hind ANNOUKHAILI.**
Thèse/ L'ATTEINTE TRICUSPIDIENNE AU COURS DES VALVULOPATHIES RHUMATISMALES. Casa 2007.
23. **Alnawaiseh K, Albkhour B, Alnaser Y, Aladwan H, Ghanma I.**
De Vega annuloplasty versus ring annuloplasty for repair of functional tricuspid regurgitation. *International Journal of Research in Medical Sciences*. 2018 Jan 24;6(2):422.
24. **I Zairi, et al**
Predictive factors of tricuspid valve regurgitation progression after surgical treatment of left hear disease. *Cardiologie Tunisienne – Volume 12 N°01 – 1er Trimestre 2016 –42-48*
25. **Khallaf AN, Saleh HZ, Elnaggar AM, Rasekh FS.**
Tricuspid valve repair by DeVega technique versus ring annuloplasty in patients with functional severe tricuspid regurge. *Journal of the Egyptian Society of Cardio-Thoracic Surgery*. 2016 Aug;24(2):131-4.
26. **Shinn SH, Dayan V, Schaff HV, Dearani JA, Joyce LD, Lahr B, et al.**
Outcomes of ring versus suture annuloplasty for tricuspid valve repair in patients undergoing mitral valve surgery. *The Journal of Thoracic and Cardiovascular Surgery*. 2016 Aug;152(2):406-415.e3.
27. **Malik A, Khalil IK, Ali SM, Khan RA, Khan A.**
DE VEGA'S TRICUSPID ANNULOPLASTY FOR SEVERE TRICUSPID REGURGITATION – EARLY AND MIDTERM FOLLOW UP. 2012;45:6.
28. **Gabriel CA, Wilfrid G, Sokhna DM, Salmane P, Mohamed L, Souleymane D, et al.**
Results of the De Vega Plasty in Tricuspid Insufficiencies Secondary to Mitral and Aortic Rheumatic Valve Diseases: Study of 58 Cases. :4. Oct 2016.
29. **Van Nooten GJ, Caes F, Taeymans Y, Van Belleghem Y, François K, De Bacquer D, et al.**
Tricuspid valve replacement: Postoperative and long-term results. *The Journal of Thoracic and Cardiovascular Surgery*. 1995 Sep;110(3):672-9.

30. **Colombo T, Russo C, Ciliberto GR, Lanfranconi M, Bruschi G, Agati S, et al.**
Tricuspid regurgitation secondary to mitral valve disease: Tricuspid annulus function as guide to tricuspid valve repair. 2001;9(4):9.
31. **Gilles D. Dreyfus et al**
Functional Tricuspid Regurgitation : a need to revise our understanding. JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY 2015
32. **Bernal JM, Pontón A, Diaz B, Llorca J, García I, Sarralde A, et al.**
Surgery for rheumatic tricuspid valve disease: A 30-year experience. The Journal of Thoracic and Cardiovascular Surgery. 2008 Aug;136(2):476-81.
33. **Han Q-Q, Xu Z-Y, Zhang B-R, Zou L-J, Hao J-H, Huang S-D.**
Primary triple valve surgery for advanced rheumatic heart disease in Mainland China: a single-center experience with 871 clinical cases. European Journal of Cardio-Thoracic Surgery. 2007 May;31(5):845-50.
34. **McCarthy PM, Bhudia SK, Rajeswaran J, Hoercher KJ, Lytle BW, Cosgrove DM, et al.**
Tricuspid valve repair: durability and risk factors for failure. The Journal of Thoracic and Cardiovascular Surgery. 2004 Mar;127(3):674-85.
35. **Sarralde JA, Bernal JM, Llorca J, Pontón A, Diez-Solorzano L, Giménez-Rico JR, et al.**
Repair of Rheumatic Tricuspid Valve Disease: Predictors of Very Long-Term Mortality and Reoperation. The Annals of Thoracic Surgery. 2010 Aug;90(2):503-8.
36. **Najah DAIFALLAH**
THESE: EPIDEMIOLOGIE DU RHUMATISME ARTICULAIRE AIGU DANS LA REGION DE MARRAKECH; FMPM 2010
37. **Omar EL MENZHI, Majdouline OBTEL, Dr Ahmed RGUIG**
Bulletin Epidémiologique des Maladies sous Surveillance. Royaume du Maroc; Ministère de la Santé. Avril 2012
38. **GHANEM DN, JROUNDI DI.**
Epidémiologie du Rhumatisme Articulaires Aigu au Maroc : Description des données de surveillance collectées entre 2000 et 2010. :33.
39. **Latifa Maazaoui**
Guide National d'Epidémiologie d'Intervention. MINISTÈRE DE LA SANTÉ. 2015

40. **Theophilus E. Owan et al**
Trends in Prevalence and Outcome of Heart Failure with Preserved Ejection Fraction. The New England Journal of Medicine. 2006;9.
41. **Guglielmo Saitto et al**
Tricuspid Valve Annuloplasty during Mitral Valve Surgery: A Risk or an Additional Benefit? Annals of Vascular Medicine & Research. 2016;5.
42. **Abdelgawad A, Ramadan M, Arafat H, Abdel Aziz A.**
Tricuspid valve repair with Dacron band versus DeVega or segmental annuloplasty. Hospital outcome and short term results. The Egyptian Heart Journal. 2017 Dec;69(4):241–6.
43. **Kunová M, Frána R, Toušek F, Mokráček A, Peší L.**
Tricuspid annuloplasty using De Vega modified technique – Short-term and medium-term results. Cor et Vasa. 2016 Aug;58(4):e379–83.
44. **Igor Gosev et al**
Should Moderate-to-Severe Tricuspid Regurgitation be Repaired During Reoperative Left-Sided Valve Procedures? Seminars in Thoracic and Cardiovascular Surgery ; 2015.
45. **Hata H, Fujita T, Miura S, Shimahara Y, Kume Y, Matsumoto Y, et al.**
Long-Term Outcomes of Suture vs. Ring Tricuspid Annuloplasty for Functional Tricuspid Regurgitation. Circulation Journal. 2017;81(10):1432–8.
46. **Chopra HK, Nanda NC, Fan P, Kapur KK, Goyal R, Daruwalla D, et al.**
Can two-dimensional echocardiography and Doppler color flow mapping identify the need for tricuspid valve repair? Journal of the American College of Cardiology. 1989 Nov;14(5):1266–74.
47. **Salma Charfeddine¹, Rania Hammami¹,&, Faten Triki¹, Leila Abid¹, Mourad Hentati¹, et al**
La plastie tricuspide annuloplastie de Carpentier versus technique de De Vega. Pan African Medical Journal 2017.
48. **Guenther et al.**
Tricuspid valve repair is ring annuloplasty superior? European Journal of Cardio-Thoracic Surgery 43 (2013) 58–65

49. **Choi JW, Kim KH, Chang HW, Jang M, Kim SH, Yeom SY, et al.**
Long-term results of annuloplasty in trivial-to-mild functional tricuspid regurgitation during mitral valve replacement: should we perform annuloplasty on the tricuspid valve or leave it alone? *European Journal of Cardio-Thoracic Surgery*. 2018 Apr 1;53(4):756-63.
50. **Chen J, Abudupataer M, Hu K, Maimaiti A, Lu S, Wei L, et al.**
Risk factors associated with perioperative morbidity and mortality following isolated tricuspid valve replacement. *Journal of Surgical Research*. 2018 Jan;221:224-31.
51. **Jang JY, Heo R, Lee S, Kim JB, Kim D-H, Yun S-C, et al.**
Comparison of Results of Tricuspid Valve Repair Versus Replacement for Severe Functional Tricuspid Regurgitation. *The American Journal of Cardiology*. 2017 Mar;119(6):905-10.
52. **Picichè M,**
BOOK: Dawn and evolution of cardiac procedures: research avenues in cardiac surgery and interventional cardiology. Milan: Springer; 2013. 353 p.
53. **Dominik J, Zacek P.**
BOOK: Heart valve surgery: an illustrated guide. Heidelberg ; New York: Springer; 2010. 415 p.
54. **Chan KMJ.**
BOOK: Functional Mitral and Tricuspid Regurgitation [Internet]. Cham: Springer International Publishing; 2017.
55. **Nelson Wang, Steven Phan, David H. Tian, Tristan D. Yan, Kevin Phan**
Flexible band versus rigid ring annuloplasty for tricuspid regurgitation a systematic review and meta-analysis. *Ann Cardiothorac Surg* 2017;6(3):194-203 .
56. **Hironori Izutani, Teruya Nakamura, Kanji Kawachi**
Flexible band versus rigid ring annuloplasty for functional tricuspid regurgitation. *Heart International* 2010;
57. **Paşaoğlu et al.**
De Vega's Tricuspid Annuloplasty Analysis of 195 patients. *Thome. cardiovasc. Surgeon* 38 (1990)
58. **Chidambaram et al.**
Long-term Results of DeVega Tricuspid Annuloplasty. *Ann Thorac Surg* 43:185-188, Feb 1987

59. **Abe et al.**
De Vega's annuloplasty for acquired tricuspid disease: Early and Late Results in 110 Patients. *Ann Thorac Surg* 1989;48:670-6
60. **Imamura et al.**
An Improved de Vega Tricuspid Annuloplasty. 0003-4975/82/120710-04\$01.25 @ 1982 by The Society of Thoracic Surgeons
61. **Schulte et al.**
Modified Annuloplasty of the Tricuspid Valve: Technique and long term results. Springer-Verlag Berlin Heidelberg 1989
62. **Guenther T, Mazzitelli D, Noebauer C, Hettich I, Tassani-Prell P, Voss B, et al.**
Tricuspid valve repair: is ring annuloplasty superior?†. *European Journal of Cardio-Thoracic Surgery*. 2013 Jan;43(1):58-65.
63. **Erin A Fender, Chad J Zack, Rick A Nishimura**
Isolated tricuspid regurgitation: outcomes and therapeutic interventions. Fender EA, et al. *Heart* 2017;0:1-9. doi:10.1136/heartjnl-2017-311586
64. **De Vega NG, De Rábago G, Castellón L, Moreno T, Azpitarte J.**
A new tricuspid repair. Short-term clinical results in 23 cases. *J Cardiovasc Surg (Torino)*. 1973;Spec No:384-6.
65. **Harlan BJ.**
BOOK: Manual of cardiac surgery. Place of publication not identified: Springer; 2011.
66. **Carrier M, Pellerin M, Guertin M-C, Bouchard D, Hébert Y, Perrault LP, et al.**
Twenty-five years' clinical experience with repair of tricuspid insufficiency. *J Heart Valve Dis*. 2004 Nov;13(6):952-6.
67. **Giamberti A, Chessa M, Ballotta A, Varrica A, Agnetti A, Frigiola A, et al.**
Functional tricuspid valve regurgitation in adults with congenital heart disease: an emerging problem. *J Heart Valve Dis*. 2011 Sep;20(5):565-70.
68. **Ho Young Hwang et al**
De Vega Annuloplasty for Functional Tricuspid Regurgitation: Concept of Tricuspid Valve Orifice Index to Optimize Tricuspid Valve Annular Reduction. *The Korean Academy of Medical Sciences*; 2016

69. **Calafiore AM, Di Mauro M.**
Tricuspid Valve Repair—Indications and Techniques: Suture Annuloplasty and Band Annuloplasty. *Operative Techniques in Thoracic and Cardiovascular Surgery*. 2011;16(2):86–96.
70. **Esenyel CZ et al.**
Evaluation of soft tissue reactions to three nonabsorbable suture materials in a rabbit model. *Acta Orthopaedica et Traumatologica Turcica*. 2009;43(4):366–72.
71. **Carlos M et al.**
The Vanishing Tricuspid Annuloplasty – A New Concept. *The journal of thoracic and cardiovascular surgery.*; 1991.
72. **Fukuda Shota et al**
Determinants of Recurrent or Residual Functional Tricuspid Regurgitation After Tricuspid Annuloplasty. *Circulation*. 2006;114[suppl 1]:I-582-I-587.
73. **Chen et al.**
The prognostic significance of tricuspid valve regurgitation in pulmonary hypertension. Department of Cardiovascular Diseases, Mayo Clinic; 2018
74. **Lin Y, Wang Z, He J, Xu Z, Xiao J, Zhang Y, et al.**
Efficiency of different annuloplasty in treating functional tricuspid regurgitation and risk factors for recurrence. *IJC Heart & Vasculature*. 2014 Dec;5:15–9.
75. **Pant AD, Thomas VS, Black AL, Verba T, Lesicko JG, Amini R.**
Pressure-induced microstructural changes in porcine tricuspid valve leaflets. *Acta Biomaterialia*. 2018 Feb;67:248–58.
76. **Bermejo J, Yotti R, García-Orta R, Sánchez-Fernández PL, Castaño M, Segovia-Cubero J, et al.**
Sildenafil for improving outcomes in patients with corrected valvular heart disease and persistent pulmonary hypertension: a multicenter, double-blind, randomized clinical trial. *European Heart Journal*. 2018 Apr 14;39(15):1255–64.
77. **Subbotina I, Girdauskas E, Bernhardt A, Sinning C, Reichenspurner H, Sill B.**
Outcome of Tricuspid Valve Surgery in Patients with Reduced Right Ventricular Function. *The Thoracic and Cardiovascular Surgeon*. 2017 Feb 3;65(S 01):S1–110.

78. **Matsumiya G, Kohno H, Matsuura K, Sakata T, Tamura Y, Watanabe M, et al.**
Right ventricular papillary muscle approximation for functional tricuspid regurgitation associated with severe leaflet tethering†. *Interactive CardioVascular and Thoracic Surgery*. 2018 Apr 1;26(4):700–2.
79. **Utsunomiya H, Itabashi Y, Mihara H, Berdejo J, Kobayashi S, Siegel RJ, et al.**
Functional Tricuspid Regurgitation Caused by Chronic Atrial Fibrillation
CLINICAL PERSPECTIVE: A Real-Time 3-Dimensional Transesophageal Echocardiography Study. *Circulation: Cardiovascular Imaging*. 2017 Jan;10(1):e004897.
80. **Yung-Tsai Lee, Chung-Yi Chang and Jeng Wei**
Anatomic Consideration of Stitch Depth in Tricuspid Valve Annuloplasty. *Acta Cardiol Sin* 2015;31:232_234
81. **Ranhel AS, Mesquita ET.**
The Middle Ages Contributions to Cardiovascular Medicine. *Brazilian Journal of Cardiovascular Surgery [Internet]*. 2016
82. **Shoja MM, Agutter PS, Loukas M, Benninger B, Shokouhi G, Namdar H, et al.**
Leonardo da Vinci's studies of the heart. *International Journal of Cardiology*. 2013 Aug;167(4):1126–33.
83. **Braile DM, Godoy MF de.**
História da cirurgia cardíaca. *REVISTA BRASILEIRA DE CIRURGIA CARDIOVASCULAR*. 2012;27(1):125–34.
84. **Ellis H et al.**
The Early Days of Heart Valve Surgery. *Journal of Perioperative Practice*. 2011 Aug;21(8):287–8.
85. **Larry W. Stephenson**
History of cardiac surgery; 1992
86. **Dominik J, Zacek P.**
BOOK: Heart valve surgery: an illustrated guide. Heidelberg ; New York: Springer; 2010. 415 p.
87. **Sabharwal N, Dev H, Smail H, McGiffin DC, Saxena P.**
Nina Braunwald: A Female Pioneer in Cardiac Surgery. *Texas Heart Institute Journal*. 2017 Apr;44(2):96–100.

88. **Antonoff MB, David EA, Donington JS, Colson YL, Litle VR, Lawton JS, et al.**
Women in Thoracic Surgery: 30 Years of History. *The Annals of Thoracic Surgery*. 2016 Jan;101(1):399-409.
89. **Pibarot P, Dumesnil JG.**
Prosthetic Heart Valves: Selection of the Optimal Prosthesis and Long-Term Management. *Circulation*. 2009 Feb 9;119(7):1034-48.
90. **Grondin P, Lepage G, Castonguay Y, Meere C.**
The tricuspid valve: a surgical challenge. *J Thorac Cardiovasc Surg*. 1967 Jan;53(1):7-20.
91. **Kay JH, Maselli-Campagna G, Tsuji KK.**
SURGICAL TREATMENT OF TRICUSPID INSUFFICIENCY. *Ann Surg*. 1965 Jul;162:53-8.
92. **Revuelta JM.**
La anuloplastia de De Vega. Perspectiva histórica. *Cirugía Cardiovascular*. 2012 Oct;19(4):351-6.
93. **Carpentier A, Deloche A, Hanania G, Forman J, Sellier P, Piwnica A, et al.**
Surgical management of acquired tricuspid valve disease. *J Thorac Cardiovasc Surg*. 1974 Jan;67(1):53-65.
94. **Duran CG, Ubago JL.**
Clinical and hemodynamic performance of a totally flexible prosthetic ring for atrioventricular valve reconstruction. *Ann Thorac Surg*. 1976 Nov;22(5):458-63.
95. **Krishnaswamy A, Navia J, Kapadia SR.**
Transcatheter Tricuspid Valve Replacement. *Interventional Cardiology Clinics*. 2018 Jan;7(1):65-70.
96. **Fawzy H, Serag A, El-Nasr MA, Warda H, El-Shora H, Mostafa A, et al.**
Tricuspid annuloplasty ring prototype testing. *Journal of the Egyptian Society of Cardio-Thoracic Surgery*. 2018 Mar;26(1):30-6.
97. **Khan JM, Rogers T, Schenke WH, Greenbaum AB, Babaliaros VC, Paone G, et al.**
Transcatheter pledget-assisted suture tricuspid annuloplasty (PASTA) to create a double-orifice valve. *Catheterization and Cardiovascular Interventions [Internet]*. 2018 Feb 6 [cited 2018 Jul 3]; Available from: <http://doi.wiley.com/10.1002/ccd.27531>

98. **Latib A.**
Transcatheter Tricuspid Valve Intervention: Addressing an Unmet Clinical Need. *Interventional Cardiology Clinics*. 2018 Jan;7(1):xiii–xiv.
99. **Carpentier A, Adams DH, Filsoufi F.**
BOOK: Carpentier’s reconstructive valve surgery: from valve analysis to valve reconstruction. Maryland Heights, Mo: Saunders/Elsevier; 2010. 354 p.
100. **Di Mauro M, Bezante GP, Di Baldassarre A, Clemente D, Cardinali A, Acitelli A, et al.**
Functional tricuspid regurgitation: An underestimated issue. *International Journal of Cardiology*. 2013 Sep;168(2):707–15.
101. **de Vlaming A, Sauls K, Hajdu Z, Visconti RP, Mehesz AN, Levine RA, et al.**
Atrioventricular valve development: New perspectives on an old theme. *Differentiation*. 2012 Jul;84(1):103–16.
102. **Bhave NM, Ward RP.**
Echocardiographic Assessment and Clinical Management of Tricuspid Regurgitation. *Current Cardiology Reports*. 2011 Jun;13(3):258–64.
103. **Luxford J, Bassin L, D’Ambra M.**
Echocardiography of the tricuspid valve. *Annals of Cardiothoracic Surgery*. 2017 May;6(3):223–39.
104. **Baumgartner H, Falk V, Bax JJ, De Bonis M, Hamm C, Holm PJ, et al.**
2017 ESC/EACTS Guidelines for the management of valvular heart disease. *European Heart Journal*. 2017 Sep 21;38(36):2739–91.



قسم الطبيب

أقسم بالله العظيم

أن أراقب الله في مهنتي.

وأن أصون حياة الإنسان في كافة أطوارها في كل الظروف

والأحوال باذلة وسعي في انقاذها من الهلاك والمرض

والألم والقلق.

وأن أحفظ للناس كرامتهم، وأستر عورتهم، وأكتم سرهم.

وأن أكون على الدوام من وسائل رحمة الله، باذلة رعايتي الطبية للقريب والبعيد،

للسالح والطالح، والصديق والعدو.

وأن أثابر على طلب العلم، وأسخره لنفع الإنسان لا لأذاه.

وأن أوقر من علمني، وأعلم من يصغرنني، وأكون أختاً لكل زميل في المهنة

الطبية متعاونين على البر والتقوى.

وأن تكون حياتي مصداق إيماني في سرّي وعلانيتي، نقيّة مما يشينها تجاه

الله ورسوله والمؤمنين.

والله على ما أقول شهيدا

النتائج قصيرة ومتوسطة المدى للإصلاح الجراحي على طريقة دي فيغا للصمام ثلاثي الشرف أثناء العلاج الجراحي للأمراض الروماتزمية لصمامات القلب

الأطروحة

قدمت ونوقشت علانية يوم 2018/ 07 /19

من طرف

الأنسة سكيبة بنباخ

المزودة في 03 غشت 1991 بمراكش

لنيل شهادة الدكتوراه في الطب

الكلمات الأساسية:

الصمام ثلاثي الشرف – اصلاح الصمام ثلاثي الشرف – اصلاح على طريقة دي فيغا

اللجنة

الرئيس

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أستاذ في أمراض القلب و الشرايين

المشرف

د. بومزيرة

السيد

أستاذ في جراحة القلب و الشرايين

ر. الحواتي

السيد

أستاذ مبرز في جراحة القلب و الشرايين

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السيدة

أستاذة مبرزة في أمراض القلب و الشرايين

ل. بندريس

السيدة

أستاذة مبرزة في أمراض القلب و الشرايين

الحكام