

Review Article

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Systolic Heart Failure Vs Diastolic Heart Failure

José F. Guadalajara Boo*

Instituto Nacional de Cardiología “Ignacio Chávez”,
México

***Corresponding Author**

José F. Guadalajara Boo, MD, Instituto Nacional de Cardiología “Ignacio Chávez”, México.

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Abstract

Heart failure is a disease in which there are multiple definitions and the concept is adopted arbitrarily by each author, in such a way that the terminology is so confusing. Now the authors of the mega trials in medical treatment of heart failure avoided this confusion and referred that this disease can be treated and have set to indicate the value of the ejection fraction <40 or 35% . The main goal of this paper is to promptly revise the concepts of contractility, ventricular function, preload, afterload myocardial oxygen consumption (MVO₂), heart failure, compensation mechanisms. These definitions and concepts are based on the original contributions of recognized researchers, in an attempt to clarify the concepts, which was born in the nomination of heart failure, and thus to avoid an erroneous interpretation, always motivated by inadequate simplification of terms, in order to scientifically explain the concept of ventricular function and heart failure.

Keywords: Ventricular Function, Heart Failure, Compensatory Mechanism

1. Systolic Heart Failure

1.1. Introduction

The analysis of research studies that, over time, have allowed us to understand the mechanisms governing ventricular function and its alteration by disease; as well as the cause of the resulting symptoms and their significance in the pathophysiology of the disease, and finally, the therapeutic measures that improve the patient's symptoms or reduce mortality; when the physician understands the disease, they move towards a rational reflection that remains grounded in scientific evidence, allowing us to reach conclusions that are valid for judgments and recommendations, especially in controversial topics that frequently cause confusion and misapplication of treatments, as well as inconclusive answers, discussions in different forums, or a lack of consensus due to a failure to understand the central issue that gave rise to the discrepancy.

In fact, today this problem is so serious that the Cardiovascular Research Journal conducted a survey in which the "definition of heart failure" was asked of 2238 expert cardiologists [1]. A total of 1018 responses were received, of which only 30 included a definition of "heart failure". Of all the versions, none offered a conceptual answer that was accepted; the conclusion was that "heart failure is the label for a cardiovascular syndrome that lacks uniform criteria for its definition" [1]. The conclusion that can

be drawn is: "If you want to know if you understand something, try to explain it; if you can't, then you don't understand it". Great researchers have given us sustenance to know the function of the heart and heart failure, in an attempt to conceptualize them in a way that it is possible to discuss the issue revised around these fundamental principles and can reach a consensus based on scientific evidence.

The heart has a prodigious function that takes place during the life of living beings that have blood circulation; in other words, the heart is the most perfect pump that the nature has created, that it is able to have 583 million, 200 thousand contractions and relaxations in a healthy 75 year old man since their internal biological systems save energy and optimize the function. No artifact created by man, which can able to perform a function that is comparable to the heart, without fault and within the time. Only when heart function is understood in all its magnitude, then the actual meaning of "heart failure" can be understood and avoid to minimize the symptoms or the response to treatment or other fragmentary pathophysiological concepts.

1.2. Definition of Systolic Heart Failure

"It is a condition in which diffuse structural damage to the myofibrils or excessive hemodynamic overload causes a decrease in the heart's contractile force (and therefore the ejection fraction)

and consequently increases ventricular volumes with normal cardiac output (compensated) or with decreased cardiac output and tissue hypoperfusion (decompensated)” [2].

1.3. Contractility

Contractility is the intrinsic capacity of the myofibril to shorten its length independent of the pre and afterload. In The isolated myofibril contractility can be measured by quantifying degree and speed of shortening to stimulate it directly to a constant initial length and without resistance to its shortening (no-load) [3,4]. The intact heart contractility is very difficult to quantify its function and is always subjected to a diastolic load (preload) and a force that has to overcome during his emptying (afterload).

1.4. Cardiac Reserve

Cardiac Reserve is the ability of the heart to increase cardiac output [3].

- A. Chronotropic reserve: is the ability of the heart to increase the cardiac output by increasing heart rate [3].
- B. Systolic reserve: “is the ability of the heart to increase cardiac

output through increasing its contractility, which depends on the anatomofunctional integrity of the myofibril [4,5] (shifts upward Starling curve) [6,7].

Today the closest way to meet the heart’s intrinsic contractile State is the end systolic relationship stress/volume or pressure/volume at which generates a curve that is extrapolated to pressure 0, this curve has been called “End systolic elastance” (E_{max}) [5]. This implies that a ventricle has more contractility when reduce the systolic volume higher magnitude to one greater afterload than another whose systolic volume is higher for the same afterload [5], unfortunately the method is little practical and difficult to achieve in clinical settings [6], but it is very useful for basic and clinic research. It’s common for the term “Contractility” is erroneously used interchangeably to refer the “ventricular function”: ventricular function refers to the relationship between contractility, and instantaneous hemodynamic load (preload and afterload), and do not mean intrinsic contractile State (heart contractility) [3,4] (Figure 1).

MAXIMUM ELASTANCE: Pressure/volume curves: It is the line that joins the convergence points of the stroke volume (SV) and the stroke stress (SS) and which is extrapolated to 0 mmHg.

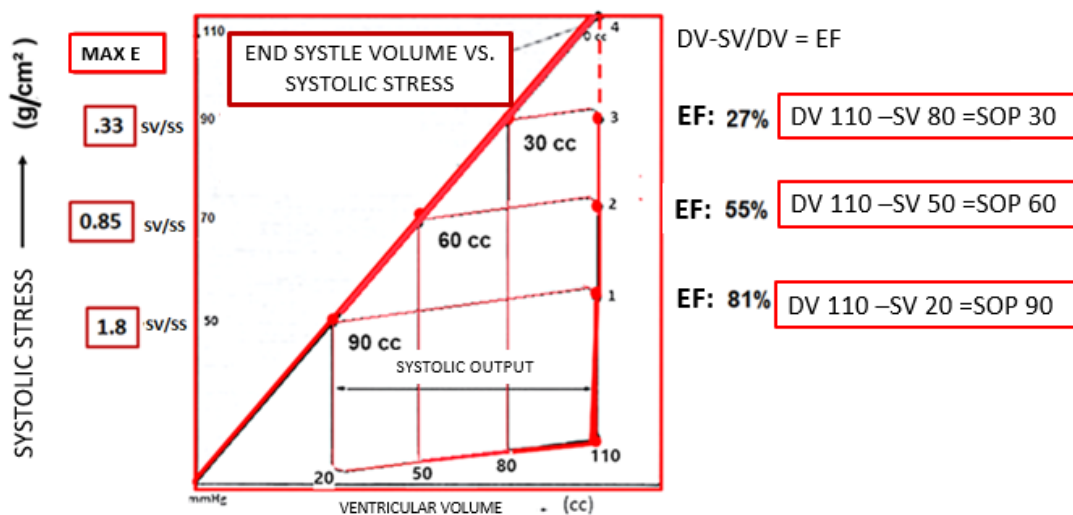


Figure 1: The heart's function depends on its intrinsic contractility and the hemodynamic loads it experiences when pumping blood. Therefore, the intrinsic contractility of the intact heart could not be determined until the concept of MAXIMUM ELASTANCE was described.

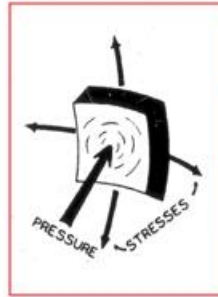
Wall stress (laplace law) [8,9](figure 2 -3).

Wall tension: is the force that tends to separate the myofibrils in length (cm) [8].

Wall Stress It is the force that tends to separate the myofibrils in area (cm²) [8].

Contractility Pre and Afterload

Stress: It is the force that tends to separate the myofibrils from each other per cm^2



$$S = \frac{P \times R}{2h}$$

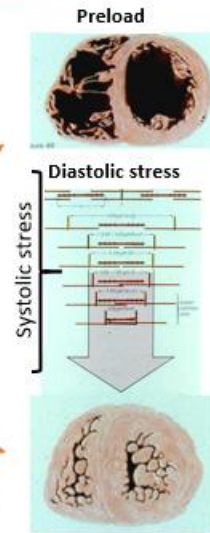


Figure 2: Afterload definition. It is the resistance per unit area of the myocardium that opposes ventricular contraction in order to raise blood pressure, open the aortic valve, and expel blood into the great vessels. It is calculated using Laplace's Law

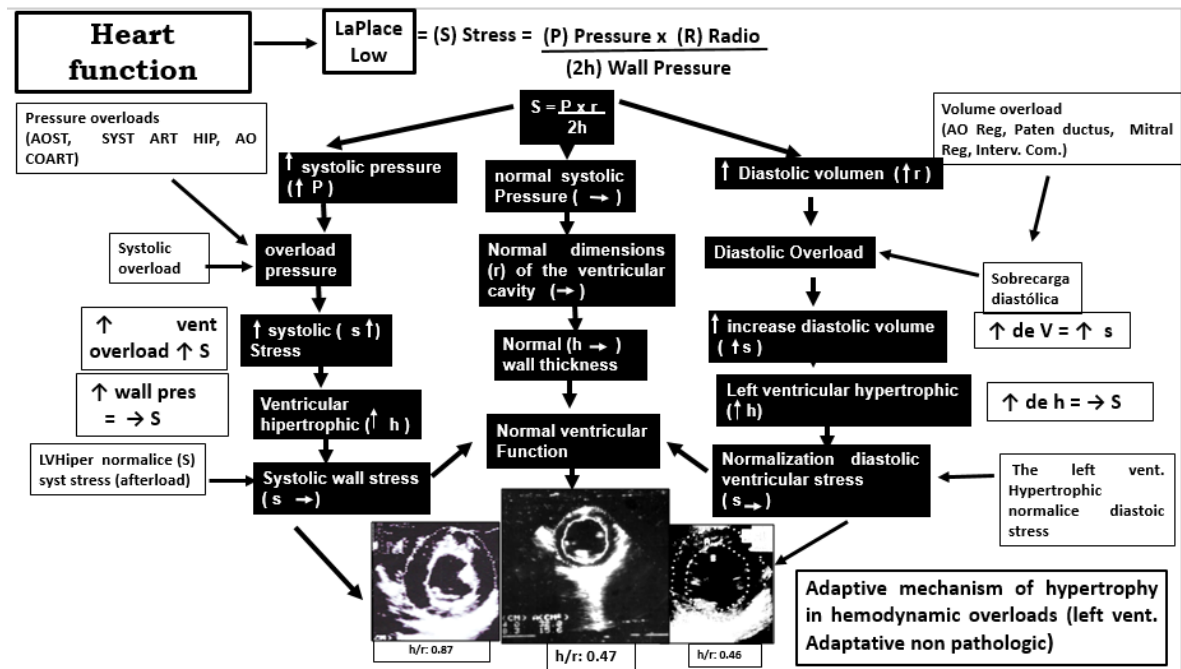


Figure 3: Heart function

1.5. Preload

It is the length of the myofibril at rest, immediately before ventricular contraction. In the intact heart is represented by the diastolic volume than in normal conditions generates a force that increases the area of the myofibril immediately before contraction (diastolic wall stress). Under normal conditions, both the preload

(diastolic volume and diastolic wall stress) and cardiac output are normal. When increases the preload: increase in diastolic volume for any reason: (aortic regurgitation or mitral insufficiency) or when using the Frank Starling mechanism to offset the fall in cardiac output heart failure (Figure 1), even without an increase in heart rate (Table 1).

Normal	Heart Failure
EDV (cc) 150	300
EDV (cc) 60	210
SV (cc) 90	90
EF (%) 60	30
HR (x min.) 75	75
CO (ml/min) 6750	6750

Table 1: Hemodynamic Disorders in Heart Failure.

1.6. Afterload Definition

It is the resistance per unit area of the myocardium that opposes ventricular contraction in order to raise blood pressure, open the aortic valve, and expel blood into the great vessels. It is calculated using Laplace's Law. In Figure 4, it is shown that the presence of heart failure, left ventricular function curve shifts downward so for a normal diastolic volume, cardiac output would be reduced

insomuch that it would be impossible to maintain an adequate, cardiac output and tissue perfusion pressure: cardiogenic shock. The use of Starling mechanism allows normalizing cardiac output at the expense of increased diastolic volume (cardiomegaly) and therefore, diastolic pressure in the left ventricle (pulmonary congestion) and its limit is pulmonary edema.

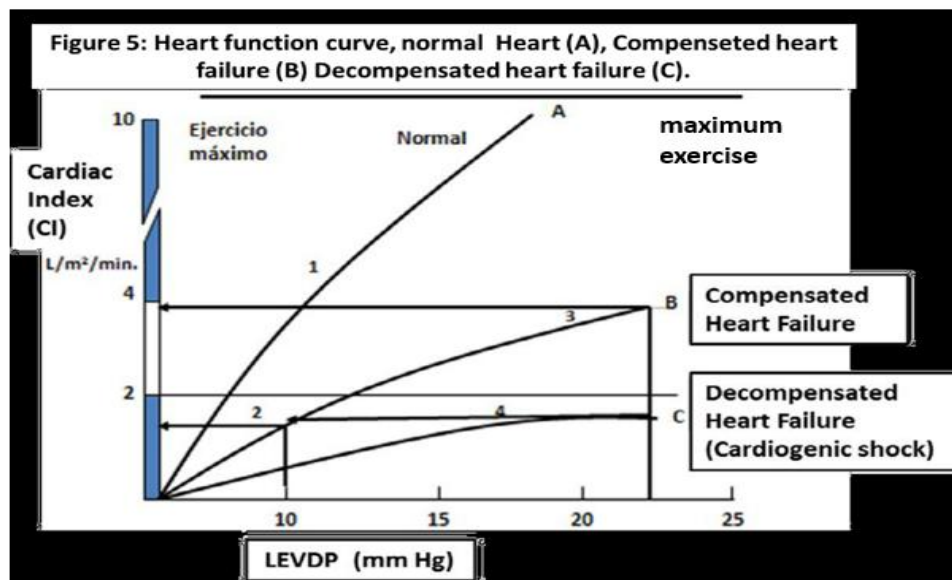


Figure 4: In a normal ventricular function curve, the cardiac index (CI) and ventricular diastolic pressure (PD) are within normal limits. In heart failure (HF), the curve shifts downward and PD increases by 27 mmHg. In cardiogenic shock, tissue perfusion and cardiac output reach levels incompatible with life.

It must be emphasized that the increase in preload (heart failure) implies increased diastolic wall stress (stretch per cm² of the myofibril), for which not all the increase in diastolic pressure implies increasing preload; as well, which brings as a consequence increased diastolic pressure. In stiff ventricles (restrictive cardiomyopathy) left ventricular diastolic pressure, increases without increasing diastolic volume, and preload remains normal.

1.7. Afterload

“Is defined as the force per unit sector area that opposes the ventricular contraction during the emptying of the heart towards the great vessels and obeys to Laplace law, so it is quantified by calculating the systolic wall stress” [8,9].

LAPLACE LAW

$$S = \frac{P \times r}{2h}$$

$$\text{STRESS} = \frac{\text{PRESURE} \times \text{RADIO}}{2 \text{ THICKNESS}}$$

Therefore, to correctly interpret the Forrester hemodynamic classification in acute myocardial infarction into four subgroups [10], patients in subgroup 2 (normal cardiac output and high wedge pressure) are not necessarily in heart failure, and an echocardiogram will be needed to confirm the diagnosis. In very severe heart failure, the patient exhibits significant cardiomegaly with acute pulmonary edema, very low EF, and a significant

elevation of venocapillary pressure.

CASE 1: Clinical Heart Failure

- Heart failure in functional class I
The patient is in heart failure and natriuretic peptides are keeping the patient in functional class I.

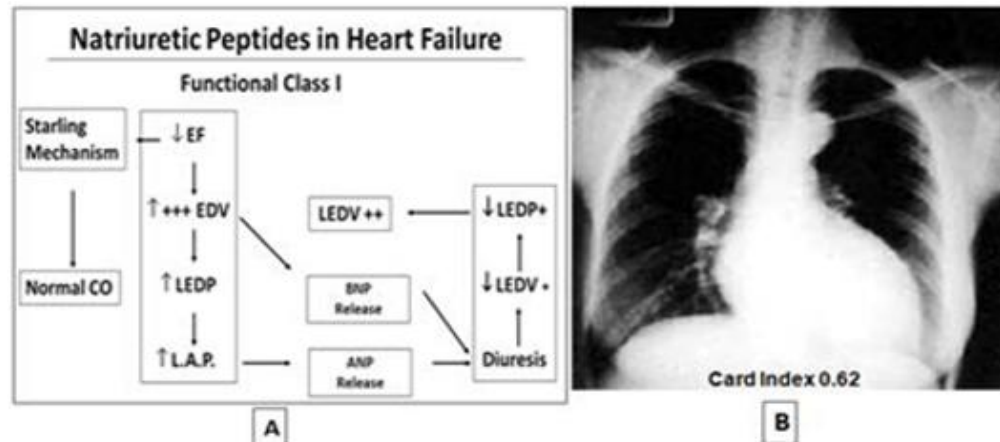


Figure 5: Heart failure in functional class I

CASE 2: Rheumatic Carditis

The treatment of rheumatic carditis should be with prophylaxis

using benzyl-methyl penicillin (1,200,000) intramuscularly every month for life [12].

ADULT RHEUMATIC CARDITIS: EVOLUTION SIX MONTHS AFTER WITHOUT TREATMENT

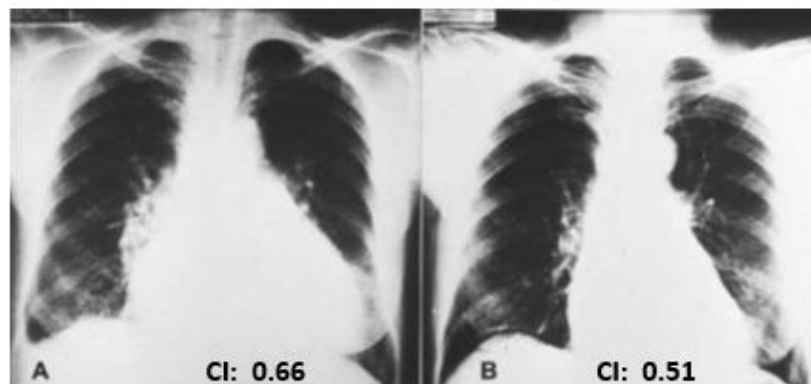


Figure 6: A) A 52-year-old patient with established double mitral valve lesion and atrial fibrillation presents with heart failure. The mitral valve lesion is clinically significant, and surgery is proposed. B) The patient misses a follow-up appointment and returns 6 months later intending to undergo surgery, now asymptomatic. Clinically, atrial fibrillation persists, and on auscultation, the double mitral valve lesion is mild. CI of 0.51. Retrospectively, a diagnosis of inactive adult rheumatic carditis was made.

CASE 3: Acute Myocardial Infarction, Functional Class IV, With Pulmonary Edema

When the isovolumic contraction intraventricular pressure increases rapidly, therefore due to wall stress; the aortic valve opens, myocardial contraction has to beat wall stress for myofibrils

shortening (afterload) [8,9,11]. higher pressure or intracavitary radio: greater systolic wall stress (higher afterload) (figure 7). Treatment: IV nitroglycerin, digitalis, furosemid, spironolactone, and captopril.

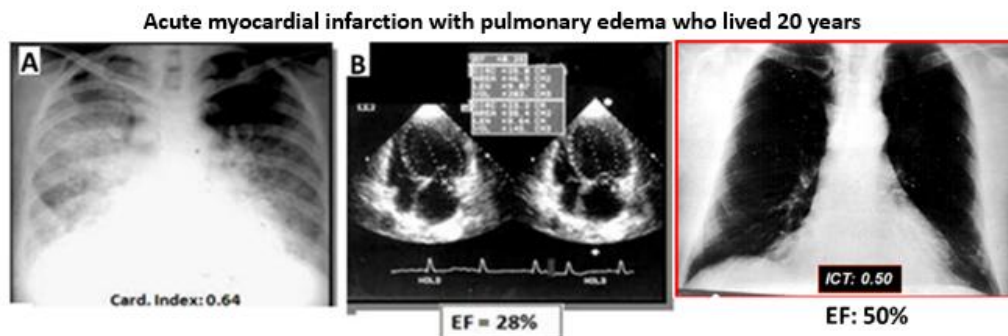


Figure 7: Acute myocardial infarction with pulmonary edema who lived 20 years.

CASE 4: Dengue Myocarditis

A 70-year-old woman, returning from a vacation in Acapulco to Mexico City, presented with unquantified fever, severe headache, myalgia, and general malaise. On December 1, 2012, she unexpectedly experienced syncope and was taken to the

Emergency Department. Her heart rate was 95 bpm, blood pressure 70/30 mmHg, respiratory rate 26 bpm, and temperature 38.6°C. On physical examination, she was conscious, lying supine, and unable to stand. The remainder of the physical examination was completely normal [13].

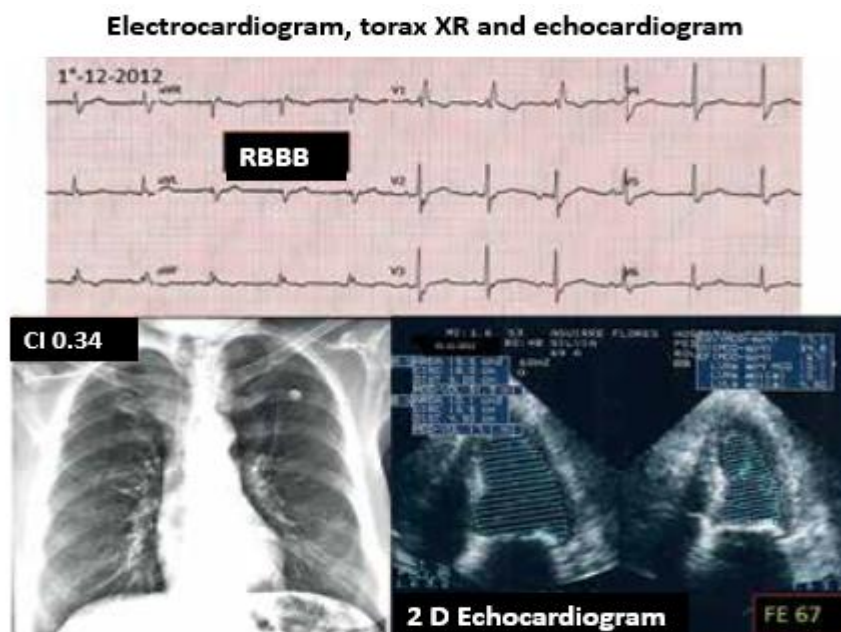


Figure 8: Acute Dengue

On the fourth day, the patient began to experience retrosternal pressure, dyspnea, and general malaise with a fever of 38.5°C. The electrocardiogram showed tachycardia with ST-segment depression, and the PA chest X-ray showed increased cardiomegaly

and signs of acute pulmonary edema (Figure 2). At that time, she was given an injection of 0.125 mg of digoxin, which significantly complicated her condition, as serious arrhythmias began to appear from the sixth day onward (Figure 3).

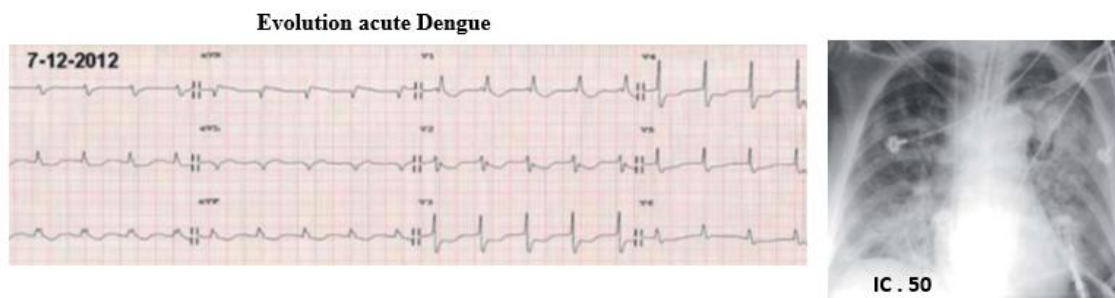


Figure 9: Evolution acute Dengue

Acute Dengue arrhythmias

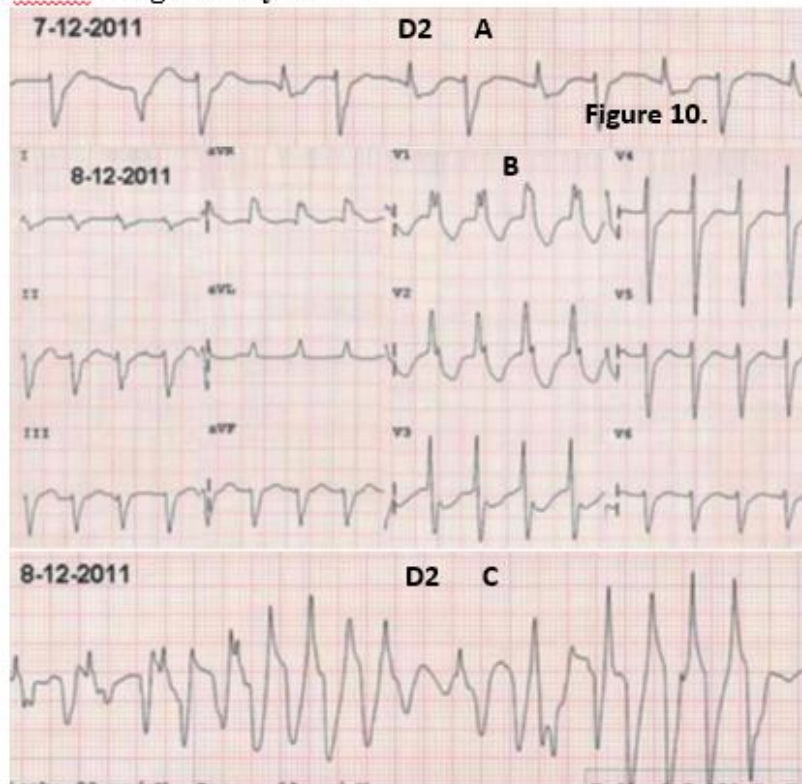


Figure 10: Rhythm disturbances. A: On the sixth day of admission, the patient is administered a dose of digoxin, and one hour later, bidirectional ventricular tachycardia appears, which resolves with the administration of phenytoin. B: On the seventh day, self-limiting, sustained monomorphic ventricular tachycardia appears. C: On the same day, polymorphic ventricular tachycardia appears(13).

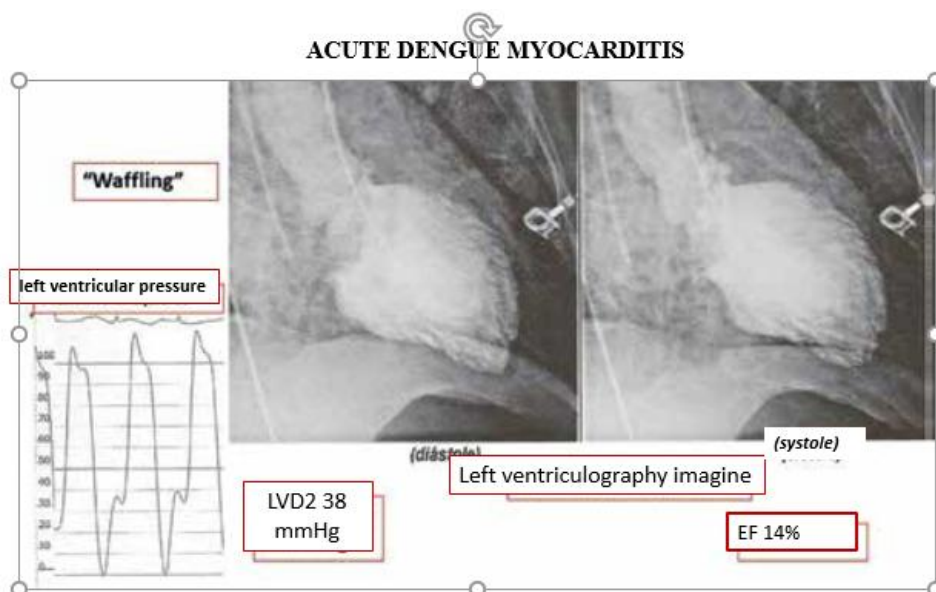


Figura 11: Cardiac catheterization. A: The left intraventricular pressure recording demonstrates a significant elevation of the left ventricular end-diastolic pressure (38 mmHg), which explains the appearance of pulmonary edema. B: Left ventriculography demonstrates an ejection fraction of 14% and mild mitral regurgitation. Note the horizontal striations in the anteroapical region of the left ventricle (waffling) which angiographically manifest myocardial edema. Rev Esp Cardiol 2014; 67 (3): 226-227.

LEFT VENTRICULAR MYOCARDIAL BIOPSY

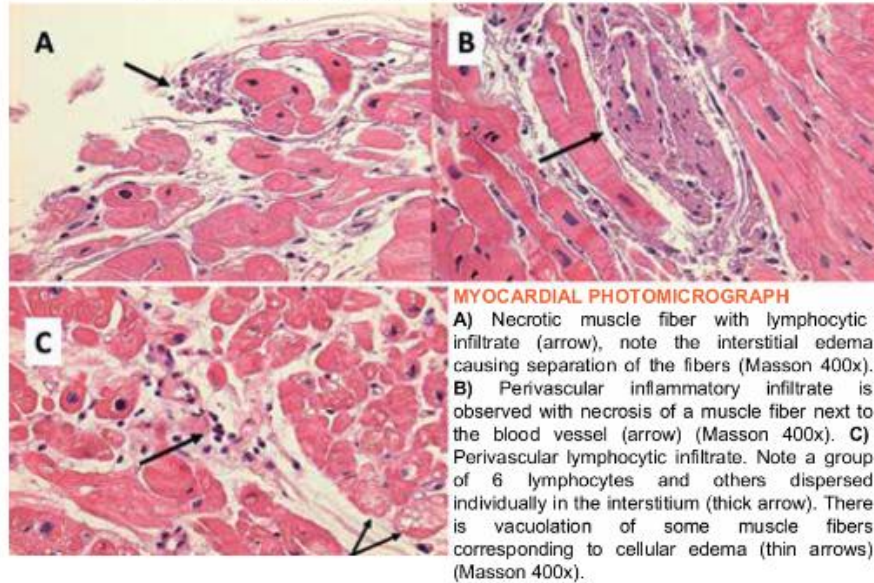


Figure 12: Left ventricular myocardial biopsy

1.8. Treatment and Evolution

Studies of biliary markers revealed. Dengue virus-specific IgM and IgG antibodies were reported positive. Oseltamivir was replaced

with ribavirin. The diagnosis of severe dengue myocarditis was made and steroid treatment was initiated with methylprednisolone (500mg I.V. bolus followed by 250 mg tid during three days).

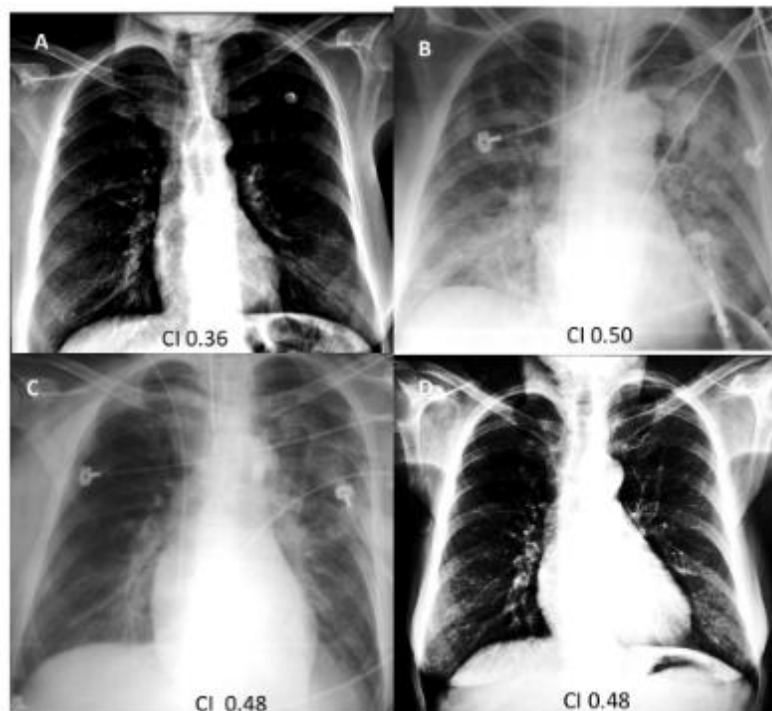


Figure 13: Patient evolution: The patient was discharged home 13 days after the onset of the clinical picture completely asymptomatic and managed to live until the age of 85 when she died of natural causes

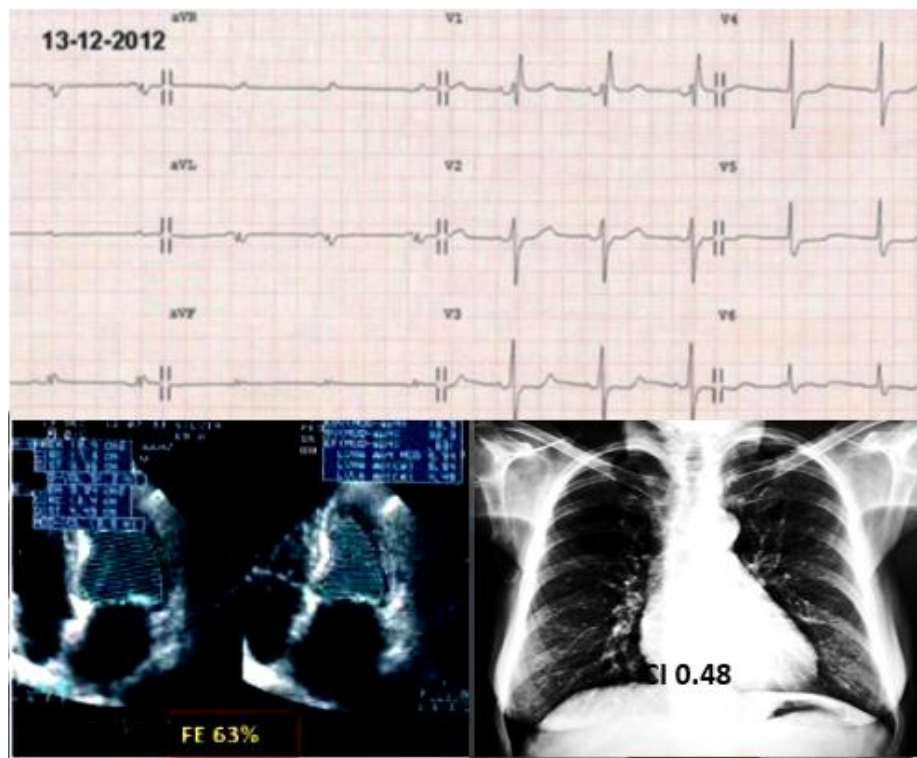


Figura 14: Hospital discharge. A: The electrocardiogram shows only mild ventricular repolarization abnormalities in the precordial leads. B: The echocardiogram shows a normal ejection fraction. C: The chest X-ray is normal.

CASE 5: ACUTE HEART FAILURE SECONDARY TO TWO VALVULAR DISEASES

(34-year-old male patient) Heart failure functional class IV with

Ao aneurysm + IAO++++, + Mitral insufficiency +++, dp/dt 646 mmHg/sec. + PAH, EF 28% and cardiac cachexia [14] (Figure 15).

(34-year-old male patient) with severe heart failure, functional class IV.

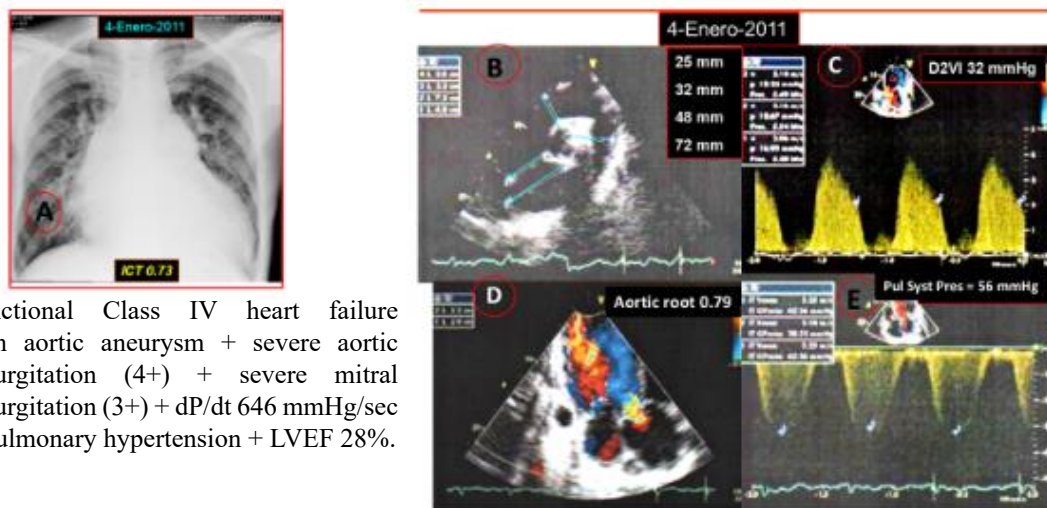


Figure 15: Aortic aneurysm + severe aortic insufficiency + D2 LV ↑ + PAH

Calculation of Systolic Stress to Assess Surgical Indication (Figure 16)

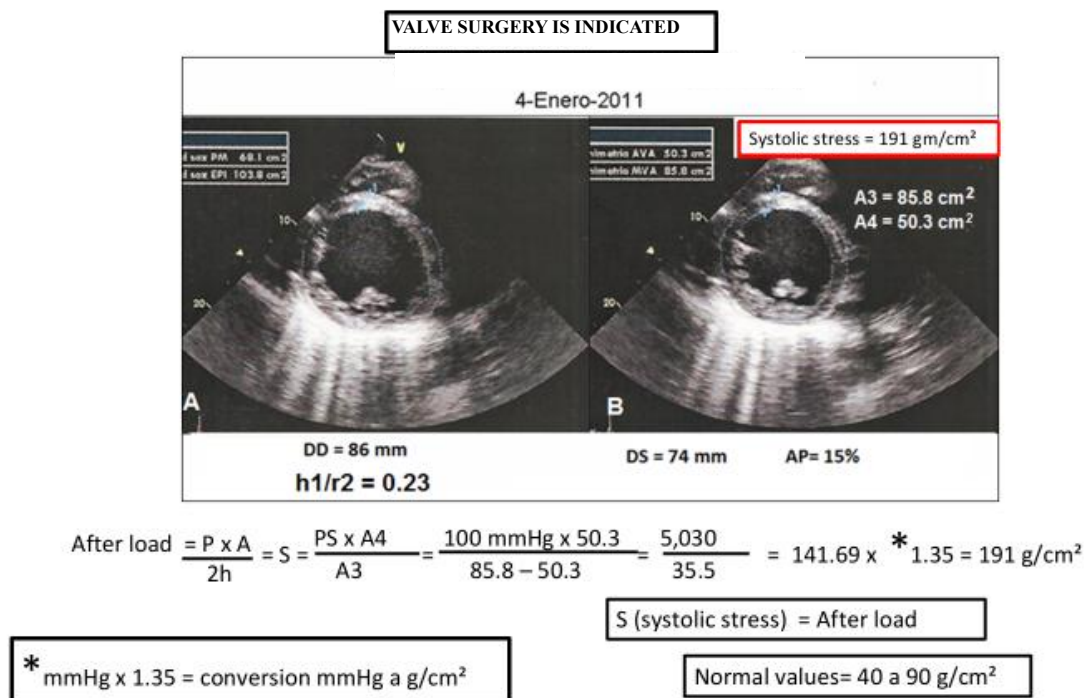


Figure 16: Calculation of systolic stress

1.9. Therapeutic Decision

1. Large aortic aneurysm with very severe aortic regurgitation.
2. Very severe mitral regurgitation.
3. Inadequate left ventricular hypertrophy.
4. Significant left ventricular dilation.
5. Severe congestive heart failure.
6. Pulmonary arterial hypertension.
7. Probable hibernating myocardium

To make the therapeutic decision and assess surgical mortality¹ and the possibilities of both postoperative survival and quality of life in the cardiologists' panel meeting, it was concluded that

mortality was very high and the chances of success were very low.

1.10. Comment

However, since 2006, a method has been described for calculating left ventricular systolic stress (afterload) in patients with severe diastolic ventricular overload, to assess the possibility of surgery. This gave us the guideline to establish the surgical indication based on the fact that the heart failure was due to excessive afterload and NOT to intrinsic myocardial damage, so this patient was indicated for complex surgery of aortic root replacement with aortic valve replacement (Bental and Bono) and mitral valve replacement (St. Jude prosthesis) 21-January-2011

Preload Compensating Mechanism

(Starling mechanism)

Normal	Heart failure
RD (cc) 150	300
SV (cc) 60	210
CO (cc) 90	90
EF (%) 60	30
HR (x min.) 75	75
SO (ml. / min.) 6750	6750

"Entendiendo la Insuficiencia Cardiaca"
Arch Cardiol Mex 2006;76:431-347

Figure 17: Preload compensating mechanism

EF and afterload correlation

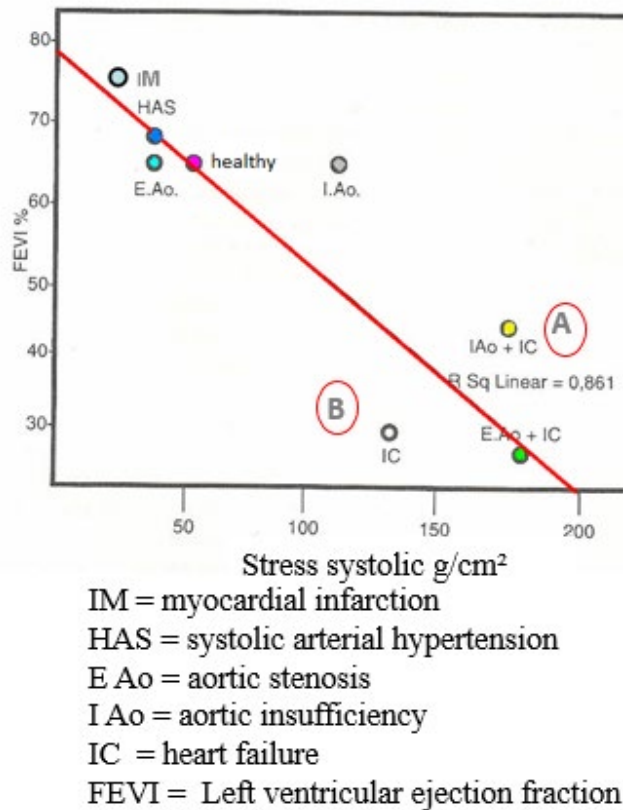
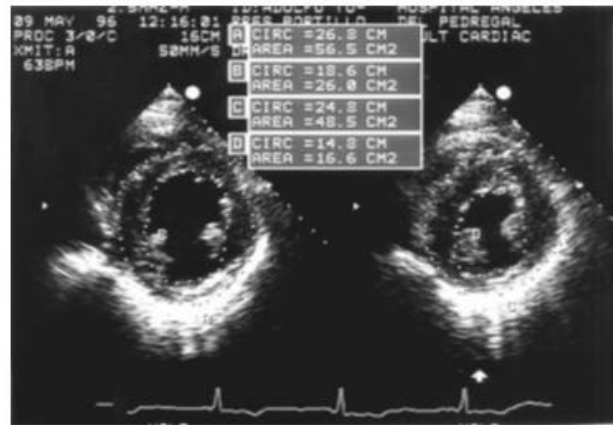


Figure 18: A) Note that aortic insufficiency (AI) constitutes increased left ventricular (LV) afterload, which explains left ventricular hypertrophy (LVH), a compensatory mechanism for this overload. B) Heart failure (HF) caused by myocardial damage does not correlate with the afterload line.

The patient progressed magnificently well to the point that he currently leads a normal life and his heart has a normal function (EF 70%) (Figure 20).

Criteria for surgical intervention in severe valvular heart disease



$$R = \sqrt{\frac{A}{\pi(3.1416)}}$$

A ₁	56.5	R ₁	4.24	R ₁ - R ₂ : h ₁	=	1.37
A ₂	26	R ₂	2.87	h ₁ /R ₂	=	0.45
A ₃	48.5	R ₃	3.92	R ₁ - R ₃ : h ₂	=	1.63
A ₄	16.6	R ₄	2.29	h ₁ /r ₄	=	0.71

Figure 19: Calculation of systolic stress and ventricular wall thickness

Long-term pre- and post-operative X-rays

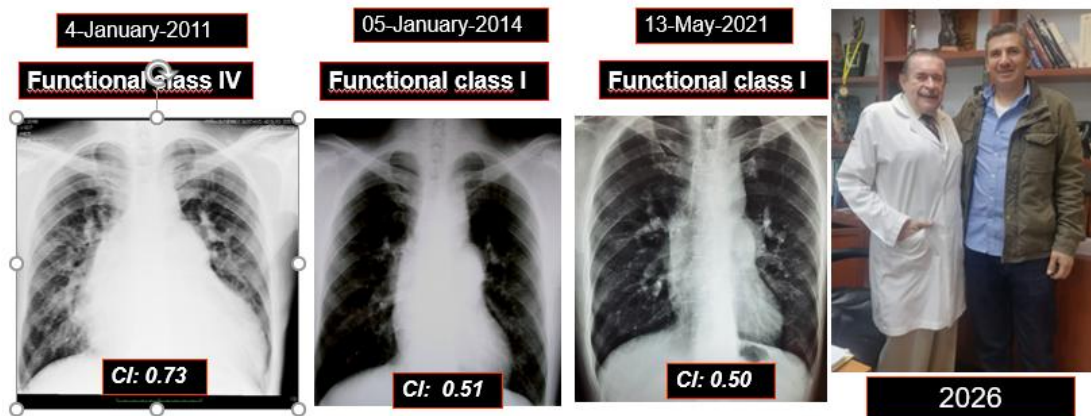


Figure 20: The image shows the extraordinary evolution of this patient who is now 49 years old in 2026, leading a normal home and work life

CASE 6: Acute Aortic Insufficiency

52-year-old male patient arrives at the emergency room with dyspnea at rest orthopnea, recumbent cough, “bloody” expectoration, heart rate 90 per minute, and Blood Pressure 80/50 mmHg, with audible pulmonary crackles at distance, and at the

mitral area strong first sound with a grade II systolic murmur with non lisent second sound.

The chest X-Ray shows important enlargement of the heart with pulmonary edema (Figure 21).

Acute aortic insufficiency



Figure 21: The chest X-ray shows marked cardiomegaly accompanied by acute pulmonary edema.

The M-mode echocardiogram demonstrates significant dilation of the right ventricle with thinned interventricular septum, great dilation of the left ventricle with thinned posterior wall, and greatly increased LV systolic diameter. The shortening fraction was 23% (Figure 22).

1.11. Conclusion: Severe Global Heart Failure.

A. The M-Mode Echo recording clearly shows the early closure of the mitral valve, which incontrovertibly establishes the diagnosis of Acute Aortic Insufficiency; I emphasize that with this simple method, the diagnosis of Acute Aortic Insufficiency can be immediately established in an exact, bloodless, fast and cheap manner, which makes it possible to clarify the precise

diagnosis to attempt the immediate treatment of this rapidly fatal disease.

B. The simultaneous recording of aortic and left ventricular pressures quantifies the exact levels of intracavitary pressures between the aorta and the left ventricle. In this case, it should be noted that the LV diastolic pressure is combined with the aortic pressure and the D2 of the LV is quantified at 40 mmHg, which explains the very serious situation that the patient presents with acute lung edema in the presence of 40 mmHg. venocapillary pressure; If this condition is not corrected immediately, the patient dies from intractable acute pulmonary edema. (Figure 23).

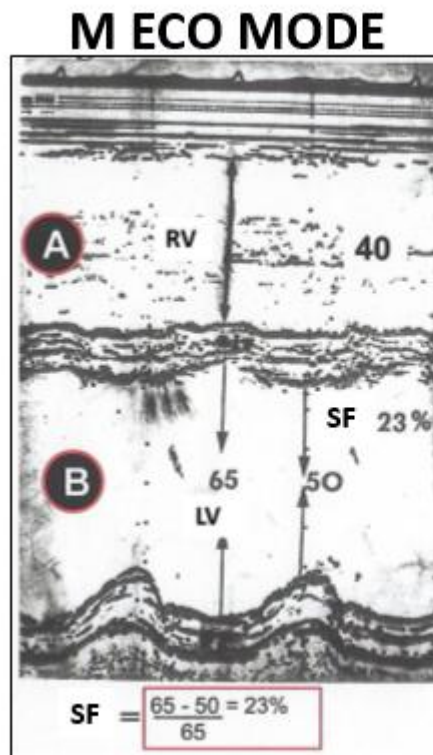


Figura 22: The study shows dilation of both ventricles and signs of severe heart failure.

The in figure 24

- The parasternal long axis view with color-coded Doppler shows that the regurgitant flow covers 90% of the aortic annulus: extreme large aortic insufficiency.
- Early closure of the mitral valve shows that it is Acute Aortic Insufficiency.
- Protodiastolic aortic regurgitant flow is demonstrated (colored mosaic). Throughout mitral closure, a mosaic of colors is

observed between the two leaflets that begins in the middle of diastole (before the R wave) and continues until the next opening of the mitral valve; That is to say, it is a Diastolic Mitral regurgitant flow: Diastolic Mitral Insufficiency. This is due to the fact that the intraventricular diastolic pressure is so high that it overcomes the mitral valve and allows blood to return during diastole.

Acute aortic insufficiency

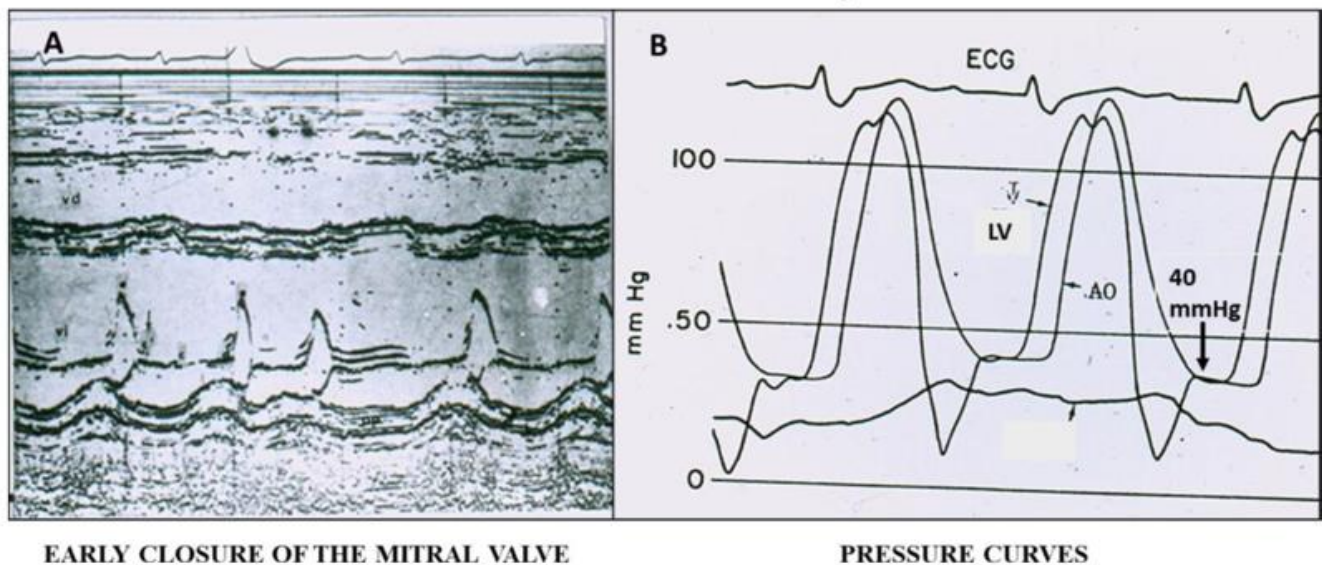


Figure 23: Acute aortic insufficiency

- Mitral regurgitation flow that begins before the R wave (see arrow) (diastolic mitral regurgitation).
- The M-mode echo shows early closure of the mitral valve (green line) that coincides with the onset of the Systolic murmur (diastolic mitral regurgitation). Clicking mitral valve closure (Ch) takes the place of a IV sound and is confused with a I sound.
- Note the mosaic of colors that appears during diastole and reflects diastolic mitral insufficiency.
- The phonocardiogram shows that the closing click is before the 1st sound which is muted, meaning that it shows that the 1st sound really appears in diastole followed by the mitral regurgitant murmur that begins in diastole until the end of the following severe aortic insufficiency [15].

Note: Mitral regurgitation is not only diastolic, see that in the phonocardiogram it is also systolic: 1st known case in which mitral regurgitation is continuous (systolic and diastolic) The patient was taken to the operating room and died during the transfer.

1.12. Conclusion

Acute Aortic Insufficiency is the sudden onset disease that can be considered the most serious in the cardiology clinic due to the rapid evolution towards death. This is why the patient must

arrive at the cardiology hospital as soon as possible and the study for diagnosis must be as quick as possible, since, as happened in this patient, all the diagnostic elements were in place until cardiac catheterization was performed to take him to the operating room [16], unfortunately we did not have time to enter surgery. Most frequent etiologies reported in the literature are; acute aortic dissection and infectious endocarditis [17], in the case presented here we had no evidence of either acute aortic dissection or infectious endocarditis, unfortunately when taken to the operating room the patient died and we were not able to have the autopsy study to know the cause of this serious illness.

CASE 7: Ventricular Assistance (Impella)

Ventricular assistance (using the IMPELLA device) refers to the process of maintaining tissue perfusion and thus the patient's life via artificial mechanical devices in cases of irreversible, end-stage decompensated heart failure (functional class IV).

This device has been used primarily in patients undergoing primary angioplasty for myocardial infarction complicated by cardiogenic shock. The entire insertion procedure takes approximately 11 minutes; the device increases cardiac output and reduces pulmonary capillary pressure. While IMPELLA was initially used for acute myocardial infarction complicated by cardiogenic shock,

these devices are now being used in an outpatient procedure.

IMPELLA is a family of intravascular micro-blood pumps that act as temporary mechanical circulatory support devices. Minimally

invasive, they draw blood from the affected ventricle and propel it into the aorta or pulmonary artery, allowing the heart to rest and recover while maintaining blood flow to vital organs [18].

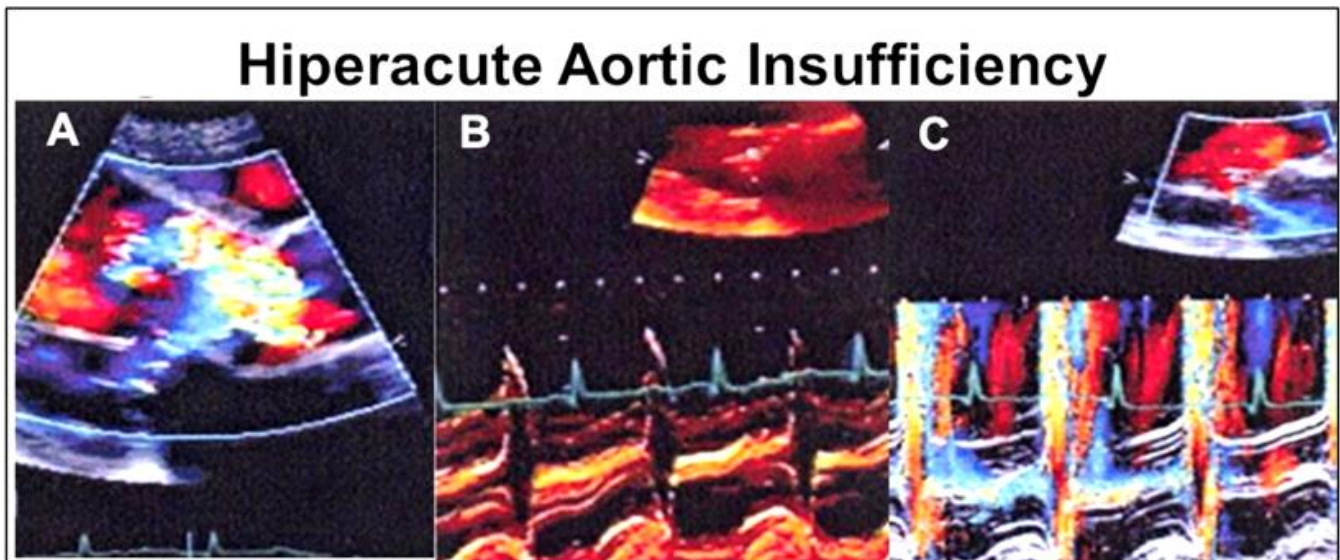


Figure 24: Hiperacute aortic insufficiency

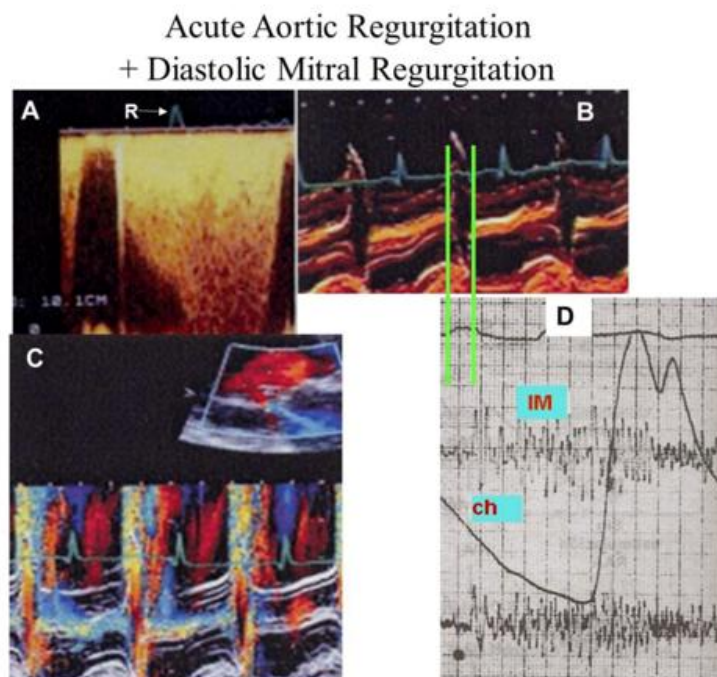


Figure 25: See text

Effect on ventricular function

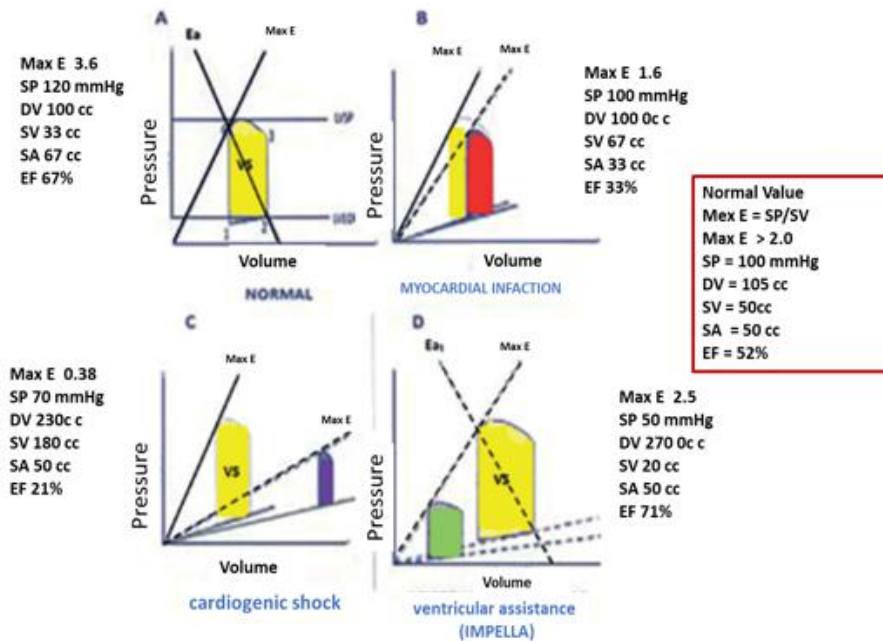


Figure 26: Impella ventricular support

2. Diastolic Heart Failure

2.1. Introduction

Regarding heart failure, there are hundreds of articles covering its definition, diagnosis, and treatment; however, certain clinical conditions—such as systemic body congestion, pulmonary congestion, or systemic venous congestion—are often attributed to it. These include the biventricular manifestations of Davis's disease (anasarca); pure mitral stenosis with pulmonary edema; African endomyocardial fibrosis (whether affecting the left ventricle or both ventricles, presenting with progressive dyspnea leading to pulmonary edema); and cases where Davis's disease affects only the right ventricle, presenting as massive cardiomegaly without pulmonary congestion. Consequently, determining whether diastolic heart failure truly exists has been a matter of great importance. Ramachandran S. Vasan y Daniel Levy, diastolic heart failure was defined as patients with symptoms suggestive of heart

failure who had an ejection fraction >50%¹⁹.

Case 1: Diastolic Heart Failure in the Elderly

An 83 year old female patient with progressive exertion dyspnea, edema and 4 months after progressed to dyspnea on minor exertion, orthopnea, and paroxysmal nocturnal dyspnea accompanied by progressive edema of the lower limbs up to anasarca with functional class III-IV, physical examination reveals rhythmic heart sounds and IV sound on auscultation, with a BP of 105/60 mmHg. Chest X-ray PA shows cardiac enlargement (CTI: 0.58) with signs of pulmonary venocapillary hypertension in the lung fields. (Figure 27-A). Electrocardiogram: Sinus rhythm: 90 x min., P wave 0.22 mm; PR space 0.28, QT 0.01 sec.; $\Delta P + 60^\circ$, $\Delta QRS + 30^\circ$, Sokolof 29 mm, left atrial enlargement, 1st degree AV block, right ventricular dilation (Figure 27-B).

DIASTOLIC HEART FAILURE



Figure 27: Diastolic heart failure

Figure 28-A demonstrates a very intense RESTRICTIVE PATTERN that contrasts with a dp/dt (Figure 28-B) and also high EF (28-C) that contrasts with the dilation of the IVC. Figure 28-D signs of DIASTOLIC HEART FAILURE. The transmitral diastolic flow shows a restrictive pattern E/A ratio is 3.5, deceleration 130

mm/sec.; Likewise, the E/E' ratio 13.25 and the TAPSE at 22 mm and the inferior vena cava shows dilation without inspiratory collapse. Calculation of the pulmonary systolic pressure, through the tricuspid regurgitant flow 50 mmHg.

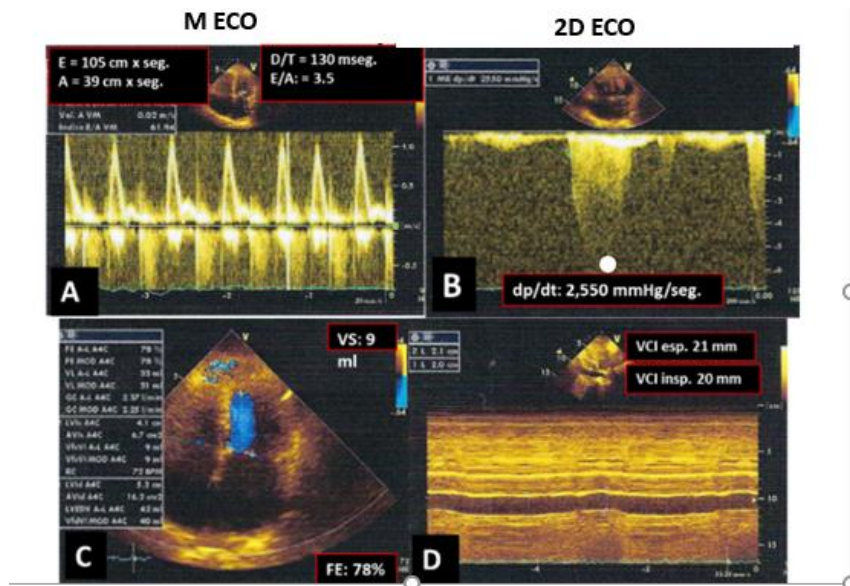


Figure 28: A) Pulsed-wave Doppler of transmitral flow showing a clearly restrictive pattern, with an E/A ratio of 3.5 and a very rapid deceleration time of 130 ms. B) Continuous-wave Doppler showing 2550 mmHg/s. C) Ejection fraction (EF) of 78%. D) Assessment of the inferior vena cava during expiration and inspiration demonstrates the presence of venous hypertension due to diastolic heart failure.

Case 2: CARDIAC AMYLOIDOSIS

A 22-year-old male patient who arrived with amyloidosis and congestive heart failure with fatigue, on exertion dyspnea, jugular plethora, and ascending lower limb edema. The diagnosis was

established with a 2D echo and myocardial scan20 (Figure29- A – B).

Simultaneous biventricular cardiac catheterization with high diastolic pressures with slow relaxation (Figure 29-C).

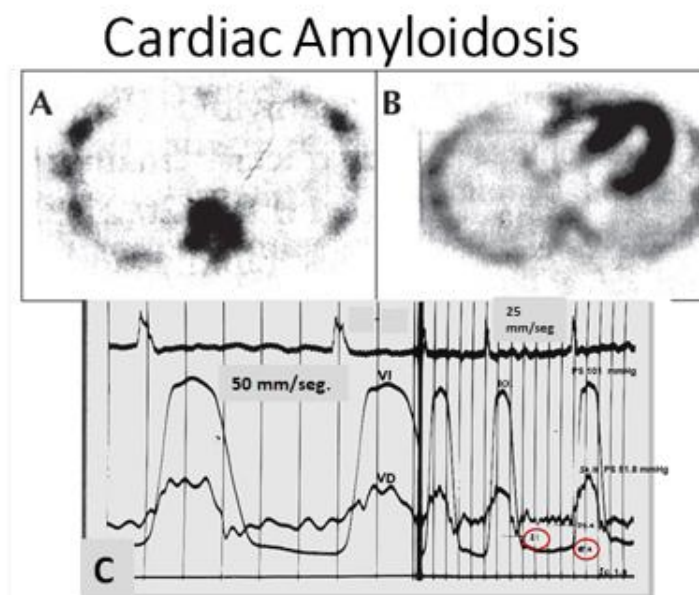


Figure 29: Cardiac amyloidosis with 99MTC-DPD. A) Negative cardiac scintigram (absence of radioisotope uptake). B) Cardiac scintigram that captures the radioisotope and demonstrates the diagnosis of cardiac AMYLOIDOSIS. C) In cases of amyloidosis the ventricular filling pattern rarely shows elevated filling pressure with a slow relaxation pattern.

Case 3: Idiopathic diffuse interstitial fibrosis

A 60 year old female patient with a 6 month history of fatigue followed by progressive dyspnea until resting, jugular plethora, progressive lower limb edema, ascites and anasarca, functional class III-IV. With M mode Echo small ventricular cavities but the fractional shortening was very low. During the initial ventricular filling, the opening of the mitral valve (E/E') but the final opening of the valve was very brief (Figure 30-A); that means that the early intraventricular pressure is low and the final pressure is very high that corresponds to diastolic pressures with morphology of dip and plateau (Figure 31-C).

To the 2D echo in 4 chambers view reveals small ventricles and the atria's very dilated (Figure 30-B), and this conditions that in diastole the blood returns from the ventricle to the atrium (diastolic mitral regurgitation) (Figure 31-A-B) and that in systole there is no valvular regurgitation and this is a consequence of the enormous elevation of left ventricular diastolic pressure that in the pressure curves demonstrates the image of dip and plateau the recording of intraventricular pressures show an image of "square root" at different levels (Figure 30-C) and that in this case the anatomical and clinical hemodynamic picture is caused by massive interstitial myocardial fibrosis (Figure 32).

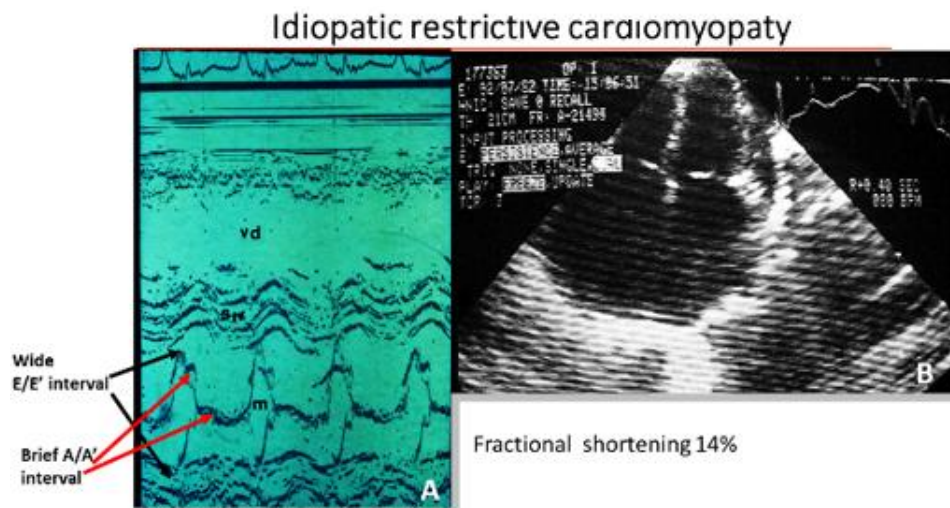


Figure 30: A) The Interval behavior E/E' and A/A' translate D1 wide and D2 the same short interval = Pressure 1< and pressure 2> +++. B) The small ventricular cavities with great dilation of both atria with a clinical picture of capillary and systemic venous hypertension are shown.

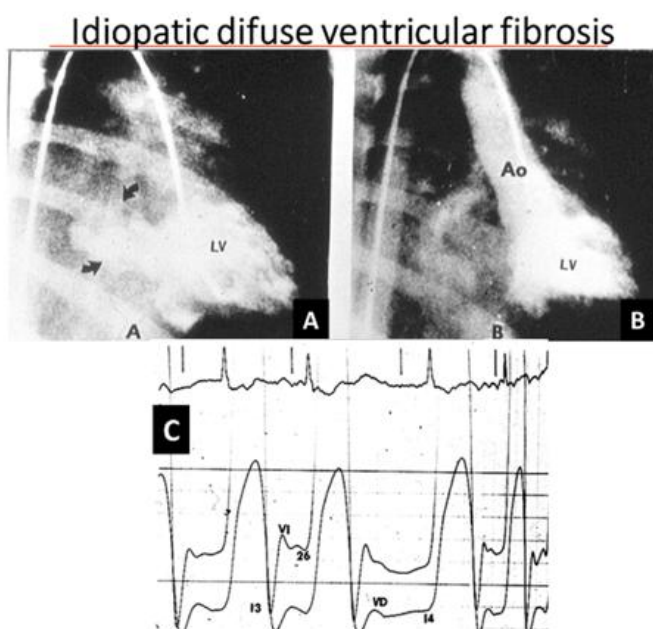


Figure 31: A) Diastolic mitral regurgitation (it means ventricular diastolic hypertension). B) In systole there is no regurgitation. C) Simultaneous pressure curves revealing Dip and Plateau at different ventricular levels.

IDIOPATHIC DIFFUSE INTERSTITIAL FIBROSIS

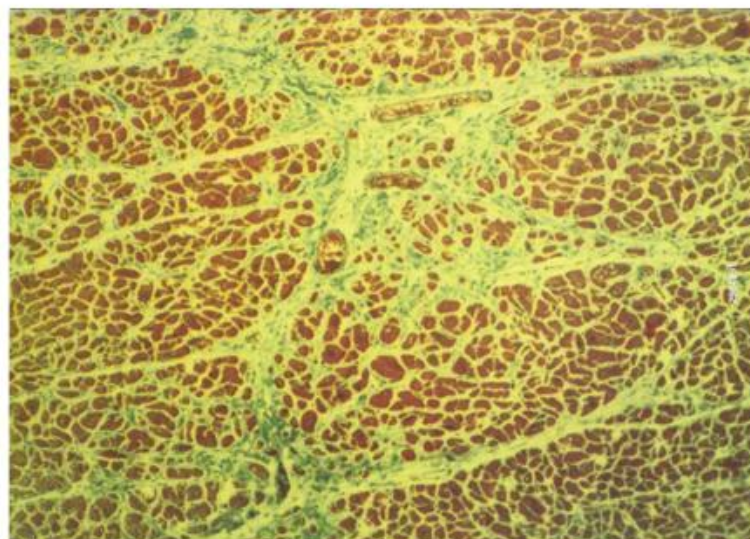


Figure 32: Idiopathic Diffuse Interstitial Fibrosis

3. Conclusion

A. Etiology: Diffuse interstitial fibrosis of the elderly, cardiac amyloidosis, and idiopathic diffuse interstitial fibrosis (see histological section).

B. Pathophysiology: Increased systolic function parameters (AP, EF, and dp/dt) [22].

1. Myocardial damage resulting from extreme myocardial stiffness, which causes a severe reduction in ventricular compliance.
2. Systolic function responds with increased LV contractility, sufficient to maintain antegrade systolic function.
3. Impaired ventricular diastolic filling renders the ventricle unable to accommodate venous return; this leads to a backup extending from peripheral veins to central abdominal veins (causing ascites, hepatomegaly, splenomegaly, and renal venous congestion), while pulmonary venous congestion progresses to pulmonary venocapillary congestion (DIASTOLIC HEART FAILURE).

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