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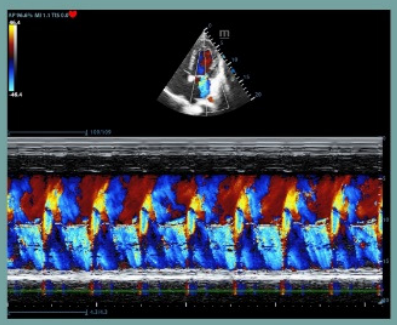

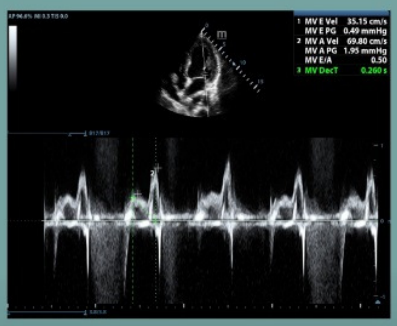

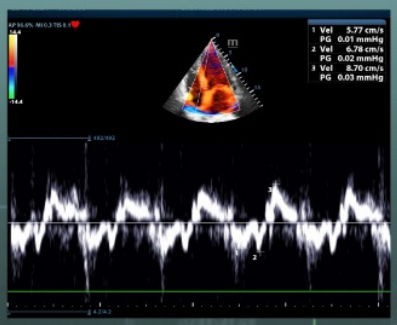

Florina Pârv
Adrian Apostol

CLINICAL TRANSTHORACIC ECHOCARDIOGRAPHY

FOR STUDENTS

MANUALE

Editura „Victor Babeș"
Timișoara, 2025

“VICTOR BABEŞ” UNIVERSITY OF MEDICINE AND PHARMACY FROM TIMIŞOARA

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CONTENTS

ABBREVIATIONS

AI	aortic insufficiency
ALT	alanin aminotransferase
Ao	aorta
APAP	average pulmonary arterial pressure
AR	aortic regurgitation
AS	aortic stenosis
AST	aspartate aminotrasferase
AV	aortic valve
AVA	aortic valve area
ATP	adenosin triphosphat
BMI	body mass index
BSA	body surface area
BP	blood pressure
CABG	coronary artery bypass graft
CO	cardiac output
CW	continuous wave Doppler
DCM	dilated cardiomyopathy
DT	deceleration time
ECG	electrocardiogram
EDD	end-diastolic diameter
EDV	end-diastolic volume
EROA	effective regurgitant orifice
ESD	end-systolic volume
GGT	gamma-glutamyl transferase
IAS	interatrial septum
INR	international normalised ratio
IVC	inferior vena cava

IVRT	isovolumic relaxation time
IVS	interventricular septum
IVUS	intravascular ultrasound
HCM	hypertrophic cardiomyopathy
HF	heart failure
HR	heart rate
IVS	interventricular septum
LA	left atrium
LAV_i	left atrial volume indexed
LDL	low density lipoprotein
LGE	late gadolinium enhancement
LV	left ventricle
LVEF	left ventricle ejection fraction
LVEDD	left ventricle end diastolic diameter
LVEDP	left ventricular end-diastolic pressure
LVESD	left ventricle end systolic diameter
LVH	left ventricular hypertrophy
LVOT	left ventricle outflow tract
MAPSE	mitral annular plane systolic excursion
MI	mitral insufficiency
MPG	mean transvalvular aortic pressure gradient
MR	mitral regurgitation
MRA	mineral receptor antagonist
MRI	magnetic resonance imaging
MS	mitral stenosis
NTproBNP	N-terminal pro-B-type natriuretic peptide
PA	pulmonary artery
PAHT	pulmonary arterial hypertension
PAP	pulmonary artery pressure

PASP	pulmonary artery systolic pressure
PHT	pressure half-time
PISA	proximal isovelocity surface area
PSAX	parasternal shortaxis
PW	pulsed wave Doppler
PWT	posterior wall thickness
Qp/Qs	pulmonary blood flow (QP)/systemic blood flow (Qs)
RAP	right atrial pressure
RA	right atrium
RV	right ventricle
RVEDP	right ventricular end-diastolic pressure
RVEF	right ventricular ejection fraction
RVH	right ventricular hypertrophy
RVOT	right ventricular outfow tract
RWT	relative wall thickness
SAP	systolic arterial pressure
SF	shortening fraction
$SGLT2_i$	sodium-glucose cotransporter-2 inhibitor
SPAP	systolic pulmonary arterial pressure
TAPSE	tricuspid annular plane systolic excursion
TI	tricuspid insufficiency
TOE	transesophageal echocardiography
TSH	thyroid-stimulating hormone
TR	tricuspid regurgitation
TVI	time-velocity integral
TTE	transthoracic echocardiography
SV	stroke volume

I. Normal transthoracic echocardiography

1. GENERAL NOTIONS. ULTRASOUND WINDOWS (ADRIAN APOSTOL)

General rules

1. Patient set-up
   - The ultrasound examination is performed with the patient in a left lateral decubitus position, with the left arm and hand placed under the head, and the right arm at the side of the body.

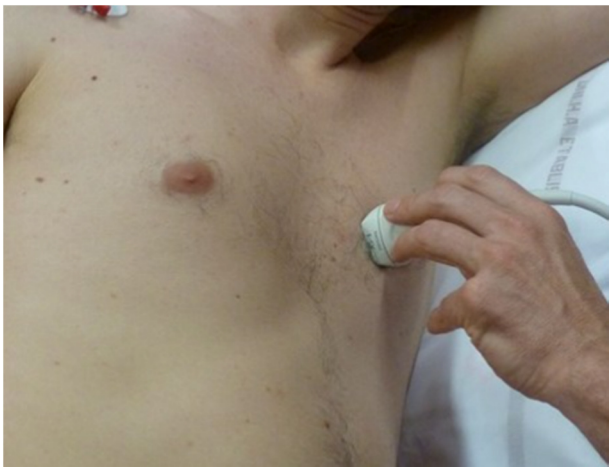
2. Examination conditions
   - Three electrodes are placed on the thorax to enable the machine to acquire an ECG trace and adapt data acquisition to the cardiac cycle.
   - The echocardiograph can be positioned on the patient's left or right, depending on the team's practice.

Main effects

Normal transthoracic echocardiography-Doppler:

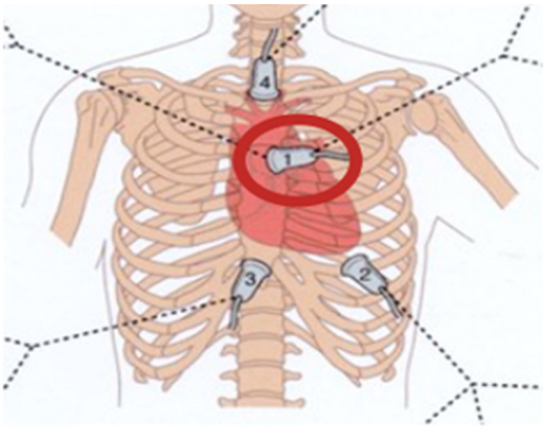
- Left parasternal window
- Apical window
- Subcostal window
- Supra-sternal window

I Left parasternal section



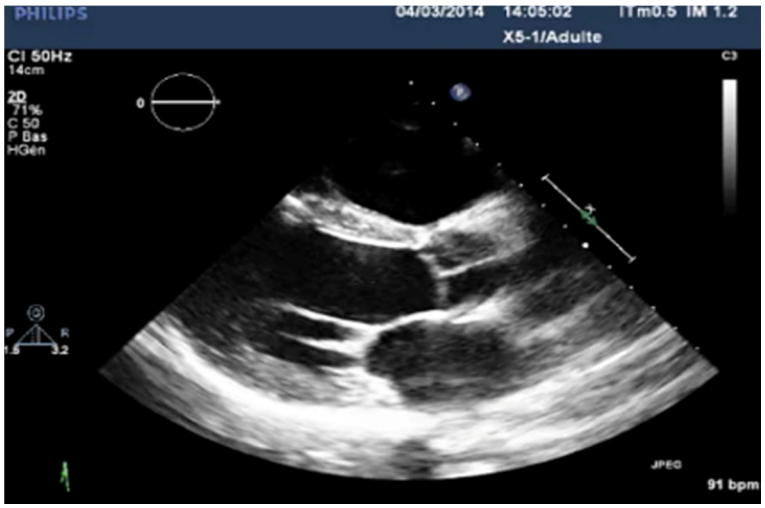
Patient positioning:

- Patient in left lateral decubitus position
- Left arm well clear of the chest, under the head
- 3rd intercostal space, left edge of sternum



Criteria for a good cut:

- Interventricular septum as horizontal as possible
- No visualization of the septal leaflet of the tricuspid valve

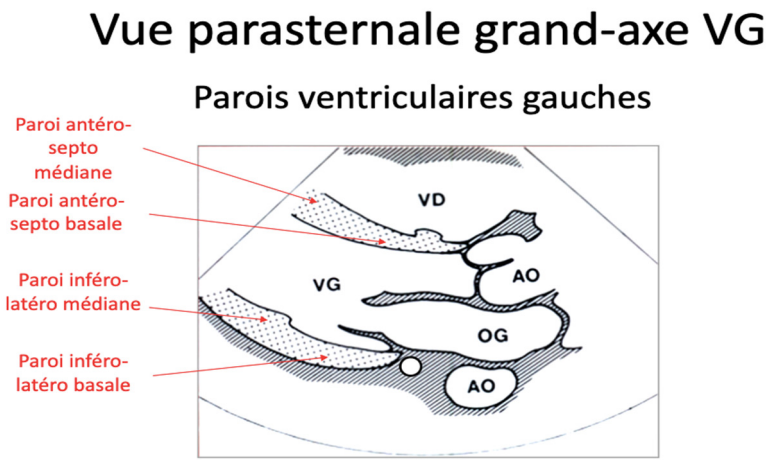


Left parasternal long-axis section - 2D mode

Visualized structures:

1. Left ventricle

- Infero-lateral wall (basal and middle segment)
- Anteroseptal wall (basal and middle segment)
- Apex generally not visible



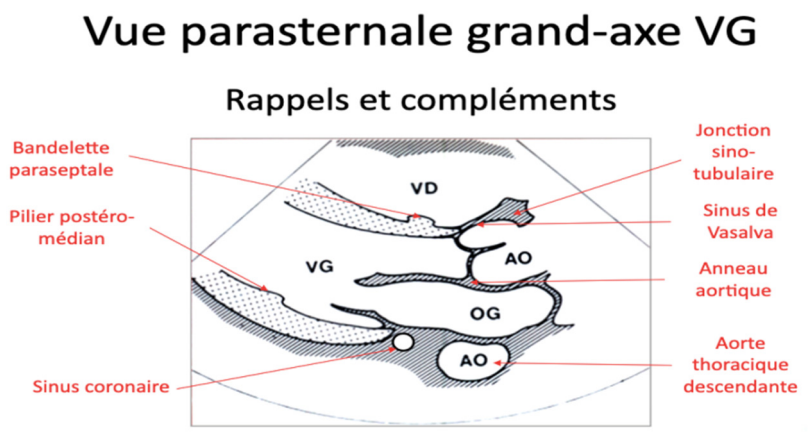
1. Left atrium
    - Anterior and posterior walls

2. Mitral valve
    - Mitral valve ring
    - Anterior mitral valve, posterior mitral valve
    - Posterior abutment or postero-median cords

3. Aorta
    - Antero-right and posterior sigmoid
    - Aortic ring (left ventricular outflow chamber)
    - Initial aorta (sinus, sinotubular junction, tubular)
    - Segment of ascending aorta

4. Right ventricle
    - Ejection tract and anterior wall of the infundibulum

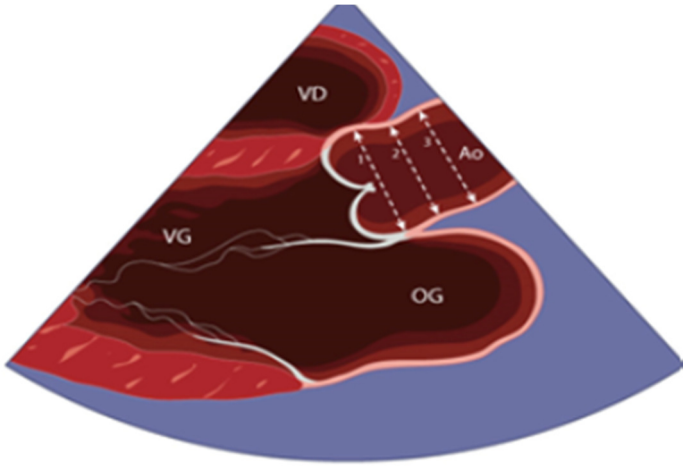
5. Pericardium
    - Anterior and posterior pericardium

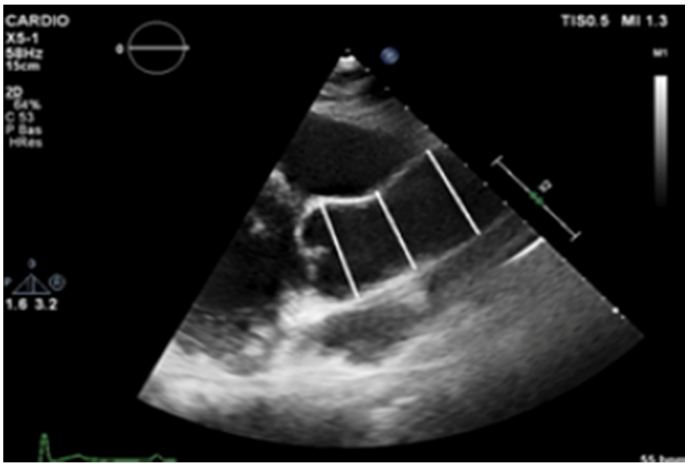


Left parasternal section long axis-2D mode: aorta

Measurement of 3 segments of the ascending aorta in diastole:

- Sinus of Valsalva
- Sinotubular junction
- Tubular aorta

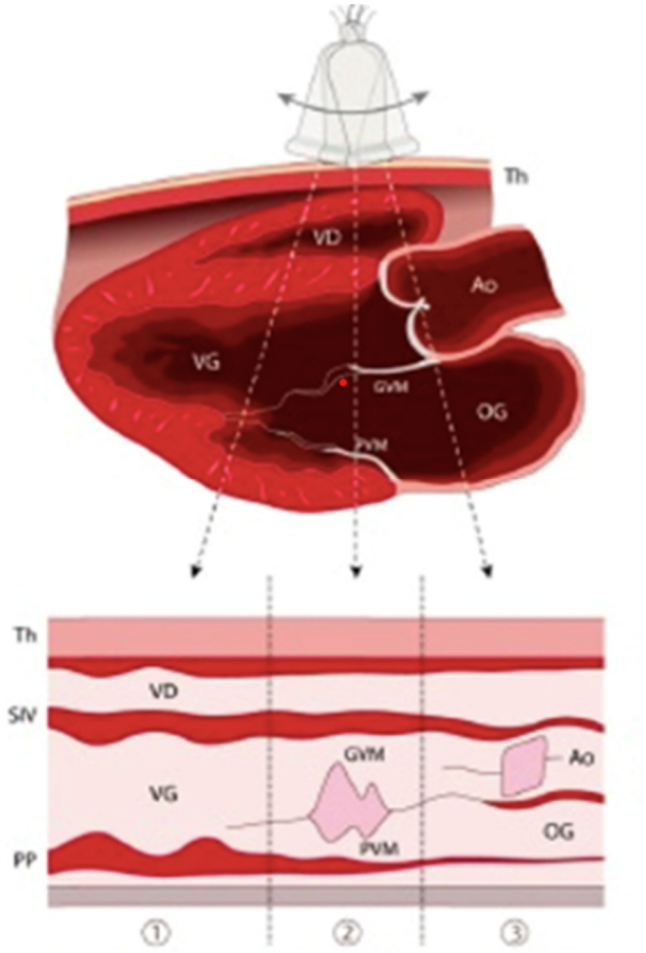




Large-axis left parasternal section-Mode TM

TM mode can be used by aligning perpendicular to:

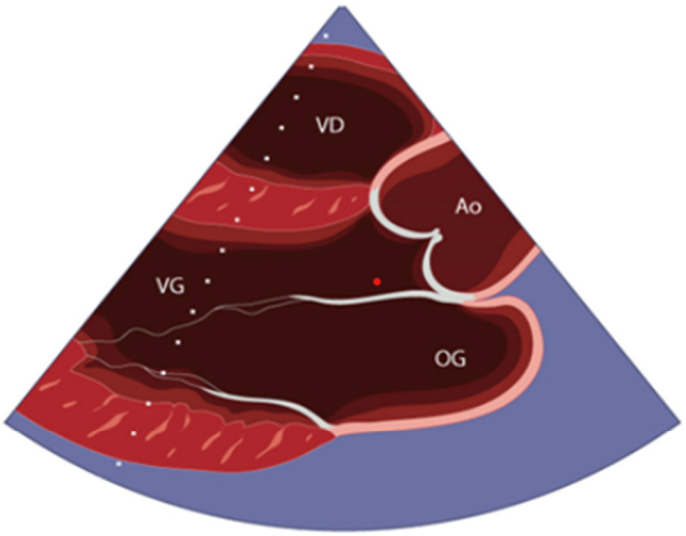
- left ventricle (1)
- mitral valve (2)
- At the aortic root and left atrium (3)



Left parasternal long-axis section - TM mode on the LV

Measurements:

- Anteroseptal wall thickness in diastole and systole
- Infero-lateral wall thickness in diastole and systole
- LV internal diameter in diastole and systole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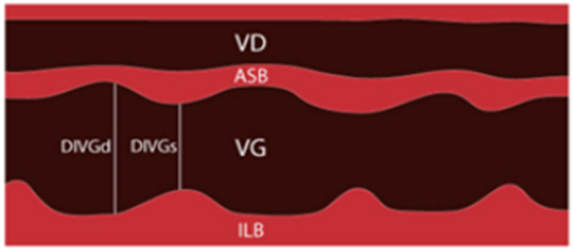
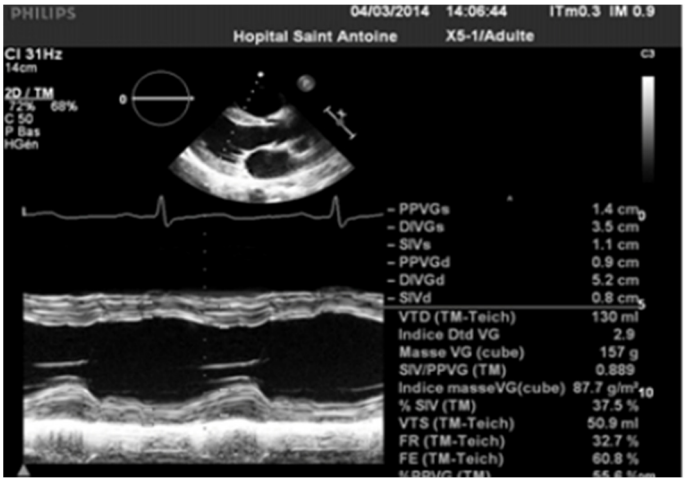
OG


VD
ASB
DIVGd
DIVGs
VG
ILB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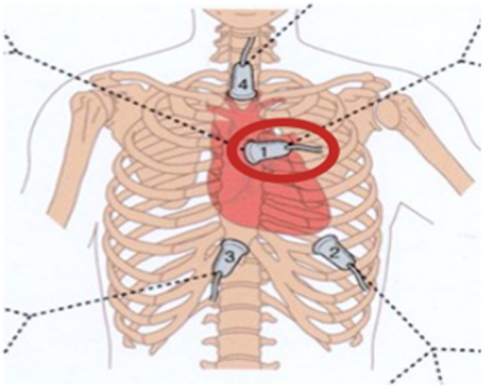
HGén
- PPVGs 1.4 cm
- DIVGs 3.5 cm
- SIVs 1.1 cm
- PPVGd 0.9 cm
- DIVGd 5.2 cm
- SIVd 0.8 cm
VTD (TM-Teich) 130 ml
Indice Dtd VG 2.9
Masse VG (cube) 157 g
SIV/PPVG (TM) 0.889
Indice masseVG(cube) 87.7 g/m²
% SIV (TM) 37.5 %
VTS (TM-Teich) 50.9 ml
FR (TM-Teich) 32.7 %
FE (TM-Teich) 60.8 %

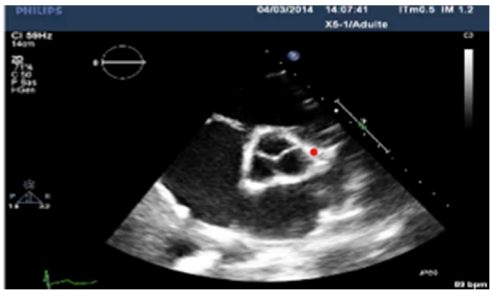
Short-axis left parasternal section



Clockwise rotation movements of 900 in relation to the left parasternal long-axis cut. 4 slices can be obtained with a sweep from the base of the Heart towards the apex:

1. Cut through the bottom of the heart
2. Transverse cut
3. Section through the pillars
4. Section at apex.

1



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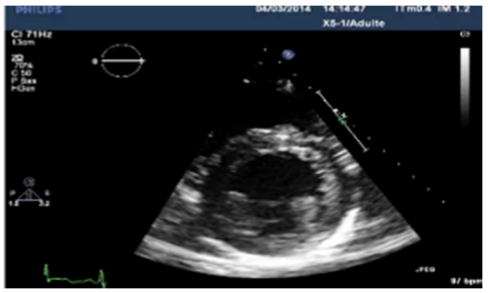


Left parasternal section, short axis passing through the lower part of the heart

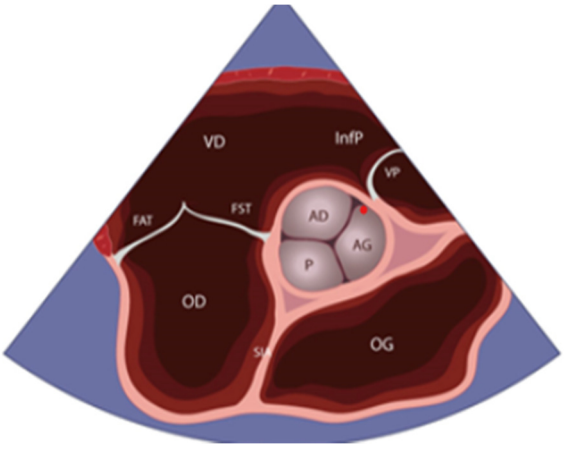
Visualized structure:

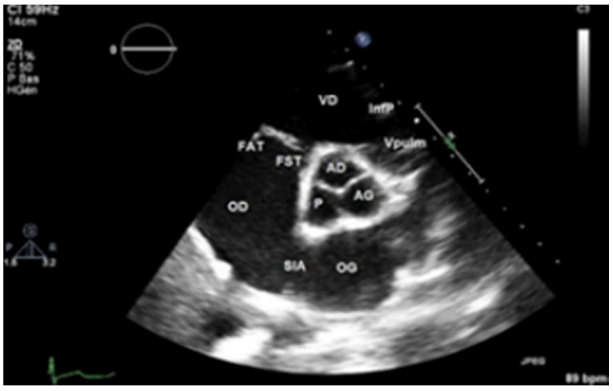
1. Left atrium

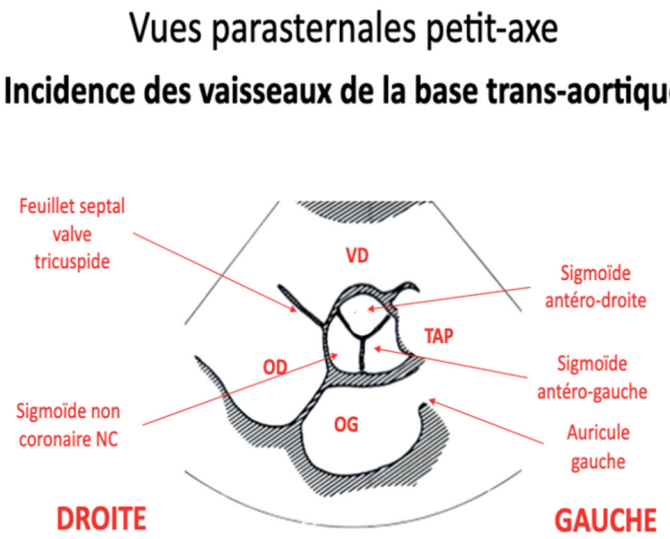
- Lateral, anterior and posterior walls
- Interatrial septum
- Left auricle

2. Aorta

- Sigmoid Antero-Right, Antero-Left and Posterior
- Aortic ring
- Ostia of left and right coronary arteries

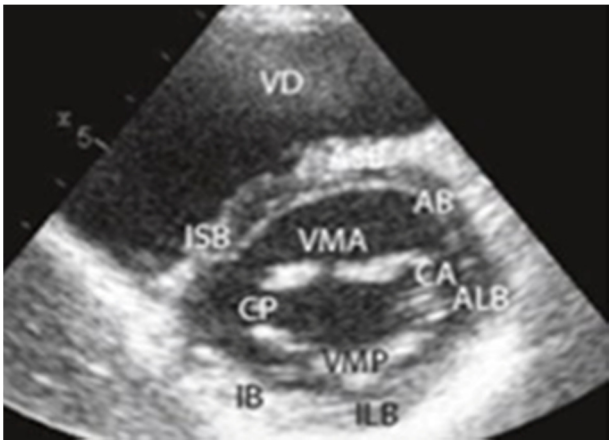






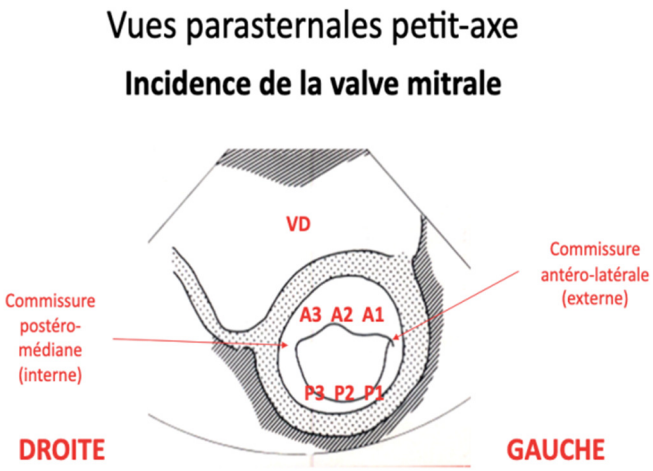
Left parasternal section, short transmitral axis

In relation to the cut at the base of the heart, incline the probe downwards, aiming at the tip of the heart.



Visualized structures:

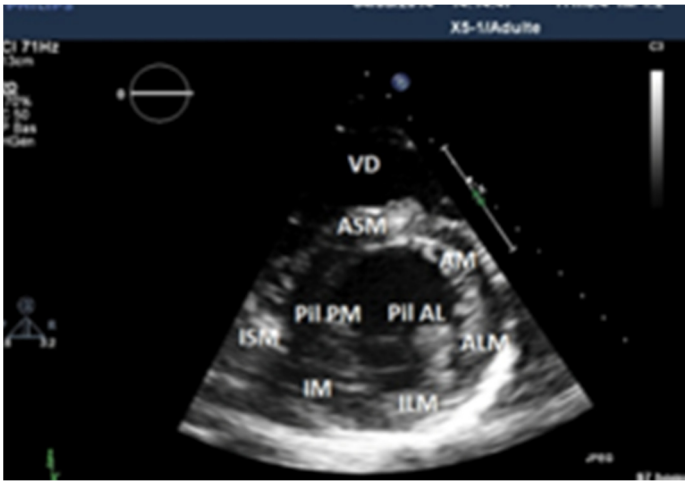
1. Left ventricle
   - The six basal segments: anteroseptal, anterior, anterolateral, infero-lateral, inferior, inferoseptal.
2. Mitral valve
   - Anterior and posterior mitral valve
   - Assessment of functional mitral surface using planimetry
3. Right ventricle
   - Filling chamber and anterior wall of the pulmonary infundibulum.
   -

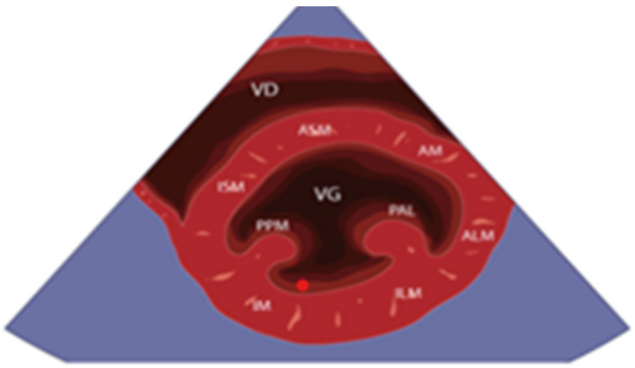


Left parasternal section, short axis through the pillars

Visualized structures:

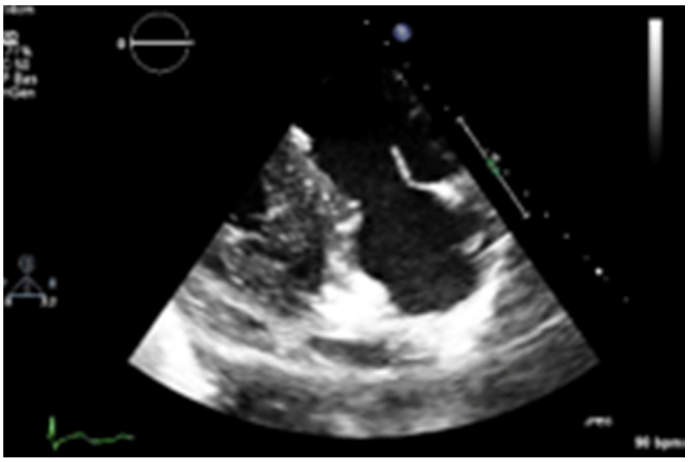
1. Left ventricle
    - The six middle segments: anteroseptal, anterior, anterolateral, infero-lateral, inferior, inferoseptal.
2. Mitral valve
    - Postero-median and antero-lateral pillar
3. Right ventricle
    - Filling chamber and anterior wall
    -

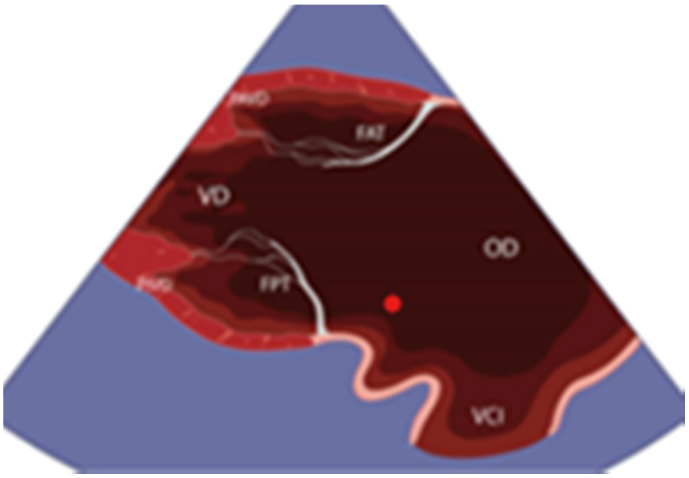




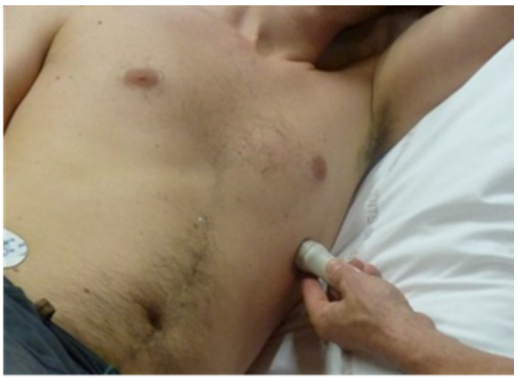
Left parasternal section, long axis of right cavities

- From the left parasternal long-axis section
- Tilting the probe down and to the left
- Clockwise rotation

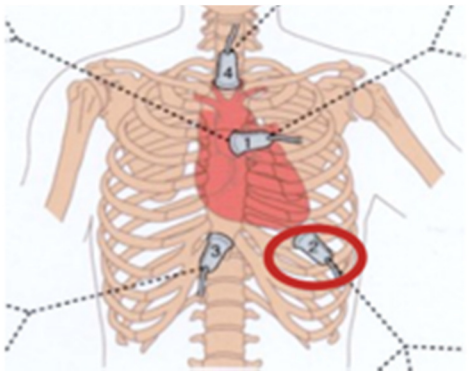


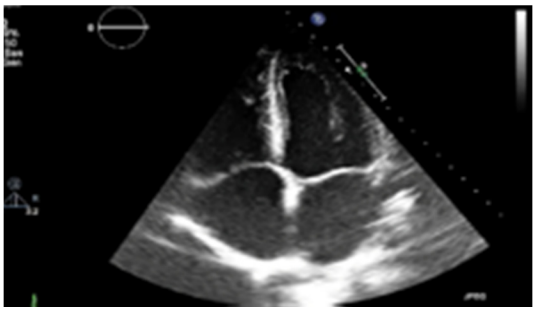


II Apical section of the four cavities



- Left lateral decubitus position
- Place probe in left submammary region, towards armpit
- You can help yourself by placing the probe at the level of the peak shock (marker on palpation)
- Patient in deep inspiration



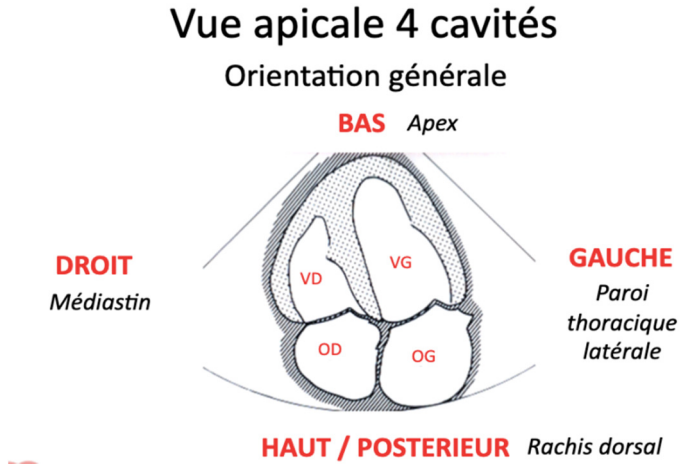


Apical section of the four cavities: 2D mode

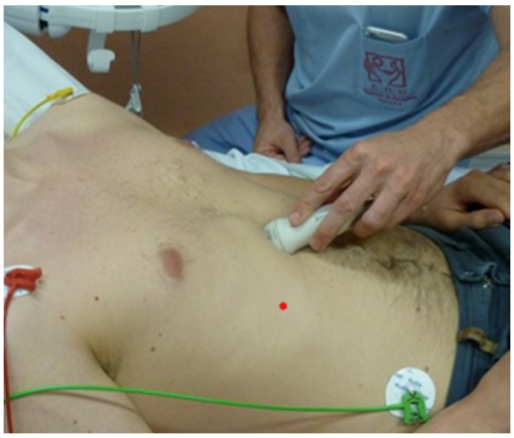
Visualized structures:

1. Left ventricle
    - Basal third and inferoseptal middle third, apical third of septum
    - Apex (17th segment)
    - Basal third and anterolateral middle third, apical third of lateral wall
    - Filling chamber
2. Mitral valve
3. Left atrium
4. Right ventricle
5. Right atrium
6. Tricuspid valve
7. Pericardium

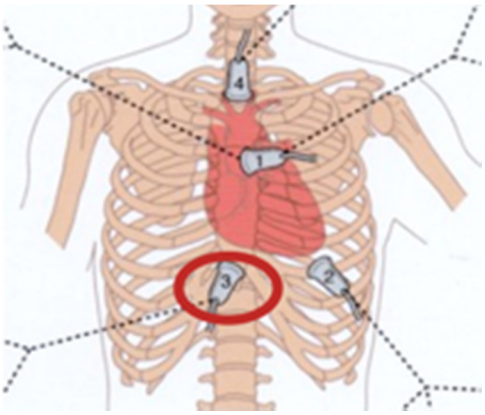
Analysis of LV segmental kinetics, analysis of LV ventricular function using the Simpson biplane method, Simpson biplane LVEF.

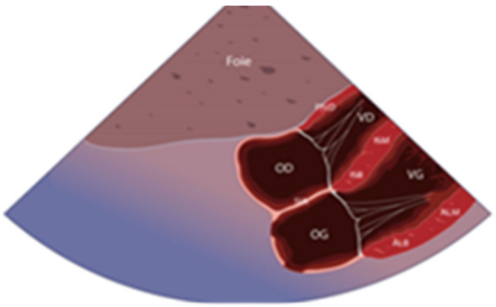


III ..Subcostal section of the four cavities



- Patient in left lateral decubitus or supine position
- Sensor positioned in the epigastric cavity

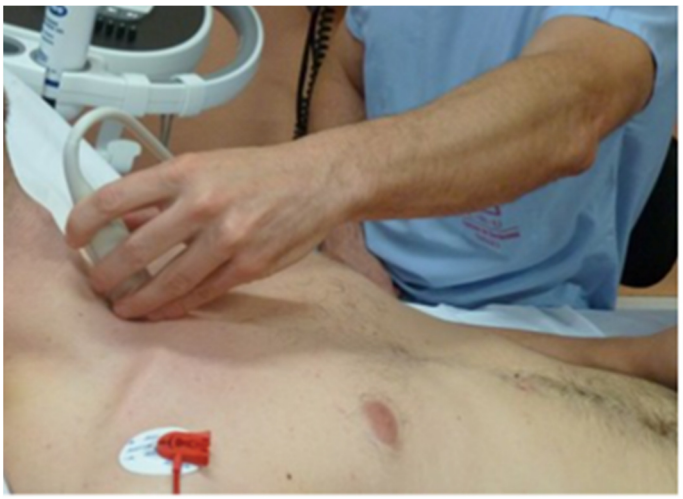




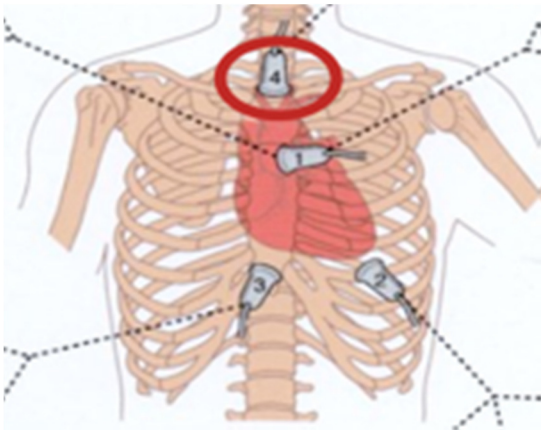
Visualized structures:

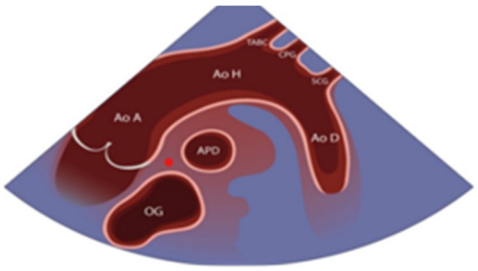
1. Left ventricle
    - Inferior septal wall, apex and anterolateral wall
2. Left atrium
    - Interatrial septum, lateral wall
3. Mitral valve
    - Anterior and posterior mitral valve
4. Aorta
    - Aortic orifice, initial aorta
5. Right ventricle
6. Right atrium
    - Interatrial septum, superior and inferior vena cava
7. Tricuspid valve
    - Septal and posterior valve
8. Pericardium

IV Suprasternal section



- Examination bed flat, patient supine, head tilted backwards and sideways, sometimes hyperextended
- Probe applied in suprasternal fossa
- Probe marker tilted slightly to the right
- Cutting plane almost perpendicular to the precordium,





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3. Sam Kaddoura Fourth Edition Echo made easy.
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2. EVALUATION OF CARDIAC CAVITIES

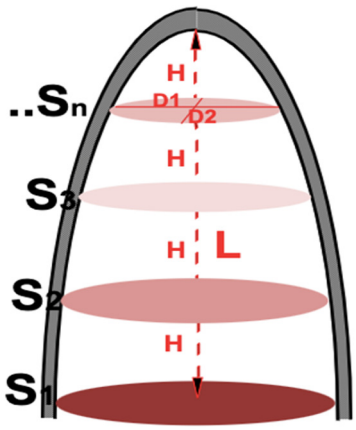
a. Left ventricle:

a.1. Systolic function (Adrian Apostol)

Assessment of LV systolic function is a fundamental step in cardiac echocardiography, and one of its most frequent indications. It can be measured in a number of ways: ventriculography, scintigraphy, MRI (reference method) and 2D, 3D, +/- contrast echocardiography. LVEF must be studied rigorously, as recommended by the Simpson biplane method, and ideally by 3D echocardiography if available.

SIMPSON BIPLAN METHOD

- Recommended reference method
- Any volume can be cut into sections of equal thickness and calculated as the sum of the volumes of these sections.



- **A modification of Simpson's rule makes it easier to use. Instead of measuring the area of each section, we calculate using two diameters, assimilating each disk to an ellipse.**

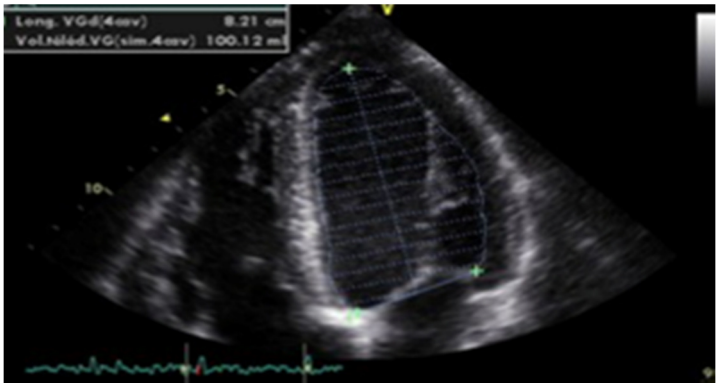
$$V = H \Sigma S = H \pi /4 \ \Sigma D_1 D_2$$

H-disk height

S- surface area of each section

D1D2-diameter of each section

In practice, endocardial contouring in apical 4 and 2-cavity incidence in telediastole and telesystole. The ultrasound machine automatically calibrates the images, mottles the LV into 10 to 20 sections, measures diameters and calculates telediastolic (LVEDV) and telesystolic (LVTSV) volumes.

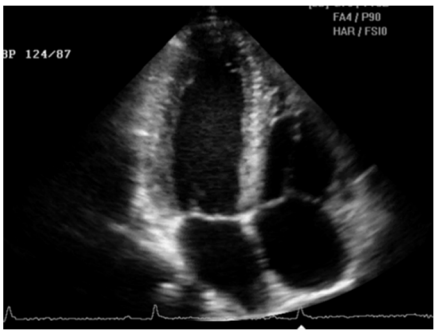


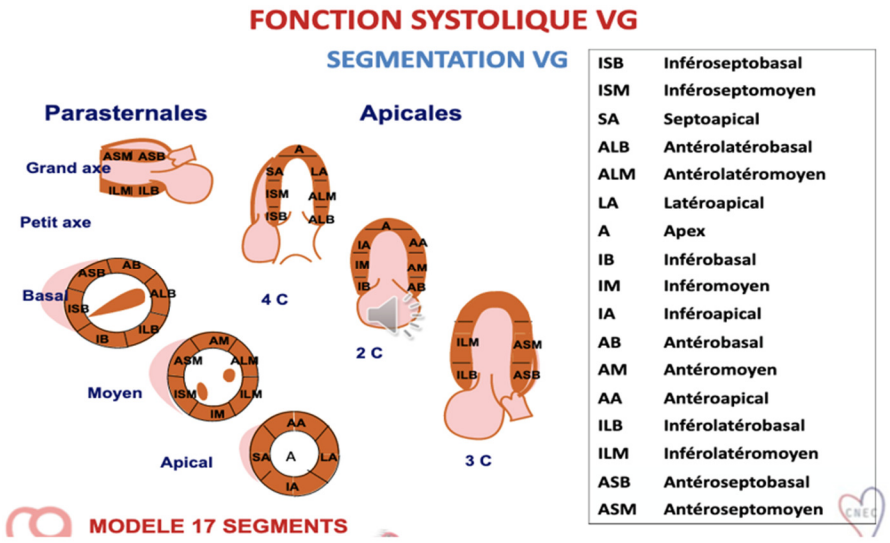
EF=(LVEDV-LVTSV) /LVEDV=V ejection/LVEDV

LVEF (%) NORMALE=52-72

Visual ejection fraction

- Most widely used in clinical practice
- Rapid method (no trace of endocardial contour), most widely used in clinical practice, but subjective, depending on experience.

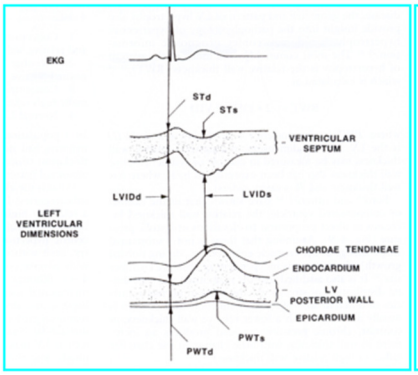


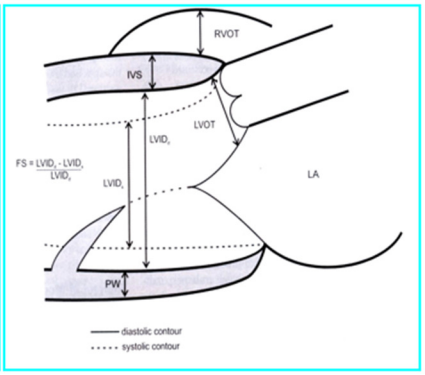


TM mode - Shortening fraction

- Left parasternal long axis incision
- TM line perpendicular to the long axis of the LV, at the tips of the mitral valve leaflets.
- Measurement of LV end-diastolic and telesystolic internal diameters
- Calculation of shortening fraction SF (%)

SF=(LVEDV-LVTSV) / LVEDV X100 (%)





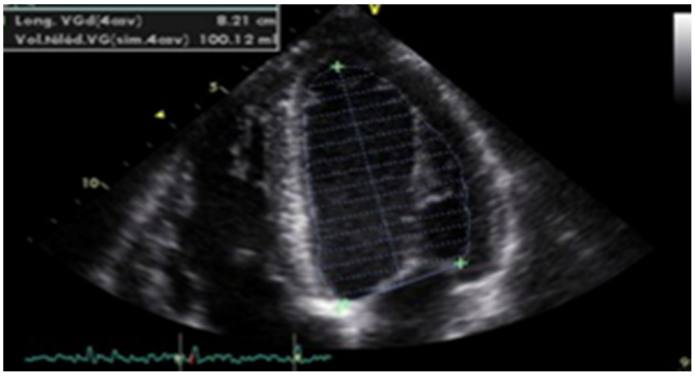
Cardiac output - volume method

- Little used in practice
- Measurement of cardiac output from volumes generally obtained using the Simpson Biplan method.

Qc=V éjection (ml) X FC

=(LVEDV-LVTSV) X FC

- In the absence of mitral or aortic regurgitation



a.2. Diastolic function (Florina Pârv)

Diastole is the period of the cardiac cycle between aortic valve closure and mitral valve closure. Normal diastolic function is divided in several time phases:

- isovolumetric relaxation
- early rapid diastolic filling
- diastasis (slow filling)
- late diastolic filling (atrial systole)

Determinants of diastole are:

- active myocardial relaxation, an active process involving ATP and calcium
- left ventricle compliance, the ratio of change in volume to change in pressure
- left atrium which represent a passive reservoir for blood during LV filling and active pump at end-diastole
- pulmonary and mitral valve characteristics
- heart rate

Diastolic dysfunction is characterised by impaired myocardial relaxation, filling, or distensibility. It is also an important criterion in defining heart failure with preserved ejection fraction.

Doppler echocardiography is the most used method for evaluating the diastolic function.

Assessment of LV diastolic function can be done in different methods.

a) In 2D and M-mode are evaluated*:*

- LV geometry

It is important to determine if LV has normal thickness or has an abnormal geometry (eccentric or concentric hypertrophy), because pathological alterations of LV geometry are associated with diastolic dysfunction. LV hypertrophy in echocardiography is defined when:

- LV mass index (LVMI) >115 g/m2 (M)
- LV mass index> 95g/m2 (W)

LV mass is measured at the end of diastole, in M-mode 2D (in normally shaped LV) and in 3D (in asymmetric hypertrophy of LV). Relative wall thickness (RWT) is a parameter useful for distinguishing between eccentric or concentric hypertrophy and it is calculated with formula: RWT= (2x PWT)/LVEDD [figure nr. a.2.1]

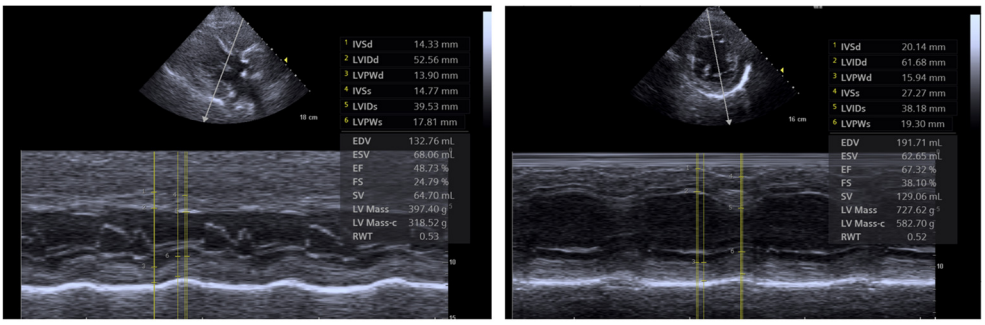


Figure nr. a.2.1 Concentric LV hypertrophy evaluated in M-mode in two different views: left-parasternal long axis (LVmass-i=318 g; RWT=0.53); right-parasternal short axis (LVmass-i=582 g; RWT=0.52);

- LA size

A dilated left atrium is considered if LA diameter in PSAX exceed 4.5 cm and LA volume index (LAVI) >34 ml/m2. [figure nr. a.2.2.]. Dilation of LA occurs as a consequence of increasing filling pressure in LV due to progressively decreasing in LV compliance.

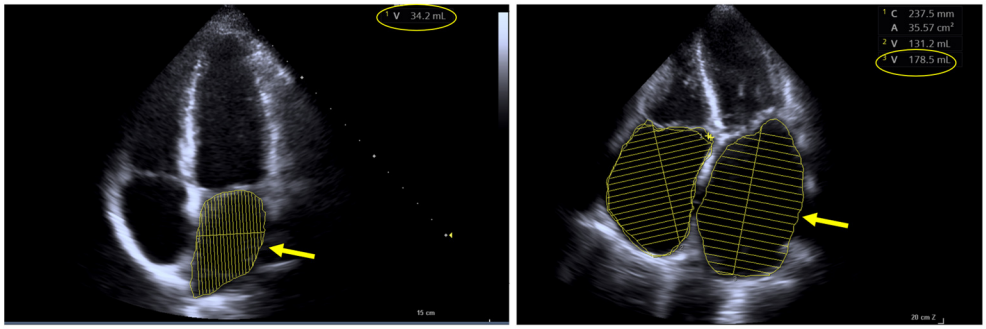


Figure nr. a.2.2. Evaluation of LA volume (arrow) in 2D, 4-chambers view. Left: normal LA (LAV= 34.2 ml; LAVi=28 ml); Right: dilated LA (LAV=178.5 ml; LAVi=105.9 ml)

LAVi is also a criterion in defining heart failure with preserved ejection fraction.

b) In PW Doppler the following are evaluated:

- Mitral inflow [figure nr. a.2.3.]

In apical-4 chamber, with volume sample at the peak of mitral valves in diastole, two waves are measured:

- *E wave*, which represent maximum velocity of early diastolic filling and corresponds to rapid LV filling. Normal values are: 0.72 (0.44-1.00) m/s

E wave decreases in conditions with reduced preload (hypovolemia, use of diuretics, venodilators, during Valsalva maneuver) and increases in conditions with high preload (hypervolemia, high LA pressure, mitral regurgitation).

- *A wave*, which is maximum velocity of late diastolic filling and represent atrial contraction. Normal values are: 0.40 (0.20-0.60) m/s.

A wave is absent in atrial fibrillation, increase in tachycardia, atrio-ventricular block 1st, and follows the inverse variation of E depending on preload.

In healthy people, E/A ratio is >1, and in elderly usually is <1 because of ageing fibrotic process of the myocardium.

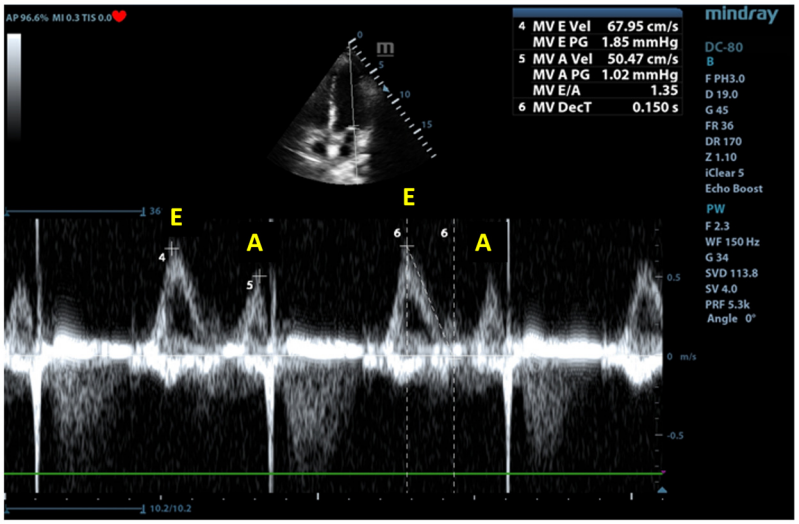


Figure nr. a.2.3. Evaluation of normal transmitral diastolic flow. E= maximum velocity of early diastolic filling (67.95 cm/s); A= maximum velocity of late diastolic filling (50.47 cm/s); E/A=1.35 (PW Doppler, apical 4-chambers view)

E and A waves have a U-shaped evolution in diastolic dysfunction, progressively with its worsening. Therefore, there are three types of mitral flow pattern, which define types of diastolic dysfunction: impaired relaxation (type 1), pseudonormalisation (type 2) and restrictive (type 3). [figure nr. a.2.4.]

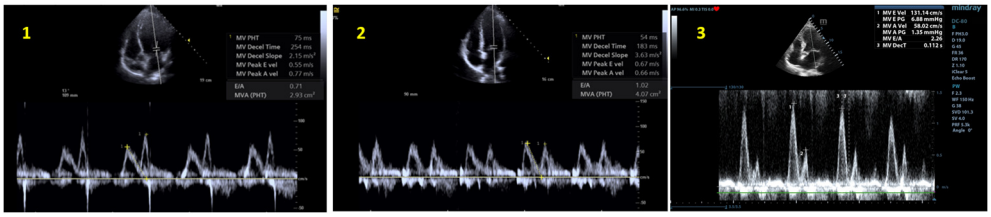


Figure nr. a.2.4. The three patterns of diastolic dysfunction: 1- type 1, E<A, E/A=0.71; 2-type 2, E>A, E/A=1.02; 1- type 3, E>>A, E/A>2; E/A=2.26 (PW Doppler)

During diastole, at transvalvular mitral level there can be recorded other important parameters [figure nr. a.2.5.]:

- early filling deceleration time (DT), integral velocity time of mitral inflow, measured between peak and end of E at baseline
- isovolumic relaxation time (IVRT), time interval between aortic valve closure and mitral valve opening

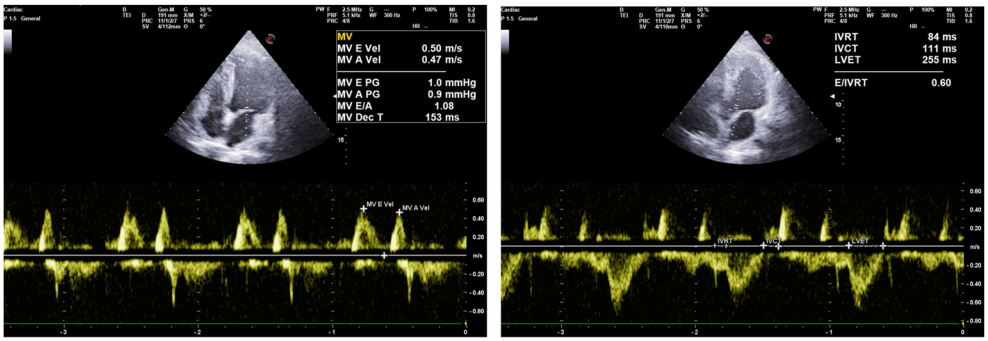


Figure nr. a.2.5. Measuring different heart cycle time intervals: DecT, deceleration time (153 ms); IVCT, isovolumic contraction time (111 ms); ejection time, LVET (255 ms); IVRT, isovolumic relaxation time (84 ms), Tei index=0.76 (PW Doppler, apical 5-chambers view)

- Pulmonary venous flow velocity offers a complex evaluation of mitral inflow by measuring:
  - Peak systolic pulmonary vein flow velocity (S) – systolic phase (LA relaxation and apical movement of mitral ring)
  - Peak diastolic pulmonary vein flow velocity (D) - early diastolic phase (LV relaxation)

    D=0.44±0.11 m/s
  - Peak atrial reversal flow velocity (AR) -for late diastole (LA contraction)

    AR=0.21±0.8 m/s
  - AR duration (ARdur)

In normal adults, with normal LA pressure, the filling of LA occurs in systole and S/D>1. As the pressure in the LA increases, blood flow occurs predominantly in diastole.

c) In CW Doppler the following are evaluated:

- Tricuspid regurgitation (see chap. RV evaluation)
- Pulmonary regurgitation (see chap. RV evaluation)

d) In PW tissue Doppler are evaluated:

- Mitral annular velocities

Myocardial tissue Doppler is useful for evaluating LV diastolic function by measuring parietal (septum and lateral wall) movement velocities in apical 4-chambers with sample size placed in basal segment above mitral annulus. Transmural velocities are similar with transvalvular velocities, but inverted. Diastole has two negative waves [figure nr. a.2.6.]:

- e' is peak early diastolic velocity which correspond to relaxation of myocardium

  Normal values are:

  septal e' (e'_s) =12±2 cm/s; average > 7 (for 40-60 yrs)

  lateral e' (e'_l)= 16±2 cm/s; average >10 (for 40-60 yrs)
- a' is peak late diastolic velocity which correspond to atrial contraction.

Normally e'/a' ratio is >1.

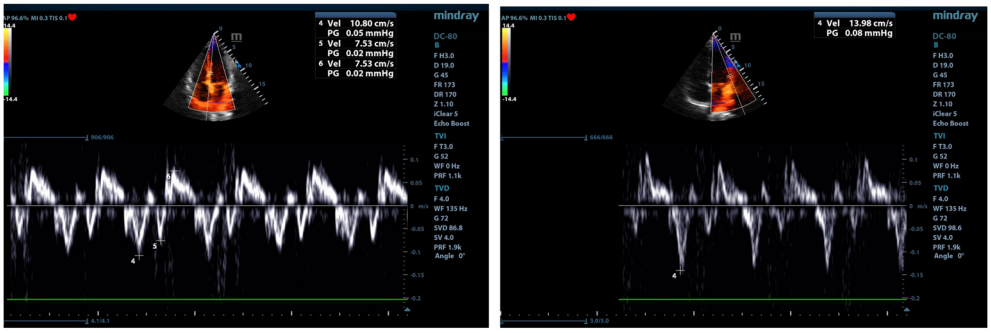


Figure nr. a.2.6. Myocardial tissue Doppler, normal findings. Left: septal measurements (e'_s= 10.8 cm/s, a'_s=7.53 cm/s; e'_s/ a'_s>1). Right: lateral measurements; e'_l=13.98 cm/s.

In diastolic dysfunction e' decreases progressively, case where the „pseudonormalisation" phenomenon does not take place, unlike in the case of the E wave. [figure a.2.7]. Average e' and E/e'ratio are parameters for diastolic dysfunction. Decreased e' is first marker for LV diastolic dysfunction.

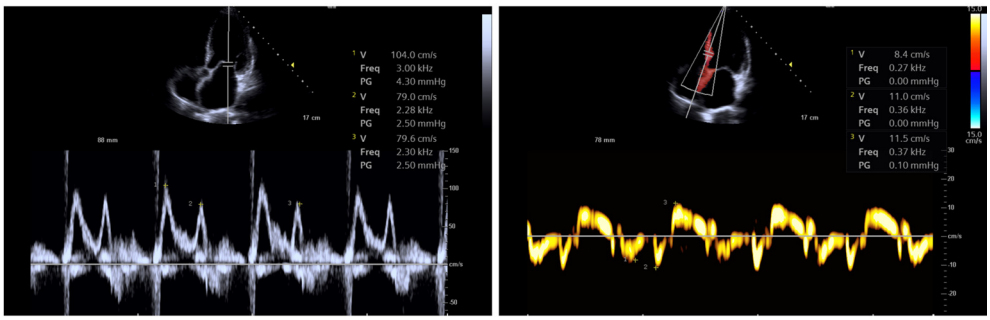


Figure nr. a.2.7. A case with type 2 of diastolic dysfunction. Left: transmitral flow indicates a E/A ratio> 1 (E=104 cm/s; A=79 cm/s) that can be false interpreted as normal (PW Doppler, 4-chambers view). Right: septal myocardium tissue Doppler indicates an e'_s/a'_s ratio < 1 revealing no „pseudonormalisation" pattern. In this case E/e'_s=12.3, a value that suggests increased telediastolic LV pressure.

E/e' ratio correlates with LV pressure and PCWP: a ratio E/e' <8 correspond to a normal telediastolic LV pressure, and a ratio E/e'_s >15 indices a high LV pressure. Between these values, there are necessary additional parameters to estimate LV filling pressure (LAVi, PAP, pulmonary vein flow evaluation)

e) *Colour-flow M-mode Doppler* evaluates blood flow propagation velocity inside the left ventricle in diastole (Vp), allowing its interrogation over the entire distance between apex and mitral annulus plane. It is measured in apical 4-chambers M mode with color Doppler with scan line aligned with the central part of blood column, between mitral valves; it has two components:
    - first wave (early diastolic relaxation)
    - second wave (atrial contraction) up to the middle of LV

In normal adults, Vp has to be greater than 45 cm/s; it decreases progressively with ageing process and in diastolic disfunction [figure nr. a.2.8.]. A low value of Vp is a marker for depressed LV relaxation.

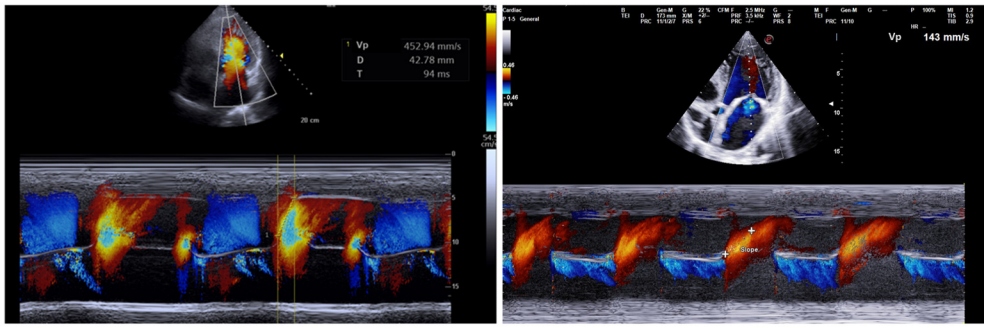


Figure nr. a.2.8. The propagation velocity of the transmitral flow (left, Vp=45.2 cm/s) in a patient with arterial hypertension and in a patient with severe diastolic dysfunction due to a restrictive cardiomyopathy (right, Vp=14.3 cm/s) (color M-mode, apical 4-chambers view)

In case of atrial fibrillation, when A wave is absent, estimation of increased LV telediastolic intraventricular pressure is made using DT (<150 ms), E/e'ratio (>15) and Vp (<45 cm/s).

	Parameter	Normal	Dyastolic dysfunction		
			Type I (abnormal relaxation)	Type II (pseudonormal)	Type III (restrictive)
Mitral flow	E/A	>1	<1	>1	>2
	DT (ms)	150-200	>220	N	<150
	IVRT (ms)	50-100	>100	N	<100
Pulmonary venous flow	S/D	≥1	>1	<1	<<1
	A_{dur} (cm/s)	<35		>35	>35
Color M-mode	Vp (cm/s)	>55	<45	<45	<<45
Mitral tissue doppler	E’		<8	<8	<8
	e’/a’		<1	<1	>1

Table. Diastolic dysfunction parameters

Assessment of diastolic function in a patient must include specifying the type of diastolic dysfunction and estimating the loading pressure of left ventricle. First, we perform interrogation of transmitral inflow and if the profile appears to be normal (E/A>1) and there are indices that there is a cardiac anomaly (enlarged LA or LV, coronary disease, cardiomyopathy), we evaluate additional parameters in tissue Doppler, pulmonary veins flow and M-color mode (table). According to the guidelines of American Society of Echocardiography and European Association of Cardiovascular Imaging, there are different algorithms for estimating LV pressure, depending on systolic function [Figure nr. a.2.9., a.2.11.].

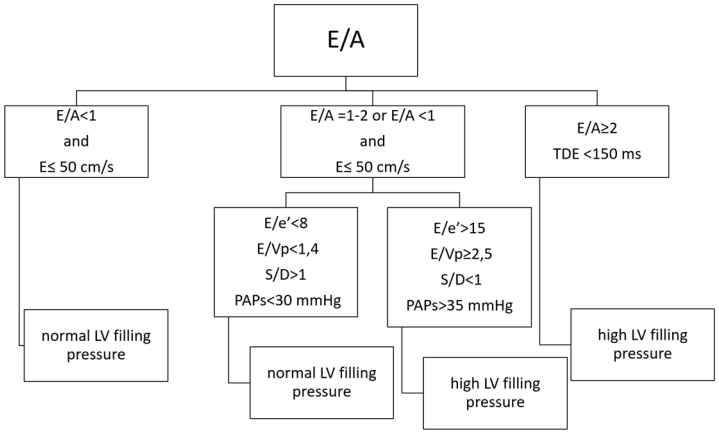


Figure nr. a.2.9. Estimating LV filling pressure in patients with reduced LVEF (adapted)

In patients with impaired systolic function and decreased LVEF, it is sufficient to evaluate transmitral flow profile , which is a good prognosis marker, for a good evolution under medical treatment of these category of patients. [figure . a.2.9., a.2.10.].

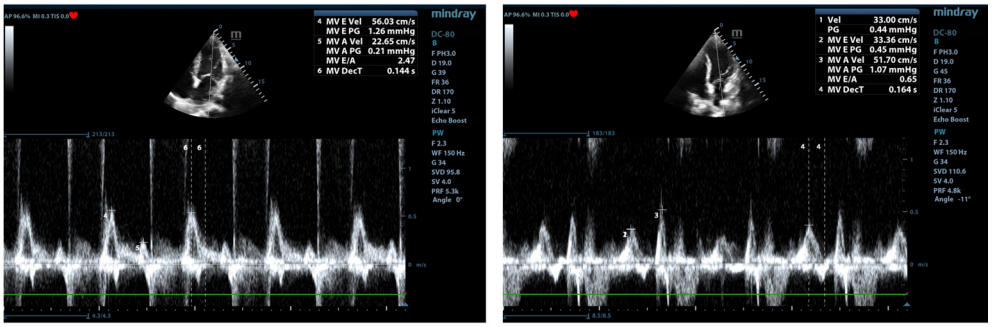


Figure nr. a.2.10. Very good evolution of a severe diastolic dysfunction in a patient with anterior myocardial infarction and depressed LV contractility (LVEF 15%). Left: initial evaluation indicated type 3 of diastolic dysfunction (E=56 cm/s, A=22 cm/s, E/A=2.47, DT=144 ms). Right: after 6 month of follow-up the patient had type 1 of diastolic dysfunction (E=33 cm/s, A=51 cm/s, E/A=0.65, DT=164 ms) (PW Doppler, apical 4-chambers view).

In patients with normal LV systolic function, evaluating the filling pressure is more complex and must include tissue Doppler as a method of evaluating the myocardial relaxation. [figure a.2.11.].

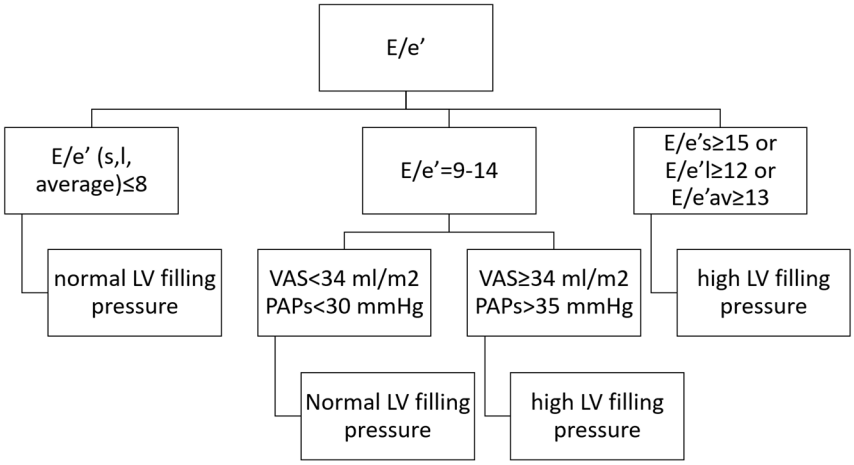


Figure nr.a.2.11. Estimating LV filling pressure in patients with normal LVEF (adapted)

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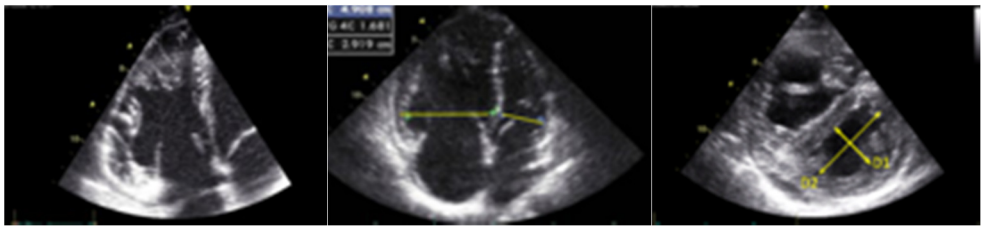
The echocardiographic images are from the own collection of dr. Florina Parv.

b. The right ventricle (Adrian Apostol)

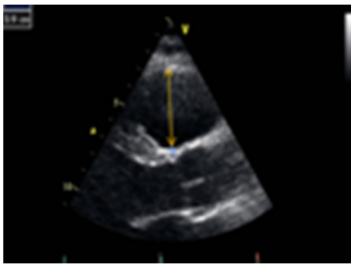
- Prognostic factor in congenital heart disease and in various pulmonary and cardiac pathologies in adults
- Most anterior chamber of the heart in superficial sub-sternal position
- Cavite has 3 components:
    1. The inlet chamber with the tricuspid valve apparatus
    2. The apical trabecular portion
    3. The ejection chamber
- Separated by the moderator strip.

Morphological analysis of the RV

- Assess kinetics, degree of dilatation and hypertrophy of the right ventricle



- Measuring the size of the right ventricle

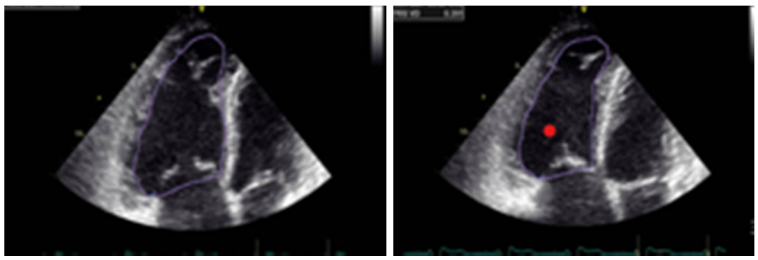


RV function parameters

- Ejection fraction
  1. Visual estimation of RVEF
  2. Fraction of RV surface shortening

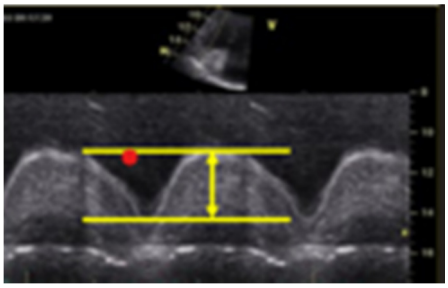
     FRSVD=(STD-STS)/STDX100

     Normal=40%



- Study of the longitudinal displacement of the RV
  1. In TM-ESPAT

     Normal=16 a 25 mm



  2. In DTI-S wave,
- Study of volumes and RVEF

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II. Transthoracic echocardiography in clinical conditions

1. VALVULOPATHIES

a. Mitral valve: mitral stenosis, mitral regurgitation (Adrian Apostol)

1. Mitral ring
2. Anterior and posterior valves
3. Rope, pillars
4. Valve segmentation

Mitral stenosis

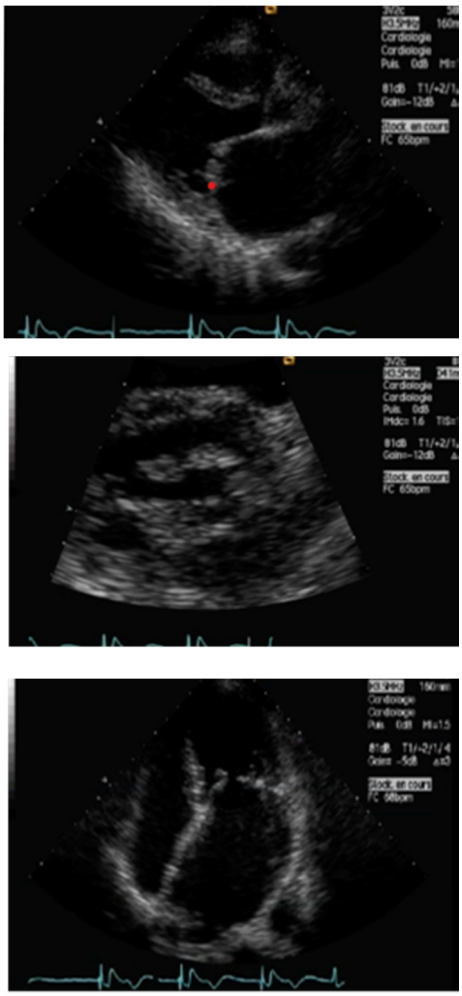
Definition- an obstacle to anterograde blood flow from the left atrium to the left ventricle due to failure of the mitral valve to open in diastole. The mitral surface area is of the order of 5 to 6 cm2. In the event of MR, it diminishes progressively and slowly. Below 1.5 cm2, the patient is generally symptomatic.

Etiology: may be congenital, but the most common etiology is rheumatic fever.

Positive diagnosis:

1. 2D echocardiography

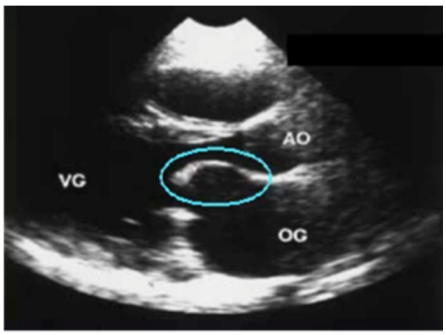
-from the different incidences: parasternal long axis (1), parasternal short axis (2), and apical (3) enables precise recognition of mitral valve damage.



Parasternal long-axis incidence:

Valvular apparatus

- The mitral valve appears thickened.
- The opening of the tips of both valves is reduced.
- Genal or field hockey stick appearance
- The small mitral valve appears retracted, rigid and not very mobile.



Subvalvular apparatus

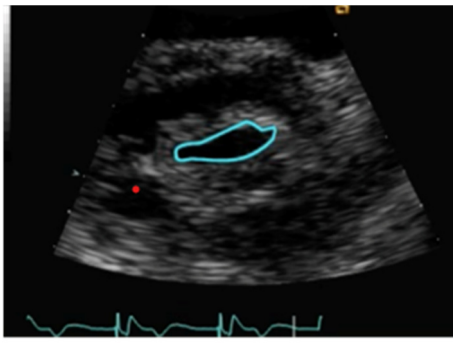
- Is classically reworked. We need to study the length of the cords, which may be normal (greater than 1 cm) or reduced, as well as the thickness of the cords, which may be normal or increased by fibrosis.

Left atrium

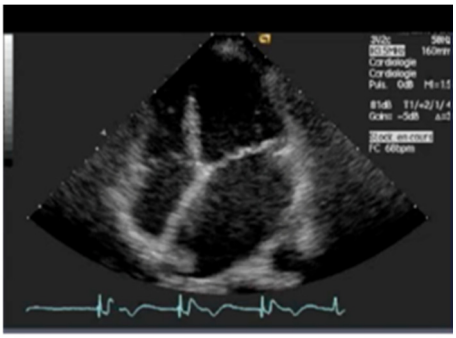
- Dilatation of the left atrium. The surface area of the normal atrium is 14.4 +/-3 cm2.

Short-axis parasternal incidence:

- Enables study of the tip of the mitral funnel.
- Mitral area is calculated by planimetry of the internal contour of the orifice, in diastole, at least three times.

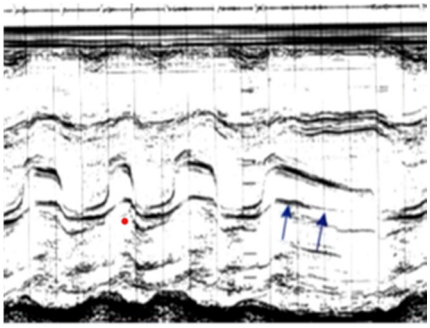


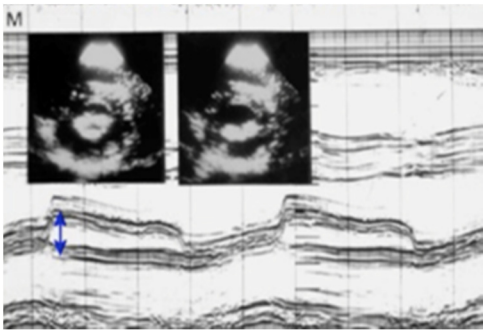
Apical incision - examination of the mitral valve apparatus reveals the lesions described above, assesses the impact on the left atrium and looks for damage to other valves.



Echocardiography TM

- Carried out in parasternal incidence, long-axis section, it reveals caricature-like changes in the morphology and play of the two valves.
- The large mitral valve is thickened in diastole, creating a crenellated appearance with reduced opening amplitude, reduced EF slope, and reduced or abolished A wave.
- The small mitral valve set is also abnormally kinetic, classically paradoxical, i.e. it obstructs the movements of the large mitral valve anteriorly, due to commissural fusion.
- The inter-valvular distance EE' is reduced, to less than 20 mm.

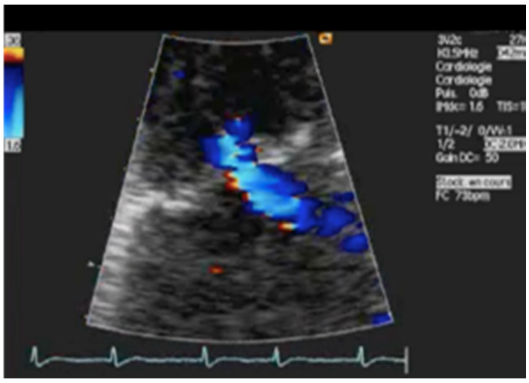




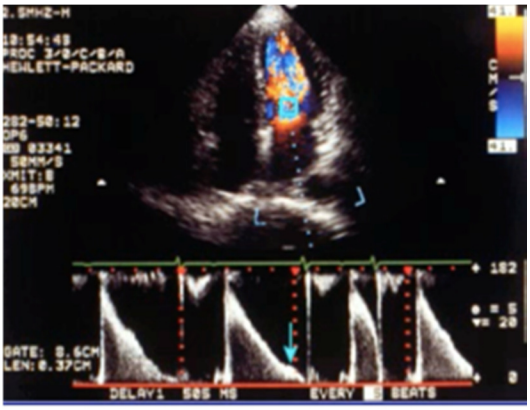
Doppler

Doppler analysis confirms the existence of flow acceleration at the level of the stenotic orifice. The ideal position for studying these flows is the apical 4-cavity position, which offers the best alignment with the blood column.

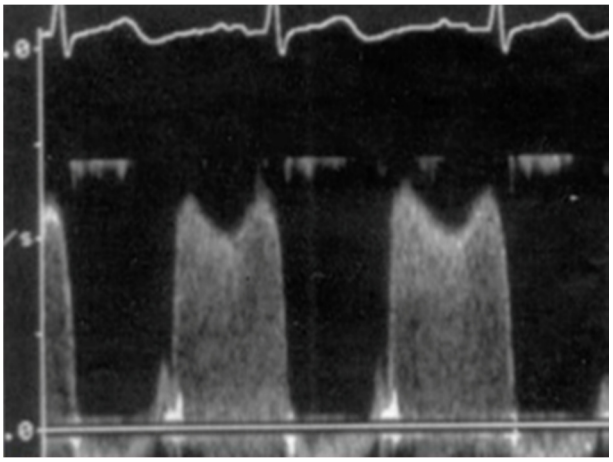
Color Doppler at the tip of the mitral funnel, a Binsen jet appears in diastole, pointing towards the apex.



Pulse Doppler, guided by color Doppler, enables recording of the highest velocities. The sampling volume should be positioned at the apex of the mitral funnel. The flow appears accelerated with a high-pitched sound, and there is a broadening of the spectral envelope.



Continuous Doppler with 2D tracking or blind Doppler allows better curves to be recorded if velocities are very high. Scanning the mitral orifice can also be used to search for associated mitral insufficiency.

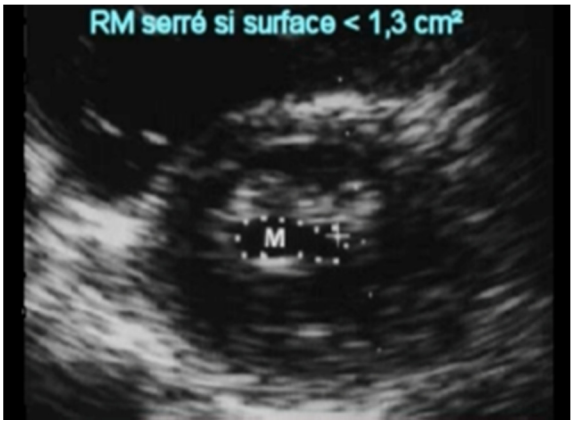


Severe diagnosis

To assess the severity of mitral stenosis, we need to determine the degree of stenosis and the extent of associated lesions.

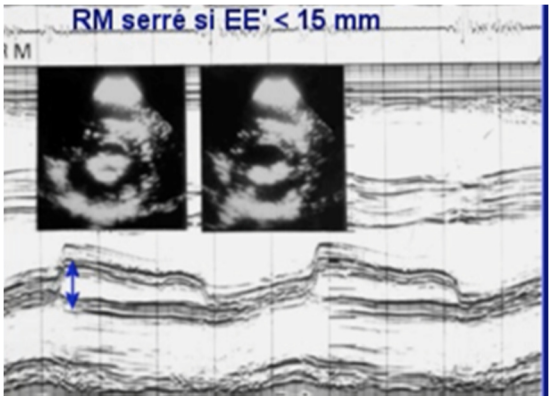
In two-dimensional echocardiography, the severity of mitral narrowing is assessed primarily on the basis of the planimetry of the mitral area measured in the parasternal short axis.

- Moderately constricted mitral stenosis, area between 1.0-1.5 cm2
- Narrowed mitral stenosis, surface area less than 1.0 cm2



On echo TM, the classical signs in favor of a mitral narrowing are an EF slope of less than 15 mm/s and an EE' intervalvular distance of less than 15 mm.

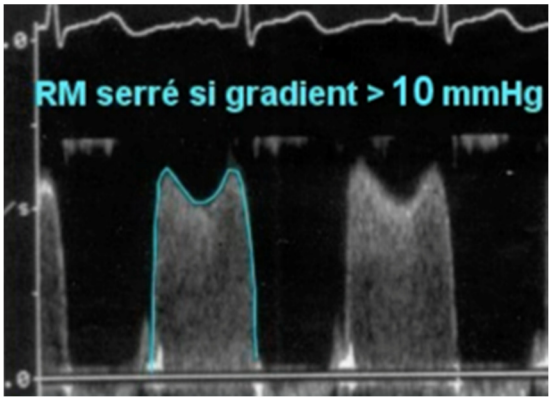
Dilatation of the left atrium is a poor criterion of severity.



Doppler - Doppler in its various modalities enables us to approach the trans-valvular gradient and the valve surface.

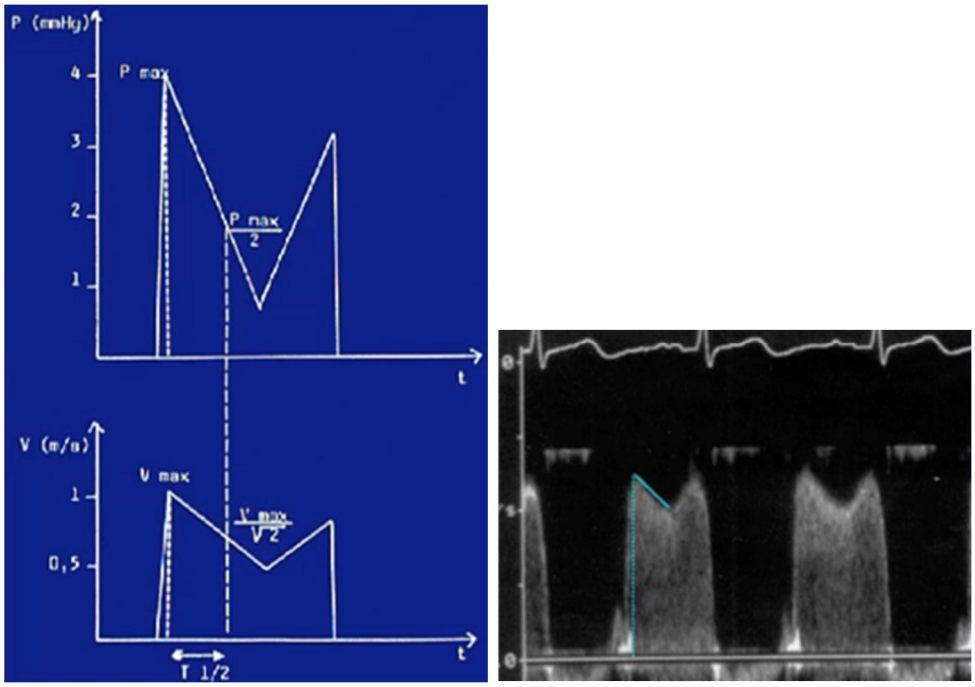
Trans-valvular Doppler gradient - we're interested in the mean gradient, which is easily obtained from the velocity curves by point-by-point quadratic transformation of the velocity curve throughout diastole.

- Medium-tight shrinkage when the mean gradient is between 5 and 10 mmHg
- Tight constriction for an average gradient in excess of 10 mmHg.



Mitral surface by Doppler

Pressure half time (PHT) is the time required for the gradient to decrease by 50% from its initial value. The PHT is obtained automatically on echocardiography after the computer has been given the maximum protodiastolic velocity and the deceleration slope. It is advisable to perform this measurement on at least three cycles. In the case of mitral narrowing, the PHT is greater than 200 ms.



Knowing that the PHT of a 2 cm2 mitral stenosis is 220 ms, Hatle proposes to calculate the mitral surface by the equation **S=220/PHT.**

Measurement of pulmonary pressures is an essential part of the examination.

- Systolic pulmonary arterial pressure is assessed on the basis of tricuspid insufficiency.
- Diastolic pulmonary arterial pressure is assessed on the basis of pulmonary insufficiency.

The assessment of mitral stenosis ends with a search for associated valvular lesions.

Table 1 Classification of Mitral Stenosis Severity

	Progressive		
	(Mild)	(Moderate)	Severe
Valve area (cm^2)	>2.5	2.5-1.6	≤1.5
Pressure half-time (milliseconds)	<100	100-149	≥150
Mean gradient (mmHg)*	<5	5-9	≥10
Systolic pulmonary artery pressure (mmHg)	<30	30-49	≥50

*At a heart rate of 60-80 beats per minute

Mitral insufficiency

Etiologies of primary MI:

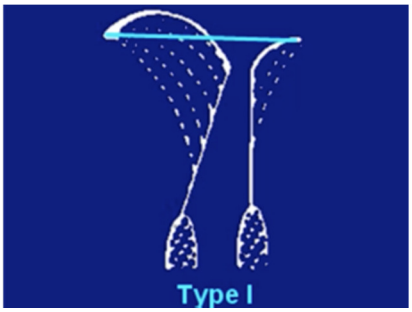
1. Dystrophic and degenerative
2. Rheumatic
3. Endocardial
4. Medication
5. Lupus
6. Ring calcification

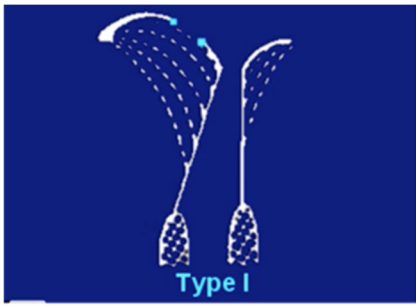
Mechanism of mitral insufficiency

The Carpentier Classification has the merit of classifying MI according to 3 pathophysiological anomalies to which different surgical attitudes correspond.

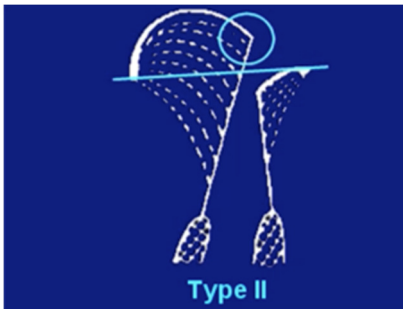
Type I :

- either ring dilatation, which is treated by annuloplasty
- or perforation, treated by direct suture or patching



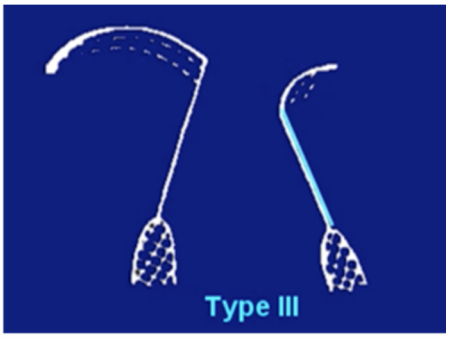


Type II- corresponds to valve prolapse. It may concern either valve.



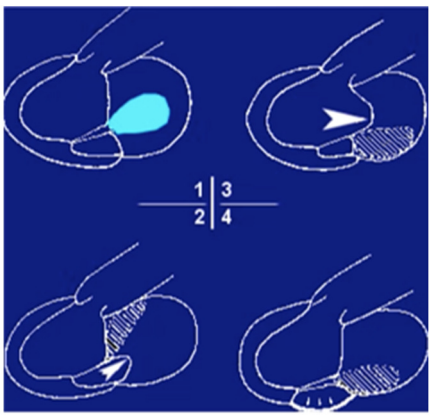
Type III -corresponds to a restriction of valvular movements and recognizes two etiologies for which reconstruction surgery is often difficult:

- Rheumatic MI
- Ischemic MI



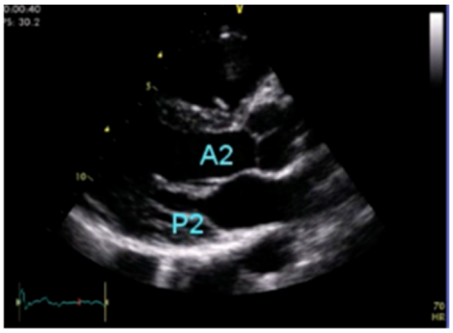
The above mechanisms directly influence the jet type of mitral insufficiency:

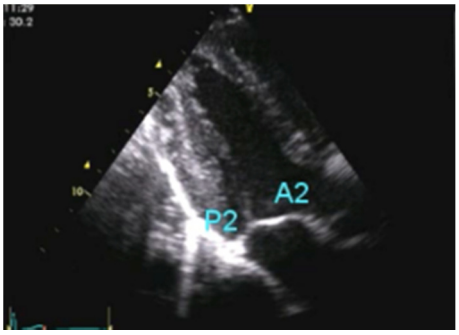
- Ring dilatation produces a central jet (1)
- Valve perforation produces a jet that originates in the middle of the valve, at a distance from the coaptation point
- Small valve prolapse produces a jet directed towards the roof of the left atrium and the inter-atrial septum (2)
- Large valve prolapse produces a jet directed towards the floor and side wall (3)
- A restrictive MI results in a jet directed towards the affected valve (4)



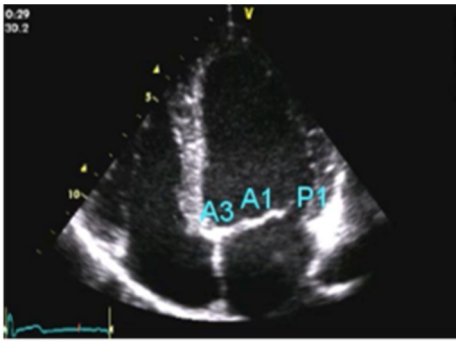
Morphological analysis of the mitral valve is possible with TTE.

- Parasternal wide-axis and apical incidence 3 cavities explore the A2 and P2 laminae

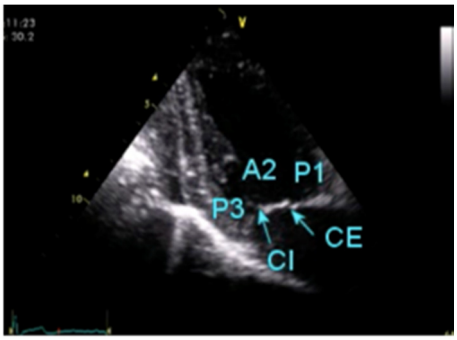




- The apical incidence 4 cavities explore the laminae A1, A2, A3, and the posterior valve at the P1-P2 junction.



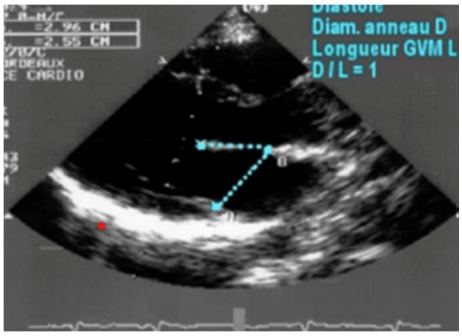
- Apical incidence 2 cavities explore the P1, A2 and P3 laminae



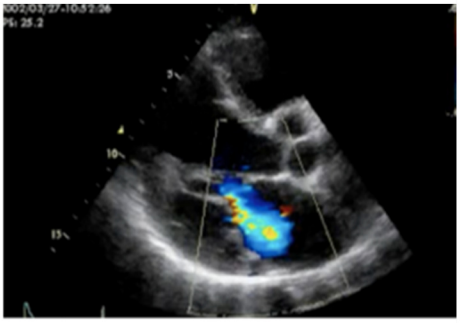
Positive diagnosis

Positive diagnosis is based on 2D, TM and Doppler echocardiography. Diagnosis is primarily based on indirect signs such as volumetric overload of the left cavities, LA dilatation or LV hyperkinesis, and more rarely on direct signs such as diastasis, valvular capping related to cord rupture or valvular prolapse.

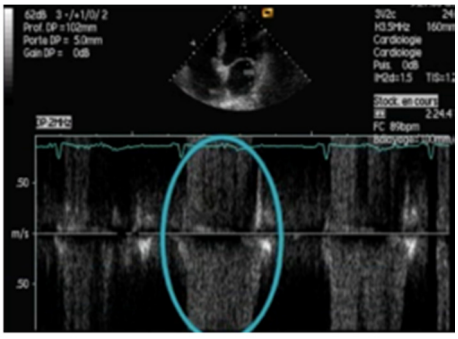
In TM, prolapse gives the trace a typical telesystolic 'louche' appearance. Cord rupture, on the other hand, gives floating, vibratory echoes in the LA. In parasternal long-axis view, it is good clinical practice to measure the diameter of the annulus and the length of the greater valve. In normal subjects, the ratio is of the order of 1.



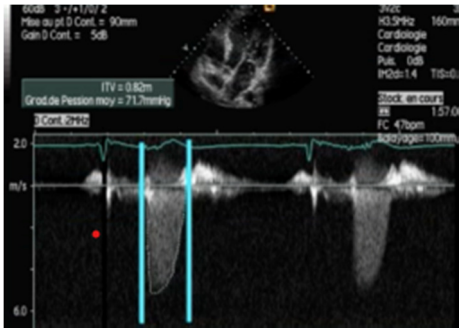
Positive diagnosis is easily made using various Doppler modalities. Color Doppler is highly sensitive for the diagnosis of MI, demonstrating a systolic color mosaic jet at parasternal long-axis or apical 4-cavity angle.



Pulse Doppler is very useful in the absence of color Doppler, and the incidences used are the parasternal long axis and the apical of the 4 cavities. MI is characterized by bidirectional systolic flow on either side of the zero line.



Continuous Doppler completes the data from color Doppler or pulse Doppler, highlighting a high-velocity negative flow of the order of 5 to 6 m/s in apical incidence of the 4 chambers, corresponding to the usual pressure gradient between the LV and the LA in systole, a holosystolic flow.



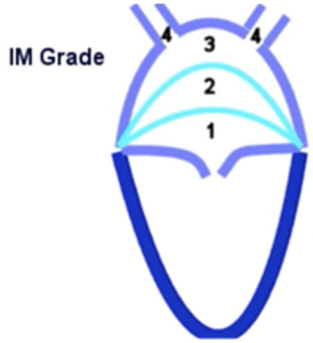
Severe diagnosis

Thanks to its different color, pulse and continuous modes, the Doppler mode provides a multiparametric approach to MI severity:

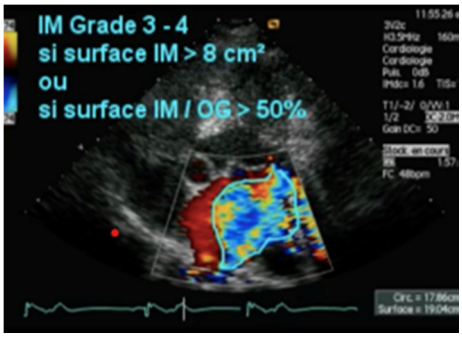
- Jet mapping
- Flow measurement
- Regurgitant orifice size
- Continuous Doppler velocity curve
- Right-sided resound.

Jet mapping studies the spatial extension of the jet using either pulse Doppler or color Doppler:

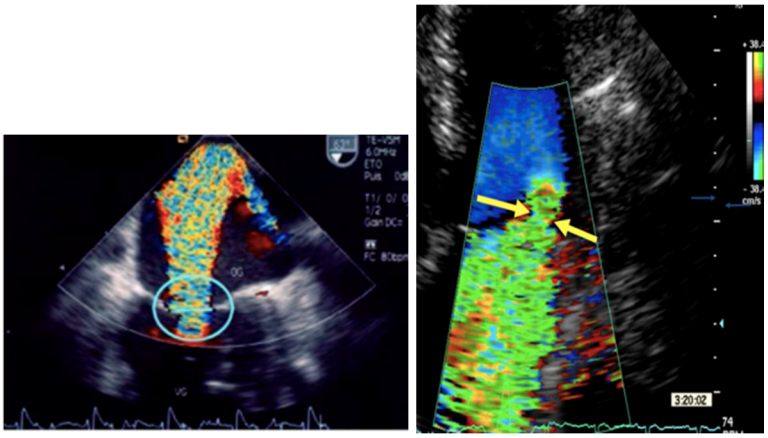
- Grade 1 when the regurgitant flow does not exceed the first third of the LA behind the plane of the mitral annulus
- Grade 2 when the jet does not extend beyond the 2nd third of the LA
- Grade 3 may involve the floor of the LA
- Grade 4 is characterized by reflux into the pulmonary veins



The same type of quantification can be adopted, and it is also possible, more easily than with pulse Doppler, to map the contours of the jet in color mosaic. A regurgitant jet surface area greater than 8 cm2 is a sign of severity (grade III-IV MI), and the ratio of regurgitant flow surface area to OG surface area can be calculated. In this case, we speak of severe MI when the ratio exceeds 50%.



The size of the regurgitant orifice can also be estimated from the diameter of the jet at origin, as assessed by color Doppler. MI is considered severe when this diameter exceeds 7 mm.



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b. Aortic valve: aortic stenosis, aortic regurgitation
(Florina Pârv)

AORTIC STENOSIS (AS)

Valvular aortic stenosis is the most frequent valvulopathy in adult. It can be diagnosed in asymptomatic patients incidentally during an echocardiographic examination or in a routine clinical examination due to its particular systolic murmur; there are also cases when diagnostic is established in symptomatic patients presenting syncope, angina or dyspnea during or immediately after an exertion or in patients with decompensated heart failure.

In AS, echocardiography is essential for evaluating disease severity, progression and for clinical decision making.

Etiology and physiopathology of AS is represented by:

1. *calcified aortic stenosis* (degenerative) – most frequent clinical form seen in adults includes a tri-leaflet valve (stellate-shaped) and bicuspid calcified aortic valve; this is an active and a progressive disease involving inflammation, lipid deposits and calcification.
2. *rheumatic disease* – usually affects both aortic and mitral valves; it is characterized by commissural fusion and thickening of the leaflet edges.
3. *congenital aortic stenosis* - can be unicuspid, bicuspid, tricuspid or quadricuspid; usually is a bicuspid with the two different sized cusps and a median raphe. It is frequently associated with other congenital defects (aortic coarctation, persistent ductus arteriosus, ventricular septal defect).

Normally, aortic valve opening area is 2.5-4 cm^2, transvalvular flow is laminar and peak velocity under 2 m/s. Obstruction due to stenotic valve, leads to damage of left ventricle (hypertrophy), left atrium (dilation of LA, mitral regurgitation, atrial fibrillation), pulmonary vasculature (systolic pulmonary hypertension, tricuspid regurgitation) and finally of right ventricle (RV

dysfunction). These consequences are present to a lesser or greater extent depending on the severity of aortic stenosis; echocardiography must evaluate and quantify all this issues.

First of all, the presence of AS is suspected based on 2D echo findings. 2D echocardiography must identify the following aspects:

1. number of cusps: in parasternal short axis in systole (round orifice in bicuspid valve and triangular in tricuspid valve) [Figure nr. II.1.b.1.]

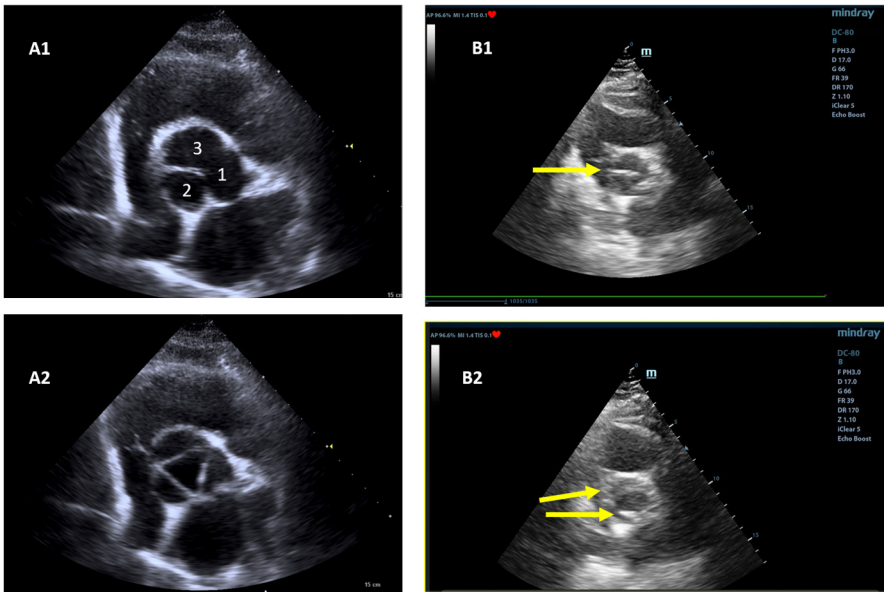


Figure nr. II.1.b.1. Normal aortic valve with 3 cusps (A1: closed normal aortic valve; 1-left coronary cusp, 2-right coronary cusp, 3-non-coronary cusp; A2: opened normal aortic valve, with a triangular appearance of the orifice) and congenital bicuspid aortic valve (B1: closed bicuspid aortic valve, with horizontal raphe; B2: opened bicuspid aortic valve with oval appearance of the orifice); (parasternal short axis view at the level of the great vessels).

2. presence or not of the fusion of commissures – more specific for rheumatic etiology
3. restricted/abnormal leaflet mobility
4. systolic doming - sign of bicuspid valve

5. reduced mobility - should be interpreted with caution in global systolic LF dysfunction

- systolic opening of aortic valves – above 15 mm excludes a severe AS [figure nr. II.1.b.2.]

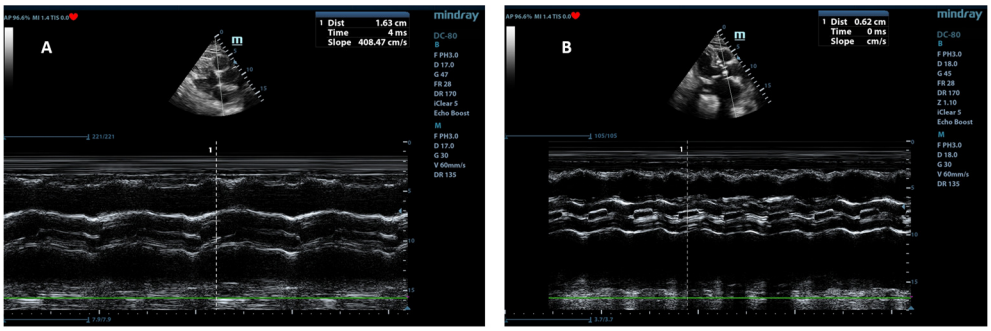


Figure nr. II.1.b.2. Normal (1.63 cm) (A) and reduced (0.62 cm) (B) distance between aortic cusps of the aortic valves during systole (parasternal long axis view, M-mode)

- extent and pattern of calcification [figure II.1.b.3.]

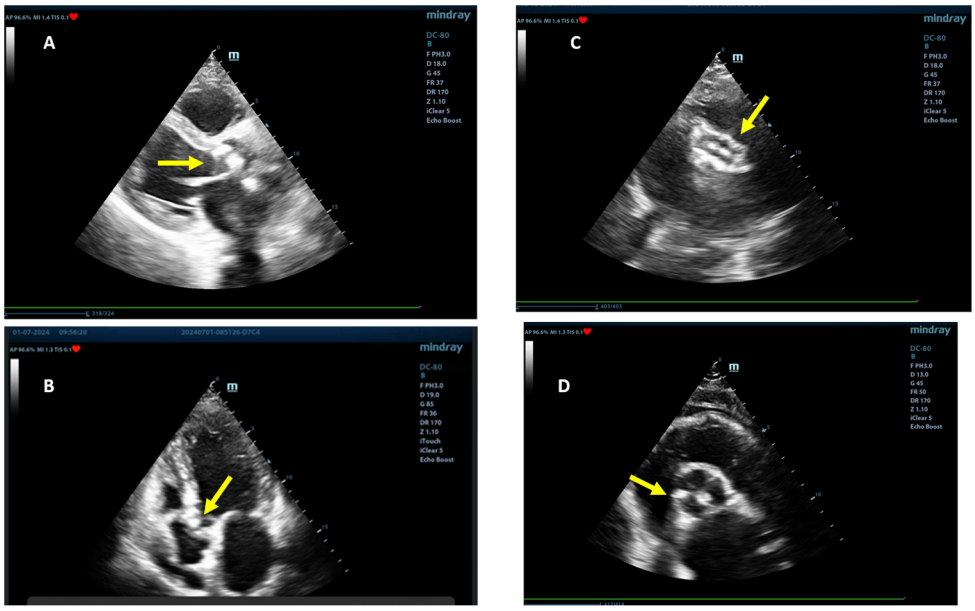


Figure nr. II.1.b.3. Extensive calcifications of the aortic valve (A-parasternal ax lung; B-5-chambers view; C-parasternal short axis-bicuspid valve; D-parasternal short axis: tricuspid valve)

- anatomic valve area (AVA) – planimetric measurement in parasternal short axis, magnified with zoom; sometime is not feasible due to massive calcifications [figure II.1.b.4.]
- LVEF
- aortic root
- aortic dilatation – sign of severe AS
- LV hypertrophy

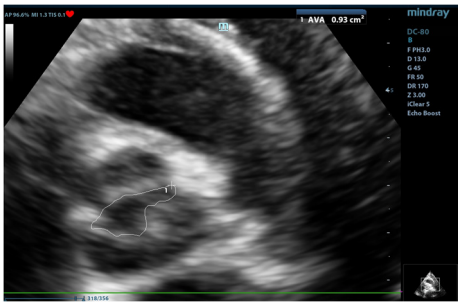


Figure nr. II.1.b.4 Aortic valve area measured planimetric (0.93 cm^2) in parasternal short axis view

Thin valves with normal movement of the cusps, a normal aortic root, normal LV wall thickness and systolic LV function indicate absence of significant AS. Thick valves, reduced leaflet motion, LV hypertrophy in appropriate clinical context are indicate severe AS.

Assessment of AS severity includes several measurements using color, PW and CW Doppler. Before any interpretation of a hemodynamic measurement, blood pressure and heart rate must be recorded.

1. *Peak velocity of the anterograde flow (V_{mxA})*

Is directly correlated with the severity of stenosis. The evaluation is made in CW Doppler, in apical 5-chamber window, with the probe aligned parallel with the blood jet direction. In sinus rhythm three measurements must be made and in atrial fibrillation, five measurements. Using simplified

Bernoulli formula ($\Delta P=4v_{mx}^2$), the maximum and mean transvalvular gradient can be obtained. V_{mx} is maximum velocity of aortic flow distally distal to the obstacle. If the peak velocity is obtained in early systole, then the AS is not severe, but if it is obtained in mezosystole, then the AS is severe. V_{mx} above 2,5 m/s indicates aortic stenosis

2. *Mean transvalvular aortic pressure gradient (MPG)* is obtained automatically when v_{mx} is evaluated. A value above 40 mmHg indicates a severe AS. [figure II.1.b.5.]

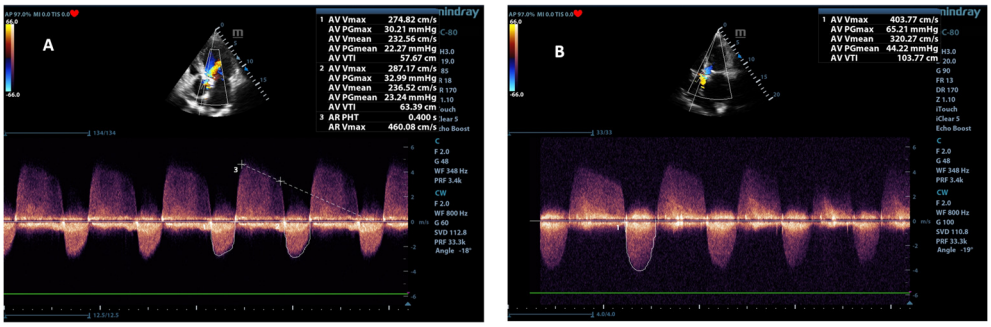


Figure nr. II.1.b.5. Transvalvular aortic velocity and mean pressure gradient in two cases: mild aortic stenosis [V_{mx}=2.7 m/s, MPG=22.27 mmHg (A)] and severe aortic stenosis [V_{mx}=4.01 m/s, MPG=44.22 mmHg (B)] (5-chambers view, continuous Doppler in patients with concomitant aortic regurgitation)

3. *Aortic valve area (AVA) by continuity equation,* represents effective functional valve area, which can be smaller than anatomic area. This takes into account velocities in LVOT and at the level of the stenotic valve. The area is calculated based on principle of conservation of the mass, assuming the outflow tract is circular. Indexing of AVA to body surface area is important in young people and adults with small height. An indexed area ≤ 0.6 cm^2/m^2 is noticed in severe stenosis. [figure II.1.b.6.]

$$AVA=SV/VTI_{AV} = \pi \times (D^2/4) \times (VTI_{LVOT}/VTI_{AV})$$

SV=stroke volume

D=diameter of LVOT; is measured in parasternal long axis, parallel with the aortic plane in mezosistole, between septal endocardium and basis of anterior mitral valve

VTI_{AV}=velocity time integral in aortic valve; is measured automatically at the same time with V_{mxA} evaluation.

VTI_{LVOT}= velocity time integral of LVOT is assessed in apical 5-chambers with pulsed Doppler, parallel with the jet flow, proximal and central to stenotic valve area

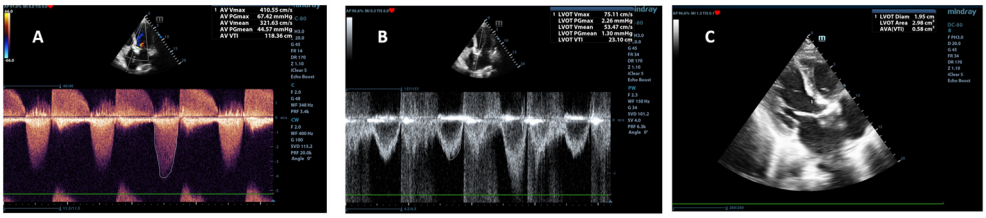


Figure nr. II.1.b.6. Calculation of aortic valve area by continuity equation (A-measuring of VTI_{AV} =118 cm with CW Doppler in 5-chambers view; B-measuring of VTI_{LVOT}=23 cm with PW Doppler; C=measuring of LVOT diameter in parasternal long axis; AVA=0.58 cm^2)

Aortic valve sclerosis is defined when aortic valves are calcified without hemodynamic significance. Severe AS is defined when peak aortic velocity is greater than 4 m/s and AVA below 1 cm^2.

Table nr. 1. Aortic stenosis severity criteria

	Aortic sclerosis	**mild AS**	**Moderate AS**	**Severe AS**
V_{mxA} (m/s)	<2.5	2.5-2.9	3-3.9	≥4
MPG (mmHg)	normal	<20	20-39	≥40
AVA (cm^2)	normal	>1.5	1-1.5	<1

There are several situations when there is a discordance between velocity and gradient area:

- a high transvalvular gradient and AVA>1 cm^2 can be found in concomitant aortic regurgitation or shunt lesions; reversible causes of tachycardia and high cardiac output (thyrotoxicosis, anemia, fever) must be recognized.
- if LVOT size is underestimated, concomitant aortic and or mitral regurgitation are present or the patient has a small stature, low values of peak velocity (<4m/s), valve area (<1 cm^2) and mean gradient can be obtained (<40 mmHg).

Severe aortic stenosis associated with LV systolic dysfunction („low flow-low gradient") is characterized by effective AVA<1 cm^2, LVEF<40% and MPG<40 mmHg. [figure II.1.b.7.] In this situation pseudo-severe AS must be differentiated by dobutamine-stress echocardiography: if a contractile reserve is present, MPG has minor modifications and the AVA increases, then AS is not severe.

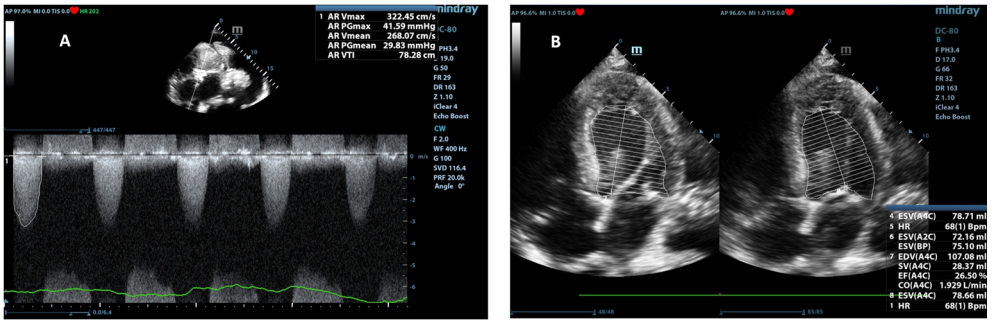


Figure nr. II.1.b.7. Low-flow low-gradient pattern of severe aortic stenosis (MPG=29 mmHg; AVA=0.47 cm^2, SV=28 ml; LVEF=26%; A: 5-chambers view CW Doppler; B:4-chambers view 2D)

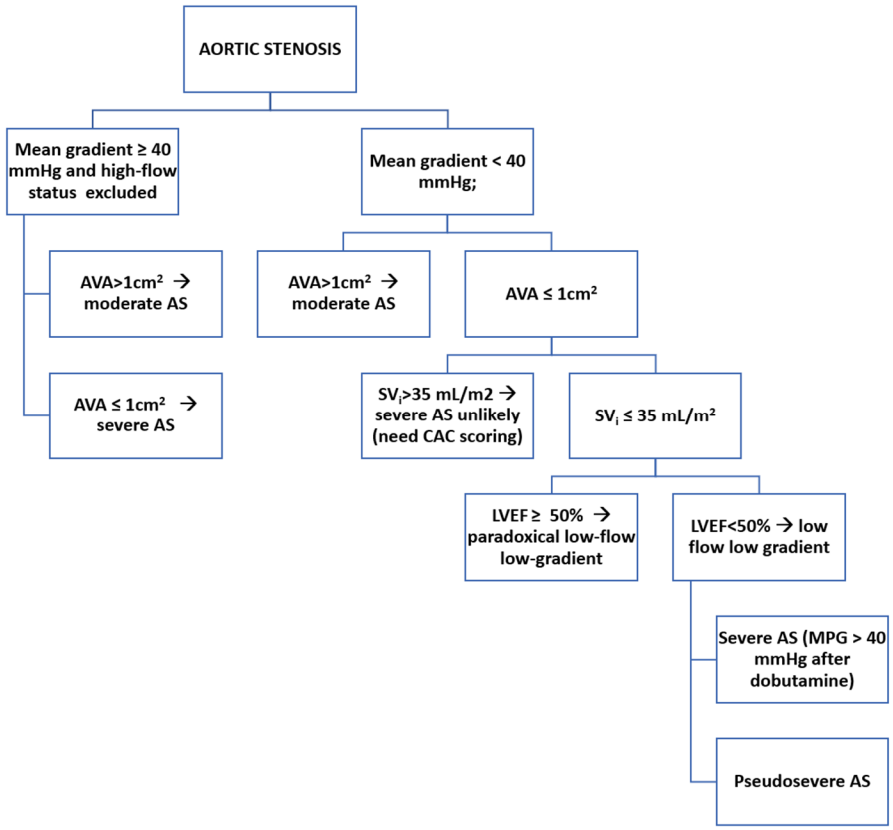
Severe aortic stenosis with preserved EF („paradoxical low flow-low gradient") is characterized by effective AVA <1 cm^2 (<0.6 cm^2/m^2), LVEF>50%, MPG <40 mmHg and an indexed stroke volume<35 ml/m^2. This

situation is seen in patients with normal/small LV and LV hypertrophy, poor LV contractility, increased global afterload and even they are categorized as having medium aortic stenosis, they have a negative prognostic.

TTE echocardiography in AS must evaluate also the consequences of AS:

- alteration of LV geometry (LV hypertrophy) and function (LVEF)
- dilation of LA (LA area, LAVi)
- pulmonary hypertension
- dilation of ascending aorta

Associated valvular lesions must be assessed also: aortic regurgitation (can overestimate the transvalvular gradient), mitral regurgitation (can underestimate the gradient), aortic coarctation (in bicuspid aortic valves).



Practical algorithm for assessment of AS severity (adapted)

A few clinical, morphologic and functional criteria (ultrasonographic), electrocardiographic and biological criteria can differentiate a severe aortic stenosis from a mild one.

Table nr. 2 Integrated clinical-echo-biological approach in AS with preserved LVEF (adapted)

	Non severe AS	Severe AS
Age	younger	older
Symptoms	-	+
LVH	-	+
MPG (mmHg)	<25 mmHg	30-40 mmHg
Longitudinal LV function	normal	reduced
AVA (cm^2)	≥0.9	≤0.8
CCT	low	high
NTproBNP	normal	high
ECG with repolarization abnormalities	-	+

Echocardiography is necessary for also monitoring the progression of AS, decision and timing for intervention; the recommended follow up is 12 months in the case of moderate AS and 6 months in severe AS. During natural history of the disease, mean AVA decrease 0.1 cm2/year, MPG increase 7 mmHg/year. Peak aortic jet velocity is directly related to AS severity and mortality. If Vmx>0.3 m/s/year, a category of very-high risk patients, a rapid progression is considered. Important calcifications are associated also with structural and hemodynamic alteration and a more rapid progression of the disease.

Indication of surgery Class I are severe AS in symptomatic patients, severe AS and LVEF<50%.

CLINICAL CASE

Patient CP, aged 47 years, with mild, treated hypertension, presented to a consultation for dyspnea and atypical precordial pain on exertion, symptomatology occurring in the last 6 months.

The physical examination revealed BMI = 28 kg/m2, rhythmic, clear, normal heart sounds, HR = 78/min, BP = 120/90 mmHg in both arms, very intense and rough systolic murmur in the aortic focus, with irradiation at the base of the neck, intense systolic murmur in the mitral focus, with irradiation in the axilla; normal pulmonary auscultatory, normal abdomen, normal pulse in the periphery.

Electrocardiogram showed sinus rhythm, 72/min, intermediate QRS axis, left ventricular hypertrophy.

Transthoracic echocardiography revealed highly calcified aortic valves [figure nr. II.1.b.8], with fused commissures, extremely reduced mobility, with an opening of 7 mm, without being able to specify whether bicuspid or tricuspid, and aneurysmally dilated ascending aorta (50 mm).

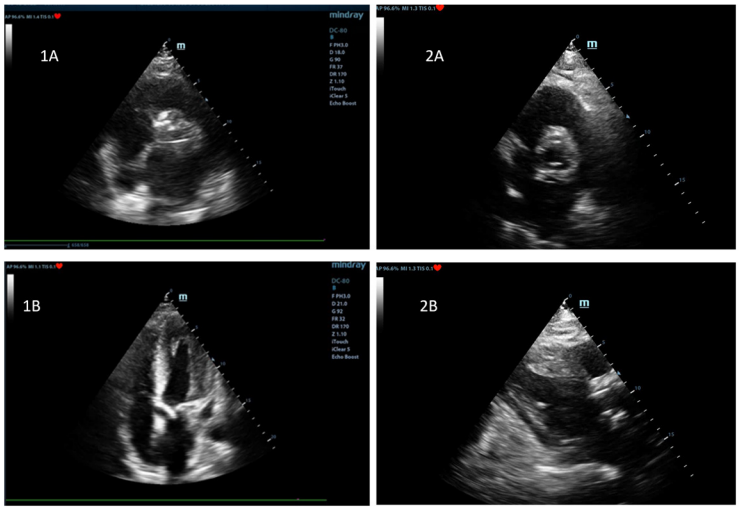


Figure nr. II.1.b.8 Morphological aspects of aortic valves before (1A: calcified valves, parasternal short axis, 1B: 4-chamber view) and after surgery (2A: normal opening of aortic prosthesis, parasternal short axis, 2B: normal opening of aortic prosthesis, parasternal long axis)

The left ventricle was non-dilated, with EDD=5 cm and EDV=126 ml, but with significant concentric ventricular hypertrophy (IVS=1.9 cm, LVPW=1.7 cm, RWT=0.42, LV mass=589 g).

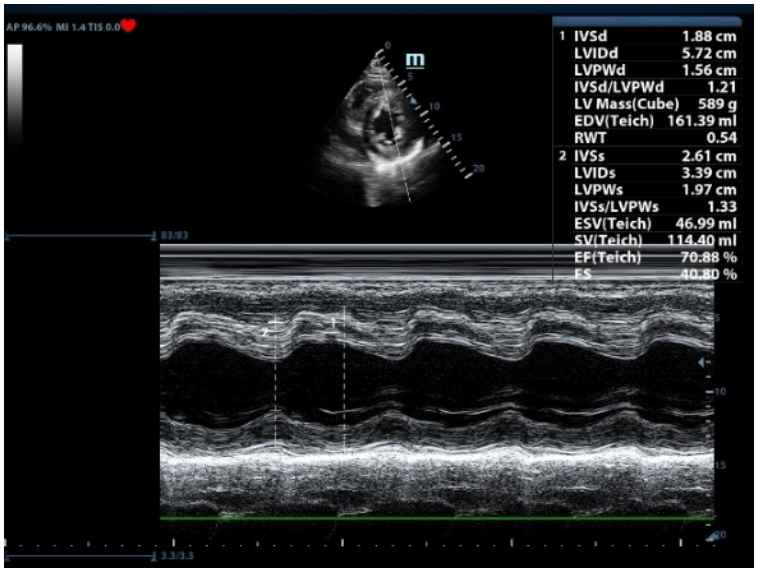


Figure nr. II.1.b.9 Concentric left ventricle hypertrophy (IVSd=1.8 cm, RWT=0.54, LV mass=589 g)

The left ventricle had normal systolic function (LVEF 52%), diastolic dysfunction type 1 delayed relaxation, and a SVi indexed flow of 57 mL. High flow status and aortic coarctation were excluded. NTproBNP, as a marker of heart failure, was elevated (7501 pg/mL). Coronary arteries were normally angiographic.

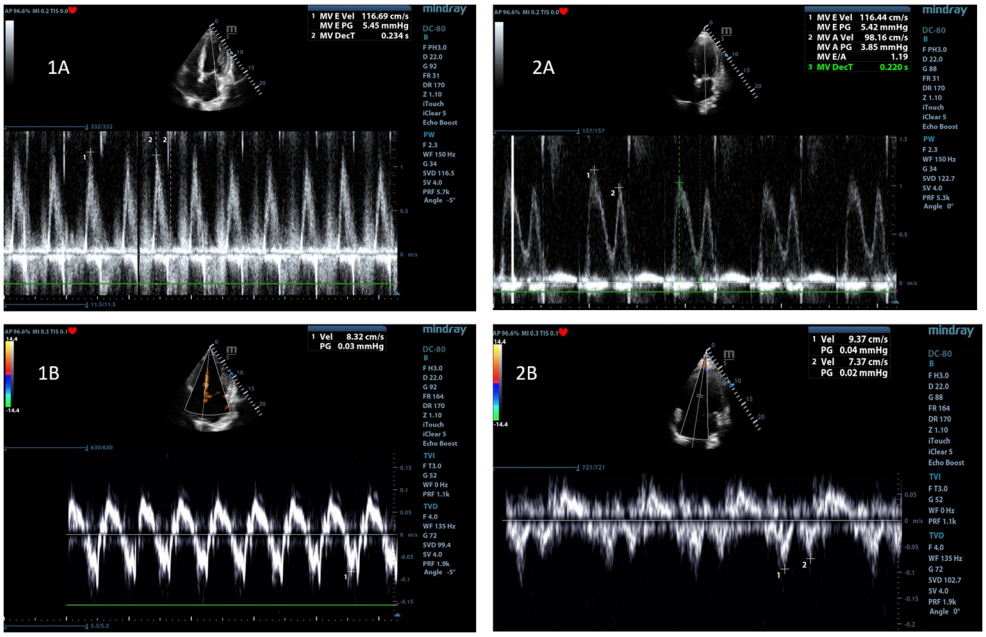


Figure nr. II.1.b.10. Evaluation of diastolic function before (1A: fused E and A waves of 116 cm/s, PW doppler 4; 1B: e'$_s$<a'$_s$ in tissue Doppler) and after surgery (2A: E>A, PW Doppler, e'$_s$> a'$_s$ in tissue Doppler)

The aortic flow interrogation showed a maximum gradient of 67 mmHg and a mean gradient of 50 mmHg; aortic valve area could not be measured planimetrically due to extensive calcifications that limited the technique and using the continuity equation, it was calculated at 0.43 cm2; at the same time, a mild aortic regurgitation with PHT = 265 ms was recorded.

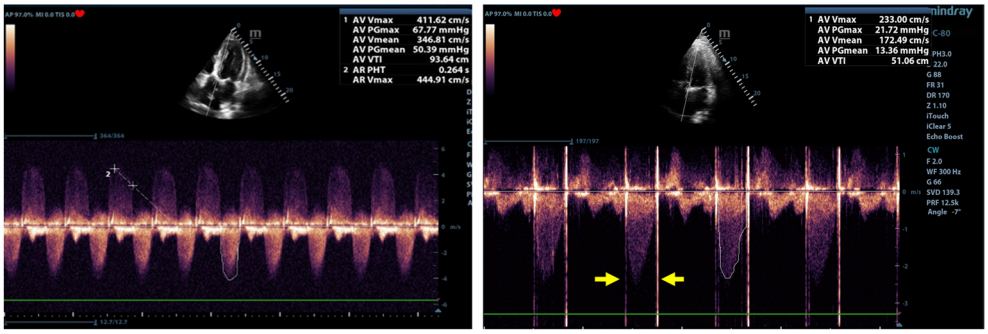


Figure nr. II.1.b.11. MPG of 50 mmHg and V_{mx}=4,11 m/s indicating severe aortic stenosis (left; CW Doppler, parasternal 5-chambers view) and normal MPG of 13 mmHg after aortic replacement with prosthetic valve (arrows shows opening and closing of the prosthesis' metallic disks) (right; CW Doppler, parasternal 5-chambers view)

The patient presented associated mitral valve lesion, a severe holosystolic mitral regurgitation, with 1.52 cm vena contracta, eccentric jet toward the lateral wall of the AS (Coandă effect).

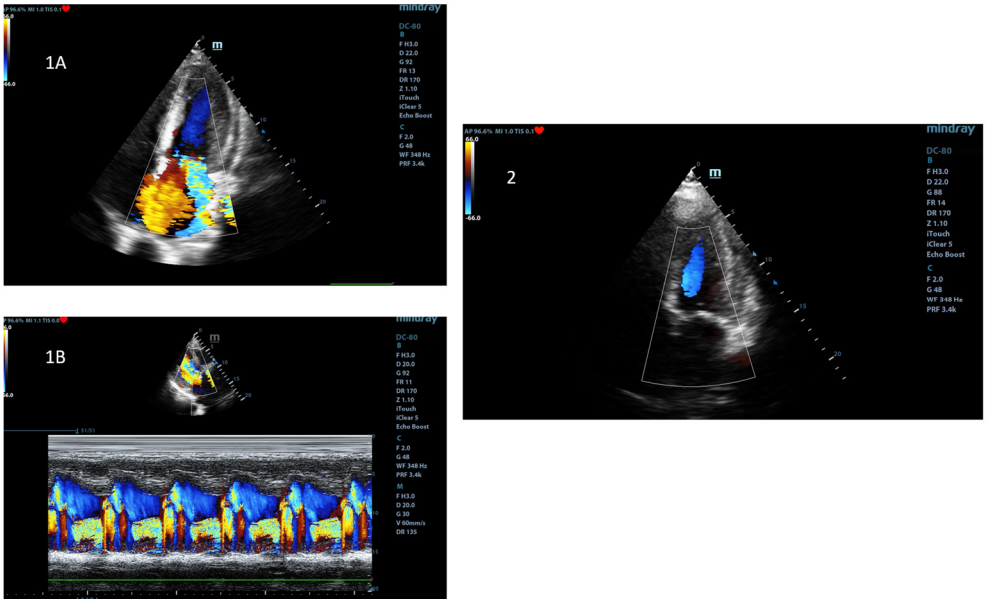


Figure nr. II.1.b.12. Severe holosystolic mitral regurgitation before surgery (image 1A: color Doppler, 4-chambers view; 1B: color M-Doppler) and no mitral regurgitation after mitral valvuloplasty (imgage 2: color Doppler, 4-chambers view)

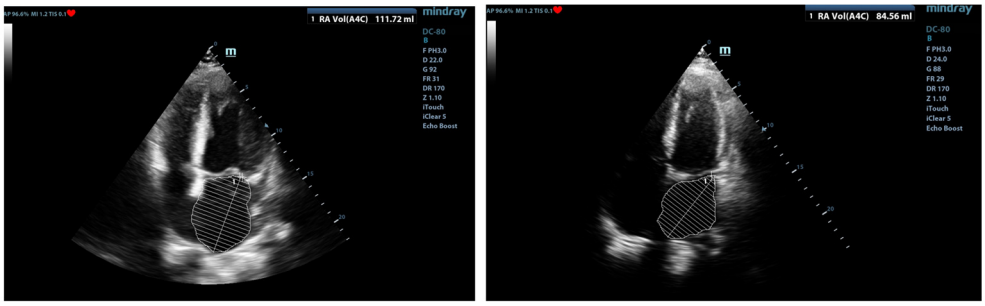


Figure nr. II.1.b.13. Small decreasing of the left atrial volume after surgery (2D, 4-chambers view)

The patient was categorized as having aortic disease (predominantly severe aortic stenosis) of probable post-rheumatic etiology, with associated severe mitral regurgitation and significant dilatation of the ascending aorta (ascending aortic aneurysm). The patient underwent a complex surgical procedure of aortic valve replacement with a double-disc mechanical prosthesis, ascending aortic replacement with Dacron prosthesis and mitral valvuloplasty. Postoperative re-evaluation at three months, indicated a normal functioning aortic prosthesis, hypertrophied LV, with normal systolic and diastolic function and no mitral regurgitation.

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The echocardiographic images are from the own collection of dr. Florina Parv.

AORTIC REGURGITATION

Aortic regurgitation (AR) is an acute or chronic condition in which, due to different etiologies that affect either aortic valves, aortic annulus or ascending aorta, a part of the ejected blood from LV into the aorta in systole returns to LV (regurgitant volume).

Etiology of AR include:

1. diseases that affect aortic valves: congenital bicuspidia, rheumatic fever, infectious endocarditis, calcifications (degenerative impairment), chest trauma, collagenases (ankylosing spondylitis, rheumatoid polyarthritis);
2. diseases that affect aortic annulus, aortic root and/or ascending aorta: collagen diseases (Marfan, Ehlers-Danlos syndrome), aortic dissection, inflammatory bowel disease, aortitis (syphilis), arterial hypertension. [figure nr. II.1.b.14.]

Mechanisms of dysfunction are enlargement of the aortic root and normal cusps, cusp prolapse with eccentric AR jet and thickened valve with reduced motion.

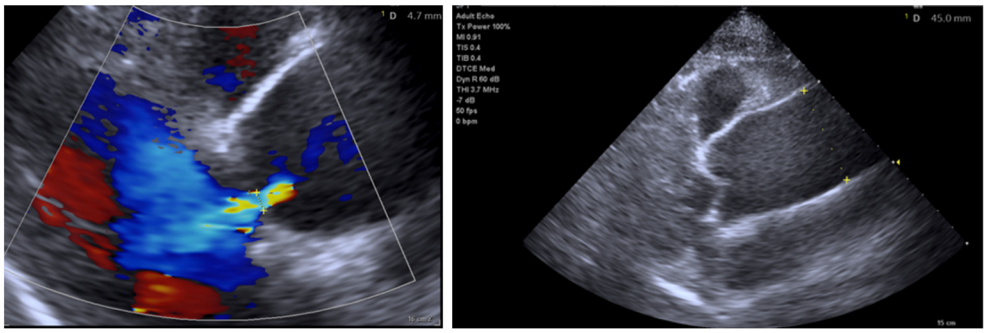

Figure nr. II.1.b.14. AR with central flow in a case with ascending aortic aneurysm (45 mm) (color Doppler and 2D, parasternal long axis view)

In *acute AR*, a rapid increase of the pressure in left atrium and telediastolic pressure intraventricular LV occurs and the result is increasing the heart rate and contractility in order to maintain cardiac output. If this mechanism fails, pulmonary edema, cardiogenic shock and myocardial ischemia appear, contributing to the haemodynamic depreciation.

In *chronic AR* the compensatory mechanisms (LV dilatation, increasing of telediastolic LV volume, LV hypertrophy) maintain a relatively decreased telediastolic LV pressure and a sufficient coronary perfusion.

Echocardiograpy is essential in AR, and must include:

- diagnostic of AR presence and elements of etiology
- estimation of AR severity
- estimating the consequences on LV and right part of the heart (RV, PAP)
- evaluation of ascending aorta

2D echocardiography must identify the following issues:

- aortic cusps aspect (number of cusps, calcifications, vegetations) and their mobility
- aortic root (dilation, dissection)
- dimensions of LV (EDD>7 cm and ESD>5 cm are indications of surgery)
- systolic and diastolic function of LV (LVEF≤50% in asymptomatic patients is indication for surgery)

Assessment of AR severity includes several measurements using color, PW and CW Doppler. Before any interpretation of a hemodynamic measurement, blood pressure and heart rate must be recorded; also, we have to take into consideration the volemic status, coexistent aortic stenosis, LV compliance, heart rate. In order to have a correct diagnosis of severe aortic regurgitation, we must evaluate several aspects:

1. AV morphology: large coaptation or flail of valve indicates severe AR.
2. Flow jet: in significant haemodynamic AR flow jet area is large in central jets (color Doppler) and has a dense signal in CW Doppler. [figure nr II.1.b.15.]

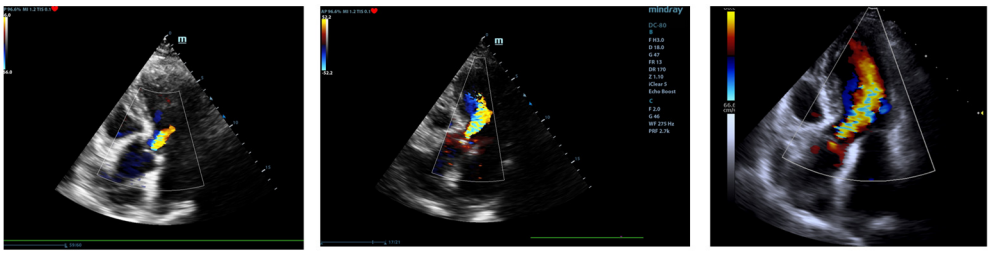

Figure nr. II.1.b.15. Different severities of aortic regurgitation (5-chambers view, color Doppler)

3. Regurgitant jet width/LVOT diameter ratio (for circular aortic regurgitant orifice) ≥ 65% indicates a severe AR

4. Vena contracta represent the narrowest part of the regurgitant jet at the level of the aortic valve and it is a semiquantitative parameter and not related to haemodynamic factors; > 6 mm indicates severe AR [figure nr. II.1.b.16.]

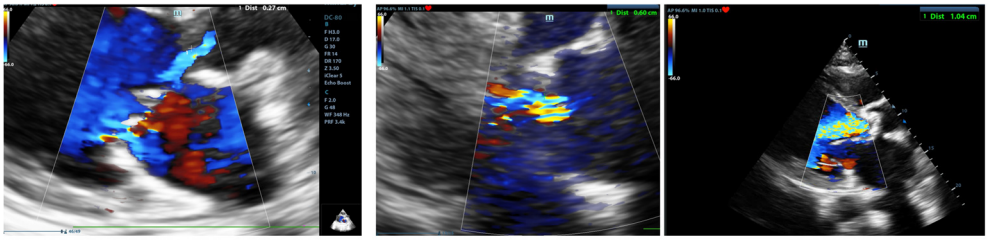

Figure nr. II.1.b.16. Different severities of aortic; left: mild AR (vena contracta 0.27 cm, flow jet is not dense and eccentric); middle: moderate AR (vena contracta 0.6 cm); right: severe AR (vena contracta 1.04 cm, intense large flow jet) regurgitation (parasternal long axis view, color Doppler)

5. PISA (proximal isovelocity surface area) method evaluates effective regurgitant orifice area (EROA) and regurgitant volume (RV) through at every heart contraction. PISA represent the surface area of the blood returning through the aorta at a given value of the aliasing velocity.

[figure nr II.1.b.17.]

$EROA = (2\pi r^2 \times Va)/peak\ velocity$

$RV = EROA \times TVI$

r = radius

Va = aliasing velocity

Peak velocity = maximum velocity of flow in CW Doppler

TVI = time-velocity integral of aortic regurgitant flow in CW Doppler

In the case of severe AR, EROA is ≥0.3 cm2 and RV≥60 mL/beat.

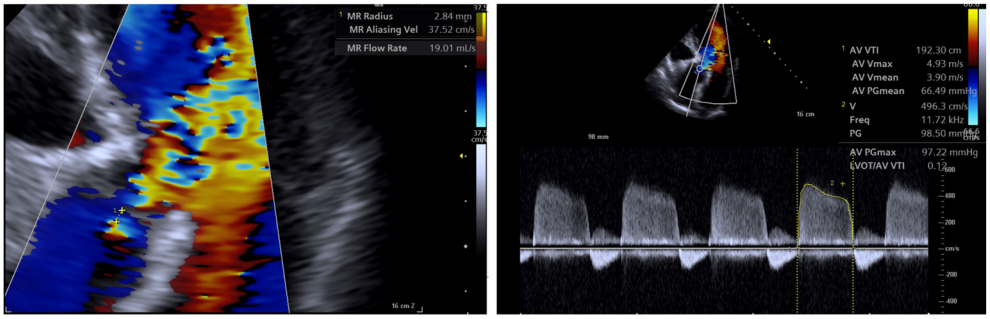

Figure nr. II.1.b.17. Measuring the EROA and RV by PISA method: left-measuring the flow rate and right: measuring the Vmx and TVI of AR jet flow (color Doppler and CW Doppler, 5-chambers view)

6. Diastolic aortic flow reversal in descending aorta, using PW Doppler, with an end-diastolic velocity >20 cm/s in severe cases. [figure nr. II.1.b.18.]

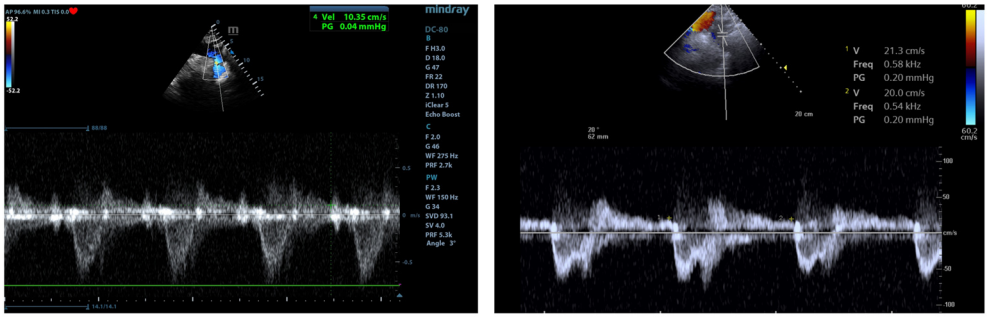

Figure nr. II.1.b.18. Evaluation of the end diastolic velocity of the aortic flow regurgitation jet in descending aorta (suprasternal view, color and PW Doppler) in a case of moderate (V = 10.3 cm/s) (left) and severe (right) aortic regurgitation (V = 21.3 cm/s)

7. PHT (pressure half-time) represent the time for peak diastolic pressure gradient to decrease by 50%; a value shorter than 200 ms indicates severe AR and a value longer than 500 ms a mild AR. [figure nr. II.1.b.19.]

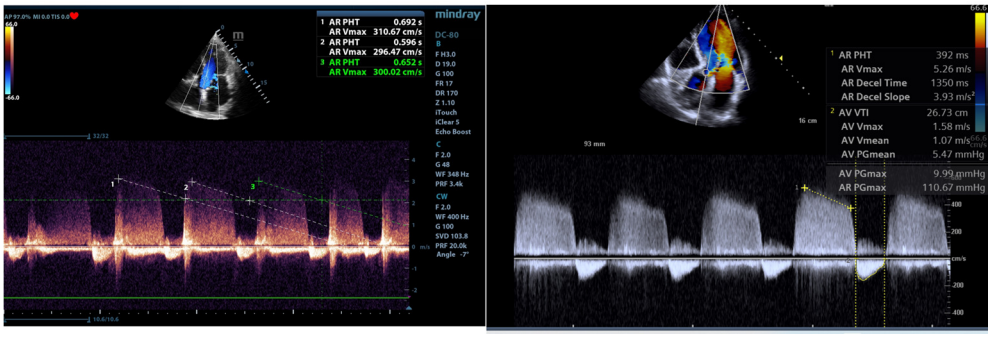

Figure nr. II.1.b.19. Estimation of AR severity by PHT method (5-chambers view, color and CW Doppler); left: mild AR (PHT = 692 ms; envelope of AR is not so intense); right: moderate AR (PHT = 392 ms; intense signal of the envelope of regurgitation flow)

Sometimes, when the regurgitant jet is quite large, it causes a vibration of the leaflet of the anterior mitral valve during diastole, called fluttering, which can lead to relative mitral stenosis. [figure nr. II.1.b.20.]

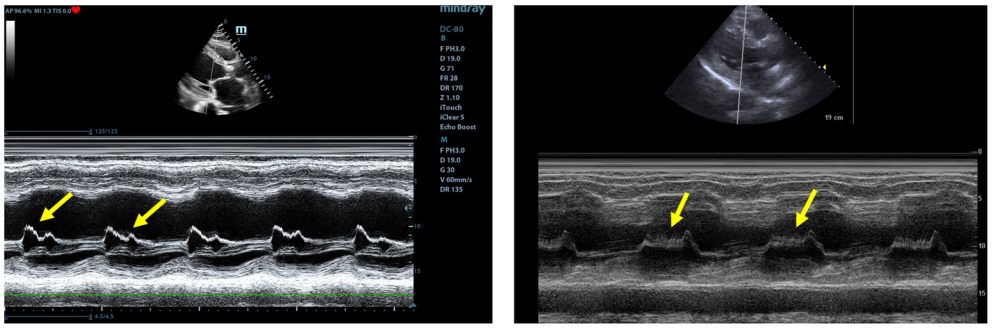

Figure nr. II.1.b.20. Fluttering of the anterior mitral valve (arrows) in a case with moderate (left) and severe (right) aortic regurgitation (parasternal long axis, M-mode)

At the same time, a dilated LV indicates a severe AR.

It is important to also evaluate the ascending aorta, according to body surface, which can be dilated; there is one phenotype with dilation of aortic root (sinuses of Valsalva > 45 mm) and another phenotype with ascending aorta dilation (aneurysm) and normal sinuses of Valsalva.

Echo-following up and surgery indications

Disease progression without intervention and the natural evolution of AR towards asymptomatic LV dysfunction is <3.5%/year and towards symptomatic heart failure >25% in patients with pre-existent LV dysfunction.

Asymptomatic patients with severe AR and normal LV function must be evaluated once a year or more often (once every 3-6 months), if changes in the cut-off values of the LV function or size appear.

Surgery is indicated in symptomatic patients with severe AR regardless LV function, in asymptomatic patients with severe AR with LVESD>50 mm (LVESD>25 mm/m2) or LEVF≤50% and in patients with severe AR which must undergo CABG, surgery of ascending aorta or another valve.

CLINICAL CASE

Patient SE, male, aged 69 years, with no cardiovascular pathology before, presented to a routine consultation at general practitioner (family doctor) without having specific cardiac symptoms.

The physical examination revealed BMI = 24 kg/m2, BSA = 1.89 m^2, rhythmic, clear, normal heart sounds, HR = 88/min, BP = 150/50 mmHg in both arms, intense diastolic murmur in the aortic focus; normal pulmonary auscultatory, normal abdomen, normal pulse in the peripheral arteries.

Electrocardiogram showed sinus tachycardia, 90/min, intermediate QRS axis, left ventricular hypertrophy.

Transthoracic echocardiography revealed normal tricuspid aortic valve, with normal mobility and normal dimensions of ascending aorta (35 mm).

A central aortic regurgitant flow with a jet width/LVOT diameter ratio of 69% and a vena contracta of 12 mm was identified in parasternal long-axis view, suggesting a hemodynamically significant aortic regurgitation. [figure nr. II.1.b.21.]

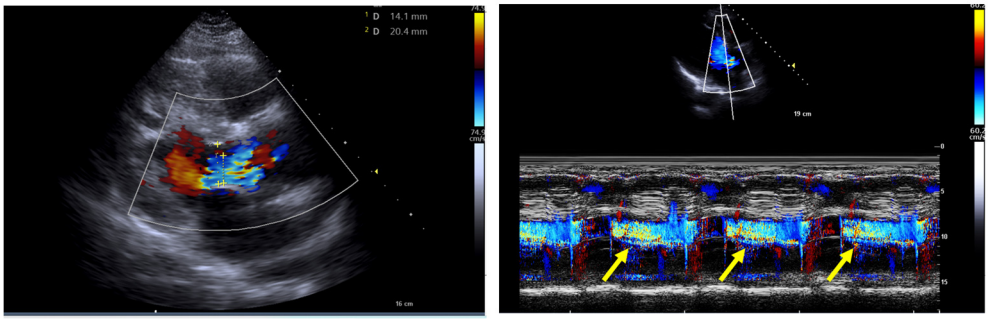

Figure nr. II.1.b.21. Significant holodiastolic (arrows) aortic regurgitant flow jet (parasternal long axis; left: color Doppler with evaluation of the flow jet/LVOT diameter ratio; right: M-mode and color Doppler showing the localisation of the regurgitant jet during the whole diastole)

In apical 5-chambers view, the identified regurgitant jet stretches across almost the entire left ventricular area, and in pulsed Doppler, the Vmx and IVT of the regurgitant flow was measured, which showed a dense, intense envelope signal. The measured PHT was 161 ms, indicating a severe regurgitation. The PISA method could not be performed technically correct, as the convergence zone was limited and difficult to identify.

A maximum telediastolic velocity of 20.1 cm/s was recorded in the descending aorta in PW Doppler, indicating significant aortic regurgitation. [figure nr. II.1.b.22.]

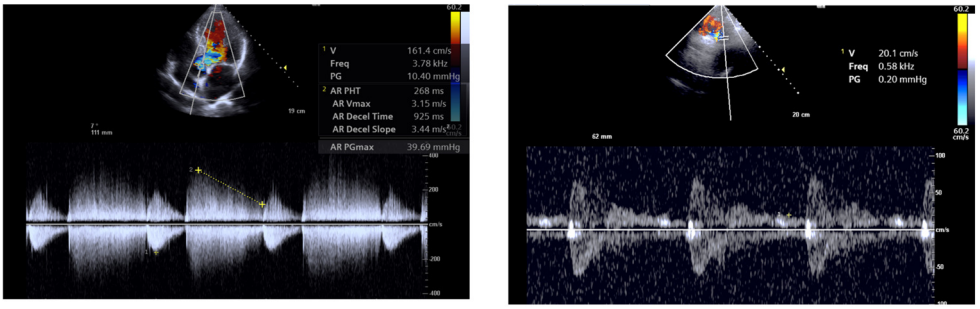

Figure nr. II.1.b.22. Parameters that estimated a severe aortic regurgitation in the presented case (left: CW Doppler, 5-chambers view showing a calculated PHT of 161 ms; right: PW Doppler, suprasternal view showing a maximum telediastolic velocity of 20.1 cm/s)

The consequences of aortic regurgitation were also evaluated, namely the LV morphology, with concentric hypertrophy (ESD = 38 mm, RWT = 0.53, increased LVmass of 727 g). [figure nr. II.1.b.23.]

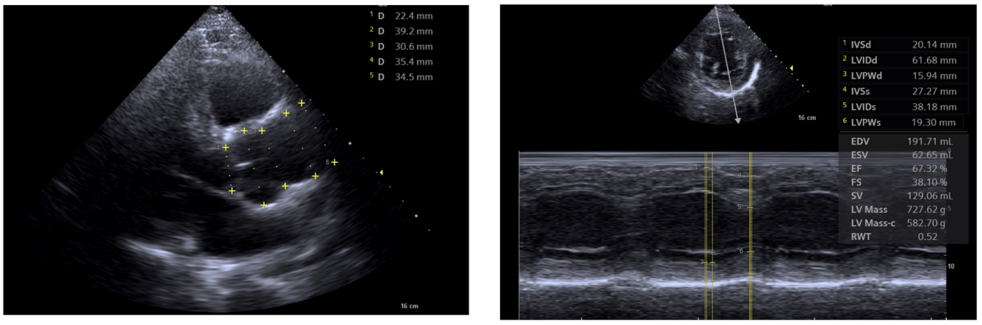

Figure nr. II.1.b.23. Left: aortic measurements (aortic root, aortic ring, Valsalva sinuses, sino-tubular junction, ascending aorta; 2D-parasternal long axis); right: LV measurements (interventricular septum, posterior LV wall and LV diameters during cardiac cycles (M-mode, parasternal short axis, middle section).

The systolic function of the LV was normal (LVEF 50%), and the diastolic function was altered, the patient presented diastolic dysfunction with delayed relaxation, type 1 pattern. [figure nr. II.1.b.24.]

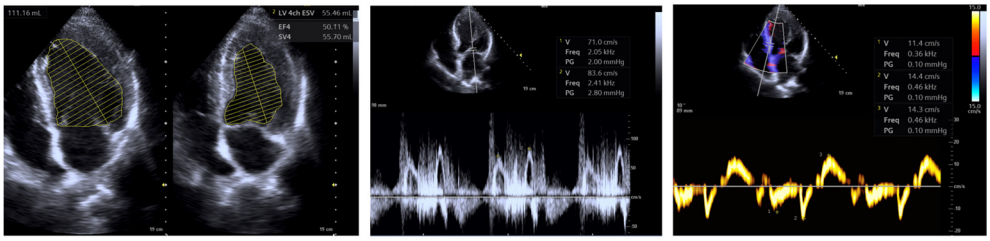

Figure nr. II.1.b.24. Evaluation of the LV function in case presented. Left: normal systolic function (2D, apical 4-chambers view); middle and right: mild type 1 diastolic dysfunction (E = 71 cm/s; A = 83 cm/s, E/e' = 7.35) (4-chambers view, PW Doppler and tissue Doppler)

The ascending aorta was not dilated and there were no secondary alterations in the right heart (right chambers of normal size, no signs of pulmonary hypertension, PAP = 24 mmHg).

From a biological point of view, routine and NT-proBNP tests were within normal limits. The patient was categorized as having asymptomatic severe chronic aortic regurgitation, with indication for clinical and echocardiographic monitoring and no definite surgical indication. Coronarography showed coronary arteries without significant stenosis.

The patient has been monitored for the last five years, during which the following clinical, biological and echocardiographic parameters have been noted. A slight increase in LVESD and decrease in LVEF have been noted, without indicating the need for surgery. [table nr. 3]

Table nr. 3. Clinical, biological and echocardiographic parameters followed over the years in presented case

Years of following-up/parameter	1	2	3	4	5
Symptoms	no	no	no	no	no
NT-proBNP (pg/mL)	NA	NA	NA	126	94
LVESD (mm)	25	27	27	28	28
PHT (ms)	243	309	310	268	161
LVEF (%)	59	58	55	52	52
VTD in descending aorta (cm/s)	14	15	15	17	20
Vena contracta (mm)	9	9	10	11	12
E (cm/s)	50	44	47	69	71
A (cm/s)	108	102	97	101	83

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The echocardiographic images are from the own collection of dr. Florina Parv.

c. Tricuspid valve: tricuspid regurgitation (Florina Pârv)

The tricuspid valve apparatus is very different from the mitral one: the tricuspid valve which generally has three leaflets of different size (anterior, posterior and septal, which is the smallest one, and has a superior insertion to the anterior mitral valve), the septal chordae which are inserted on the interventricular septum, and the tricuspid ring which has a non-planar elliptical shape and whose dimensions vary on age, sex, size of the right cavities. The size of the tricuspid annulus is 19±2 mm/m2.

Tricuspid regurgitation represents the return of blood from the RV to the RA during the systole. During routine echocardiographic examinations, minor tricuspid regurgitation can be incidentally discovered in healthy subjects in a fairly high percentage (60-70%) being considered without pathological significance if the valves are morphologically normal, the RV is not dilated and the regurgitation jet is short, protosystolic with maximum velocity below 2.3 m/s. Pathological tricuspid regurgitation is etiologically and pathogenically classified into:

- *Secondary* (functional) to the dilation of the tricuspid ring due to the overload of volume and or pressure in the RV (pulmonary hypertension, most frequently secondary to left heart diseases, septal defects) [figure nr. II.1.c.1., II.1.c.3.].

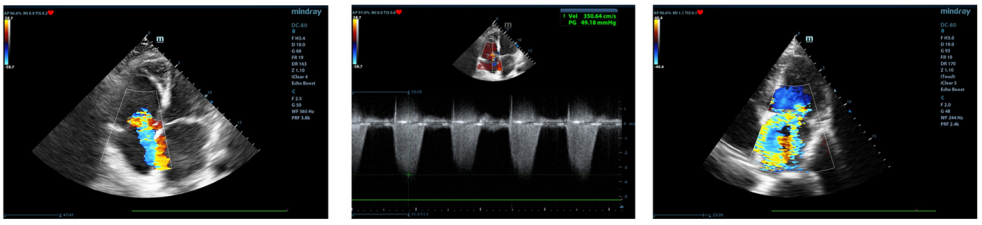

Figure nr. II.1.c.1. Moderate tricuspid regurgitation (left and middle image; Vmx=3.5 m/s; color and CW Doppler at tricuspid valve level, 4-chambers view) due to secondary pulmonary hypertension consecutively to a left cardiac pathology: severe mitral regurgitation (right image-color Doppler, 4-chambers view)

- *Primary* (organic) caused by anatomical abnormalities of the tricuspid valve apparatus (rheumatic disease, infectious endocarditis [figure nr. II.1.c.2.], Ebstein's disease, carcinoid syndrome, infiltrative diseases, post-irradiation, collagenosis, cardiovascular implantable electronic devices);

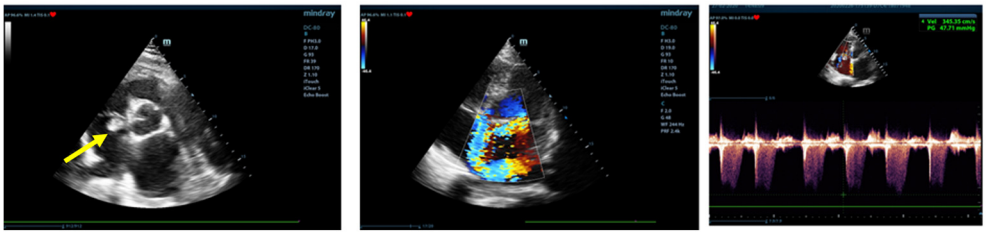

Figure nr. II.1.c.2. Moderate tricuspid regurgitation in a case with concomitant left and right heart pathology: left-tricuspid valve endocarditis; a large vegetation attached to tricuspid ring (arrow) (2D, parasternal short axis at great vessel level); middle-severe asymmetric mitral regurgitation (color Doppler focused on left chambers, 4-chambers view); right-tricuspid regurgitation and calculation of right interatrial-ventricular gradient Pmx=47 mmHg, CW Doppler, 4-chambers view).

- *Atrial* caused by remodeling of the tricuspid ring or the right atrium as a result of aging or atrial fibrillation.

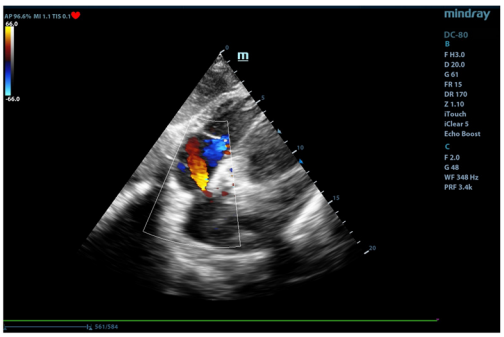

Figure nr. II.1.c.3. Tricuspid regurgitation in a case with atrial septal defect (color Doppler, subxiphoid view)

Pathological tricuspid regurgitation is associated with increased morbidity and mortality, therefore not only its early detection and evaluation, but also the conservative medical and the interventional (surgical or endovascular) treatment appropriate for the stage of the disease are very important.

In 2D ultrasound, the following aspects are evaluated:

- *the morphology and mobility* of the tricuspid valves: in rheumatic and carcinoid disease they are thickened (even the chordae) with reduced mobility, in Ebstein's disease the septal cusp is displaced apically and the anterior cusp has a shape of a "sail"; the mobility is exaggerated in the case of valve prolapse.
- *coaptation abnormalities* of the valves, frequently observed in the case of severe regurgitation, the presence of intracavitary leads and less often in Ebstein's disease. An increased distance of the coaptation plane (>1 cm) indicates a risk of regurgitation relapse postoperatively.
- *the size of the tricuspid ring*; in adults in the 4-chamber apical view in protodiastole it is 28±5 mm, and values >3.5 cm (21 mm/m^2) indicate severe TR. It can be underestimated in 2D ultrasound.
- RA size - frequently RA is dilated
- RV size - RV is dilated in moderate and severe TR
- the *morphology of the LV* in parasternal short axis - can be modified in conditions of increased pressure in the right cavities, having the shape of the letter "D" [figure nr. II.1.c.4.]
- *paradoxical movement of the interventricular septum*

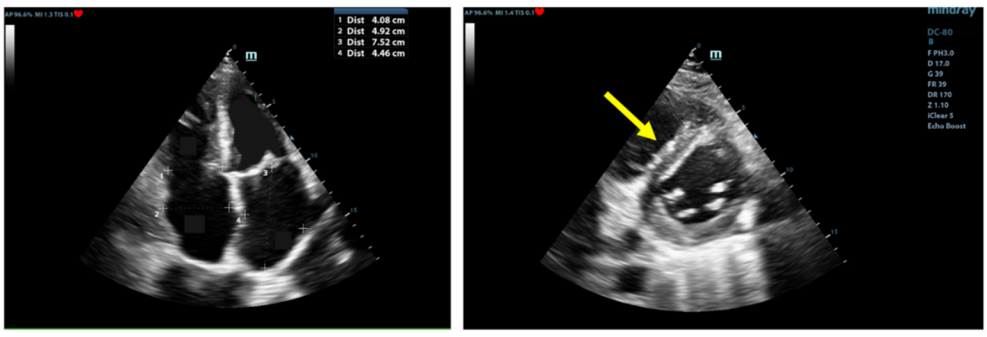

Figure nr. II.1.c.4. Dilated right ventricle with a large tricuspid ring (4.08 cm) (left - 2D, apical 4-chambers view) and flattened „D"-shaped interventricular septum (arrow) in a case with severe pulmonary hypertension (right - parasternal short axis, middle level)

A dilated inferior vena cava (>2.1 cm) and with respiratory variability <50% indicates increased pressure in the right atrium. (10–20 mmHg); A non-dilated IVC with inspiratory collapse by more than 50% indicates a normal RA pressure (0–5 mmHg); [figure nr. II.1.c.5.]

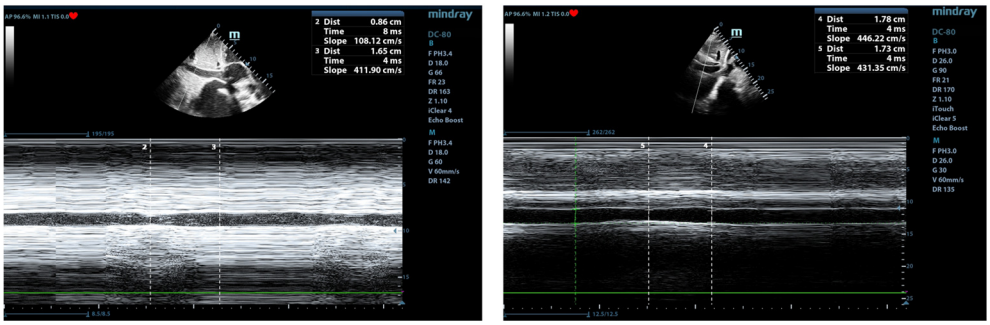

Figure nr. II.1.c.5. Left: inferior vena cava with normal diameter and normal respiratory variability (M-mode, modified subxiphoid view); Right: inferior vena cava with normal diameter and reduced respiratory variability (3%)(M-mode, modified subxiphoid view).

Using color Doppler ultrasound, it is possible to quantify the area of the regurgitation jet (only the turbulent portion) compared to the area of RA and also vena contracta (which indirectly reflects the area of the regurgitant orifice); an area ≥40% and a vena contracta ≥0.7 mm indicates a severe TR. Eccentric jets, those with the Coandă effect, can be underestimated. The PISA method quantifies the area of the regurgitant orifice and the regurgitant volume.

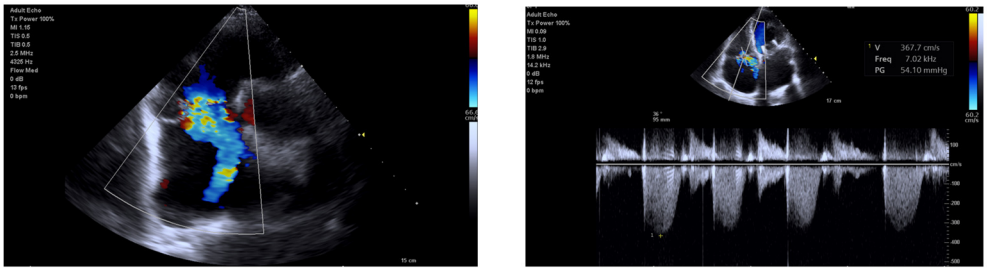

Figure nr. II.1.c.6. Severe tricuspid regurgitation and calculating of pulmonary systolic arterial pressure (54 mmHg) (left: color Doppler, parasternal short-axis; right: CW Doppler, 4-chambers view)

Pulsed and continuous Doppler ultrasound assesses:

- the shape and density of the regurgitation envelope; in severe TR the jet is turbulent, the envelope is dense and has a triangular shape, with an early peak
- tricuspid diastolic flow velocity; an E-wave velocity ≥ 1m/s indicates severe TR.
- estimation of the systolic pressure in the pulmonary artery ($4v^2$ + average RAP) (in the apical 4-chambers view, by measuring the tricuspid systolic blood flow velocity and estimating the RAP pressure in the right atrium according to the diameter of the inferior vena cava and its respiratory variability). Concomitant with dilation of inferior vena cava, suprahepatic veins are also dilated, and the blood reflow into them can be visualized in color Doppler [figure nr. II.1.c.7.]. An increased velocity of the regurgitation jet does not corelate with the severity of the regurgitation. The lack of identification of a tricuspid regurgitation does not indicate the absence of pulmonary hypertension, in which case other ultrasound aspects will be carefully evaluated (RV hypertrophy, shortening of the acceleration time at the pulmonary valve level).

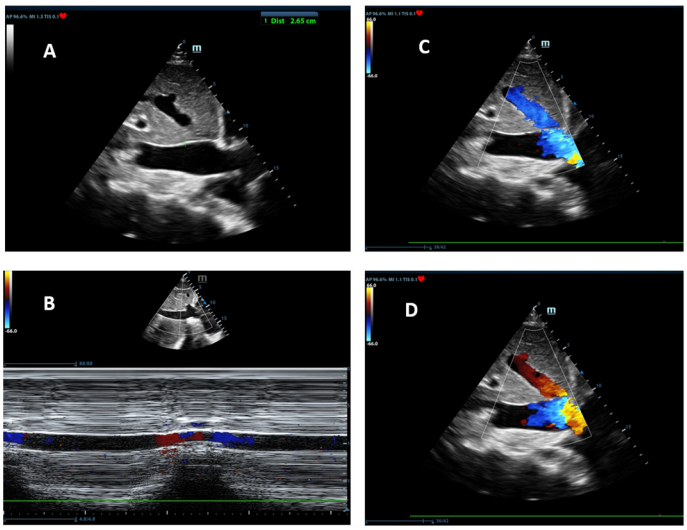

Figure nr. II.1.c.7. Dilated inferior vena cava (2.65 cm) (A) with reduced respiratory variability (B) and dilated suprahepatic veins with flow reversal revealed by blue (C) and red (D) color during cardiac cycle (2D, M-mode, color Doppler, subxiphoid and subcostal views).

- the aspect of the flow in the hepatic veins (in the subcostal incidence in severe TR there is a retrograde systolic flow) [figure nr. II.1.c.8.]

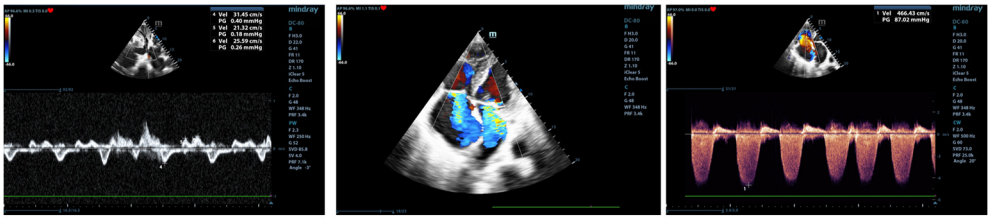

Figure nr. II.1.c.8. Reversal systolic flow in hepatic veins in a case with severe tricuspid regurgitation (left: PW Doppler; middle: color Doppler 4-chamber view and right: CW Doppler; maximum velocity of 4.66 m/s and maximum gradient 87 mmHg; PASP estimated at 102 mmHg).

The currently criteria of ultrasound diagnosis for severe tricuspid regurgitation are qualitative, semi-quantitative and quantitative. [table nr. 1.] [figure nr. II.1.c.9]

Table nr.1. Echocardiographyc criteria for severe tricuspid regurgitation

Qualitative	
Tricuspid valve morphology	Abnormal/flail
Colour flow regurgitant jet	Very large ventral/eccentric impinging jet
CW signal of regurgitant jet	Dense, triangular with early peaking
Semiquantitative	
Vena contracta	>7 mm
Hepatic vein flow	Systolic flow reversal
Tricuspid inflow	E wave ≥1 m/s
PISA radius	>9 mm
Quantitative	
Effective regurgitant oriffice area (EROA)	≥40 mm^2
Regurgitant volume	≥45 mL/beat
Enlargement of RV, RA, IVC	

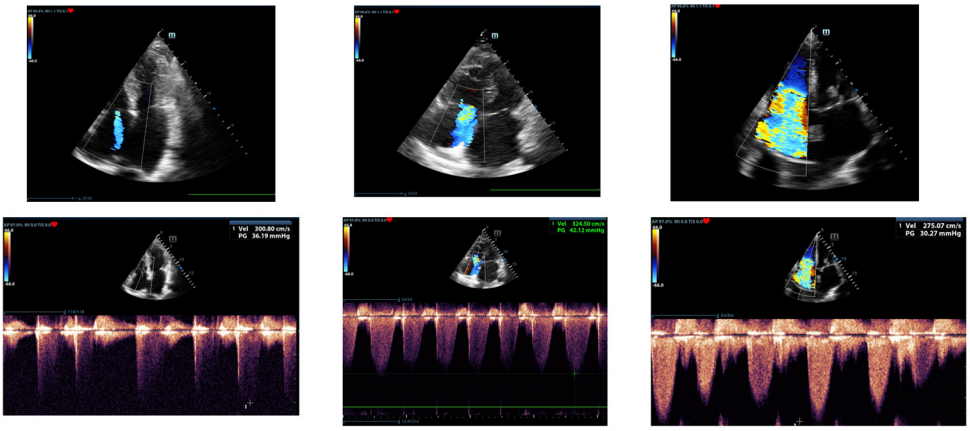

Figure nr. II.1.c.9. Mild, moderate and severe tricuspid regurgitation (upper row: color Doppler, 4-chambers view and bottom row: CW Doppler, 4-chambers view)

The classification of tricuspid regurgitation strictly based on quantitative methods (mild, moderate, severe) currently has two new classes: massive regurgitation (vena contracta 14-20 mm, EROA 60-79 mm2) and torrential (VC≥21 mm, EROA≥80 mm2).

In pathology that includes tricuspid regurgitation, mitral valvulopathies must also be correctly evaluated; also with time, in the case of severe regurgitation, there is a remodeling of the RV and the tricuspid valve apparatus, which worsens the regurgitation, thus the decision for surgical treatment is made accordingly.

Indications for surgical treatment (repair or replacement) in tricuspid regurgitation are individualized, generally addressed to severe forms and depend on the etiology and consequences of regurgitation:

- in patients who require surgery on the left heart valves (aortic and/or mitral), with severe or moderate primary/secondary tricuspid regurgitation, but with a dilated tricuspid ring

- in patients who do not require surgery on the left heart valves, with severe primary tricuspid regurgitation symptomatic or asymptomatic, but with RV dilatation
- in patients who do not require surgery on the left heart valves, with severe secondary tricuspid regurgitation without severe RV/LV dysfunction and without severe secondary pulmonary hypertension.

CLINICAL CASE

A 45-year-old woman, smoker, without prior pathology, presented for about a month progressive dyspnea on exertion, precordial pain with atypical angina and in the last week, rapid palpitations, sweating, pronounced vertigo, nocturnal dyspnea, and malleolar edema, reasons for which she was presented to the emergency department. The clinical status was altered, BP=90/60 mmHg, HR=135/min, arrhythmic heart sounds, bilateral basal stasis rales, SaO2=94%. The resting electrocardiogram showed atrial fibrillation with rapid rhythm and mild diffuse ST-segment depression. Biological investigations showed normal hemogram, creatinine 1.3 mg%, AST=54 U/L, ALT=88 U/L, GGT=108 U/l, INR=1.3, LDL cholesterol=180 mg%, NTproBNP=4556 pg/ML, TSH=0.01 μUI/mL.

The positive diagnosis consisted of symptomatic hyperthyroidism, tachycardia-induced cardiomyopathy, angina pectoris, newly diagnosed atrial fibrillation, NYHA III heart failure, hypercholesterolemia, hepatic cytolytic syndrome. Subsequent endocrinologic investigations established Basedow disease as the etiology of hyperthyroidism.

An echocardiography was performed, which revealed a slightly dilated VS with EDD = 5.5 cm, normal wall thickness, with global hypokinesia and

an apparently normal LVEF of 50%, but appreciated as systolic dysfunction in the context of mitral regurgitation, probably chronic and tachycardia-induced cardiomyopathy due to prolonged adrenergic stimulation. In the apical 4-chamber apical view, a normal morphologic appearance of the mitral and tricuspid valves, a mitral regurgitant jet of mild severity (4 mm vena contracta, slightly eccentric jet towards the lateral wall of the AS), and a tricuspid regurgitant jet of medium severity (6 mm vena contracta, slightly dense and Ü"-shaped envelope of the regurgitant in CW Doppler). RV and AD were dilated: tricuspid annulus 4 cm, RA volume of 80 ml. Inferior vena cava was dilated 2.2 cm, with reduced respiratory variability of 45%. Pulmonary artery systolic pressure was estimated to be 48 mmHg, framed as mean pulmonary hypertension [figure nr. II.1.c.10.].

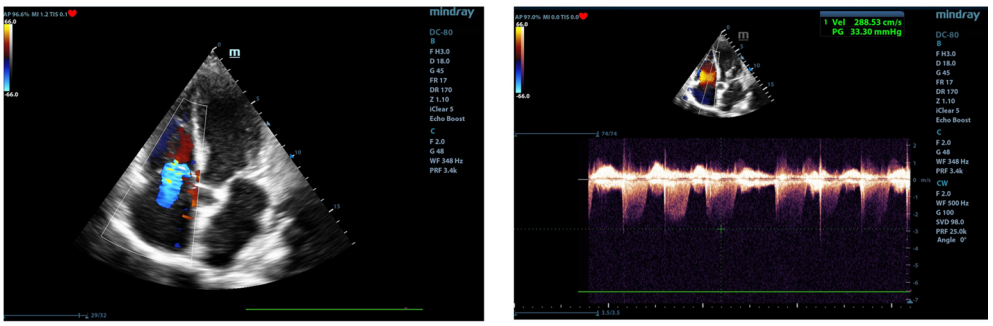

Figure nr. II.1.c.10. Moderate tricuspid regurgitation (left, color Doppler, 4-chambers view) and maximum gradient between right atrium and right ventricle of 33 mmHg (right, CW Doppler, 4-chambers view)

The ultrasonographic surprise came when a small interatrial communication, an atrial septal defect (less than 0.5 cm) with right left shunt, with QP/QS ratio <1 was found, which was identified in several incidents [figure II.1.c.11]. LA was dilated with an LA volume of 90 ml.

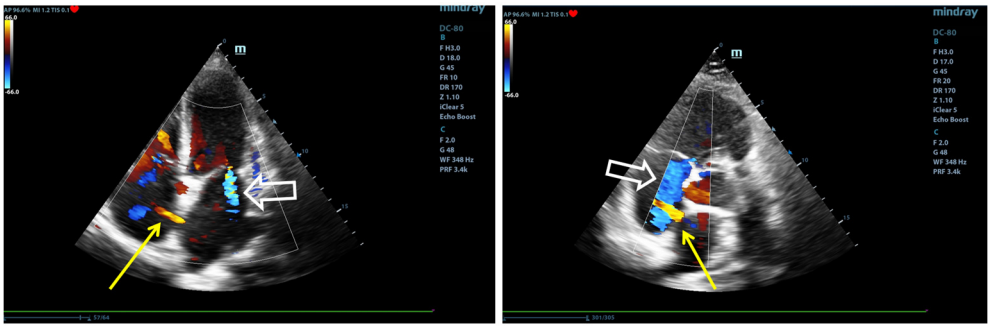

Figure nr. II.1.c.11. Left: mild mitral regurgitation (empty arrow) and left-to-right shunt at interatrial septum (slender arrow) color Doppler, 4-chambers view. Right: moderate tricuspid regurgitation (empty arrow) and left-to-right shunt at interatrial septum (slender arrow), color Doppler, parasternal short axis, great vessels level.

The tricuspid regurgitation was thus thought to be due to both mechanisms: pressure and volume overload in the right atrium, secondary to mitral regurgitation, but also to the left atrium-right atrium shunt.

Treatment was initiated with synthetic antithyroid medication and treatment for neglected complications of thyroid disease (atrial fibrillation, tachycardia-induced cardiomyopathy, heart failure): beta-blocker in adjusted dose, loop diuretic and MRA, SGLT2 inhibitor, anticoagulant. Clinical evolution was slow but favorable, with complete disappearance of heart failure symptoms after three weeks. After three months of follow-up the patient was still in atrial fibrillation with moderate heart rate, but with normal exercise capacity, NTproBNP reduced to 560 pg/mL; echocardiographic findings were mild mitral regurgitation, LVEF 55%, normal VS kinetics, EDD of 5 cm, mild tricuspid regurgitation and PASP 35 mmHg. She remained on treatment with synthetic antithyroid in decreasing doses, beta blocker, anticoagulant, diuretic in minimal dose until the next medical visit.

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The echocardiographic images are from the own collection of dr. Florina Parv.

d. Pulmonary valve: pulmonary stenosis; pulmonary regurgitation (Florina Pârv)

Pulmonary stenosis

Pulmonary stenosis represents the narrowing of the opening of the pulmonary valve, due to valvular structural changes, usually in congenital heart diseases (tetralogy of Fallot, isolated pulmonary stenosis) and also in acquired conditions (carcinoid syndrome, rheumatic fever); the consequence is RV hypertrophy, decreased systolic flow in the pulmonary artery and its dilation. Sometimes there are extrinsic compressions that narrow the pulmonary valve, without morphological alterations (e.g. extrinsic compression due to mediastinal pathology, cardiac tumors, Valsalva sinus aneurysm).

Transthoracic echocardiography must describe:

- the consequences of pulmonary stenosis:
    - RV hypertrophy (wall thickness >5 mm in diastole) and subsequent RV dilatation
    - dilation of the pulmonary artery
    - increased systolic pulmonary artery pressure
- the morphology and mobility of the pulmonary valves: thickened, with systolic bulge and reduced movement of the cusps (2D, parasternal short axis)

Increased transvalvular systolic velocity >4 m/s, valvular area < 1 cm^2/m^2 and a peak pressure gradient greater than 60 mmHg indicate a severe pulmonary stenosis (CW Doppler) [figure nr. II.1.d.1.]

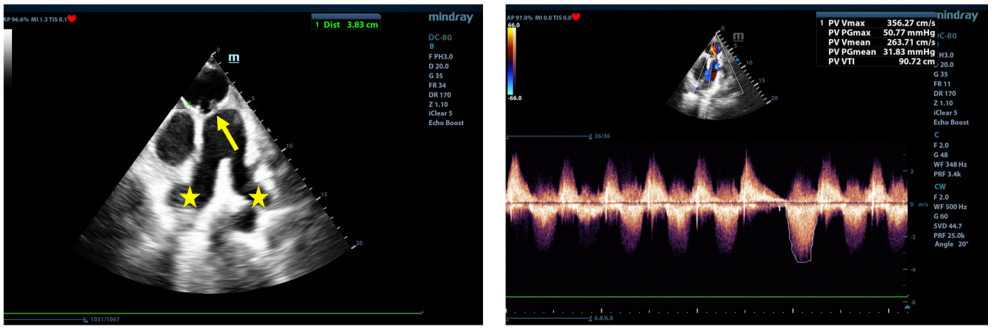

Figure nr. II.1.d.1. Pulmonary (re)stenosis 70 years after a pulmonary valvulotomy performed for tetralogy Fallot: markedly dilated pulmonary artery (3.83 cm) and of its principal branches (starred) (left, 2D, parasternal short axis) and increased pulmonary peak velocity V_{mx}=3.56 m/s with maximum gradient of 50 mmHg (right, color and CW Doppler)

Pulmonary regurgitation

Pulmonary regurgitation represents an abnormal return of blood flow in diastole from the pulmonary artery to the ejection tract of the RV and into the RV. As in the case of tricuspid regurgitation, minor pulmonary regurgitation is discovered incidentally in a large percentage of healthy individuals. Pathological pulmonary regurgitation is present in most cases of primary or secondary pulmonary hypertension. Other causes that can structurally alter the pulmonary valve and cause regurgitation are: congenital malformations, bacterial endocarditis, iatrogenic (after cardiac catheterization or post-interventional), carcinoid syndrome, Marfan syndrome.

Transthoracic echocardiography documents the following aspects:

- signs of pulmonary hypertension (RV, RA dilatation; flattened interventricular septum and with paradoxical movement)
- dilation of the ejection tract of the RV and the pulmonary artery
- morphology of the pulmonary valve: thickened or degenerated myxomatous cusps, vegetations (in the case of endocarditis), with incomplete closure

Quantification of pulmonary regurgitation is rather semi-quantitative taking into consideration the thickness of the jet and the intensity of the spectral Doppler signal of the regurgitation envelope. [figure nr. II.1.d.2].

The echographic characteristics of a hemodynamically significant pulmonary regurgitation are:

- dense, triangular envelope
- (holo) diastolic reflux with premature end
- rapid deceleration slope of diastolic velocity
- antegrade presystolic flow
- turbulent diastolic jet with a significant thickness (>65% of RVOT)

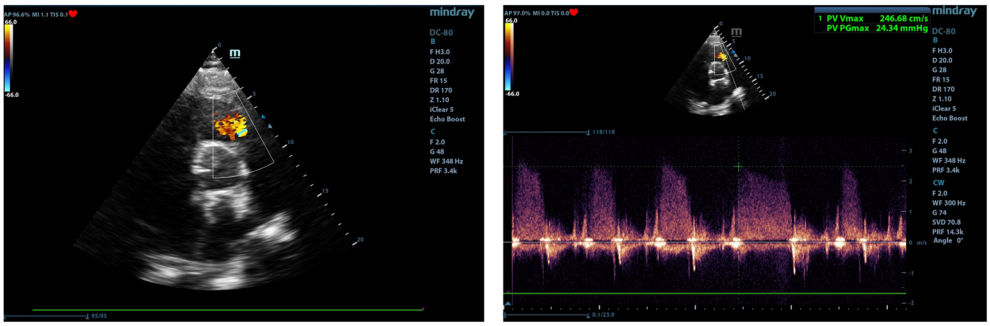

Figure nr. II.1.d.2. Moderate pulmonary regurgitation (left: turbulent diastolic flow; color Doppler; right: slow deceleration slope CW Doppler, both in parasternal short axis view) in a patient with severe pulmonary hypertension

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The echocardiographic images are from the own collection of dr. Florina Parv.

e. Valve prostheses (Adrian Apostol)

TTE is the method of choice for the initial assessment and monitoring of all cardiac valve prostheses.

- Systematic TTE 1 month after surgery (hemodynamically stabilized) to establish reference parameters for the prosthesis.
- Biological prostheses: TTE every year after the 5th year.
- Mechanical prostheses: systematic inspection every 3 to 5 years.

There are many types of prosthesis:

- 4 bioprosthesis profiles: stented, stentless, catheter-delivered and homograft;
- 3 types of mechanical valves: with tilting disc (60-80° angle), with 2 hemidisks (2 fins, 70-90° opening angle), with ball contained in a cage built with metal arches.

Valve prosthesis assessment

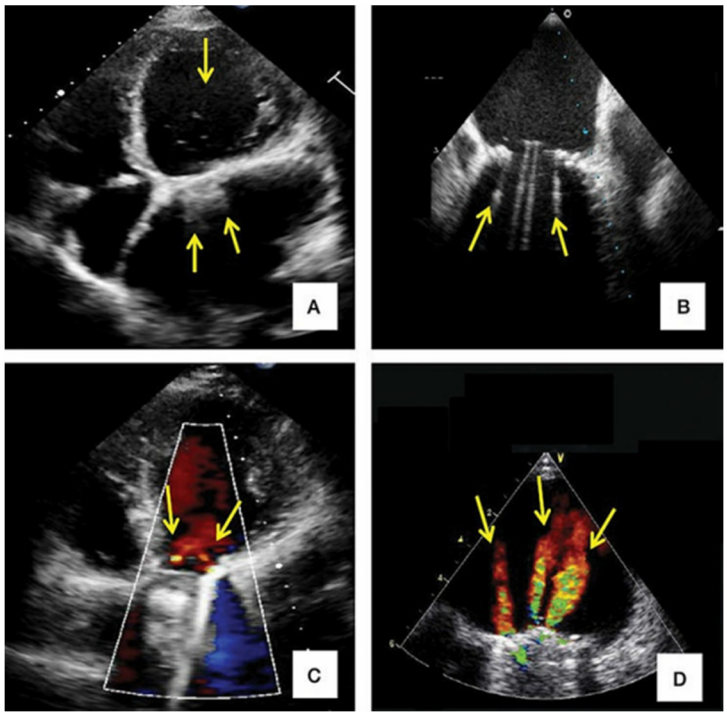
Aortic valve prosthesis

2D, TM

1. Mechanical prostheses
   - Difficult evaluation due to artifacts and shadow cones.
   - Hyperechoic prosthetic ring, passive systolodiastolic movement without paraprosthetic dehiscence.
2. Bioprostheses
   - Hyperechoic prosthetic ring without paraprosthetic dehiscence.
   - Cusp appearance: thin (2 mm), not thickened or calcified.
   - Cusp mobility: normal opening in 2D and TM, without restriction or prolapse.

Color Doppler

- Parasternal and apical 3- or 5-cavity incidences in TTE and short-axis aortic and long-axis incidences in TOE.
- Visualization of minimal physiological washout leaks, single or multiple, depending on the type of mechanical prosthesis (disk or fins)



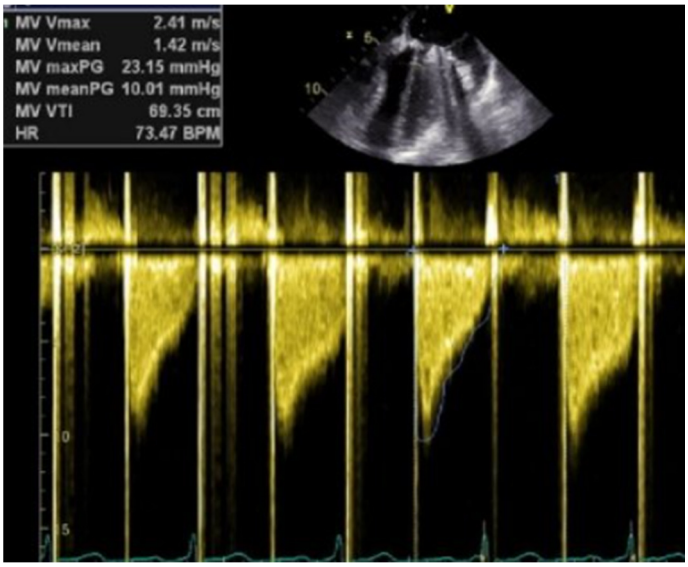
A, B- Posterior shadow cones of a mechanical mitral prosthesis

C- two physiological aortic leaks from a mechanical prosthesis

D-3 physiological leaks from a mechanical mitral prosthesis

Pulsed and continuous Doppler

- Vmax < 3 m/s;
- average gradient < 20 mmHg;
- triangular aortic flow pattern with early peak;
- SVE > 1,1 cm^2

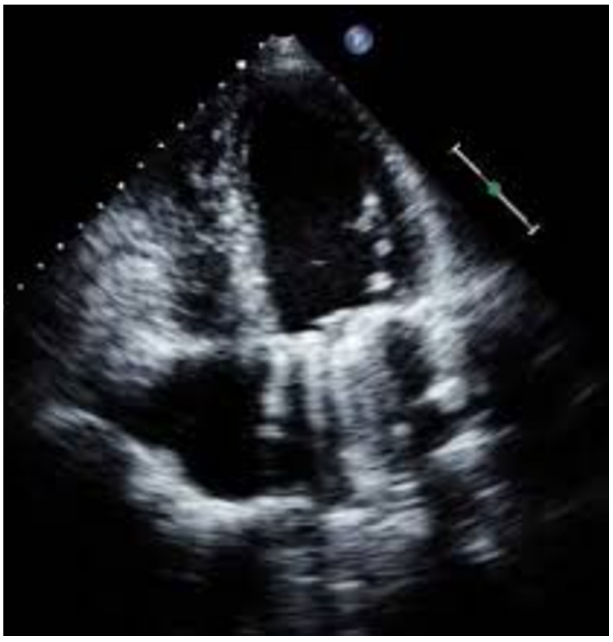


Mitral valve prosthesis

2D, TM

Mechanical prostheses

- Difficult 2D apical analysis due to artifacts (posterior shadow cones)



Pulsed and continuous Doppler

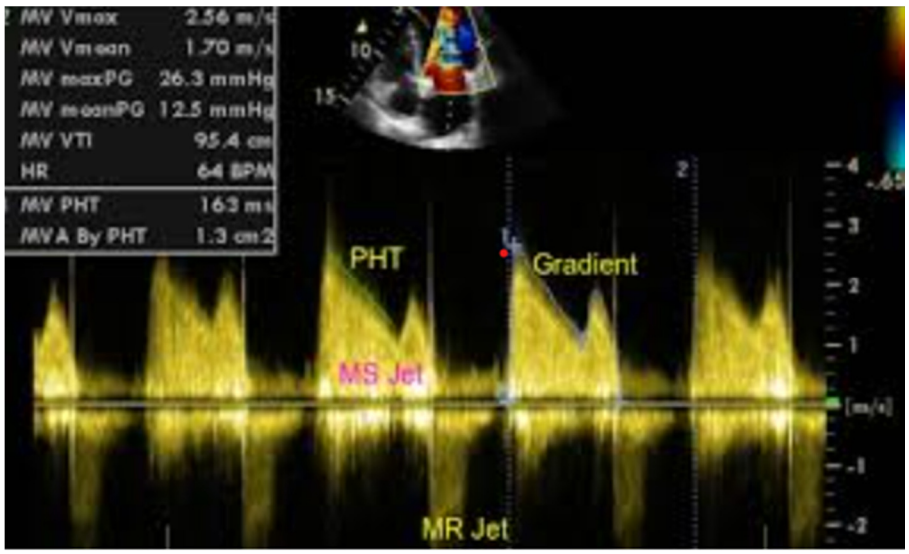
- TTE apical incidence for continuous Doppler.
- In the case of several mechanical prostheses, use the chronological analysis of closing and opening clicks to identify the different flows.

Hemodynamic assessment: record the hemodynamic parameters used in the stenosing native mitral valve study:

- Vmax E mitral;
- prosthetic mean gradient;
- PHT;
- Mitral SVE by continuity equation.

Normal values :

- Vmax E < 1,9 m/s;
- medium gradient ≤ 5 mmHg;
- PHT < 130 ms;
- SVE > 2 cm^2



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3. Sam Kaddoura Fourth Edition Echo made easy.
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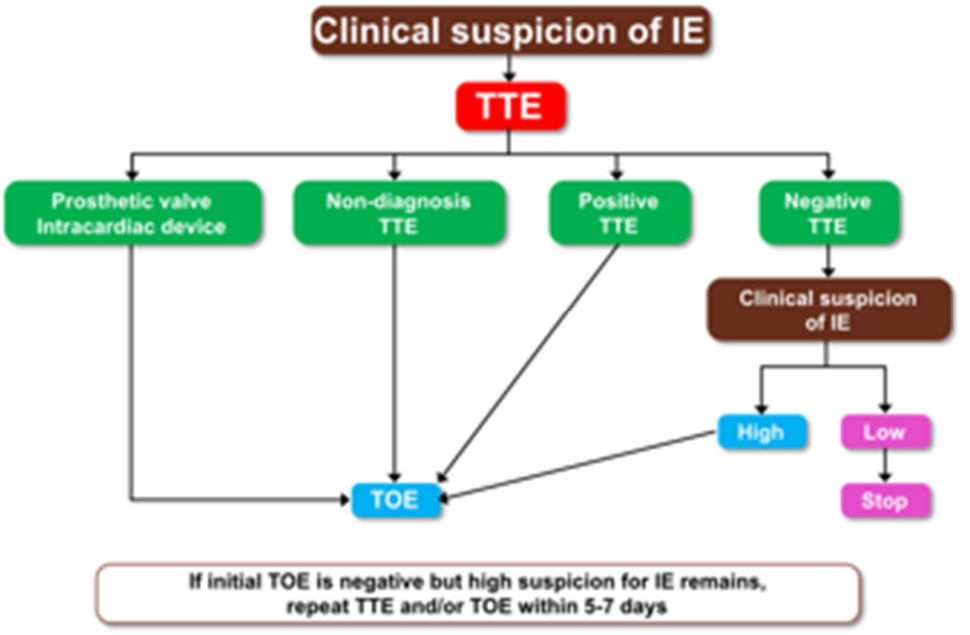
5. Ravi Rasalingam Manualul Washinton de Ecocardiografie

The echocardiographic images are from the own collection of dr. Adrian Apostol.

f. Infective endocarditis (Adrian Apostol)

Infective endocarditis (IE) is a difficult diagnosis, with often atypical presentations. It is also associated with high mortality and serious complications (hemodynamic, embolic and infectious). Echocardiography plays a central diagnostic and prognostic role in its management. Indeed, the search for lesions (vegetation, abscesses, recent valve leakage) is one of the two major diagnostic criteria of the Duke criteria.

The steps to be taken when IE is suspected are summarized in the figure (recommendations of the European Society of Cardiology). Transthoracic echocardiography (TTE) is the first line of defence.

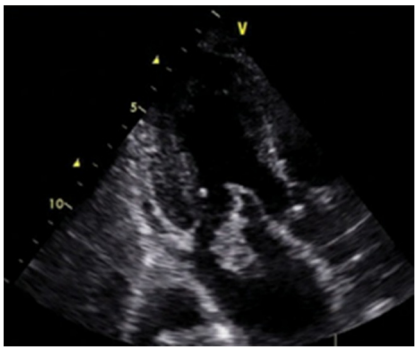


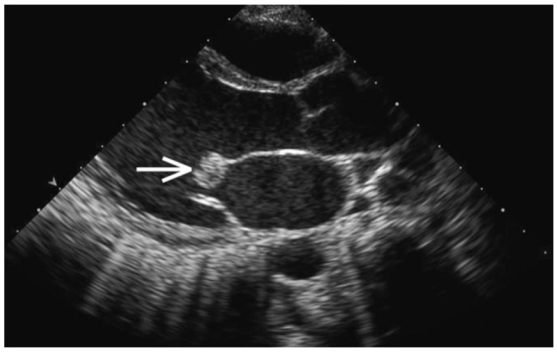
Echocardiography in IE

Recommendations	Class	Level
A. Diagnosis		
TTE is recommended as the first-line imaging modality in suspected IE.	I	B
TOE is recommended in all patients with clinical suspicion of IE and a negative or non diagnostic TTE.	I	B
TOE is recommended in patients with clinical suspicion of IE, in case of prosthetic valve or intracardiac device.	I	B
Repeat TTE/TOE within 5–7 days is recommended in case of initially negative examination when clinical suspicion of IE remains high.	I	C
Echocardiography should be considered in *Staphylococcus aureus* bacteraemia.	IIa	B
TOE should be considered in the majority of adult patients with suspected IE, even in cases with positive TTE.	IIa	C

Injuries:

6. Vegetation. This is a mass :
   - variable shape, vibratile, most often located on the free edge of a heart valve, but can be implanted on the endocardium or on intracardiac material (pacemaker lead or defibrillator)
   - whose mobility is often independent of the structure to which it is attached





It is essential to carry out a systematic study of each valve and each structure, and to give a clear description of the vegetation, specifying :

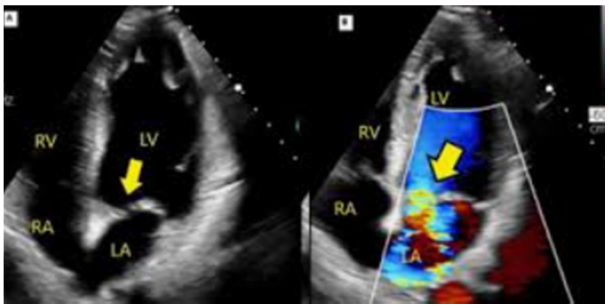
- size (measured in all incidences)
- mobility: not mobile, not very mobile, moderately mobile or very mobile
- location of vegetation

7. Abscesses and perivalvular lesions

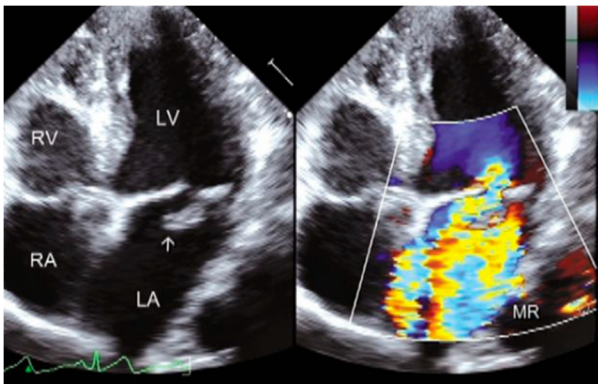
- The abscess is defined by a perivalvular cavity containing necrosis and purulent material that does not communicate with the lumen of the heart chambers. A neocavity then forms. Abscesses most often involve the aortic orifice.
- formation of a pseudoaneurysm (frequent, most often due to communication with the LV hunting chamber): pulsatile, circulating, echo-empty perivalvular cavity communicating with the adjacent cavity.
- formation of a fistula, which corresponds to a communication between two adjacent cavities visible on color Doppler.

8. Recent valve leakage (native or prosthetic)

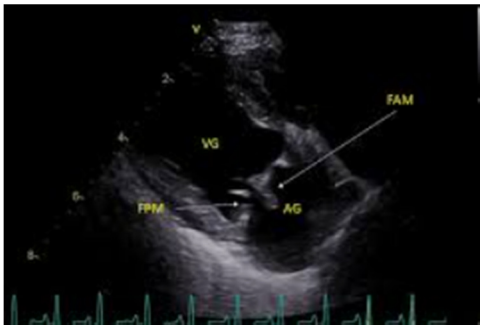
- valvular perforation



- tearing of a native valve or a heterograft cusp: capsizing of a valve fragment in the LV outflow tract during diastole (aortic valve) or in the left atrium during systole (mitral valve)



- mitral cord rupture



- prosthetic displacement

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The echocardiographic images are from the own collection of dr. Adrian Apostol.

2. CARDIOMYOPATHIES (FLORINA PÂRV)

a. Dilated cardiomyopathy (DCM)

Dilated cardiomyopathy is a primary myocardial pathology characterized by left ventricle dilatation and systolic LV dysfunction in the absence of explainable abnormal filling conditions (hypertension, valvular heart disease) or coronary artery disease. Echocardiographically, the defining criteria are LVEDD > 58 mm, $LVEDV_i \geq 75$ mL/m^2 in males and LVEDD > 52 mm, $LVEDV_i \geq 62$ mL/m^2 in females [pinto] and a LVEF <50%. In addition to the defining criteria, transthoracic echocardiography in DCM is also intended to evaluate the anatomy of the LV, right ventricular function, valve anatomy and hemodynamics, and left atrial anatomy, both for positive and differential diagnosis of conditions that may lead to dilatation of the VS, without signifying DCM.

The etiology of DCM is multifactorial. There are familial, inherited or overlapping forms. Familial forms are diagnosed in situations with at least two affected members of the same family (genetic testing is essential). The overlapped forms are those that are triggered by epigenetic or inherited factors (toxic, including alcohol, pregnancy, hypertension). The non-genetic causes are varied and they should be carefully investigated initially in any suspected case of DCM:

- viral (echovirus, Coxsackie virus, SARS-COV-2, enterovirus), bacterial (Lyme disease), parasitic, fungal infections
- toxic (alcohol, drugs, steroids, heavy metals)
- endocrinopathies (thyroid disorders, acromegaly, diabetes, Cushing's disease)

- nutritional deficiencies

- peripartum

- autoimmune diseases (collagenosis, celiac disease, inflammatory bowel diseases, myasthenia gravis)

- medications (chemotherapy, psychotropic medication)

In DCM, echocardiography is useful not only for positive diagnosis, to understand the pathophysiology, in the differential diagnosis, but also to follow up on the patients and monitoring the treatment.

1. *LV dilatation* is documented in 2D mode (parasternal long-axis, short-axis and apical 4-chamber views) by measuring the telediastolic and telesystolic diameters, sphericity index (longitudinal diameter/transverse diameter < 1.5), wall thickness (which is usually normal) and volumes (which are increased and should be related to body surface area).

2. Evaluation of the *systolic function of LV* by calculating the LVEF using the Simpson biplane method, the most correct method considering the geometric deformation of the LV. [figure nr. 2.a.1.]. Other parameters that assess the systolic performance of the LV are: Tei index of global myocardial performance, decrease in amplitude and duration of mitral valve leaflet closure, dP/dT ratio calculated on the mitral regurgitation envelope [figure], stroke volume, mitral annulus velocity in tissue Doppler (<7.5 cm/s), and speckle tracking strain. MAPSE (mitral annular plane systolic excursion) evaluates ventricular longitudinal function; a value <8 mm indicates reduced systolic performance.

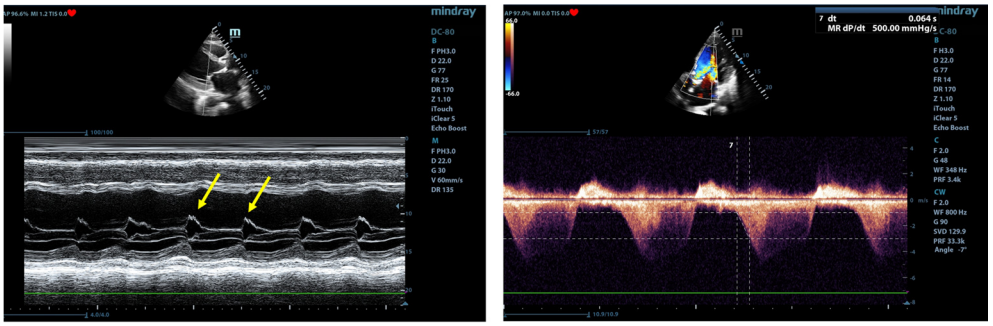

Figure nr. 2.a.1. Left: early closure of the mitral valves (arrows) (M-mode, parasternal long axis). Right: dP/dT ratio of 500 mmHg/s indicating severe systolic LV dysfunction in a case of idiopathic dilated cardiomyopathy (calculation is made on mitral regurgitation envelope between 1m/s and 3 m/s; apical 4-chambers view, color and CW Doppler)

Cardiac output (CO) can be calculated with formula: CO = stroke volume x heart rate, where stroke volume (SV) is $Area_{LVOT}$ x TVI_{LVOT} and $Area_{LVOT} = \pi \times D_{LVOT}^2/4$, in absence of an aortic regurgitation. D_{LVOT} is the diameter of LV outflow tract measured in systole in parasternal long axis. Cardiac output is influenced by the heart rate. Initially, the stroke volume is maintained by LV dilatation for compensatory purposes, despite reduced systolic function.

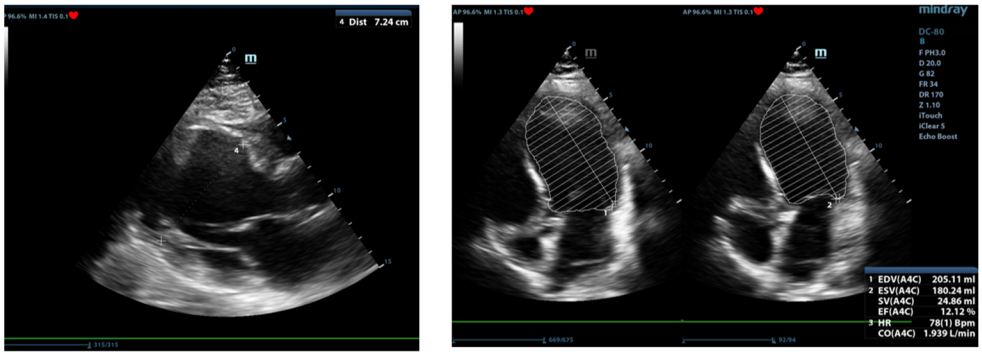

Figure nr. 2.a.2. Dilated LV and severe systolic dysfunction in a case of alcoholic dilated cardiomyopathy (left: EDD=7,24 cm; 2D, parasternal long axis view; right: increased teledyastolic volume EDV=205 ml, decreased stroke volume (24.8 ml) and cardiac output (1.9 L/min); 2D, apical 4-chambers view, Simpson's biplane method)

3. Evaluation of the *diastolic function of the LV* by all the parameters presented in Chap. Diastolic LV dysfunction. Diastolic dysfunction may be present in any pattern: type 1 delayed relaxation, type 2 pseudonormalization or type 3 restrictive. Negative prognosis is determined by a DT<150 ms, E/A ratio>2, increased E velocity>50 cm/s, increased E/E'ratio>20, low Vp <600 mmHg/s, E/Vp≥2.5 [figure nr 2.a.3.] [figure nr. 2.a.4.].

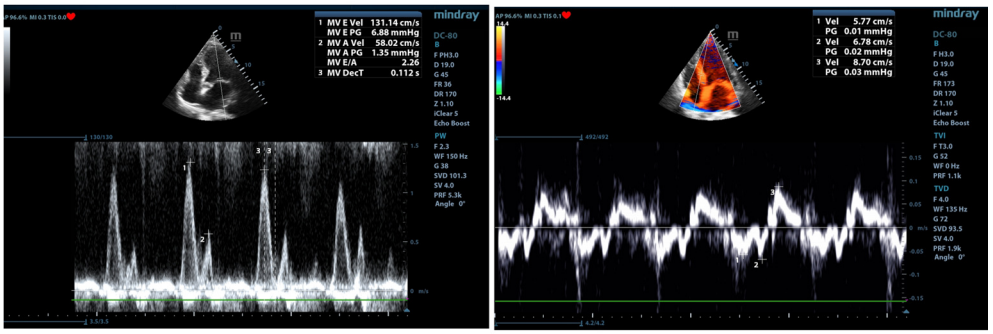

Figure nr. 2.a.3. Type 3 (restrictive pattern) of a diastolic dysfunction with increased E velocity of 131 cm/s, E/A ratio=2.26 and E/e's=22.7 (left: transmitral diastolic flow-PW Doppler; right: septal tissue Doppler, 4-chambers view)

4. *Evaluation of the right ventricular* function and size (see Chap. Evaluation of the RV) is always important, also in biventricular involvement. Fractional area change, TAPSE and RV myocardial performance index are determined.

5. Assessment of the presence and severity of the *consequences* of systolic dysfunction and left ventricular dilatation:

a) *mitral regurgitation*, usually functional due to mitral annulus dilatation, apical and lateral displacement of the papillary muscles. In 2D ultrasound, the mitral valve morphology, mitral annulus size, and in color and CW Doppler the severity of regurgitation are assessed. Usually the jet is central, and the color M-mode highlights the holosystolic character, with presystolic onset and accentuated proto- and telesystolic flow [figure nr. 2.a.4.].

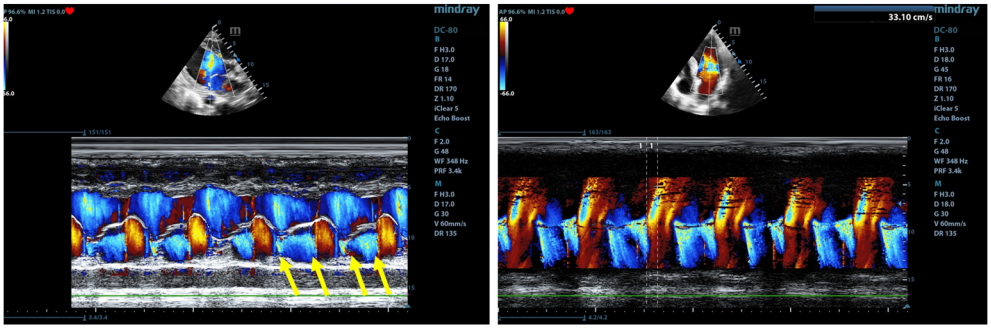

Figure nr. 2.a.4. M-color flow Doppler showing the blood flow during cardiac cycles in two cases with dilated cardiomyopathy (left: holosystolic mitral regurgitation flow accentuated in proto- and mezosystole (arrows); parasternal long axis; right: decreased propagation velocity of the diastolic mitral flow (Vp=33.1 cm/s) and holosystolic mitral regurgitation; 4-chambers view)

b) *pulmonary hypertension* is common in the presence of RV dilatation and functional tricuspid regurgitation; it is a negative prognostic factor.

c) the presence of *thrombi* in the LV, most commonly apical, in severe contractile dysfunction [figure nr. 2.a.5.].

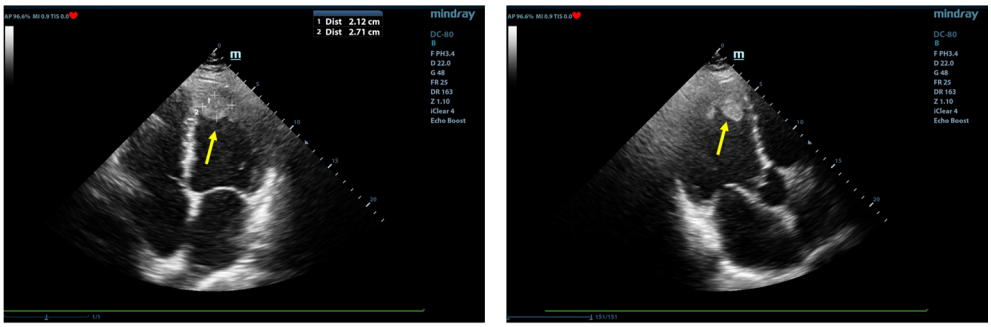

Figure nr. 2.a.5. Hyperechogenic apical LV thrombus (arrow) in a young man with familial dilated cardiomyopathy and LVEF of 20% (2D, 4 and 3-chambers view)

d) *ventricular dyssynchrony* is appreciated by rocking movement of LV apex, septal flash, or in tissue Doppler.

Negative prognostic factors detected by transthoracic ultrasound are:

- LVEDD $\geq$ 38 mm/m^2

- LEVF ≤ 35%
- Restrictive diastolic filling pattern
- RV dysfunction, TAPSE<14 mm
- LA dilatation
- Severe mitral regurgitation
- Maximum systolic transtricuspid peak systolic velocity >3 m/s

CLINICAL CASE

Male patient, 28 years old, without cardiovascular history, without cardiovascular risk factors presented dyspnea and progressive exertional fatigue, dry cough, predominantly nocturnal, symptoms installed for about 4 weeks; in addition, in recent days he presents paroxysmal nocturnal dyspnea. The first medical consultation was pneumologic because in the medical history, four months prior he had a respiratory virosis with mild symptoms, subfebrile, myalgias, headache, asthenia. The following tests were performed: a spirometry which showed normal values of lung volumes and capacities; usual biological tests, that were in normal ranges except for a minor inflammatory syndrome and a chest X-ray which revealed cardiomegaly, for which the patient was referred to cardiologic consultation. At the time of the consultation, the patient presented BMI=22.65 kg/m2; general condition relatively good, BP=110/75 mmHg, HR=98/min, rhythmic, tachycardic heart sounds, LV gallop, bilateral basal stasis rales, SaO2=95%. Left heart failure was suspected and ECG (sinus rhythm), echocardiography and additional biological investigations (inflammatory markers, immunologic and NTproBNP=8788 pg/mL) were performed. The first echocardiography showed dilated left chambers: dilated left ventricle with a telediastolic diameter of 38.37 mm/m2, a telediastolic volume of 95 mL/m2, dilated left atrium (LAVi=60 mL)[figure nr. 2.a.5.][figure nr. 2.a.6.].

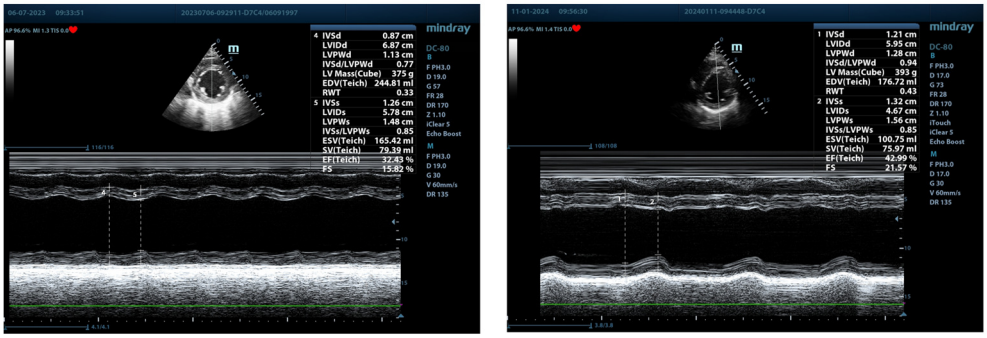

Figure nr. 2.a.5. Measurement of the left ventricle dimensions at first echocardiographyc evaluation (left) (LVEDD = 6.87 cm, LVESD = 5.78 cm, reduced contractility of the inferior wall) and after 6 months (right) (LVEDD = 5.95 cm, LVESD = 4.67 cm) (M-mode, parasternal short axis view, middle section)

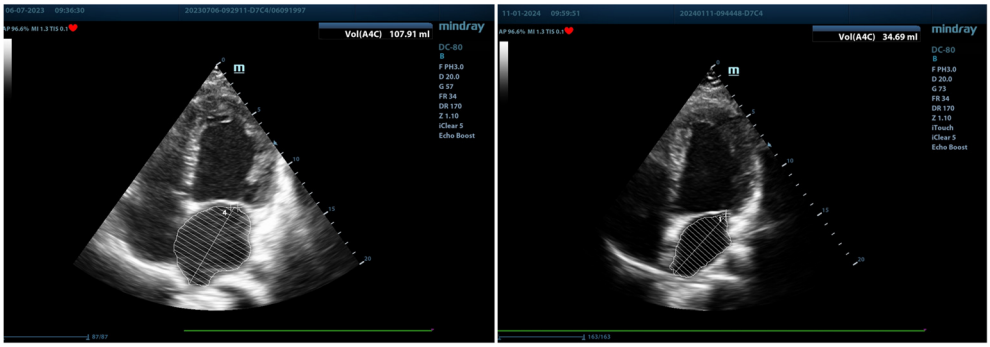

Figure nr. 2.a.6. Evaluation of the left atrial volume at first echocardiographyc evaluation (left: LAV=107 ml) and after 6 months (right: LAV=34.69 ml) (2D, 4-chambers view)

The patient had also a moderate holosystolic mitral regurgitation due to dilation of the left ventricle and consequently of the mitral annulus. Mitral leaflets had normal 2D echo aspect and normal mobility. Mitral regurgitation was evaluated in color and CW Doppler [figure nr. 2.a.7.]; the jet of the flow was central, the envelope had intense spectral signal and parabolic shape, vena contracta was 5 mm and regurgitant volume was 45 ml.

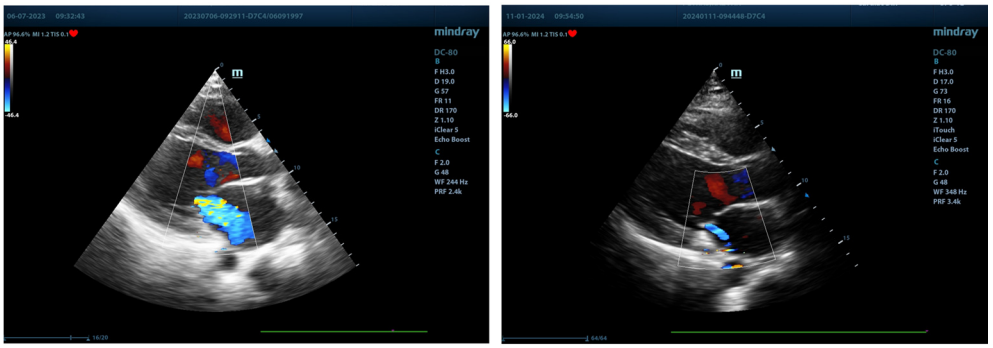

Figure nr. 2.a.7. Mitral regurgitation in presented case at first echocardiographyc evaluation (left: moderate MR) and after 6 months (mild MR) (color Doppler, parasternal long axis view)

Transthoracic echocardiography evaluation of the systolic LV performance showed a decreased LVEF (22.52%) in Simpson' biplane volume method and dP/dT under 800 mmHg/sec indicating severe systolic dysfunction [figure nr.2.a.10.][figure nr.2.a.8.]. The global performance of the LV was decreased with a global index Tei of 0.49, global moderate hypokinesia, and particulary severe hypokinesia of infero-lateral wall and reduced global longitudinal strain -15%.

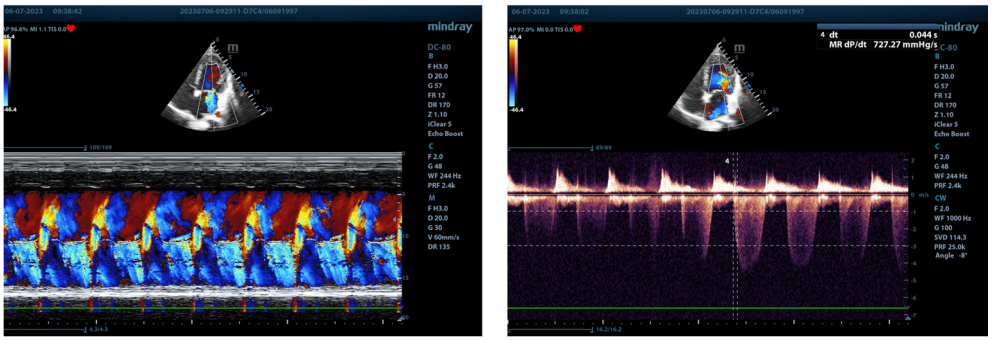

Figure nr. 2.a.8. Left: holosystolic mitral regurgitation (M-color mode, 4-chambers view); right: calculating dP/dT = 727 mmHg/sec (CW Doppler, 4-chambers view)

Assessment of diastolic function initially showed a severe diastolic dysfunction, restrictive type 3 with predominant E wave (105 cm/s), E/A ratio

>2, a very low transmitral diastolic deceleration time (80 ms), low septal e' 4 cm/s, increased E/E'septal ratio = 22, all these parameters indicating increased left intra-atrial filling pressures. [figure nr.2.a.9.]

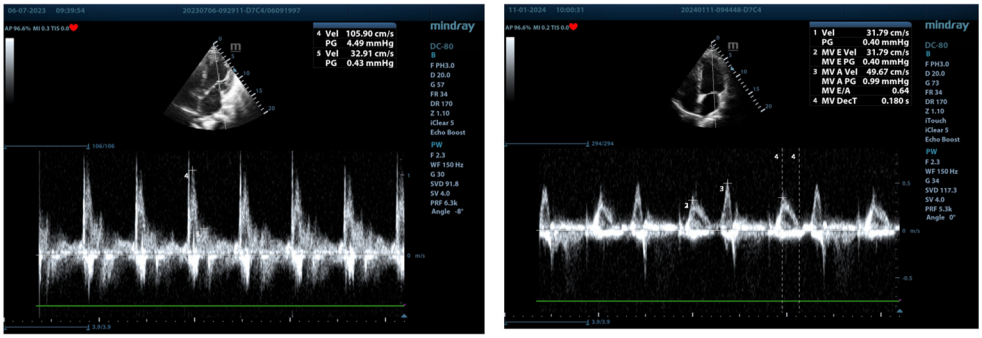

Figure nr. 2.a.9. Evaluation of the diastolic function in presented case at first echocardiographyc evaluation (left: type 3 dyastolic dysfunction pattern, E/A= 3.19) and after 6 months (right: type 1 dyastolic dysfunction pattern, E/A= 0.64) (PW Doppler, 4-chambers view)

Although stroke volume was reduced (SVi=21.22 ml/m2), cardiac output was maintained by compensatory tachycardia. [Figure nr.2.a.10.]

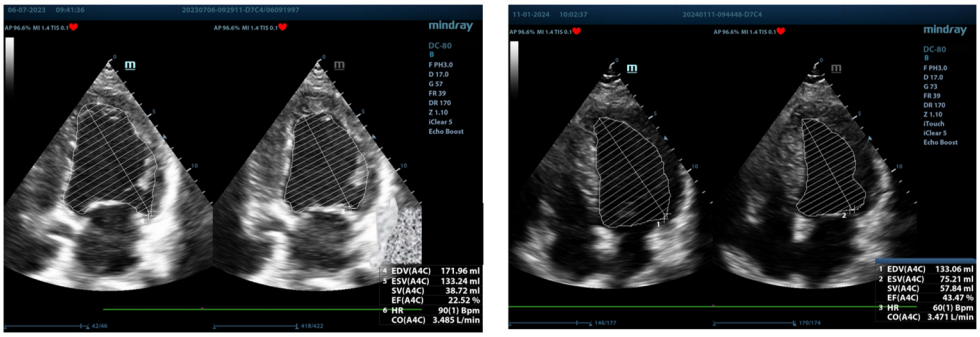

Figure nr. 2.a.10. Evaluation of the LV volumes and LVEF by Simpson method in presented case at first echocardiographyc evaluation (left: EF=22.52%, LVEDV=171 ml) and after 6 months (right: EF=46.47%, LVEDV=133 ml) (2D, 4-chambers view)

The patient had no dilatation of right heart chambers and no pulmonary hypertension.

Specific drug treatment for heart failure was gradually started with loop diuretics and mineralocorticoid receptor blocker, beta-blocker, ARNI, SGLT2 inhibitor, If channel blocker. At a later stage, cardiac MRI was performed and showed a specific pattern of post-viral inflammation in the inferolateral wall. The patient was diagnosed with post-viral dilated cardiomyopathy.

The patient was monitored clinically, biologically and echocardiographically, with adjustment of the drug treatment regimen according to the evolution. After 6 months, the clinical condition was good, the symptoms had completely disappeared, NT proBNP was 638 pg/mL. Echocardiographic evaluation at 6 months showed improved systolic performance with an increase in LVEF to 46%, slight reduction in LV size, respectively a 13% decrease in LVEDD and a 22% decrease in LV telediastolic volume, normalization of AS volume. The diastolic function had a very good evolution, thus, the diastolic dysfunction initially of restrictive type became of type 1 delayed relaxation. [figure nr. 2.a.9., 2.a.10.].

b. Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a genetic disease of the cardiac muscle caused by sarcomeric protein damage and characterized by symmetric or asymmetric left ventricular hypertrophy. It is necessary to exclude secondary causes leading to LV hypertrophy (hypertension, aortic stenosis, athlete's heart, metabolic diseases or infiltrative cardiomyopathies). It may or may not imply left ventricular outflow tract obstruction; rare cases of obstructive HCM in young people are at risk of sudden cardiac death.

Transthoracic echocardiography is important for:

a) quantifying the extent of *myocardial thickening* at septal level (IV septum/posterior wall ratio >1.3), of papillary muscles, apex, anterolateral or global wall (2D, parasternal long/short axis and apical chamber-view): thickness >1.5 cm, with LV telediastolic diameter within normal limits. [figure nr. 2.b.1.]. Inferolateral wall is not involved (differential diagnosis with hypertensive cardiopathy). Determination of the severity of interventricular septal hypertrophy and increased VS mass usually is made in M-mode.

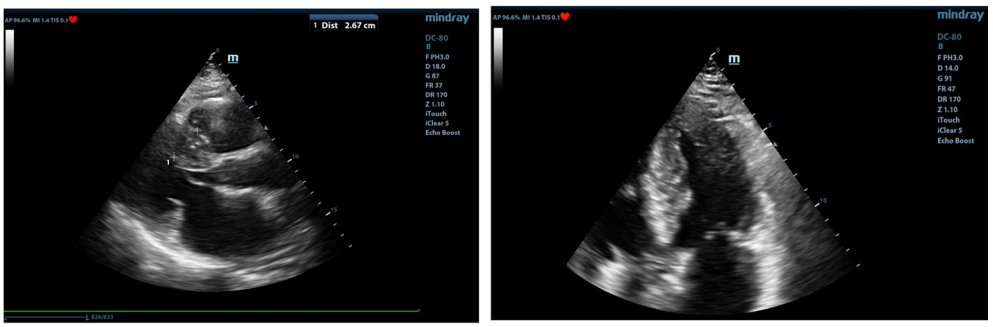

Figure nr. 2.b.1. Marked myocardial hypertrophy localised at the interventricular septum (2.67 cm) and LV apex (2D echo, parasternal long axis and 4-chamber view)

b) determination of the *presence and degree of obstruction in the outflow ejection tract of the LV* (LVOT), narrowing which occurs due to septal hypertrophy, displacement of the mitral apparatus and mitral leaflets elongation.

- in color Doppler: turbulent flow in LVOT [figure nr. 2.b.2.]

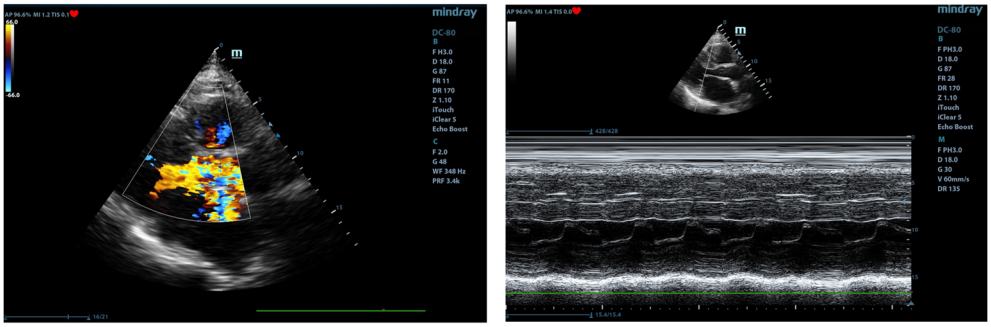

Figure nr. 2.b.2. Turbulent flow in LVOT (left: color Doppler) and contact of anterior mitral leaflet with interventricular septum (right: M-mode; parasternal long axis view)

- in 2D and M-mode: systolic anterior motion of the mitral anterior valve (SAM): can be mild (<10 mm distance of systolic contact with the septum), moderate or severe (prolonged septal contact, >30% of systolic)

- in CW Doppler the maximum velocity in the LVOT is measured, at the level of the maximum degree of obstruction of the dagger-shaped envelope, with a concave downward slope (due to the initial rapid increase) and with a late peak; then the maximum gradient is assessed, according to Bernoulli's law ($\Delta P=4v2$), a gradient $\geq$ 30 mmHg at rest in LVOT being significant. [figure nr. II.b.3.]. The obstruction is dynamic during systole, accentuated meso-telesystolic and can be amplified by Valsalva maneuver, nitroglycerin administration, in orthostatism, or by hypovolemia.

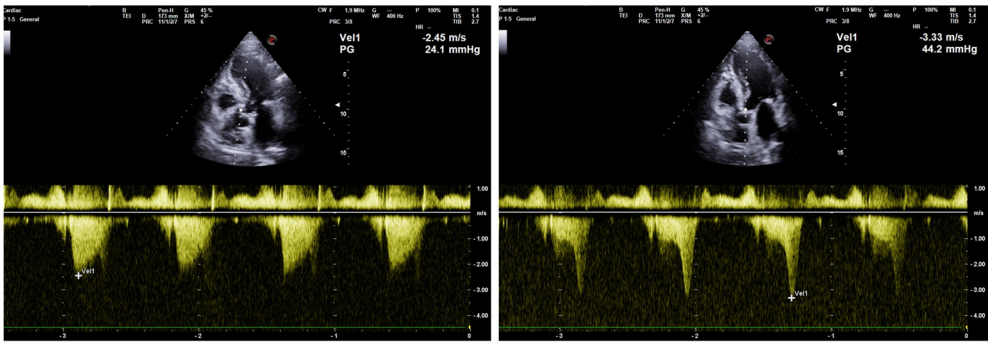

Figure nr. II.b.3. CW Doppler in a patient with obstructive HCM: left image-recording of transvalvular aortic flow with a maximum gradient of 24.1 mmHg; right image-recording of flow in LV outflow tract with a maximum gradient of 44.2 mmHg

- in PW Doppler the site of the obstruction is determined.

c) evaluation of *mitral regurgitation* which is caused by the inability to achieve complete coaptation the mitral valve leaflets, due to the contact of the anterior leaflet with the septum and which is particularly posteriorly oriented; it allows the determination of the gradient in LVOT using formula: $(4xV_{MR}^2 + LAP) – SPB$

where:

V_{MR}=mitral regurgitation maximum velocity

LAP=left atrial pressure

SPB=systolic blood pressure

e) assessment of *diastolic dysfunction* which is present in all cases; if the pattern is normal, the diastolic impairment can be demonstrated by the Valsalva maneuver. It is always necessary to also evaluate by tissue Doppler, which indicates low velocities and in the pulmonary veins, too (increased Ar duration and velocity). [Figure nr. II.b.4.]

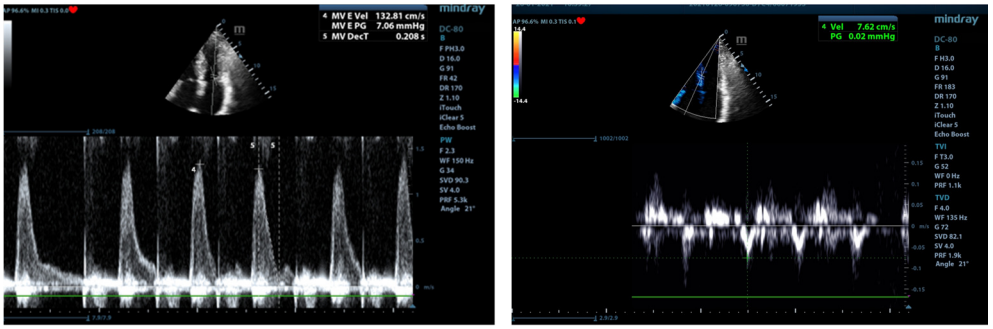

Figure nr. II.b.4. Restrictive diastolic filling patern (E=132 cm/s; e'$_s$=7.62 cm/s; E/e'=17.36) in a patient with HCM and atrial fibrillation

f) assessment of the *dimensions of LA* (usually dilated), *VS* (undilated; later becomes enlarged) with normal or supranormal LVEF [figure nr. II.b.5.]

g) longitudinal dysfunction (low MAPSE)

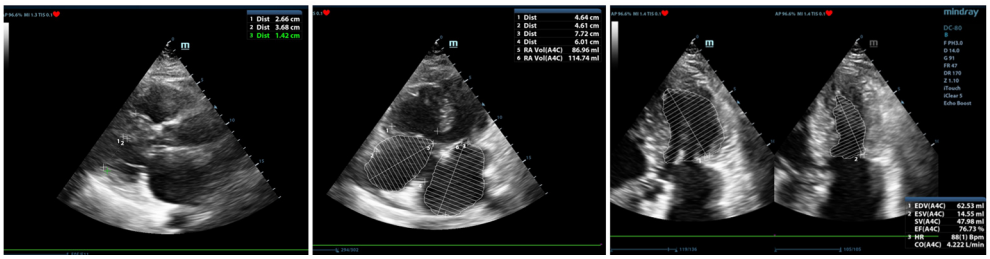

Figure nr. II.b.5. Nondilated LV with EDD=3.68 cm (left; parasternal long axis), dilated LA (114 ml) (middle; apical 4-chamber 2D) and supranormal LVEF=76% (left; apical 4-chamber view)

In elderly patients, the LV hypertrophy is frequently localized in the anterior or posterior basal septum, and the mitral regurgitant jet is central when associated with calcifications of the mitral annulus. A similar envelope of obstruction flow in LVOT and a midventricular gradient may be identified in patients with hypovolemia and positive inotropic support.

CLINICAL CASE

The 68-year-old female patient, without significant cardiac antecedents, presented for about 6 months progressive exertional dyspnea, culminating with dyspnea on minimal exertion and nocturnal dyspnea and rapid palpitations in the last days. The objective examination showed BMI = 30.47 kg/m2, BSA = 1.813 m2, BP = 110/70 mmHg, HR = 110 bpm, tachycardic and arrhythmic heart sounds, mitral systolic murmur, pulmonary stasis, abolished right basal vesicular murmur. The resting electrocardiogram showed atrial fibrillation and LV hypertrophy and the chest X-ray showed elongation of the lower left arch and moderate right pleural fluid. Biological investigations showed increased NT proBNP, mild nitrogen retention, normal hemogram, normal liver and thyroid function.

Echocardiography was performed and showed normal morphological valves with normal mobility, marked hypertrophy of the interventricular septum (2.6-2.7 cm), a less hypertrophied posterior wall and a LV with normal intracavitary dimensions (EDD=3.68 cm), and increased mass (380 g, $LVmass_i$ = 209 g). [figure nr. II.b.6.]

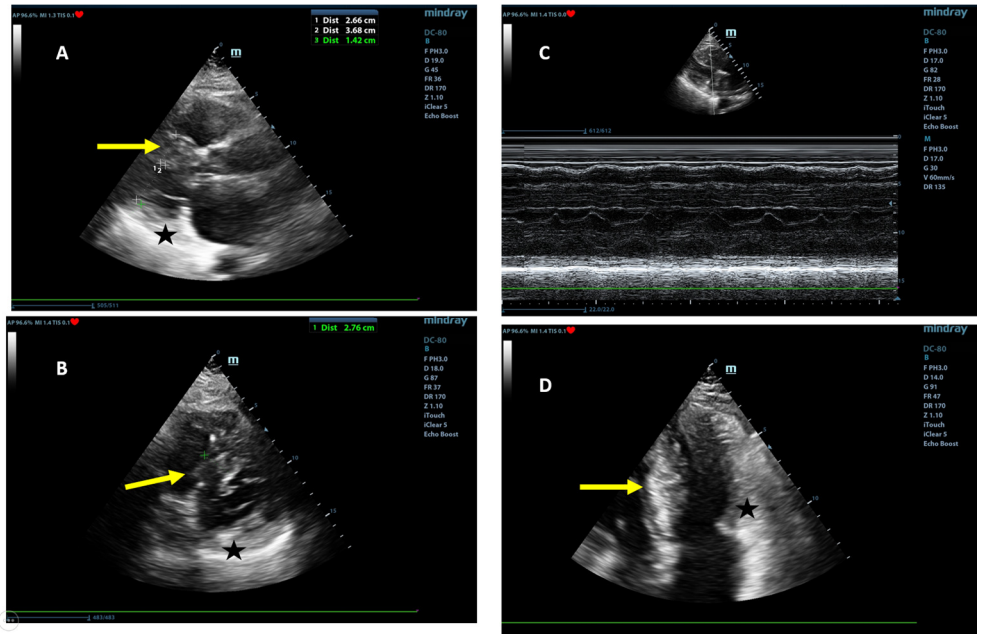

Figure nr. II.b.6. Important hypertrophy of the interventricular septum (2.66-2.76 cm, arrow), comparing with posterior, inferior and lateral LV walls (star) (images A: parasternal long axis, B: parasternal short axis, D: 4-chambers views) and lack of the SAM sign suggesting lack of obstruction (C: M-mode, parasternal long axis view)

Assessment of systolic LV function showed a supranormal LVEF (76%), small ventricular volumes, and assessment of diastolic function showed significant dysfunction, with an E/E'ratio>13. [figure nr. II.b.7.]

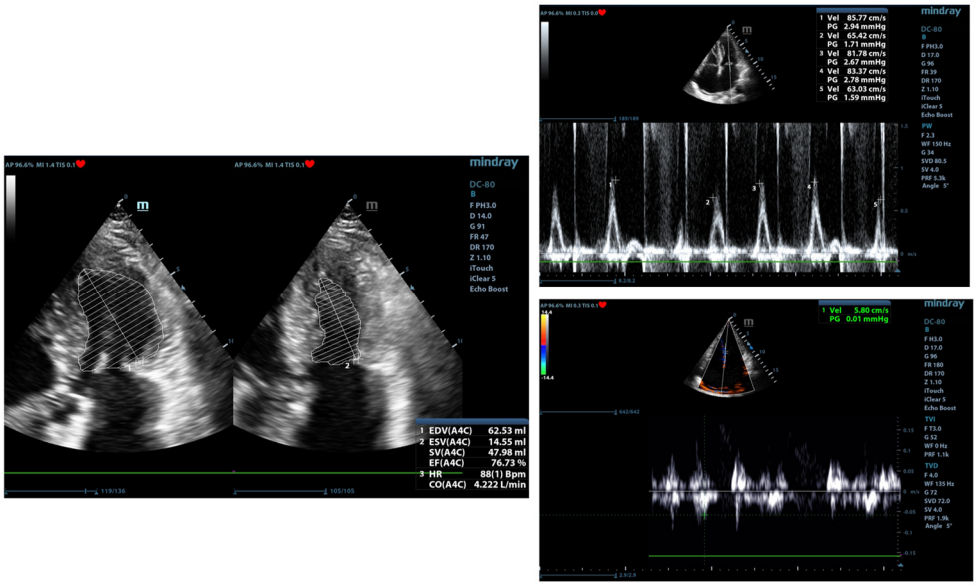

Figure nr. II.b.7. Left: supranormal LV systolic function (76%) and normal to small LV volumes (LVEDV=62 ml; LVESV=14 ml) (2D, 4-chambers view); Right: diastolic dysfunction in case presented (average E=75 cm/s, average e'_s=5; E/E'=15) (CW and tissue Doppler, 4-chambers view).

We looked for signs of potentially associated LVOT obstruction, but the patient showed no systolic anterior motion of the anterior mitral valve, and the measured LVOT gradient was 22 mmHg, without change on Valsalva maneuver. [figure nr. II.b.6.][figure nr. II.b.8]

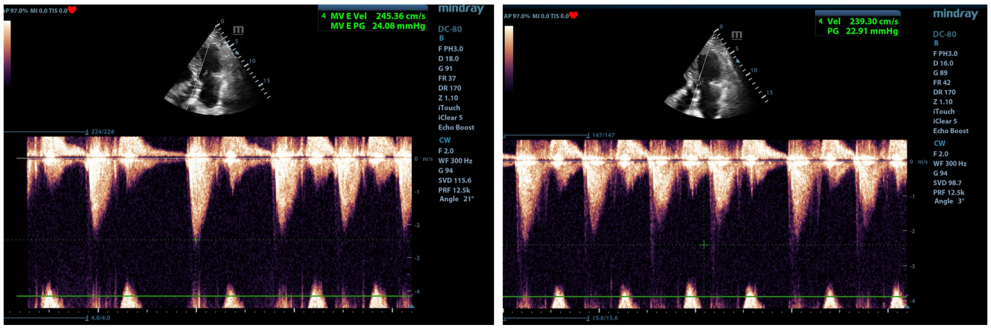

Figure nr. II.b.8. Evaluation of maximum gradient at transaortic valve level 24 mmHg (left) and at LVOT level 22 mmHg (right) (CW Doppler, apical 5-chambers view)

Associated there was also moderate mitral regurgitation with central jet, a dilated left atrium and mild tricuspid regurgitation, but without secondary pulmonary hypertension. [figure nr. II.b.9]

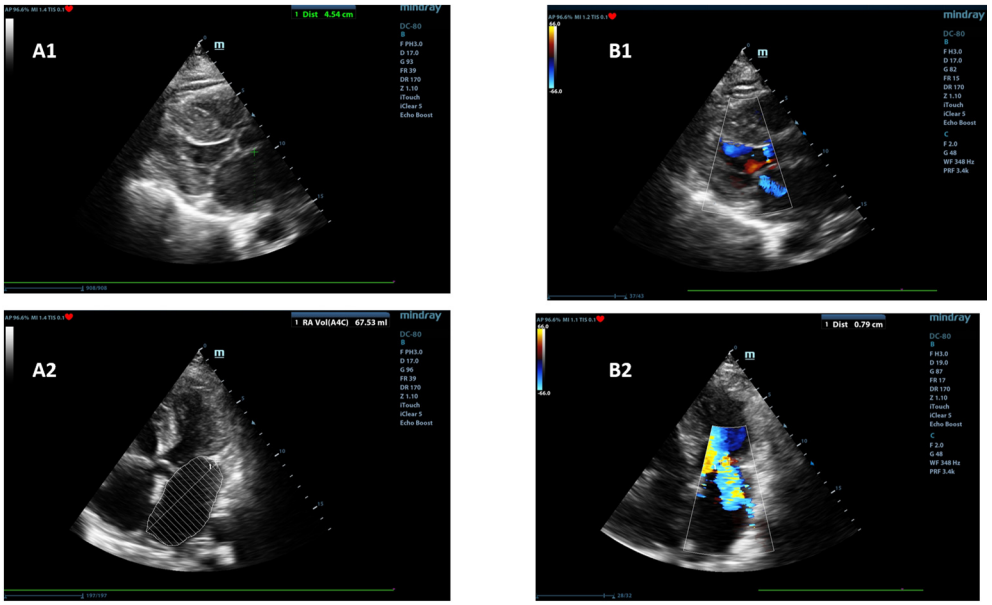

Figure nr. II.b.9. Left: dilated left atrium with antero-posterior diameter of 4.54 cm (A1: 2D-parasternal long axis view), LAVi=37 ml/m^2 (B2: 2 D, 4-chambers view modified for LA). Right: moderate mitral regurgitation (B1: color Doppler, parasternal long axis view, B2: color Doppler, 4 chambers view)

In the meantime, we discovered that the patient presented a small patent foramen ovale with left-to-right shunt, without hemodynamic significance. [figure nr. II.b.10]

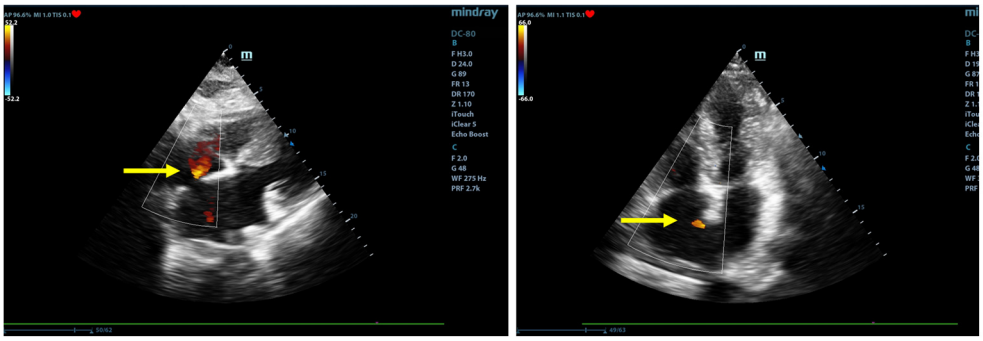

Figure nr. II.b.10. Patent foramen ovale (arrow) in presented case (color Doppler; left: subxiphoid view, right: apical 4-chambers view)

The patient was diagnosed with non-obstructive hypertrophic cardiomyopathy, predominantly septal, moderate mitral regurgitation, atrial fibrillation and NYHA III failure. She underwent right thoracocentesis, the pleural fluid was biochemical transudate and she was treated with high-dose beta-blocker, anticoagulant, diuretics, SGLT2 inhibitor, with a rapid favorable evolution.

c. Restrictive cardiomyopathy

Restrictive cardiomyopathy is a disease of the myocardium (uni- or biventricular) characterized by the abnormality of the diastolic function in the conditions of a normal systolic function. The primary consequence is the increase in intraventricular end-diastolic pressures and dilation of the atria, but with normal ventricular volumes.

According to the etiology, restrictive cardiomyopathy can be:

- non-infiltrative (idiopathic)
- infiltrative (amyloidosis)
- secondary to some storage diseases (hemochromatosis, Fabry disease)
- secondary to pathologies that affect the endomyocardium (endomyocardial fibrosis, eosinophilic syndrome, carcinoid syndrome, scleroderma, post-irradiation, drug toxicity)

The transthoracic echocardiography shows alteration of the diastolic function (any of the three patterns can be found, but most of the times it is discovered in stage 3 of the restrictive type), a normal systolic function and the consequences of the increase of intracavity filling pressures.

In the 2D and M mode are observed:

- normal intraventricular diameters
- dilatation of LA and RA [figure nr. 2.c.1.]

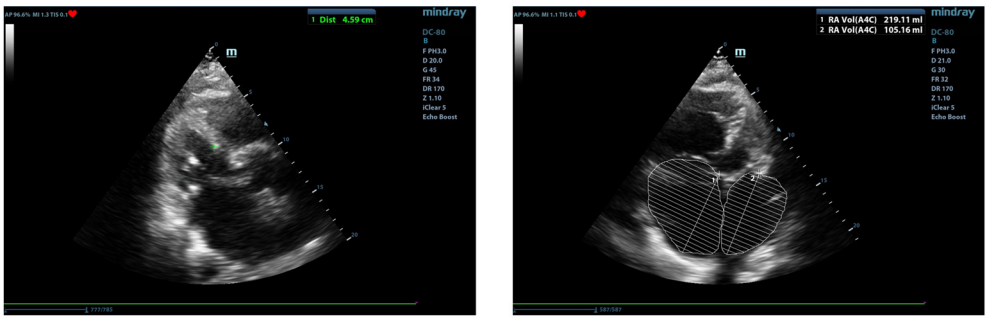

Figure nr. 2.c.1. Dilation of both left (105 ml) and right atrium (219 ml), nondilated left ventricle in a case with restrictive cardiomyopathy (left 2D, parasternal long axis view; right 2D, apical 4-chambers view)

- dilation of the IVC

- normal LVEF

- reduced velocity of blood flow propagation (Vp <45 cm/s (expression of myocardial damage).[figure nr. 2.c.2]

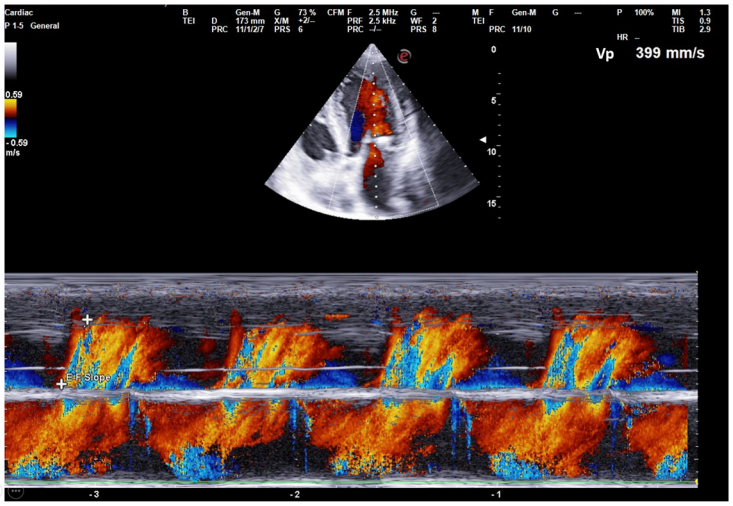

Figure nr. 2.c.2. Diastolic dysfunction indicated by a decreased velocity of blood flow propagation (Vp=39 cm/s) (color M-mode, apical 4-chambers view)

In Doppler ultrasound are noticed:

- at the mitral transvalvular level, the restrictive profile of diastolic dysfunction with E/A ratio>1, E wave>1m/s, lower DT <160 ms, low IVRT <60 ms is most frequently encountered.

- in tissue Doppler (4-chambers view at septal or lateral corner of mitral annulus), the relaxation deficit is indicated by a reduced e-wave (<8cm/s), E/E'average ratio ≥13. [figure nr. 2.c.3.]. Restrictive filling pattern is not specific only to restrictive cardiomyopathy; it can be noticed also in other cardiomyopathies (dilated, hypertrophic) or hypertensive cardiopathy in final stages.

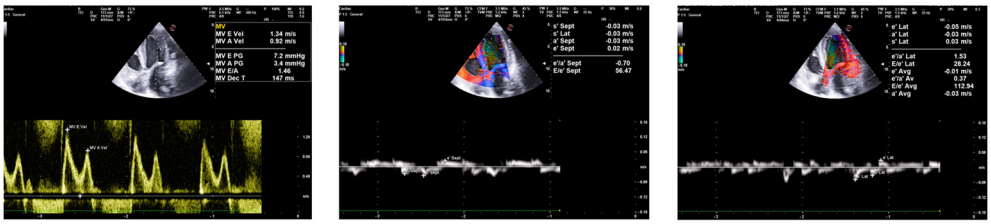

Figure nr. 2.c.3. Diastolic dysfunction in a case with restrictive cardiomyopathy due to amyloidosis. Left: E/A ratio 1.46, E=1.34 m/s, DT=147 ms (PW Doppler, apical 4-chambers view). Middle and right: reduced velocities of myocardium movement e's=3 cm/s, e'l=5 cm/s, E/E'av=56 (tissue Doppler, apical 4-chambers view)

- a low S/D ratio (<0.5), the increase in the amplitude and duration of the Ar are recorded in the pulmonary veins

- at the transtricuspid level there are no respiratory variations as in constrictive pericarditis, the appearance of the diastolic function being similar to that at the transmitral level [figure nr. 2.c.4.]

- in the suprahepatic veins, an increased *a* wave and a systolic velocity much lower than diastolic one

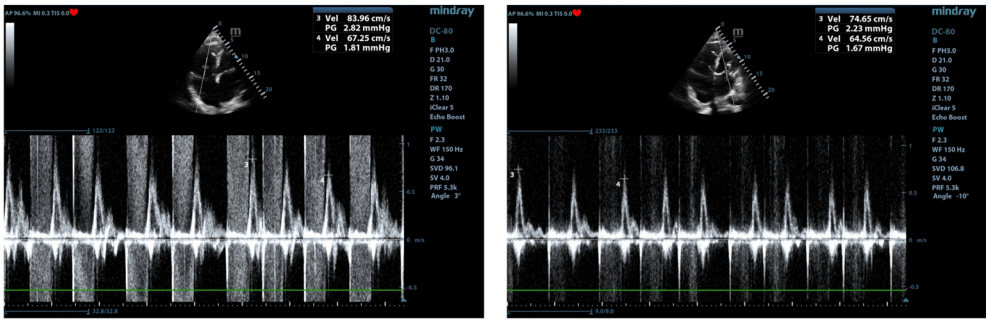

Figure nr. 2.c.4.Transtricuspid (left) and transmitral flow (right) with respiratory variations <25% in a case with restrictive cardiopmyopathy (PW Doppler, 4-chambers view)

In amyloidosis in particular, the myocardium has a shiny granular appearance, the thickening of the valves and of the interatrial septum are associated. [figure nr. 2.c.5.]

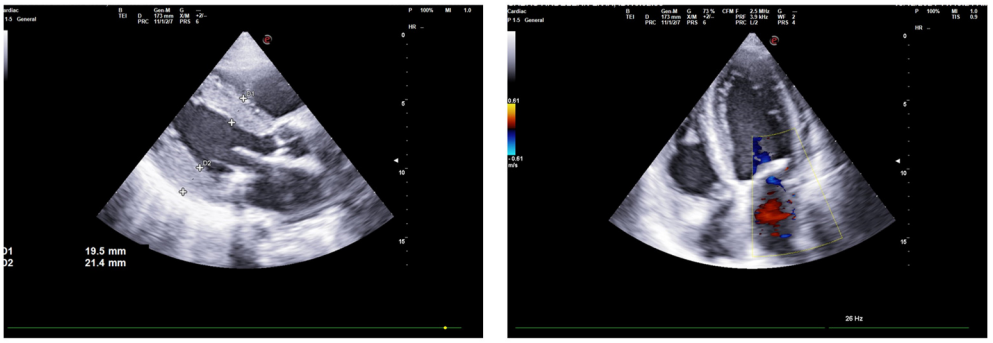

Figure nr. 2.c.5. Concentric left ventricle hypertrophy, normal intraventricular diameters, thick mitral valves and granular appearance of the myocardium in a case with restrictive cardiomyopathy due to amyloidosis (left: parasternal long axis view; right: apical 4-chambers view)

In cardiomyopathy, due to **non-compaction of the LV** (which is a particular genetic condition, sometimes familial) there is a compact myocardial layer adjacent to the endocardium and a non-compact layer adjacent to the endocardium in a ratio of 1:2, the non-compaction changes, respectively the trabeculation and the recesses between the trabeculae at the LV level being more visible at the apex and mid-ventricular level. [figure nr. 2.c.6.]

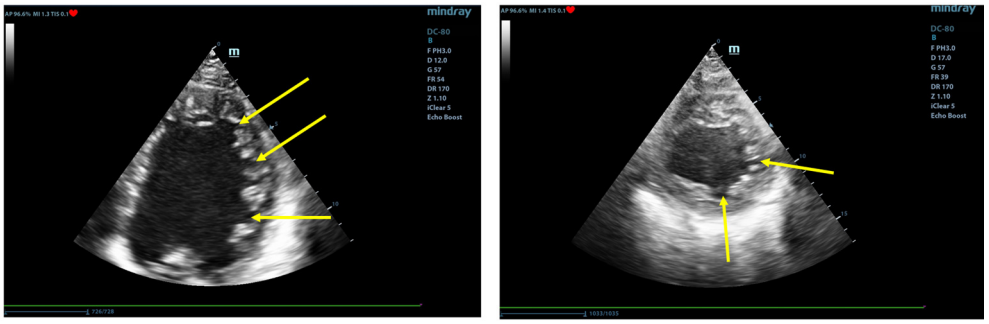

Figure nr. 2.c.6. Deep recesses (arrows) between left ventricle trabeculae visible very clearly at apical and lateral wall (left image: 2D, magnified parasternal 4-chambers view; right image: 2D, parasternal short axis view at apical level) in a case with non-compaction cardiomyopathy

In color Doppler the color is noticed deeply into intertrabecular recesses.[figure nr. 2.c.7.].

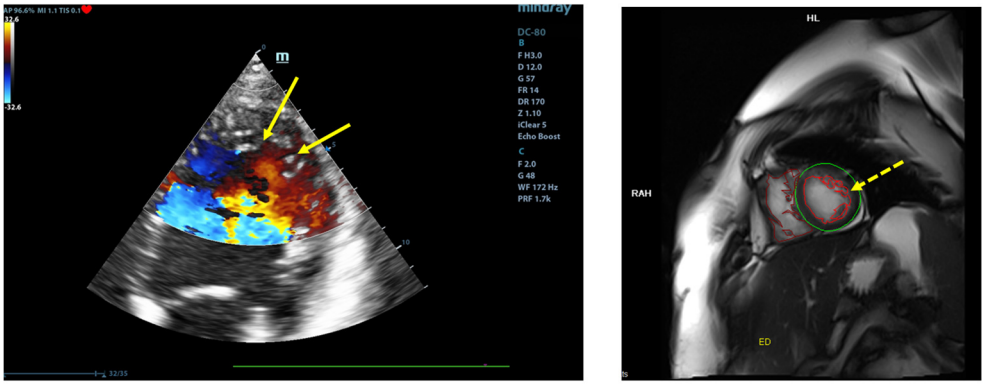

Figure nr. 2.c.7. Color Doppler useful to distinguish recesses between prominent trabeculations (left image, continuous arrows) in a case with non-compaction cardiomyopathy confirmed by cardiac MRI (right image, dotted arrow)

In most cases, the systolic dysfunction, LVEF globally reduced and global hypokinesia are found, and it is often necessary to perform cardiac MRI for the positive diagnosis [figure nr. 2.c.7].

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The echocardiographic images are from the own collection of dr. Florina Parv.

3. EVALUATION OF MYOCARDIAL ISCHEMIA (FLORINA PÂRV)

Myocardial ischemia is assessed by several complimentary diagnostic methods (ECG, stress test, echocardiography, scintigraphy, coronarography, cardiac MRI, IVUS), each one having its indication, contraindications and limitations. Transthoracic echocardiography is the most accessible one and is important both in emergency and common situations.

In ischemia there are abnormalities of myocardial kinetics in the area where blood is supplied by the affected coronary artery and directly visualized by endocardial motion and parietal systolic thickening. TTE cannot differentiate between acute or chronic kinetic abnormalities, but its advantage is that it can evaluate the consequences of myocardial ischemia: mitral regurgitation, mechanical complication of myocardial infarction (interventricular septum or wall rupture, remodeling of LV myocardium, systolic and diastolic dysfunction, intraventricular thrombosis, pericarditis).

Normally, at the same time with systolic myocardium contraction, there is a systolic thickening and a displacement of the endocardium with 4-10 mm. There are three types of motion abnormalities:

- hypokinesia, characterised by a reduction in systolic thickening and endocardial displacement < 4 mm in systole
- dyskinesia represents paradoxical movement of endocardium outward during the systole
- akinesia, absence of endocardium motion and systolic thickening.

In aneurism, both systolic and diastolic outer displacement of endocardium are present, in a region of dilated and very thinned myocardium.

It is important to differentiate other situations when kinetic abnormalities exist, but not having an ischemia etiology, like bundle brunch block, RV pacing, myocarditis, Takotsubo cardiomyopathy, dilated

cardiomyopathy. Medical history, symptomatology, ECG, laboratory findings are very useful and decisive.

For echo-analysing every myocardial segment a 17-segment anatomical model (former 16-segment model plus apex) that correspond to arterial distribution was made.[figure 3.1.]. The anterior, anterolateral, anteroseptal and apical segments correspond to the left anterior descending artery, the lateral segments correspond to circumflex artery area and the inferolateral wall to posterior descending artery (which has its origin into the right coronary artery in 80% of population). The right ventricle walls are irrigated by right coronary artery and its branches.

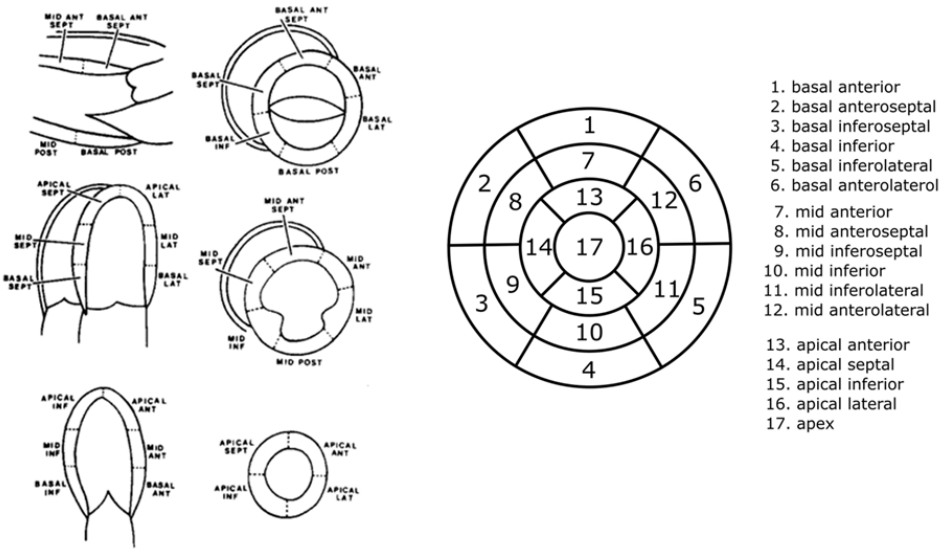

Figure nr. 3.1. Segmentation of left ventricle myocardium for analysing regional wall abnormalities of kinetics (adapted)

Myocardial ischemia can be evaluated by assessing:

- myocardial velocities in tissue Doppler: they are reduced compared to normal values of the normal ones of the same segment

- myocardial strain (either longitudinal or global) in apical 2,3 or 4-chamber view; pathological strain is characterised by reducing of systolic deformation, paradoxical and postsystolic deformation. It is transitory (during ischemia) and depends on ischemia localisation, presence of collaterals and the degree of mural involvement. [figure nr. 3.2.]

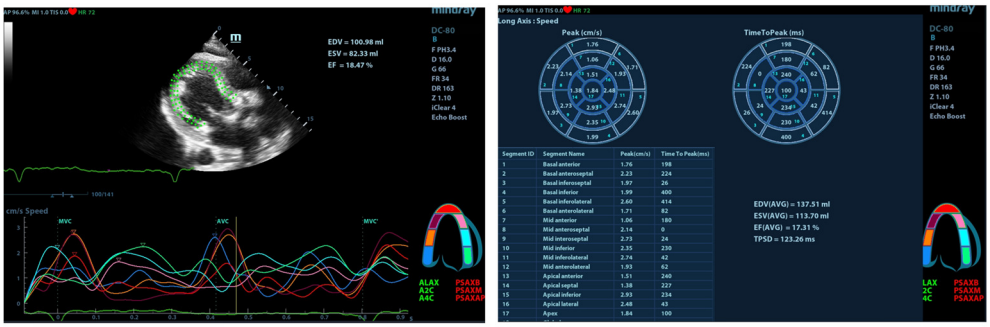

Figure nr. 3.2. Evaluation of myocardial ischemia in the 17-th segments using strain method

- coronary flow in modified echo windows, by recording a turbulence flow (in color Doppler), intracoronary velocities and flow pattern (anterograde or retrograde distal to a total occlusion) (in PW Doppler).

In myocardial ischemia there can exist also a diastolic dysfunction pattern, irrespective of a normal LVEF.

Dobutamine stress echocardiography is a noninvasive, cost-effective method, not only of diagnostic and localisation of myocardial ischemia, but also for prognostic, myocardial viability, preoperative risk and coronary spasm evaluation. It is indicated also in patients that cannot perform an exercise test (with locomotor difficulties of different etiologies, obese, left bundle branch) and is contraindicated in acute/severe cardiac pathologies (severe aortic stenosis, acute cardiac syndromes, pulmonary embolism, pericarditis,

myocarditis, uncontrolled hypertension, high degree AV block, hypertrophic cardiomyopathy, decompensated heart failure), severe dyselectrolytemia. Besides clinical, electrocardiographic and hemodynamic (BP, HR) evaluation, myocardial ischemia is appreciated by:

- visualization of hypokinesia during and after stress
- absence of normal hyperkinesia at effort

In interpreting the severity degree of ischemia, it is important to take into account the moment of ischemia appearance during the test (stress threshold), recovery time of myocardial wall kinetics. Other echo parameters can indirectly suggest myocardial ischemia: dilation of LV during effort, decreasing of EF, aggravation of diastolic LV disfunction, mitral regurgitation, RV contractility dysfunction.

ETT is very useful to evaluate acute and late consequences of myocardial infarction. In acute myocardial infarction, besides ischemic signs like hypokinesia (decreasing of systolic endocardium motion and thickening of myocardium), there also is hyperkinesia of other nonischemic segments that have a normal thickness. In chronic myocardial infarction a scar can be identified by thinning of involved myocardial segment wall, absence of hyperkinesia, presence of akinesia and sometimes a paradoxical motion.

Echocardiography has an important role in diagnosis of the RV myocardial infarction because anatomical variations of its own vascularization, atypical clinical signs and few electrocardiographic data. Besides wall motion abnormality of the free RV wall, there can be observed dilation of RV, reduced TAPSE<12 cm/s or other secondary signs (paradoxical sept motion, tricuspid regurgitation, bowing of the interatrial septum into the left atrium, dilation of inferior vena cava with reduced collapse during respiratory movements)[3].

In the same time echocardiography is important in evaluating the hemodynamic status (hypovolemia, LV and RV contractility) and in diagnostic of the complications of myocardial infarction:

Acute mechanical complications

- Acute mitral regurgitation (MR), sign of severe myocardial infarction, multivessel involvement and with negative prognostic, can be produced by:
  - rupture of a papillary muscle (posteromedial) during the process of necrosis due to the infarction of the adjacent myocardium and in this situation, MR is irreversible
  - intensively sustained episode of ischemia, situation in which MR is mostly transient

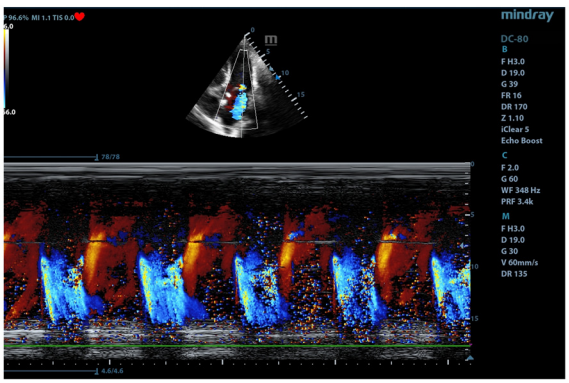

Figure nr. 3.3. Mitral regurgitation due to sustained ischemia (color M-mode, apical 4-chambers view) in a case with LV posterolateral myocardial infarction

- Rupture of myocardial wall is most often seen in large anteriorly or laterally infarcts at the junction between the necrotic and healthy myocardium; in 2D echo there can be observed abnormal contractility and diffusely distributed pericardial fluid in variable amounts; clinically pericardial tamponade may occur. In color

Doppler the blood flow oriented from LV towards the pericardium is observed.

- Rupture of interventricular septum ether apical or basal [figure nr. 3.4.] is recognised in 2D echo directly or indirectly (signs of RV overload, RA dilation, bulging of interatrial septum towards left atrium) and in color and PW/CW Doppler by visualising of a turbulent blood flow with left-to-right shunt.

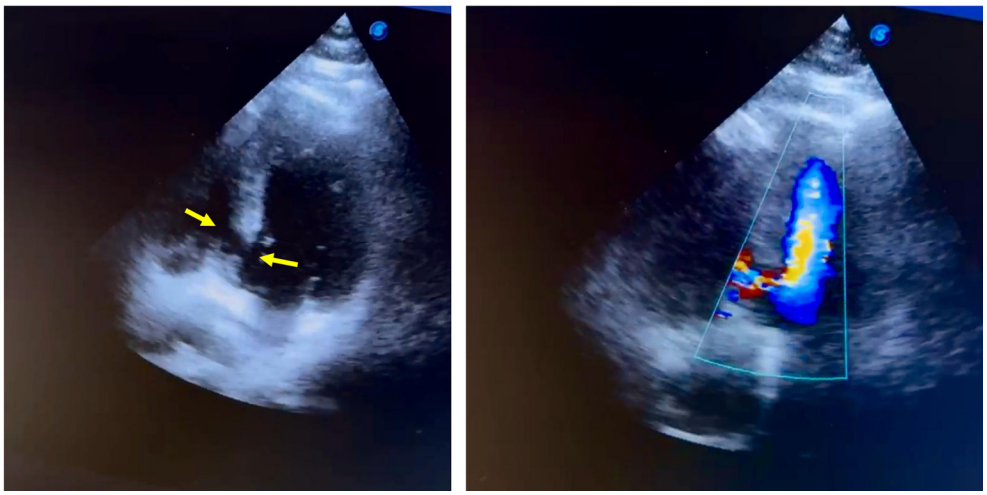

Figure nr. 3.4. Rupture of interventricular basal septum (arrows) in a case with acute myocardial infarction (2D and color Doppler, apical 4-chambers view)
Other complications

- LV aneurysm [figure nr. 3.5.] is visualized in 2D echo as a deformation of the infarcted myocardial wall, which is very thin, sometimes with increased echogenicity and with paradoxical motion. The size of the aneurysm in relation to the rest of the myocardium, the distance from mitral valve and presence of a thrombus most frequently located at the apex are important to be evaluated, which can be a source for distal emboli.

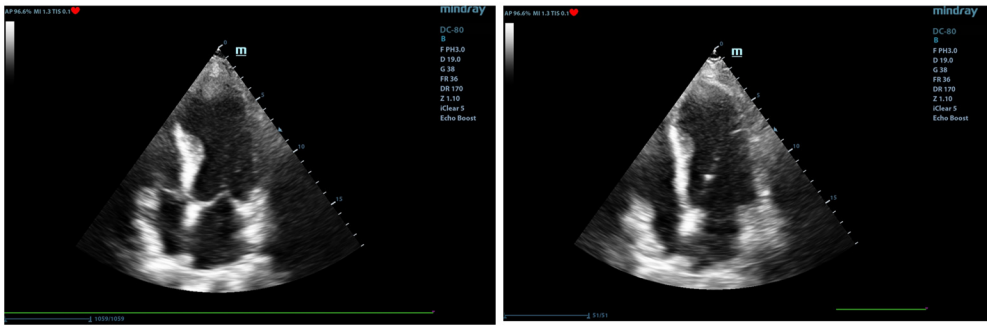

Figure nr. 3.5. Apical aneurysm noticed in a case with myocardial infarction ten days later (2D, apical 4-chambers view)

- Intraventricular thrombus [figure nr. 3.6.] is a homogenous isoechogenic mass (similar to the myocardium), sometimes mobile - when it is recent, and heterogenous hyperechogenic, adherent to the wall when it is old.

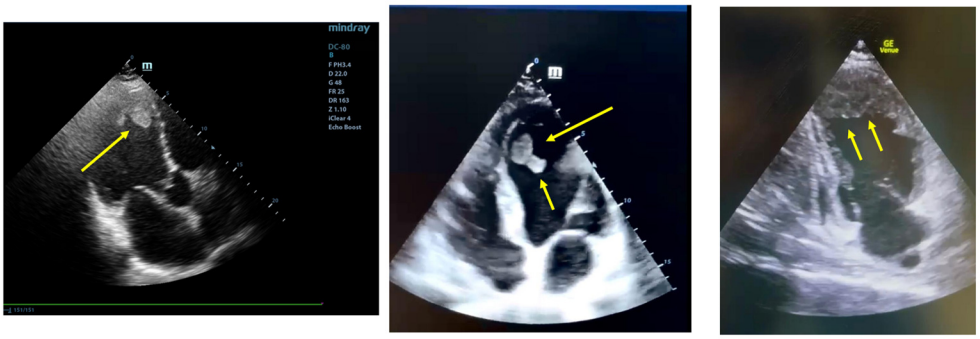

Figure nr. 3.6. Three different cases with apical LV thrombi (arrows) formed at apical level after a myocardial infarction and severe hypokinesia of the apex (2D, 3-chambers, 4-chambers and 2-chambers views)

- Pericardial effusion – can be observed as an echofree area between the layers of the pericardium; the fluid appears in smaller (postinfarct pericarditis, Dressler syndrome) or larger amounts (in myocardial rupture).

- Haemodynamic consequences: LV/RV reduced contractility, decrease in the CO and of SV (stroke volume), hypovolemia, ratio E/e' (if it is increased ≥15 correspond to PCP>20 mm/Hg) [figure nr. 3.7.]. Stroke volume can be calculated using formula: $SV=A_{LVOT} \times IVT_{LVOT}$ or LV volumes.

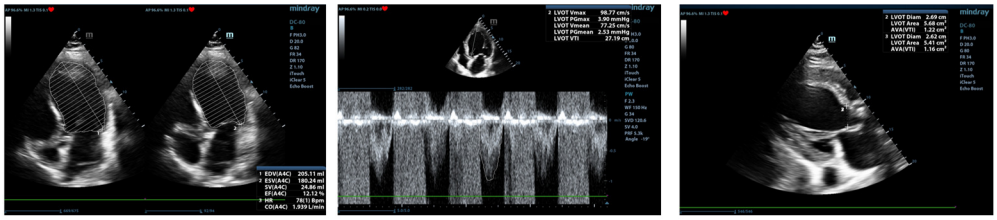

Figure nr. 3.7. Reduced stroke volume (SV=24 ml) and cardiac output (CO=1.9 L/min) in a case with myocardial infarction

- Chronic mitral regurgitation due to myocardial ischemia is the result of an imbalance between the closing and traction forces that act on the mitral valves[6,7]. In 2D restricted movement of the posterior mitral valve during systole is observed and in color Doppler the color jet oriented to the lateral wall of the left atrium. In most cases, the mechanisms are complex. Other causes can also be involved (e.g. decreased contractility and/or dilation of LV).

In the emergency department, in the case of patients with acute chest pain, TTE is helpful to differentiate or exclude other causes than myocardial ischemia: aortic dissection, pulmonary embolism, pericarditis.

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The echocardiographic images are from the own collection of dr. Florina Parv.

4. PERICARDITIS (FLORINA PÂRV)

The pericardium is a complex structure that surrounds the heart and is made up of the visceral pericardium, a thin membrane that comes in direct contact with the heart and the parietal pericardium, which is a fibrous membrane; between the two sheets, there is a space with 15-50 ml serous liquid important for the physiology of the heart.

Pericarditis represents the inflammation of the pericardium, a clinical situation in which a pathological accumulation of pericardial fluid may or may not be present; thus, pericarditis can be fibrinous or exudative. Regarding the onset of clinical symptoms, pericarditis can be acute or chronic. A rare form of pericarditis is the constrictive one in which the fibrous pericardium does not allow the expansion of the heart.

In transthoracic echocardiography the pericardial fluid is visualized the easiest. It can be placed circumferentially or it can be loculated. Its presence is not necessary for the diagnosis of pericarditis. The fluid often is the consequence of an inflammation, a proximity systemic or malignant process, it can be due to an endocrine disease, aortic dissection or trauma. Fluid accumulation takes place between the visceral and parietal layers of the pericardium; the rate of accumulation gives the severity of the condition. The main consequence is the increase of intrapericardial pressure, which leads to the decrease of intracardiac filling pressures, initially with effect in the right cavities. The most severe clinical form is represented by cardiac tamponade.

The etiology of pericarditis is very diverse (infectious, inflammatory, immune, neoplastic, post-traumatic, systemic diseases, aortic dissection) and usually requires multiple investigations.

Echocardiography is the most effective method for diagnosing and following the evolution of pericarditis, being specific, non-invasive, and easy to perform.

a. Exudative pericarditis

In 2D ultrasound, **pericardial fluid** is visualized as a transonic space between the two pericardial layers and sometimes requires differentiation from epicardial fat (which has a more hypoechoic appearance, sometimes granular, more commonly visible lateral to the right ventricle free wall).[figure nr. 4.a.1.]

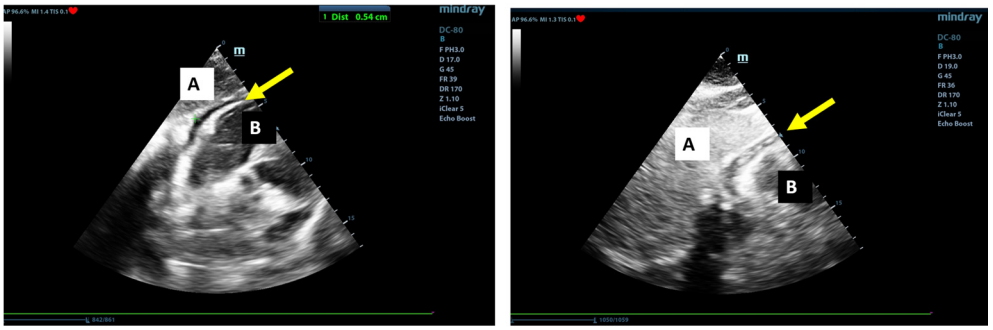

Figure nr. 4.a.1. Left: small amount of pericardial fluid 5.4 mm with transonic aspect (arrow) between the two pericardium layers; right: pericardial fat with granular, izo- and hyperechoic aspect(arrow); A=liver; B=right ventricle; (2D, subxiphoid view)

Other clinical situations or other pericardial pathologies that can be identified echographically and that can create diagnostic difficulties can be: pleural effusion [figure nr.4.a.2.], hiatal hernia, pericardial tumor, pericardial cyst, left ventricle pseudoaneurysm.

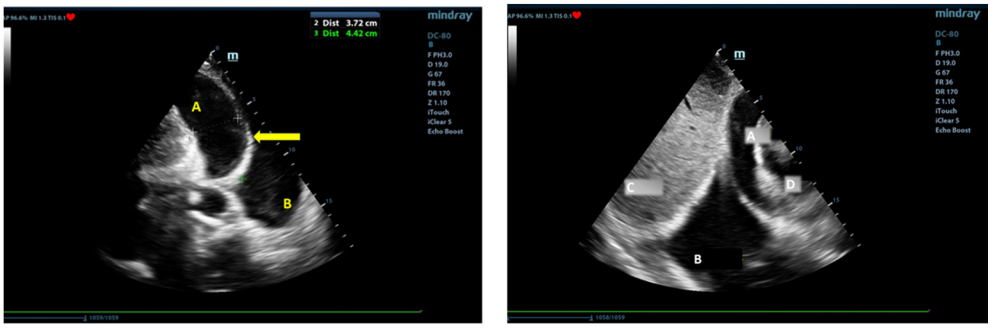

Figure nr. 4.a.2. Left: Large amount of pericardial fluid (3.72 cm) posterolateral of the left ventricle (A) and important left pleural effusion (B) separated by diaphragm (arrow) (2D, modified 4-chamber view). Right: Medium amount of pericardial fluid located lateral to the right ventricle (A) and important right pleural effusion (B); C=liver; D=heart (2D, modified subxiphoid view).

Sometimes the pericardial fluid is hemorrhagic and then the ultrasound image is no longer perfectly transonic and has a denser appearance.

In other situations, intrapericardial thrombi or fibrin can be visualized. [figure nr. 4.a.3.]

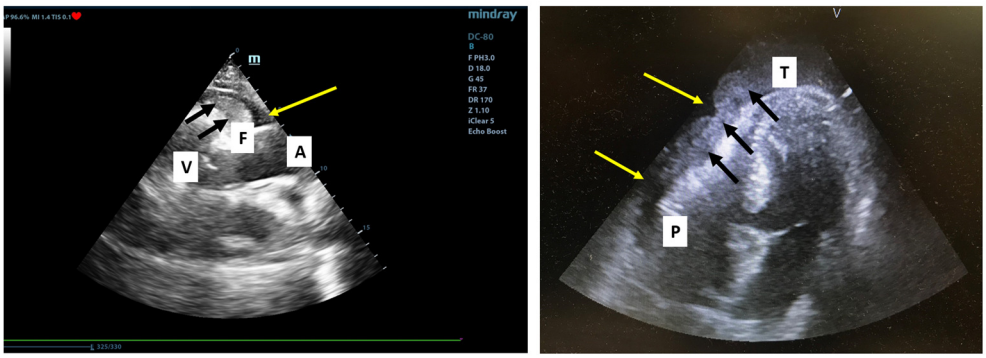

Figure nr. 4.a.3. Left: a case with small quantity of pericardial fluid (transonic, yellow arrow) and intrapericardial fibrin (isoechoic homogenous structure, black arrows); F=fibrin, A=aorta, V=right ventricle (2D, parasternal long axis); Right: a case with cardiac tamponade with medium amount of pleural fluid (transonic structure, yellow arrows) and a big thrombus inside and RV collapse; T=thrombus, P=pericardium (hyperechoic structure) (2D, apical 4-chambers view)

Evaluation of the amount of pleural fluid is appreciated at the end of diastole, as the distance between the sheets of the pericardium, in parasternal long axis, 4-chambers, subxiphoid views. The amount of fluid is small if the measured distance is <0.5 cm, medium between 0.5-2 cm and large at > 2 cm. [figure nr. 4.a.4.]. In pericarditis with a large amount of fluid, the rocking movement of the heart called "swinging heart" can be observed.

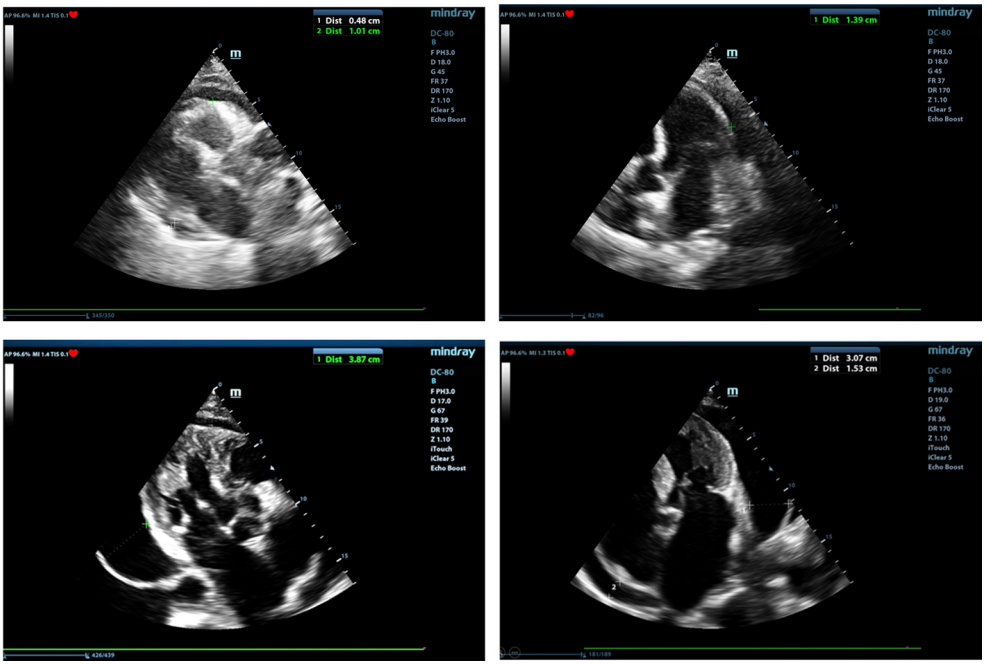

Figure nr. 4.a.4. Upper row: Circumferential pericardial fluid located posterolateral of the left ventricle (0.48 cm) and anterior of the right ventricle (1.01 cm) (2D, parasternal long axis view), and posterior of the left ventricle (1.39 cm) (2D, 4-chambers view).
Bottom row: Circumferential pericardial fluid located posterolateral of the left ventricle (3.07 cm) and posterior of the right atrium, without free wall collapse (1.53 cm) (2D, parasternal long axis view) and large amount of pericardial fluid (3.87 cm) posterior of the left ventricle and small amount fluid anterior of the right ventricle, without free wall collapse (2D, 4-chambers view).

b. Pericardial tamponade

Pericardial tamponade is a particular pathophysiological situation in which ventricular filling is obstructed due to excessive intrapericardial pressure, simultaneously with a decrease in cardiac output. The diagnosis is mainly clinical (hypotension, increased central venous pressure, paradoxical pulse, state of shock), but ultrasound has an essential role because it allows the dynamic visualization of transvalvular hemodynamic changes.

In 2D and M-mode ultrasound, the following aspects need to be noted:

- presence of pericardial effusion
- evaluating the amount of liquid is very important and requires dynamic monitoring.
- the paradoxical movement of sept
- AD systolic collapse (if it is present in more than one third of the systole, it suggests an increased intrapericardial pressure and imminent tamponade)

diastolic collapse of the free wall of a RV with normal compliance (sign of increased intrapericardial pressure) [figure nr. 4.b.1.]

- the diameter of the inferior vena cava and its respiratory variability: a diameter >2 cm and variability <50% indicates increased pressures in RA

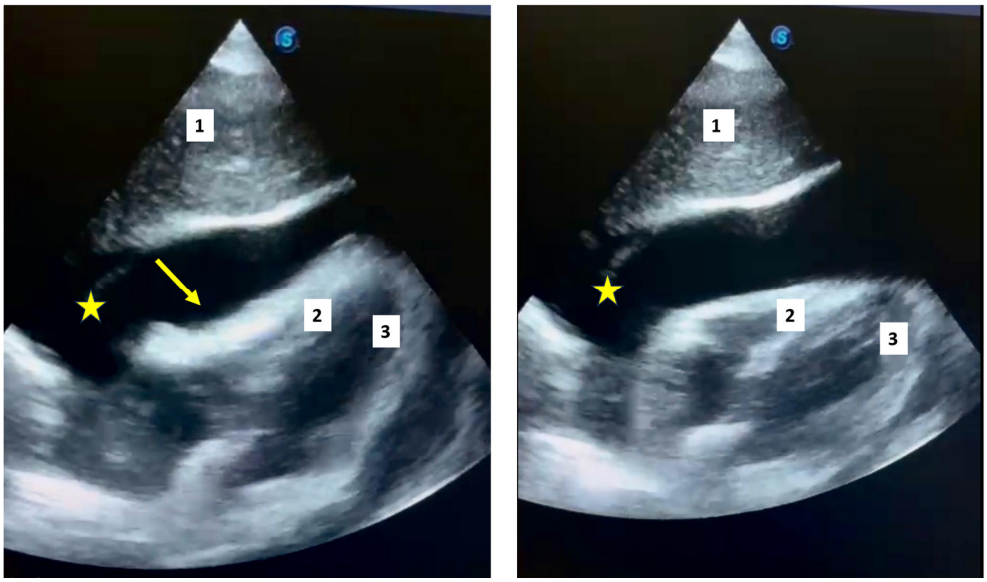

Figure nr. 4.b.1. Collapse of the free wall of a RV (arrow) in a case with cardiac tamponade. (star=pericardial fluid, 1=liver, 2=right ventricle, 3=left ventricle; 2D, subxiphoid view)

In the pulsed doppler ultrasound, the following items must be evaluated:

- respiratory variation of the transtricuspid flow (flow velocity increases during inspiration)
- respiratory variation of transmitral flow (flow velocity decreases during inspiration)

A respiratory variability greater than 25% at the transmitral flow level and 40% at the transtricuspid level occurs in pericardial tamponade, but also in large intrapleural fluid accumulations or pneumothorax that compress the mediastinum and lead to an extrinsic increase in pericardial pressure. [figure nr.4.b.2.]

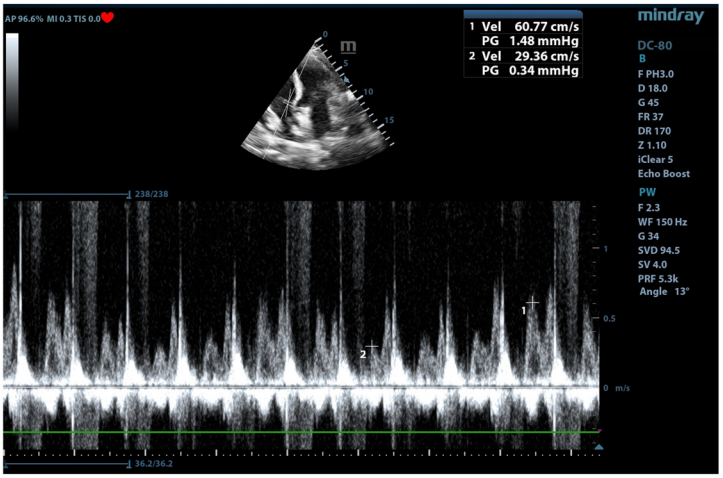

Figure nr. 4.b.2. Respiratory variability of 51% of transtricuspidian E-wave recorded in a patient with cardiac tamponade (PW Doppler, 4-chambers view)

c. Constrictive pericarditis

Constrictive pericarditis is a form of pericarditis in which the pericardium has undergone an infectious-inflammatory histopathological process followed by fibrosis; due to the rigid pericardium, the distensibility of the myocardium is limited and the filling of the cardiac cavities is affected. Clinically, it can be asymptomatic in the initial stages, later it manifests itself with signs of right heart failure. Ultrasound has an important role in the positive and differential diagnosis, especially with restrictive cardiomyopathy.

In 2D and M mode, the following characteristic aspects can be identified:

- hyperechoic pericardium, thickened, with calcifications
- absence of pericardial fluid
- dilated atria
- normal sized ventricles, usually normal LVEF
- abnormal end-diastolic movement of the interventricular septum

- Vp (transmitral diastolic flow propagation velocity) increased >100 cm/s [figure nr. 4.c.1.]

- IVC dilation, with reduced respiratory variability, dilation of the hepatic veins. [figure nr. 4.c.1.]

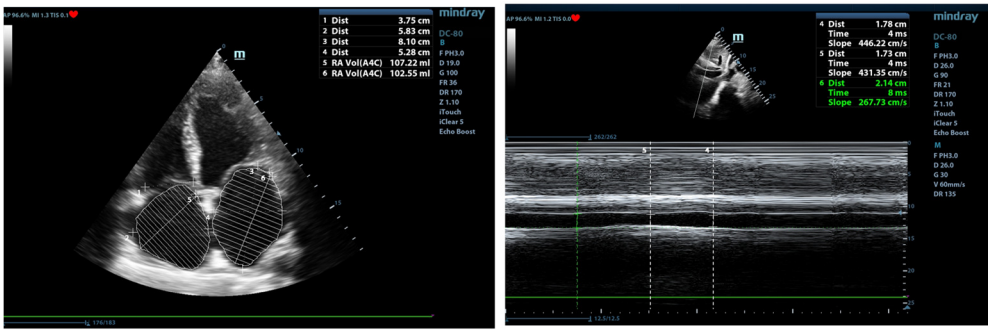

Figure nr.4.c.1. Biatrial dilation (left, 2D, apical 4-chambers view) and dilated IVC with reduced respiratory variability (right, M-mode, subxiphoid view)

In the pulsed doppler echography respiratory variations of the transmitral flow (in inspiration the E wave velocity decreases by >25%) and transtricuspid flow (in inspiration the E wave velocity increases by >35%), an E/A ratio>2, but with preserved myocardial relaxation (in tissue doppler with normal e's and ratio e'l/e's<1) are identified. [figure nr. 4.c.2]

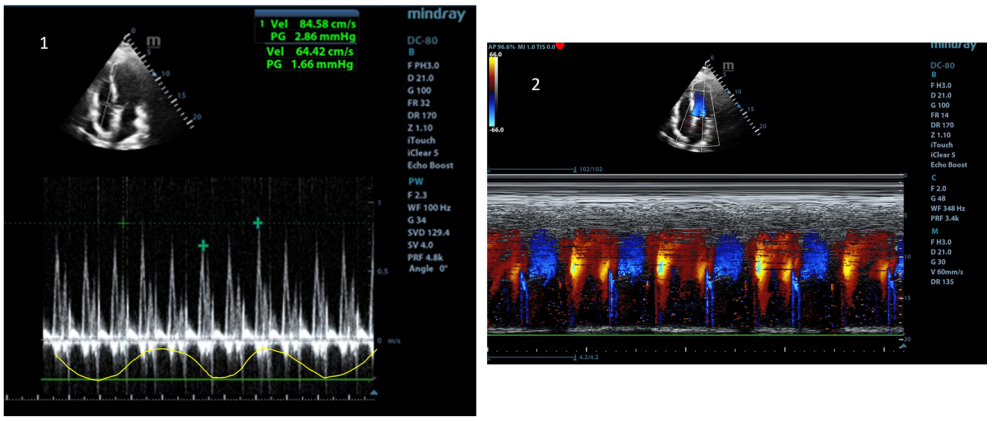

Figure nr. 4.c.2. Left (1): respiratory variations of the E-wave of transmitral flow (PW Doppler, apical 4-chambers view); Right (2): increased transmitral diastolic flow propagation velocity (color M-mode, apical 4-chambers view)

CLINICAL CASE

A male patient aged 69 years with cardiovascular (essential arterial hypertension, ischemic coronary artery disease, bifascicular block, atrial fibrillation, compensated heart failure), metabolic (diabetes mellitus) and oncologic (colon cancer operated, chemo and radio therapy, with treatment completed) history was evaluated 5 years ago when he was diagnosed with exudative pericarditis with small amount of fluid, the patient being asymptomatic from this point of view. At that time, the etiology of pericarditis was investigated using complex imaging and biological, biochemical, immunologic, bacteriologic, and immunologic tests. Neoplastic, infectious (viral, bacterial), autoimmune, chronic inflammatory, metabolic, endocrinologic causes were ruled out. A period of anti-inflammatory treatment with NSAIDs and colchicine was followed, and the same minimum amount of fluid was maintained at periodic re-evaluations at 1, 3, 6, 12 months. As a potential cause that could not be excluded was late-onset post-radiation pericarditis. After another 5 years, he returned for cardiologic re-evaluation presenting signs of cardiac decompensation. The objective examination showed dyspnea, orthopnea, exertional asthenia, leg edema, BP=140/70 mmHg, HR=64/min, SaO2=88%, bilateral basal stasis rales, arrhythmic heart sounds, soft abdomen, painless.

The electrocardiogram showed atrial flutter, major left bundle branch block, and the chest X-ray showed global cardiomegaly, with characteristic "flask-shape" or carafe appearance of the heart and also central and peripheral vascular stasis. [figure nr. 4.c.3]. Current investigations showed normal values of hemogram, inflammatory and autoimmune markers, TSH, tumor markers, and negative fecal occult blood test.

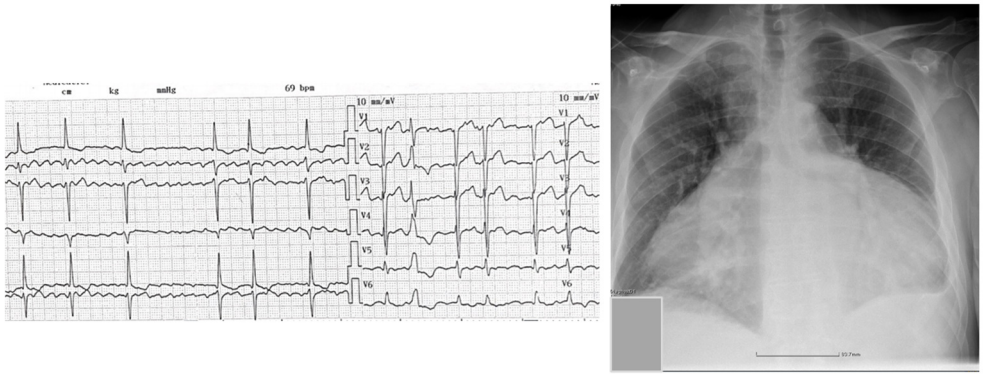

Figure nr. 4.c.3. Electrocardiogram (left) and X-ray (right) of the patient showing typically enlarged cardiac silhouette with an increased transverse cardiac diameter with a globular configuration

Transthoracic echocardiography was performed which showed heart valves with calcifications, with preserved mobility, with mild mitral, tricuspid and aortic regurgitation. [figure nr. 4.c.6].

The left ventricle presented eccentric hypertrophy with interventricular septum of 13.5 mm and posterior wall of 16 mm, RWT=0.44, but also with dilatation, with a volume of 200 ml, respectively 111 ml/ m2 sc. The LA was dilated with an anteroposterior diameter of 5.14 cm and a volume of 176 ml, indexed 98 ml/m2 sc. The right cavities were dilated, RV diameter of 4.4 cm, RA diameter of 5 cm and volume of 100 ml. The systolic pressure in the pulmonary artery was estimated to be 45 mmHg. The pericardium was ultrasonographically mildly hyperechoic and a large amount of fluid was noticed between the pericardial leaflets, relatively uniformly distributed, but with no signs of collapse in the right atrium or right ventricle and no Doppler signs of pericardial constriction. [figure nr. 4.c.4.] [figure nr. 4.c.5]

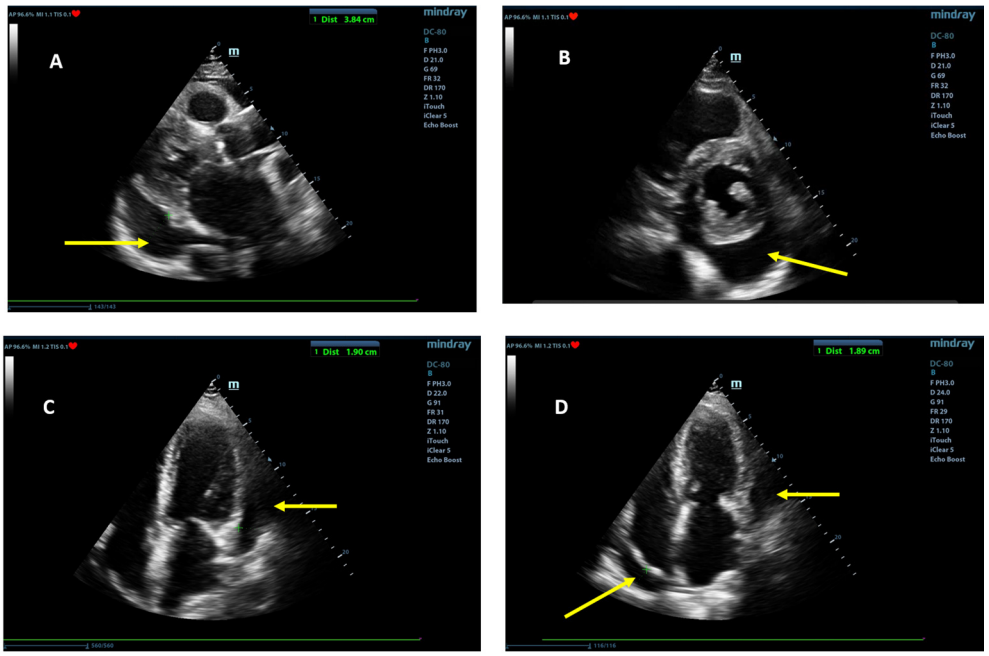

Figure nr. 4.c.4. Large amount of pericardial fluid (arrow) noticed as a transonic, echo-free space between pericardium sheets noticed between heart and aorta (3.84 cm, image A: 2D, parasternal long axis view), posterior to the left ventricle (image B: 2D, parasternal short axis view), posterolateral to the left ventricle (1.9 cm; image C: apical 4-chambers view), posterior to the right atrium (1.89 cm, image D: 2D, apical 4-chambers view)

The inferior vena cava was dilated (2.7 cm), and its respiratory variability reduced, below 50%. The systolic function of the LV was slightly reduced (LVEF was 45-50%), and the diastolic function assessment showed diastolic dysfunction with an E wave of 143 cm/s, e's of 10.27 cm/s, an E/e' ratio of 14. [figure nr 4.c.5.]

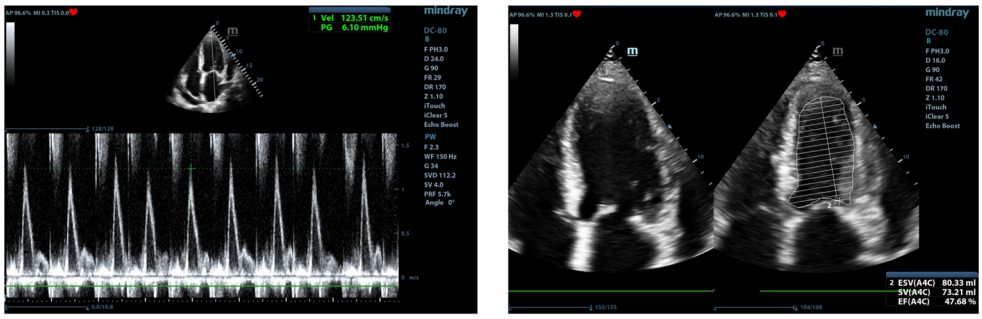

Figure nr. 4.c.5. Evaluation of diastolic (left: transmitral valves PW Doppler, E wave respiratory variations of 13%; apical 4-chambers view) and systolic function (right: LVEF 47%, apical 4-chambers view) of the left ventricle in the presented case

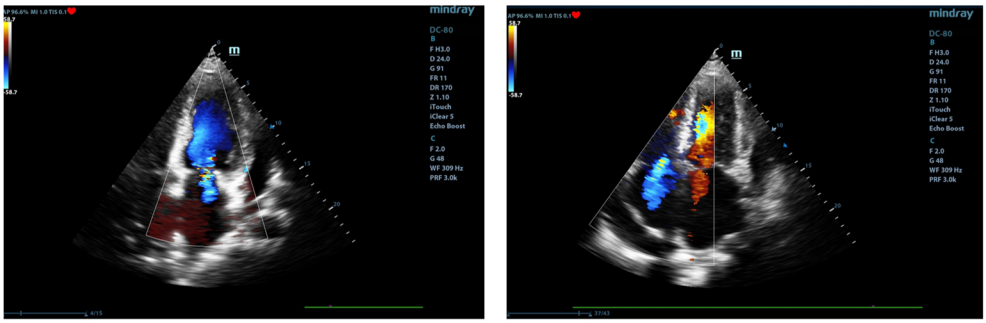

Figure nr. 4.c.6. Mild mitral and tricuspid regurgitation associated in the case presented (color Doppler, apical 4-chambers view)

The patient was diagnosed as having chronic exudative pericarditis with a large amount of fluid, and underwent specific treatment of the underlying disease, accordingly to renal glomerular function, with diuretics, vasodilators, beta-blockers, insulin, SGLT2 inhibitor.

In addition, cardiac MRI was performed which confirmed the presence of pericardial fluid and ruled out constrictive pericarditis. [figure nr. 4.c.7.]

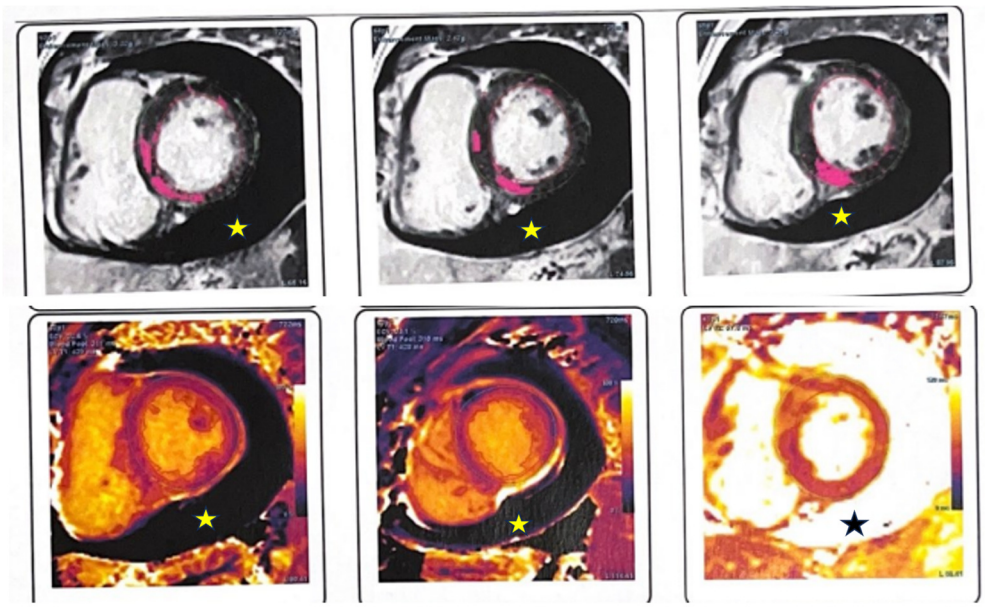

Figure nr. 4.c.7. Cardiac MRI in presented case showing exudative pericarditis with large amount of fluid (star), without thickening of the pericardial sheets and nonischemic scarring (probably postmyocarditis) in the LGE sequences, in the anterolateral, inferolateral and inferior walls of the basal and medioventricular LV.

The patient was referred to the cardiac surgery department where pericardiocentesis, pericardial biopsy (with non-specific inflammatory findings) and pleuropericardial window were performed, with favorable outcome.

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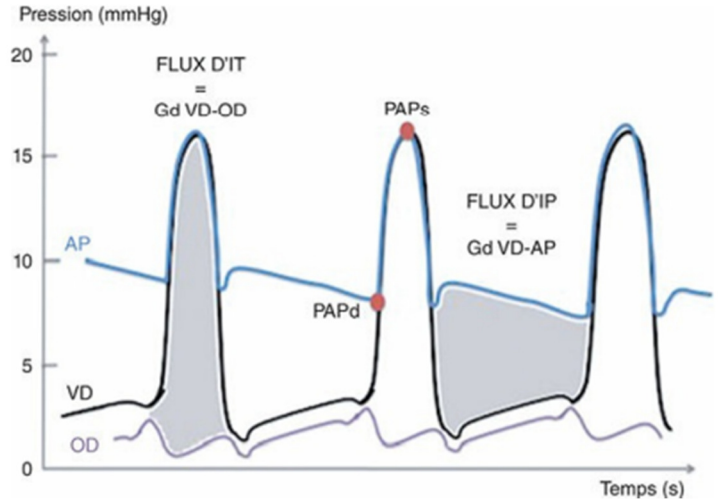
1. Lancelotti P et al. The EACVI Textbook of Echocardiography. 2-nd Ed.Oxford, 2017
2. Catherine M Otto. Textbook of clinical echocardiography. Ed.4 Elsevier Saunders, 2009

The echocardiographic images are from the own collection of dr. Florina Parv.

5. PULMONARY HYPERTENSION (ADRIAN APOSTOL)

Pulmonary arterial pressure (PAP) varies during the cardiac cycle between a maximum value in systole (PAPs) and a minimum value at the end of diastole (PAPd). Pulmonary hypertension (PH) is defined by a mean PAP (PAPm) ≥ 25 mmHg at rest. Right heart catheterization is the gold standard for measuring PAP. Echocardiography is a method of estimating PAP, and is widely used routinely to detect PH.



Estimation of pulmonary pressures

Estimation of pulmonary pressures relies essentially on measurement of flow velocity, which must therefore be rigorous and precise.

Tricuspid insufficiency (TI) flow velocity

- Most commonly used method in practice.
- TI most frequently present in PH.
- Reflects the pressure gradient between RV and RA in systole.

Measuring method

- 2D
- Color Doppler

- Pulsed Doppler
- Continuous Doppler

Estimation of right atrial pressure:

- Estimation of gradient (Gd) max (RV-RA) by Bernoulli equation :

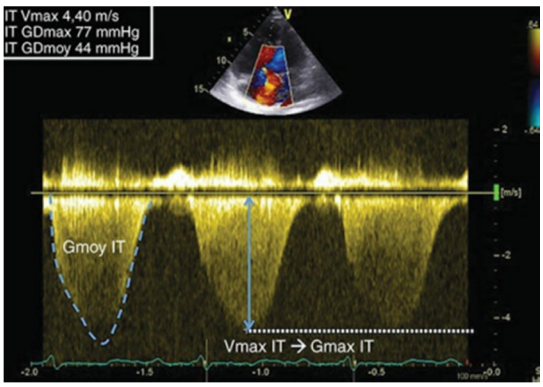
  Gdmax (RV-RA) = 4 Vmax TI 2

- Estimation of right atrial pressure (RAP) according to size and degree of inspiratory collapse of the IVC:

  PAPs = Gdmax (RV-RA) + RAP

- Estimation of mPAP (less reliable than sPAP): estimation of mean Gd (RV-RA) by contouring the spectral envelope of TI flow

  PAPm = Gd med (RV-RA) + RAP



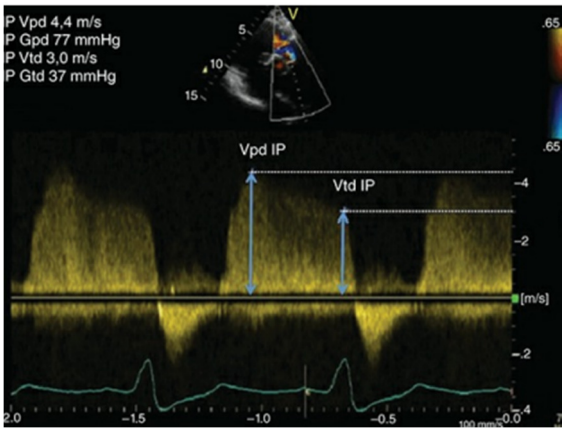
Pulmonary insufficiency (PI) flow velocity

- Reflects the pressure gradient between PA and RV during systole.

Measuring method :

- 2D
- Color Doppler

- Continuous Doppler
- Estimation of mPAP: mPAP = protodiastolic Gd + RAP.
- Estimation of PAPd: PAPd = Gd telediastolic + POD.
- Estimation of PAPs: formula of thirds: PAPm = 1/3 PAPs + 2/3 PAPd.
- PAPs = 3 PAPm - 2 PAPd

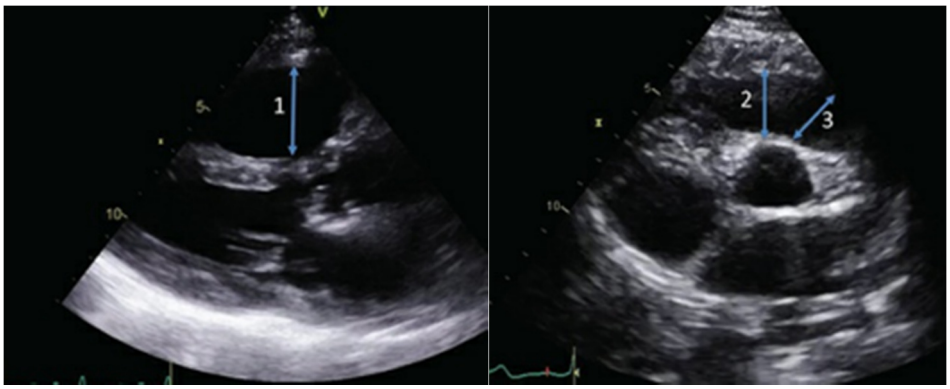


Assessing the impact of pulmonary hypertension

1. **Dilation of the right ventricle**

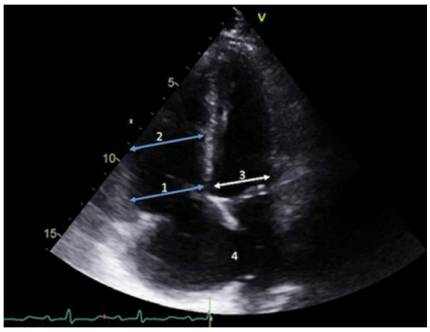
   Chambre de chasse du RV

   - Proximal diameter of RV hunting chamber > 33 mm
   - Parasternal long-axis section: measured vertically, between the anterior wall of the RV and the IVS; - parasternal short-axis section centered on the basal vessels: measured vertically, between the anterior wall of the RV and the aorta.



RV inlet chamber. Apical section centered on the right cavities:

- maximum transverse diameter of the basal third of the RV > 42 mm
- transverse diameter of middle third of RV in telediastole > 35 mm
- ratio of RV to LV basal transverse diameters > 1



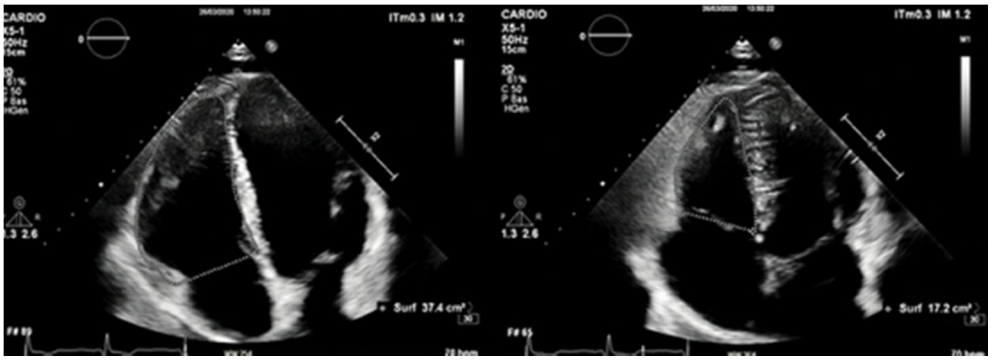
2. Hypertrophy of the right ventricle

- RV wall diameter > 5 mm (subcostal slice; parasternal slice)

3. Impaired right ventricular function

Surface shortening fraction

- Ratio (telediastolic area - telesystolic area)/telediastolic area, apical section 4 cavities
- Normal value: 46-52%. RV dysfunction if < 35%.
- Ratio of surfaces, not volumes.



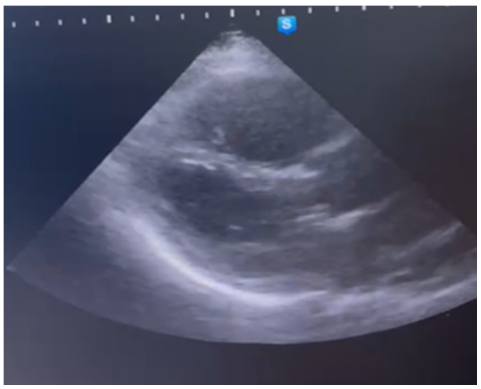
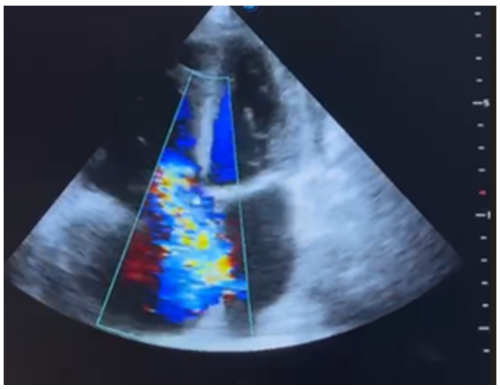
Tricuspid annulus systolic excursion (TAPSE)

- Obtained on apical 4-cavity incidence, in TM mode with cursor at the junction of the tricuspid annulus and lateral wall of the RV.
- Measured between telediastole and mesosystole (figure 4.6).
- Normal value: 24 ± 4 mm.
- RV dysfunction if TAPSE < 17 mm.

Pulmonary embolism

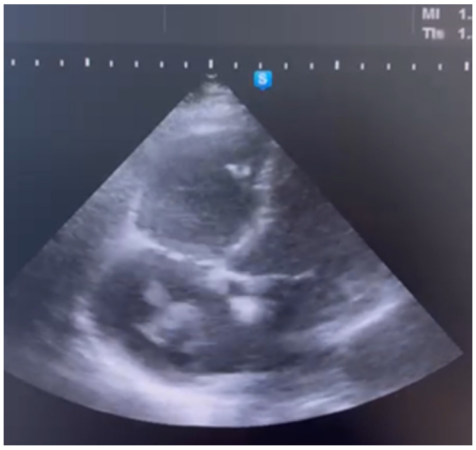
- **Signs of acute pulmonary heart disease** - Significant and abrupt PH.

1. Dilation of the right heart chambers
   - dilated PA trunk if massive PE
   - moderate to severe RV dilatation, depending on severity of PE: RV/LV ratio > 0.6
   - more marked dilatation of the RV apex

2. Absence of RV hypertrophy.
3. Contractile abnormalities :
   - convex appearance of RV anterior wall
   - altered RV anterior wall contractility
4. Changes to the IVS :
   - bulging of the IVS towards the LV
   - paradoxical septal movement.
5. Frequent functional IT.
6. TI Vmax > 2.7 m/s.
7. Left cardiac output initially increased by tachycardia, decreased in severe PE by reduced LV preload.

- **Presence of thrombi in the right heart chambers.**



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5. Ravi Rasalingam Manualul Washinton de Ecocardiografie

The echocardiographic images are from the own collection of dr. Adrian Apostol.

6. DISEASES OF THE AORTA (ADRIAN APOSTOL)

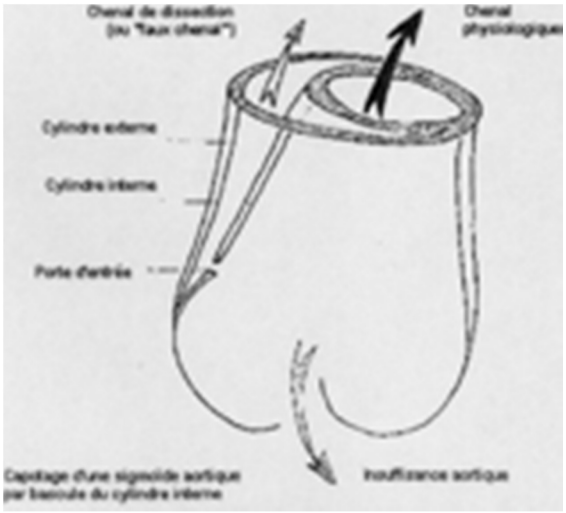
a. Aortic dissection

- The greatest aortic tragedy
- Responsible for 1% of sudden deaths
- Fundamental role of early diagnosis and optimal management

Definition - longitudinal cleavage of the aortic wall at the level of the media, from a 'portal of entry'.

-acute within 14 days of 1st symptom

-Chronicle beyond.

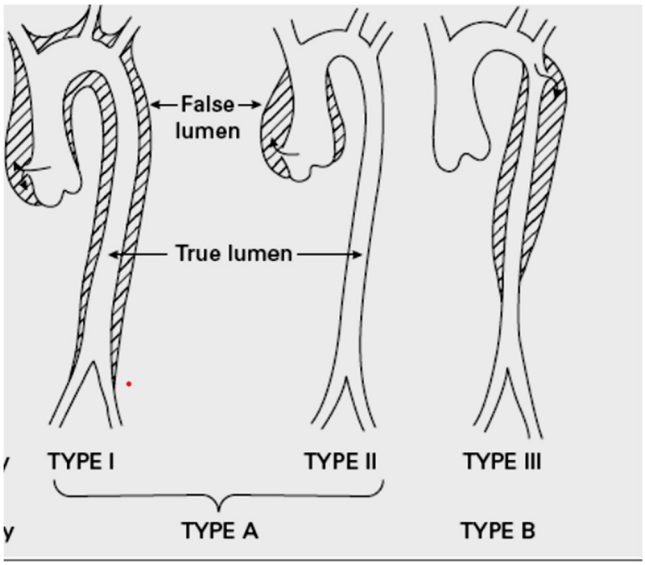


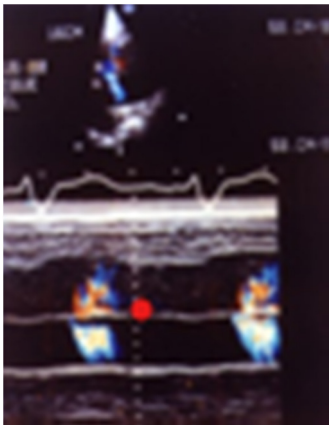
Pathophysiology

- Two factors:
  1. Anatomical: media necrosis Cystic aortic wall
  2. Hemodynamics: HP, cardiac inotropism
- Two clinical contexts:
  1. Diseases of elastic tissue (Marfan)
  2. Hypertensive subjects

Types and entrances:

- De Bakey : Type I, Type II, Type III
- Stanford : Type A, Type B

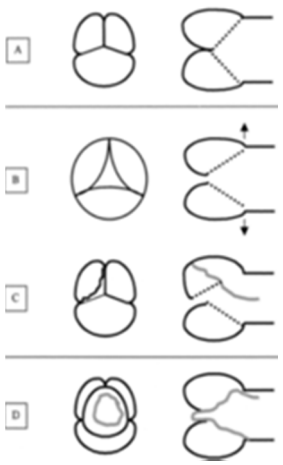




Positive diagnosis

4 signs :

- INTIMAL FLAP" intimal veiling
- Aortic dilatation, but inconstant
- Aortic insufficiency, frequent in type A



A. Normal

B- incomplete closure due to dilatation of sino-tubular area

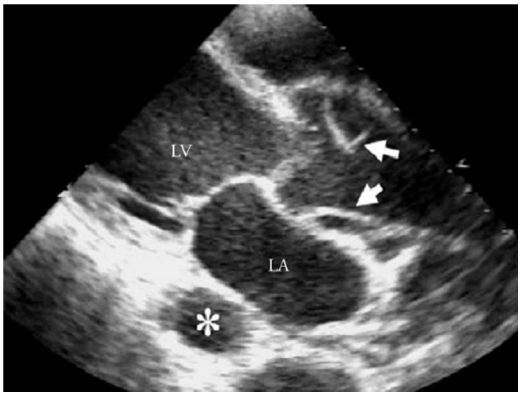
C- sigmoid prolapse by valve attachment dissection

D- intimal veil prolapse

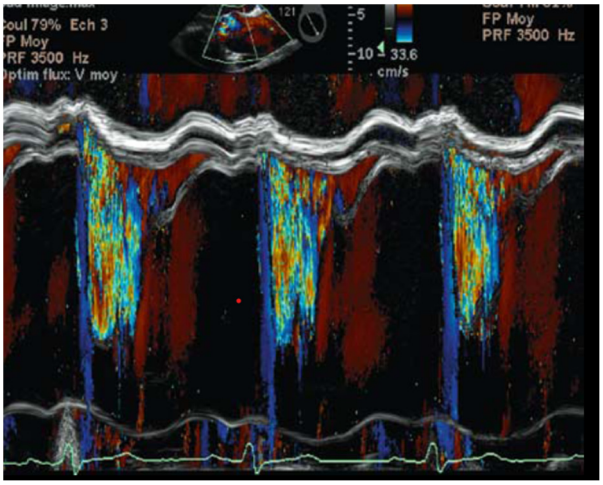
- Pericardial effusion
- Pleural effusion

Entrance:

- Echo: continuity solution at the level of the intimal web



- Color Doppler: aliasing between true and false channels.

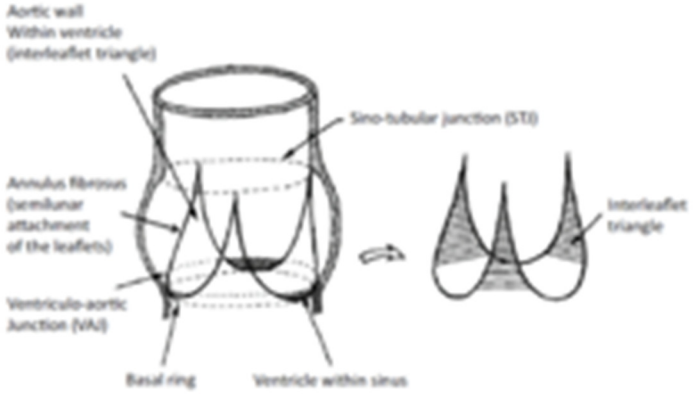


b. Aneurysm of the ascending aorta

Main etiologies :

- Degenerative
- Atherosclerotic
- Congenital (sinus Valsalva aneurysms, aortic bicuspidity)
- Dystrophic (Marfan's disease, elastic tissue disease)
- Inflammatory (Takayasu, Horton's disease, ankylosing spondylitis)
- Infectious (Syphilis)
- Traumatic.

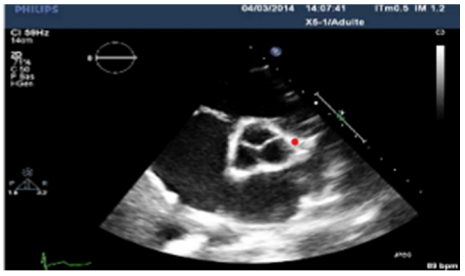
The aortic valve is inserted into the aortic culot, and is a three-dimensional structure.

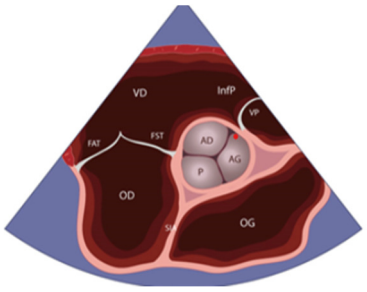


Sinus of Valsalva :

- 3 sinuses
- 3 valves
- 2 coronary ostia

1

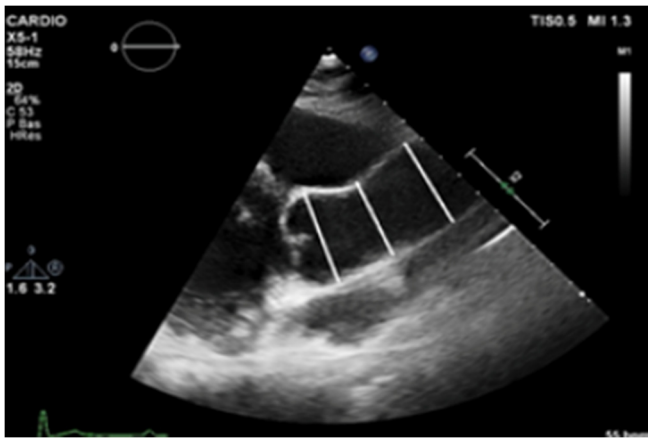


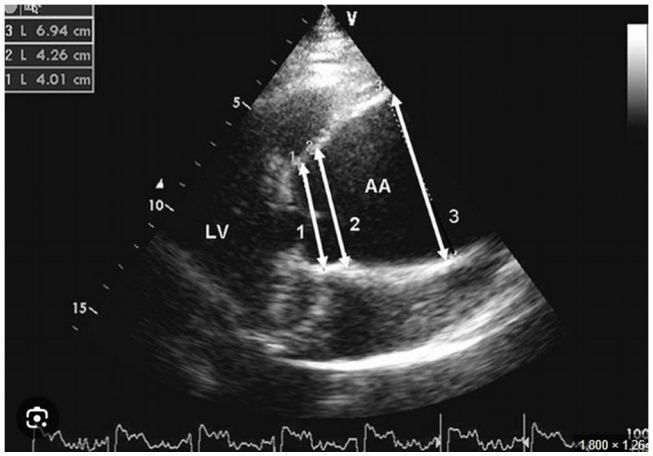


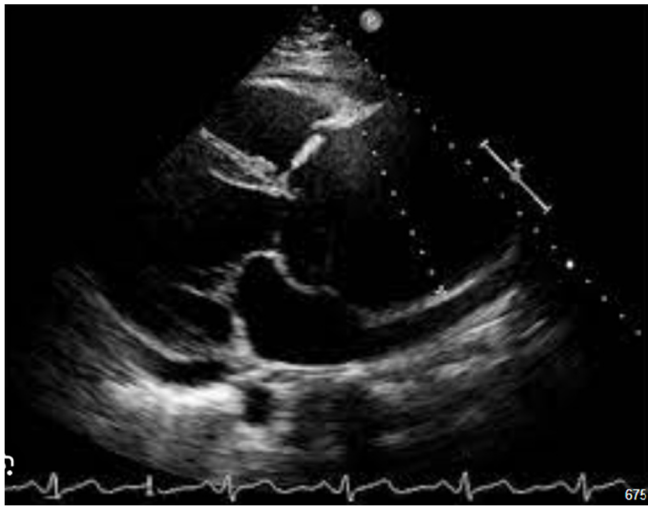
Left parasternal long-axis section - 2D mode

Measurement of 3 segments of the ascending aorta in diastole:

- Sinus of Valsalva
- Sinotubular junction
- Tubular aorta


CARDIO
X5-1
TIS0.5 MI 1.3


3 L 6.94 cm
2 L 4.26 cm
1 L 4.01 cm
LV
AA
1
2
3



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