Echo Facts

Includes over 480 illustrations and more than
180 online video examples

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Foreword

This book has evolved from a simple companion syllabus for our 123sonography Masterclass to a textbook that now stands on its own. Our users' feedback revealed that while learning online is highly effective, it is equally important to have a hardcopy book in your hands. Having a compendium version online was very important because it enabled us to collect our users' feedback. The latter was of paramount importance in writing this book.

As the title of the book implies, we provide relevant facts that you need to know if you are practicing echocardiography and wish to go beyond. We have included chapters on stress echo, contrast echo, 3D echo, and deformation imaging.

We used as little text as possible, listing the contents in tabular form so that the book can be viewed as a study guide and a reference book. We took care to include all relevant reference values, formulas, and checklists that will help in your daily practice. The book is richly illustrated: it contains more than 300 figures, most of which have been taken from our online 123sonography.com Masterclass course. Importantly, we have included many practical notes on how to image. These will help to improve your imaging skills and, ultimately, your diagnostic yield.
The book may also be viewed as an atlas of echocardiography. In contrast to our workbook, we have incorporated more than 180 echo examples. The cine videos are available on the web (http://123sonography.com/echofacts). After all, how else can you learn echocardiography than by viewing images of the moving heart?

Our 123sonograpahy echo project has grown tremendously since we launched the website over three years ago. We have become very attached to our users and friends.
We hope this book will serve as a step forward towards our goal of improving the quality of echocardiography in all parts of the world and making you a better echocardiographer.

Thomas Binder

Georg Goliasch

Acknowledgments

Collecting the large number of images for this book was a joint effort on the part of all the people working at our lab. The book would not have been possible without the assistance of our sonographers Beatrix Buschenreithner, Ulrike Grojer, Regina Schlossnickel, and Andrea Schuckert, and many of our residents who were always on the lookout for suitable loops and cases. We are sure they will recognize many of their contributions.

Bernhard Richter was responsible for the corrections and improvements made while publishing this version of the book. Bernhard possesses the rare skills needed to edit a book from the perspective of an expert as well as a student. His efforts had a tremendous impact on its quality.

Thanks to Sujata Wagner for her reliability and hard work. She converted many of our awkward Germanic phrases into fluent and comprehensible English.

To Oliver Hübler for his support and programming, which permitted us to put up the web-based atlas that complements this book. No programming hurdle is insurmountable for him.

Saskia Erbschwendner who collected, sorted, and categorized all the user feedback that was so valuable to us. And for setting us up so that the work could actually go into print.

Georg Greutter, "the man on the drums", for paving the way in planning and marketing the book. He transformed us into a publishing company.

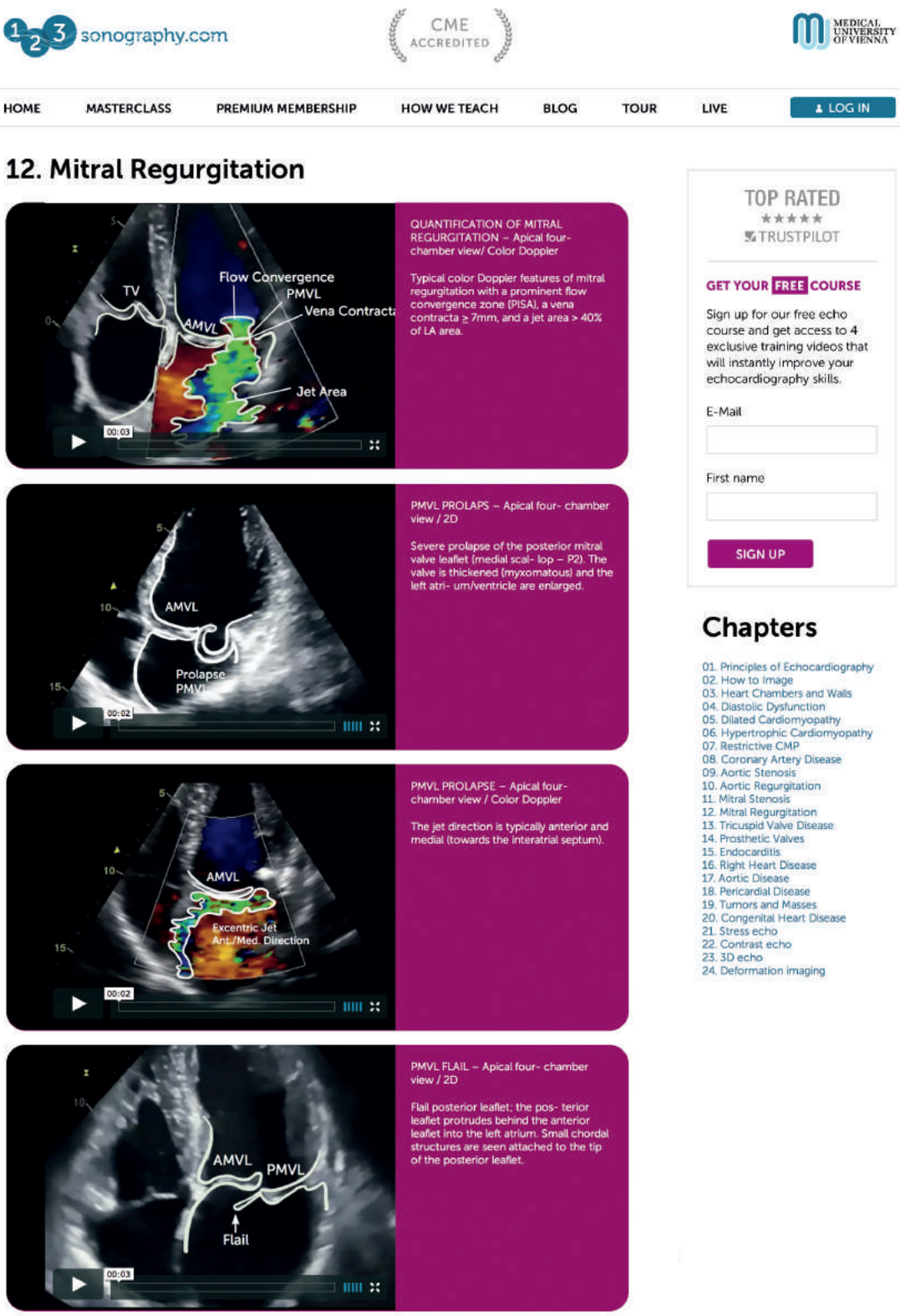
Karin Dreher and Inge Vorraber, who enthused the book with life. They are responsible for its unique style, layout, and graphics.

Thanks to our mentors and supporters: Helmut Baumgartner, Massoud Zangeneh, Gerald Maurer, Partho Sengupta and Senta Graf. They taught us much of the knowledge that we now share with you.

Most of all, our thanks to the many users who provided us with their valuable feedback, and those who are embarking on this fruitful and rewarding journey of learning echocardiography.

Free Access to the Videos at 123sonography.com/echofacts

Echo Atlas



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001 // Principles of Echocardiography

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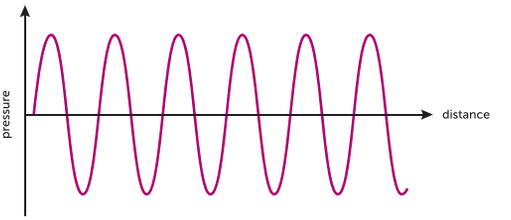
PHYSICS OF ULTRASOUND

Ultrasound Wave



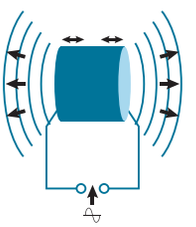
Wave propagation occurs through compression and decompression of tissue.

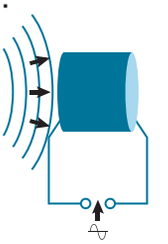
The velocity of ultrasound is 1540 m/s in tissue and 1570 m/s in blood.

The higher the ultrasound frequency, the better the resolution. However, you lose penetration.

Medical Ultrasound

Frequencies between 2 – 10 MHz are used.





Alternating current applied to piezoelectric crystals generates ultrasound waves..

Received ultrasound waves (echoes) cause the piezoelectric crystals to generate an electric signal which is transformed into an image..

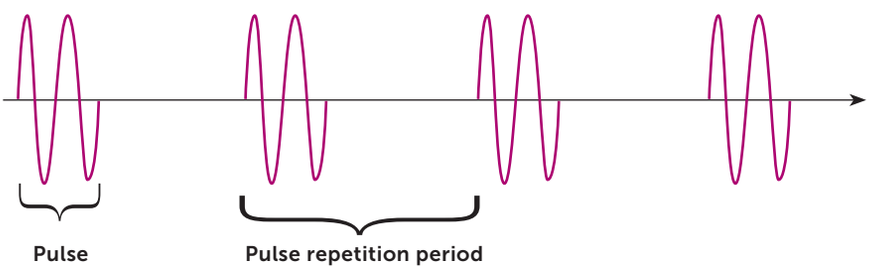
Diagnostic ultrasound has no adverse effects.

Safety of Ultrasound

Physical effects of ultrasound:

- Thermal effect (depends on US intensity)
- Cavitations

Ultrasound Pulse

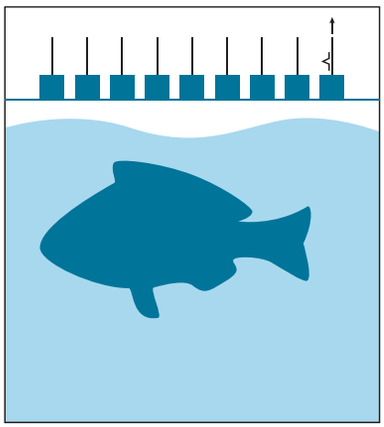


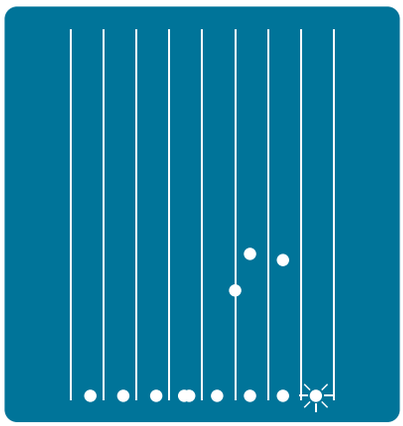
The higher the US frequency, the higher the pulse repetition frequency.

The higher the pulse repetition frequency, the higher the frame rate and image resolution.

2D IMAGE

2D Image





Types of Probes

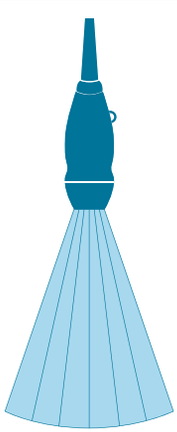
Image Quality

What determines overall resolution?

- Spatial resolution – lateral
- Spatial resolution – axial
- Contrast resolution
- Temporal resolution

Determinants of Spatial Resolution

Lateral resolution	Axial resolution
Beam width/line density	Ultrasound frequency
Ultrasound frequency	Pulse repitition frequency
Gain	Gray



NOTES

Ultrasound is a cut-plane technique. Several elements are used to generate a 2D image.

In echocardiography we use curvilinear probes. The advantage of such probes is their small "footprint". Thus, it is easier to image from small intercostal spaces.

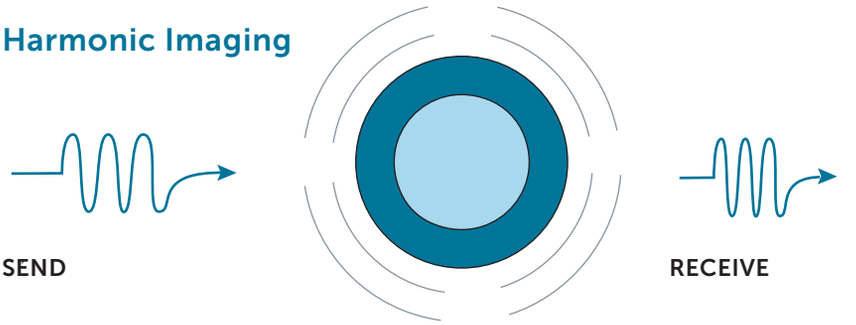
Image quality increases with higher scan line densities.

NOTES

2D IMAGE

Harmonic Imaging

Harmonic imaging uses the resonance characteristics of tissue. The advantage is less artefacts, improved spatial and contrast resolution, leading to better image quality.



Legend: The signal returned by tissue includes the transmitted "fundamental" frequency as well as signals of other frequencies. In harmonic imaging one uses those frequencies that are a multiple (harmonic) of the fundamental (sending) frequency.

Frame Rate – Influence

Aim for high frame rates. They allow the study of rapid motion when using the image review function.

The frame rate describes the number of frames/sec that are displayed.
Frame rate depends on:

- Sector width
- Scan lines
- Frequency
- Depth

Limitations of 2D Imaging

- Attenuation
- Tissue properties (fibrosis, calcification)
- Artefacts
- Limited penetration (obesity, narrow imaging window)

Attenuation

Definition: Decrease in amplitude and intensity as the ultrasound wave travels through a medium

Attenuation may be caused by:

- Absorption (proportional to frequency)
- Refraction
- Transfer of energy from the beam to tissue
- Reflection
- Shadowing
- Pseudoenhancement

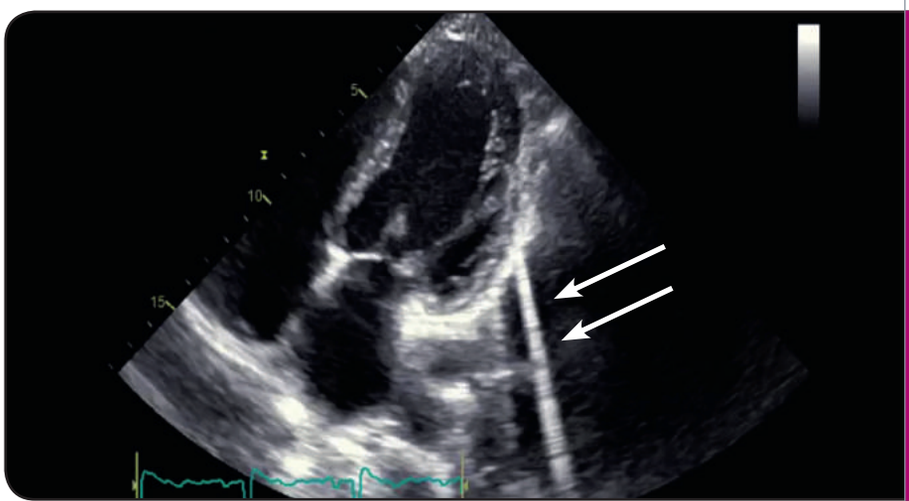
Enemies of Ultrasound

Air (reflection of ultrasound) and bone (absorption of ultrasound)
In both conditions you cannot see what is behind.

ARTEFACTS

Types of Artefacts

- Near field clutter
- Reverberation
- Acoustic shadowing
- Mirror imaging/double images (caused by refraction)
- Side lobe artefact
- Beam width artefacts
- Attenuation artefacts

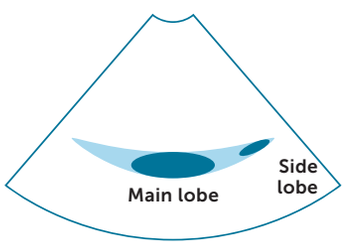


REVERBERATION –
apical four-chamber view/2D

Highly echogenic pericardium leading to reverbations

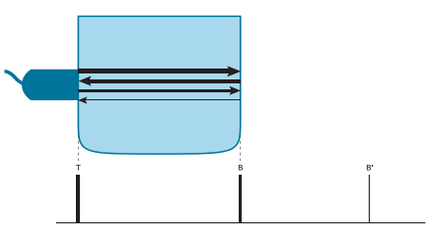
Specific Forms

Side lobes



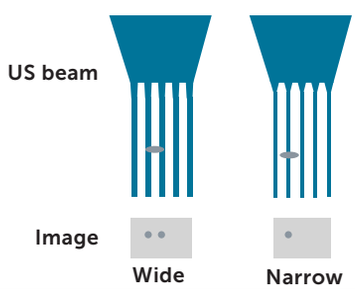
Side lobes usually occur at strong reflectors (e.g. prosthetic material). Power density is higher in the central beam than in side lobes. This may lead to the edge effect, which makes structures appear wider than they actually are.

Reverberation



Reverberation occurs when the echo bounces back and forth several times – sometimes between a structure and the surface of the transducer.

Beam width artefact



Beam width artefacts occur when the beam width is wide and unfocused.

NOTES

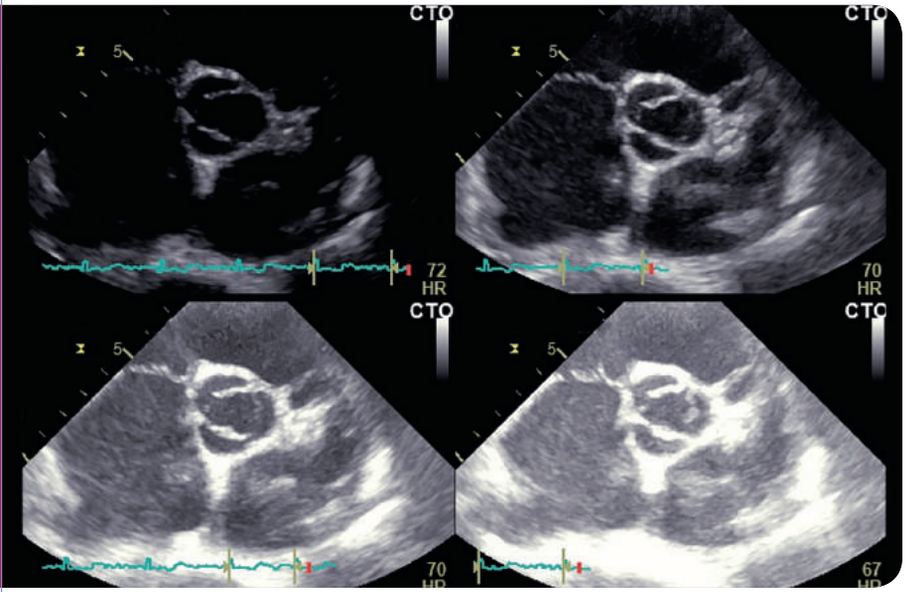
Imaging is difficult in patients with small intercostal spaces (bone) and in patients with COPD (air).

NOTES

ARTEFACTS

GAIN SETTINGS – PSAX/2D

Different gain settings in the same patient. Structures are missed when gain settings are too low (upper left). Delineation of different gray scales (tissue characteristics) is impaired when the gain is set to high (lower right).



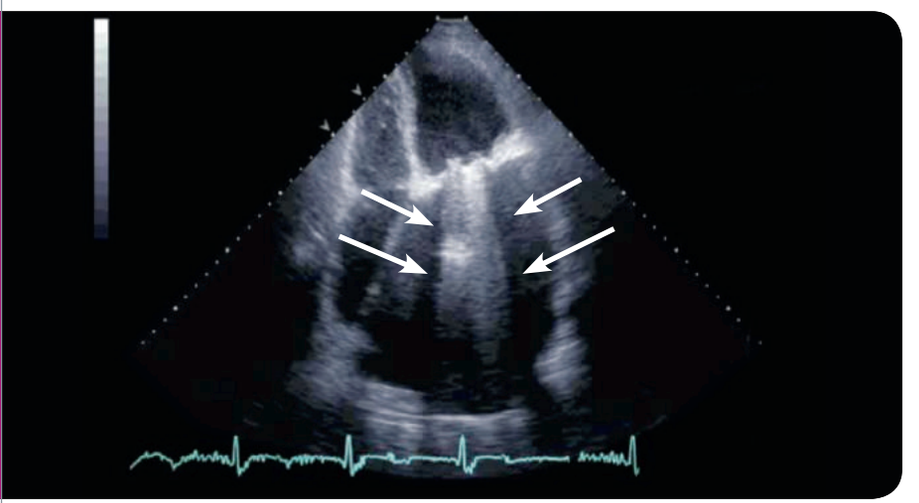
Artefacts are inconsistent.

When Do Artefacts Occur?

- Good image quality (e.g. mirror artefacts)
- Poor image quality
- Strong reflectors (e.g. calcification, prosthetic material)
- More frequent in fundamental imaging

ARTEFACT IN PROSTHETIC VALVE – apical four-chamber view/2D

Shadowing and reverberations of the left atrium caused by a mechanical mitral valve prosthesis.



Tips to Avoid Artefacts

- Know the pitfalls
- Know the anatomy
- Be cautious of strong reflections
- Use multiple views

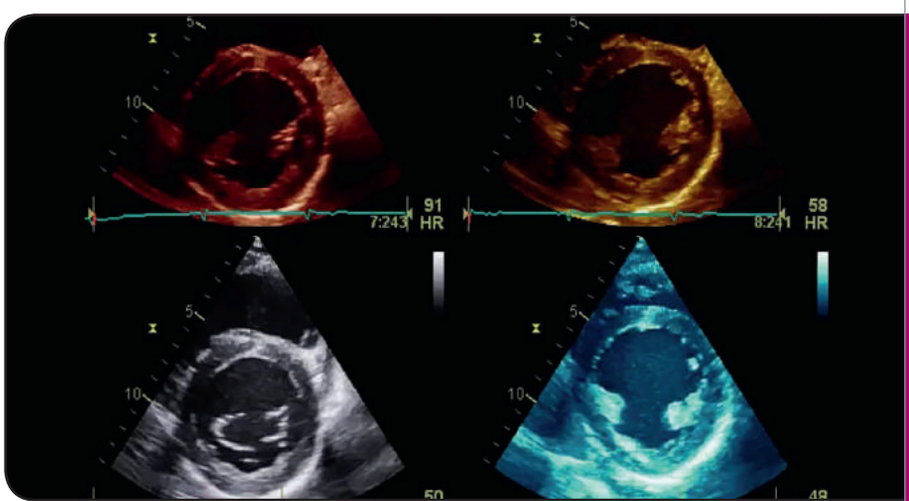
OPTIMIZING THE 2D IMAGE

Important Settings

- Gain
- Time gain compensation (TGC)
- Sector width
- Depth
- Imaging frequency
- Focus

Post-Processing

- Gray scale
- Contrast
- Compression
- Color maps



COLOR MAPS – PSAX/2D

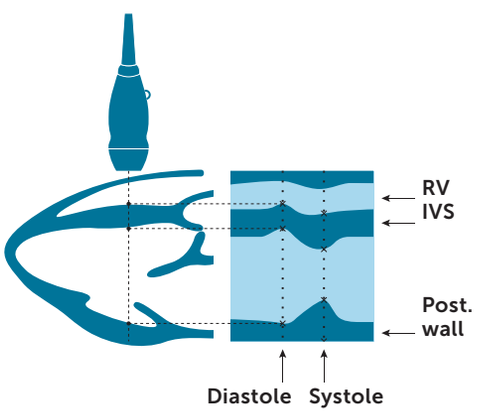
Different 2D color maps for individualized 2D display.

MMODE

MMode

Advantage

- High temporal resolution
- Good for certain measurements
- Allows measurement of time intervals
- Timing of events



Where is it used?

- Aorta/left atrium (measurements, opening of the aortic valve)
- Left/right ventricle (measurements, LV function)
- Mitral/prosthetic valve (type of valve)
- Endocarditis (motion of suspected vegetation)
- Tricuspid annular plane systolic excursion (TAPSE) for RV function
- Mitral valve (mitral stenosis)
- Mitral valve annular excursion (MAPSE) for longitudinal LV function
- Display of mid-systolic notching (flying W) of the posterior pulmonary valve cusp

NOTES

Know your echo machine!

Use predefined settings for specific situations (i.e. patients who are difficult to examine) and for specific modalities (i.e. standard echo, contrast).

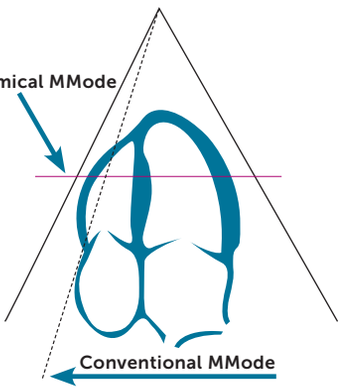
MMode has lost much of its importance, but is still valuable in certain situations.

NOTES

MMODE

Other Forms of MMode

Anatomical MMode	Freedom of axis
Color Doppler MMode	Timing of flow (i.e. flow propagation)
Tissue Doppler MMode	Myocardial function, timing of events
Curved MMode	Functional information along a variable MMode line



SPECTRAL DOPPLER

The measured velocity greatly depends on the angle between blood flow and the ultrasound beam. Always try to be as parallel to blood flow as possible. Use color Doppler to visualize the direction of flow.

Doppler Formula

$$\Delta d = 2\frac{v}{c} f_0 \cos\alpha$$

Δd = frequency alteration between S and E (=Doppler shift)(Hz)
f_0 = transmitting frequency (Hz)
v = blood flow (m/s)
c = sound propagation velocity (1550 m/s)
α = Doppler irradiation angle

The Doppler formula allows us to calculate velocities (i.e. blood and tissue), based on the Doppler shift between the send and the receive signal.

Doppler

Pulsed wave (PW) – Doppler	Low velocity (< approx. 1.5 m/s) (site specific)
Continous wave (CW) – Doppler	High velocity (> approx. 1.5 m/s) (site unspecific)
Tissue Doppler	Lower velocity, higher amplitdue

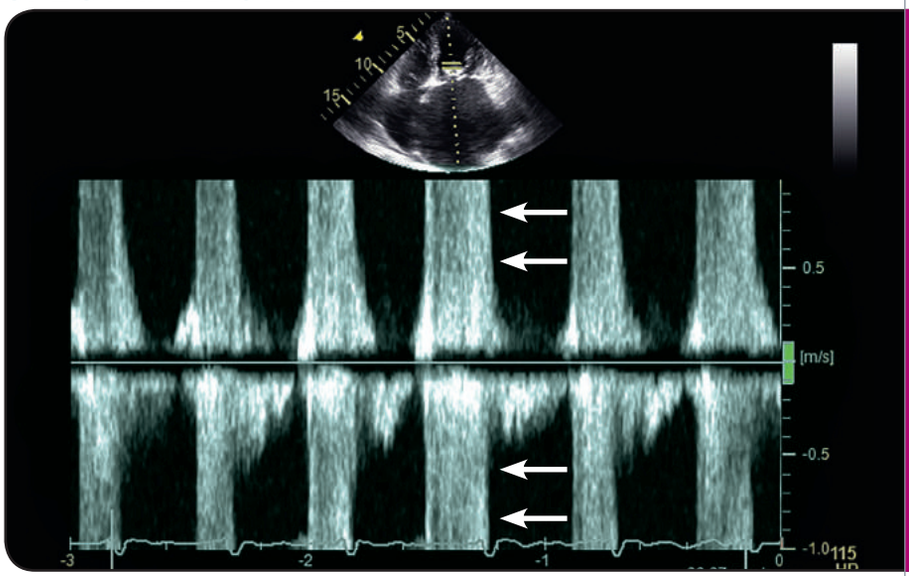
Aliasing will occur when blood flow velocity exceeds the Nyquist limit. The Nyquist limit is equal to a half of the pulse repetition frequency. Use the baseline shift to "stretch" the Nyquist limit.

Doppler Aliasing

Depends on

- Depth
- Velocity
- Width of sample volume
- Doppler frequency

SPECTRAL DOPPLER



NOTES

PW DOPPLER ALIASING – apical four-chamber view/PW MV

Pulsed-wave Doppler in a patient with mitral stenosis. The maximum velocity exceeds 2.5 m/s and exceeds the aliasing limit. Velocity profiles are noted both above and below the zero line.

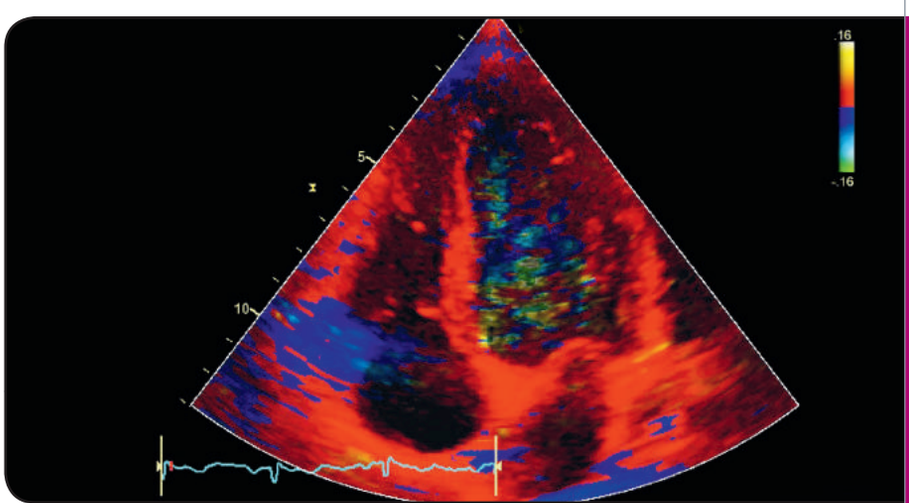
Tissue Doppler Imaging

Information

- Myocardial velocity
- Displacement
- Strain
- Strain rate

Tissue Doppler is angle dependent.

PW spectral tissue Doppler measures deformation and velocities at a specific site (within the sample volume).



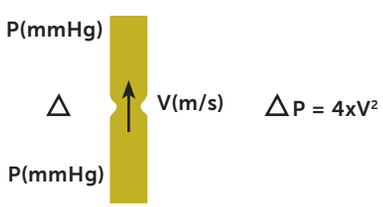
TISSUE DOPPLER – apical four-chamber view

Tissue Doppler color display of the heart during early systole. Red indicates myocardial motion towards the transducer.

FLOW DYNAMICS

Bernoulli Equation

The simplified Bernoulli equation permits easy estimation of pressure gradients from velocities.

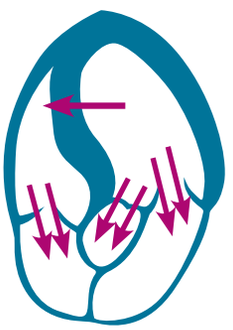


NOTES

FLOW DYNAMICS

Where Can You Apply the Bernoulli Equation in the Heart?

Direct applications (gradients)	Indirect applications (pressure decay)
Valvular stenosis	Aortic regurgitation quantification
Defects (i.e. VSD, coarctation, PDA)	Diastolic function (deceleration time)
Tricuspid regurgitation signal (sPAP)	dP/dt (contractility)
Prosthetic valves	Mitral stenosis (pressure half-time method)



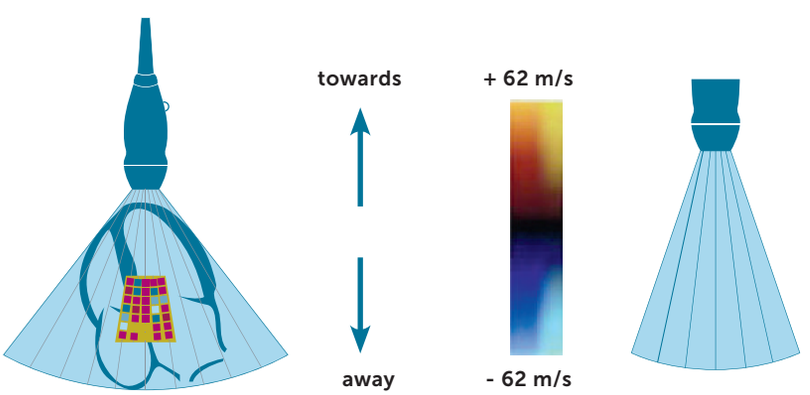
Sites where Gradients can be measured.

COLOR DOPPLER

The manner of displaying flow, flow velocities or turbulant flow is determined by the color map. Most scanners allow you to change the color map. Check your machine setings.

Color Encoding

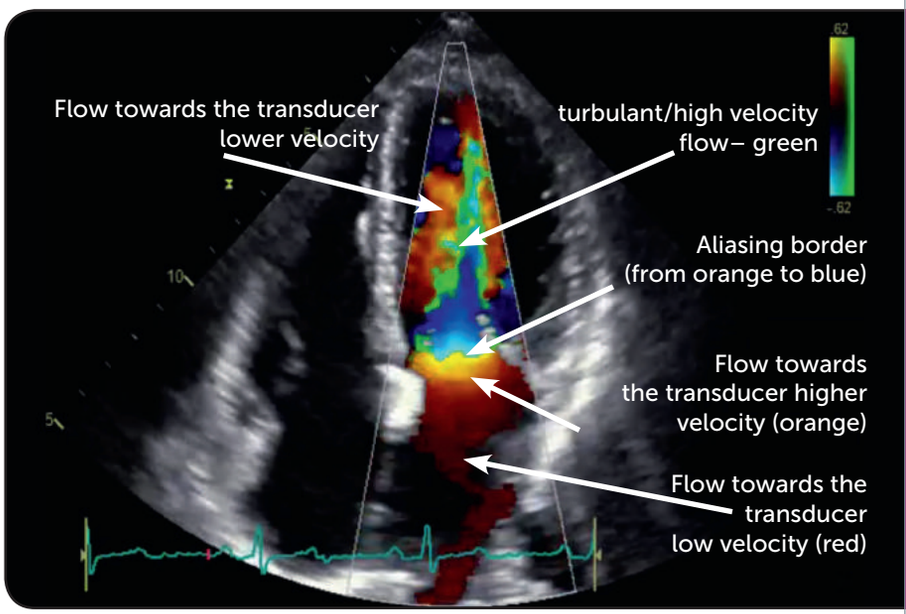
Flow towards the transducer is coded in red, and flow away from the transducer in blue.



COLOR DOPPLER

Color Doppler and Aliasing

Once the Nyquist limit is reached, the color changes abruptly (red to blue, or blue to red). The color Doppler display will show a mosaic pattern. Some color maps also display variants of velocity in green (high variants in velocities indicate turbulent flow).



COLOR DOPPLER ALIASING– apical four-chamber view/ Color Doppler

Patient with mitral stenosis. The color Doppler of mitral valve inflow shows the typical pattern of a high velocity jet. Red color denotes the direction of flow towards the transducer. The sudden change from yellow to blue depicts the region where aliasing occurs.

Color Doppler Frame Rate

- Scan line density
- Emphasis (2D vs. color)
- Sector width (2D)
- Sector width (color)
- Pulse repetition frequency
- Depth

NOTES

The phenomenon of aliasing provides good delineation of jets (e.g. PISA).

Always aim for a high color Doppler frame rate.

Try to use the same settings for quantification of regurgitation in all patients (maps, aliasing limits, color gain).

NOTES

002 How to Image

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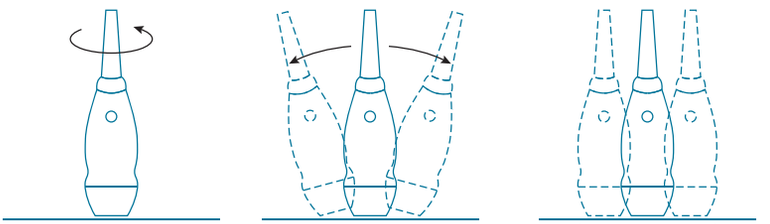
NOTES

Use enough ultrasound gel.

HOW TO MOVE THE TRANSDUCER



Displacement | Rotation | Angulation

IMAGING WINDOWS

Use as many views as possible, including atypical views. Always image so that the pathology of interest is seen best.

Parasternal	2nd–4th intercostal space left sternal border
Apical	4th – 5th intercostal space, lateral
Subcostal	Below xiphoid
Right parasternal	2nd–4th intercostal space, right sternal border
Suprasternal	Suprasternal notch

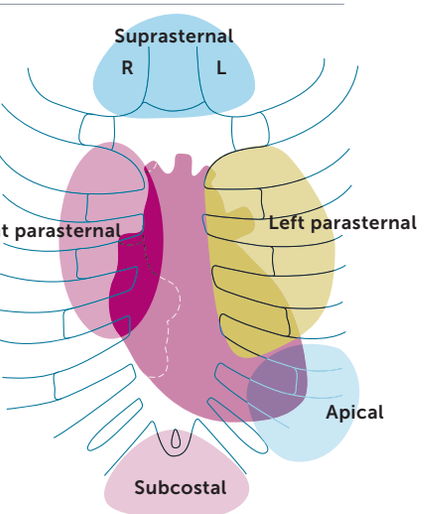
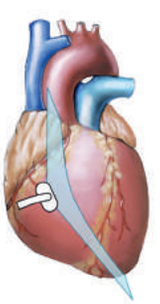
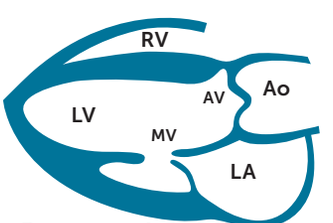


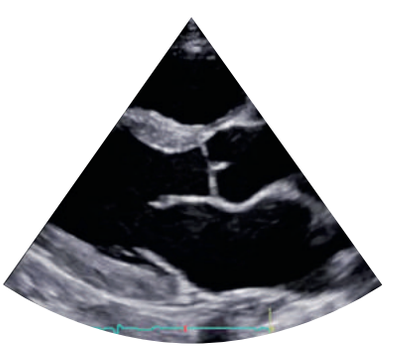
IMAGE VIEW

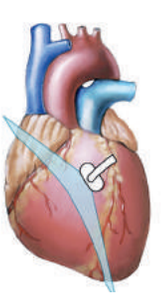
Parasternal Long-Axis Views

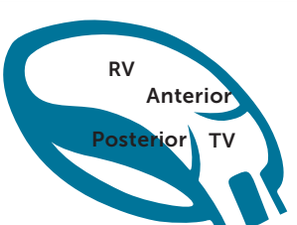




Parasternal long-axis view







Right parasternal long axis

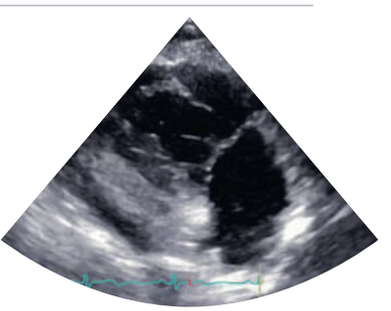


IMAGE VIEW

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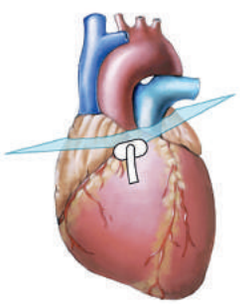
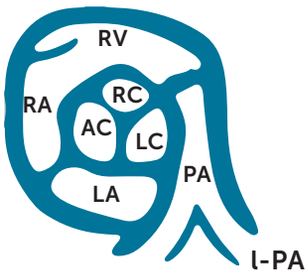
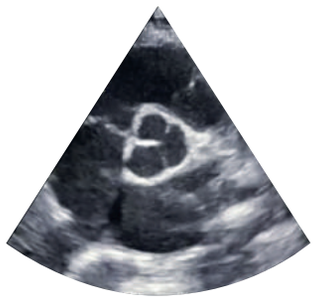
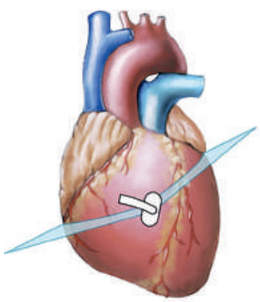
Parasternal Short-Axis Aiews

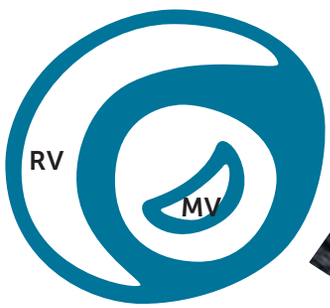


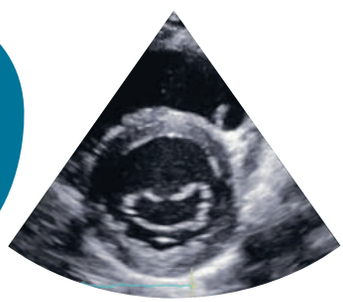




Parasternal short axis – base

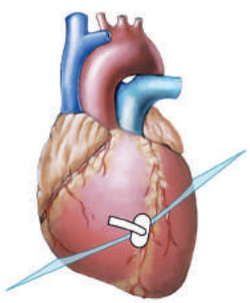


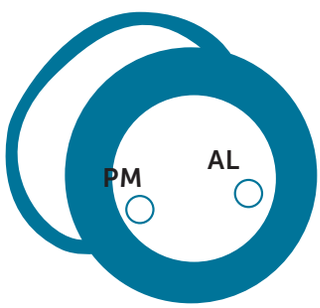


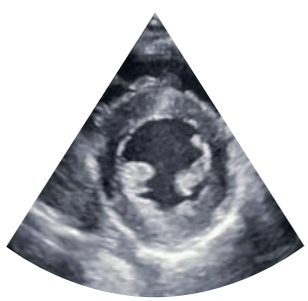


Parasternal short axis – mitral valve

Move down one intercostal space to obtain good image quality and a “more” spherical (round) configuration of the distal parts of the left ventricle.







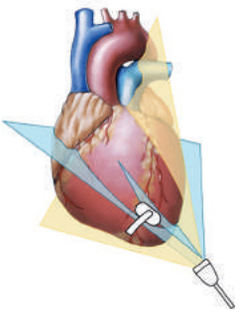
Parasternal short axis – mid-ventricle

NOTES

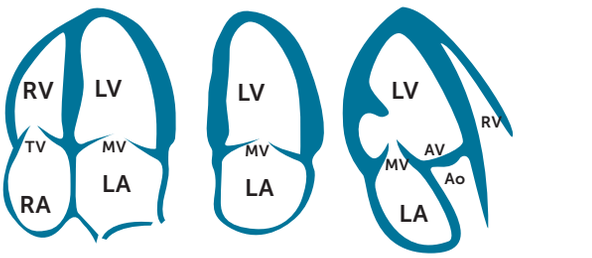
Use a medial position (A) to visualize the lateral wall of the LV and a lateral position (B) to visualize the RV.

IMAGE VIEW

Apical Views

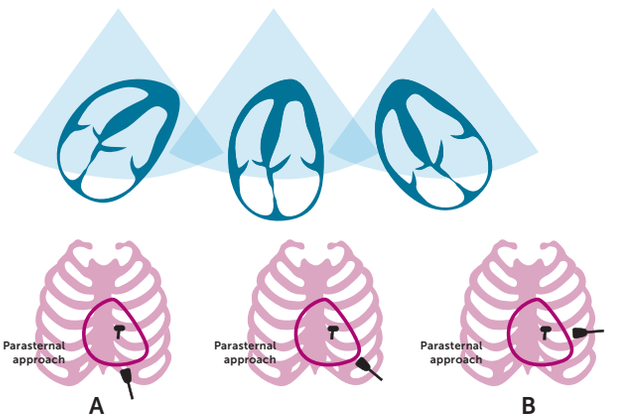


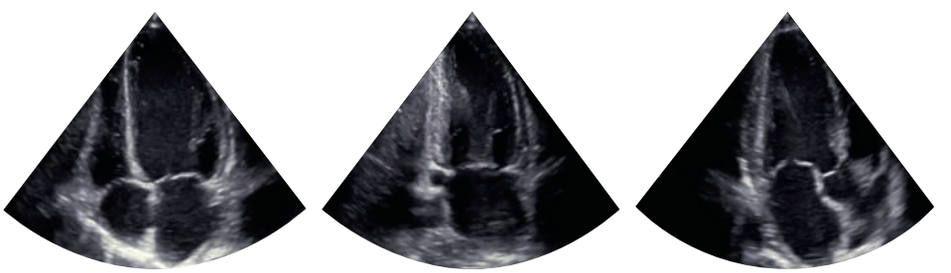
Rotate counterclockwise



4-chamber view 2-chamber view 3-chamber view

The orientation of the septum indicates whether you are in lateral or medial position relative to the true apex. Use all views to fully examine all aspects of the left and right ventricle.





Four-chamber view Two-chamber view Three-chamber view

Orientation of the Apical Views

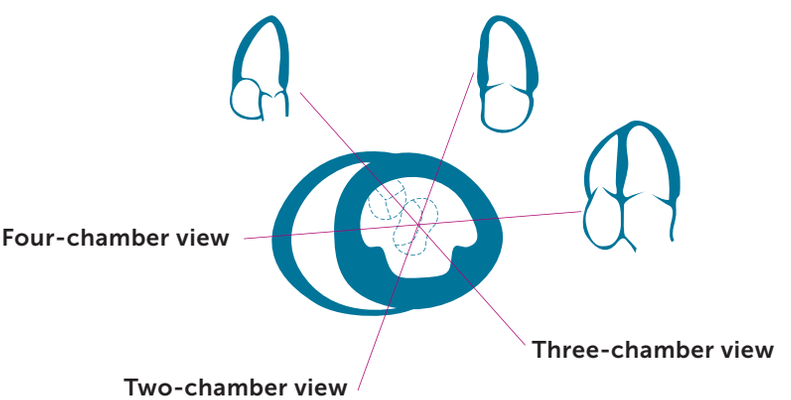
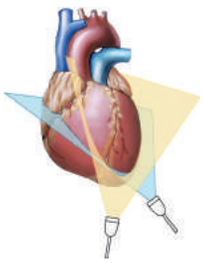
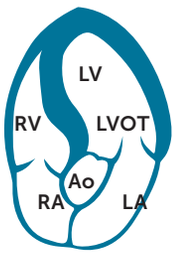
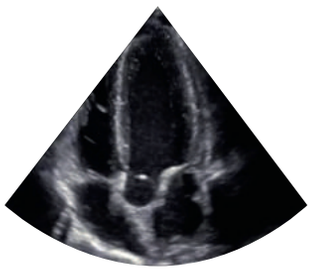


IMAGE VIEW

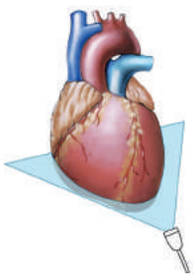
Five-chamber view

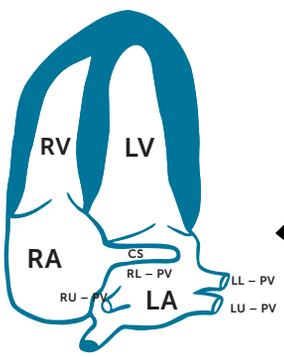


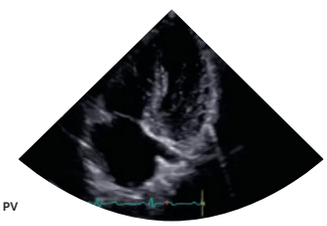




Coronary sinus view

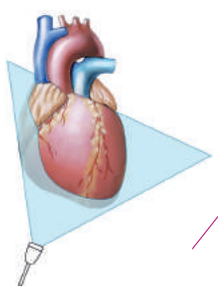


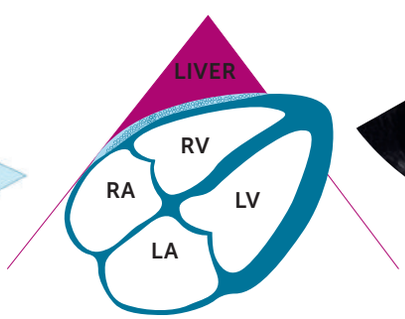


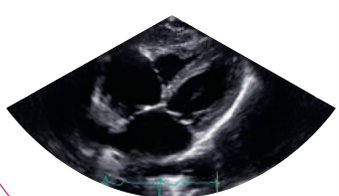


Subcostal Views

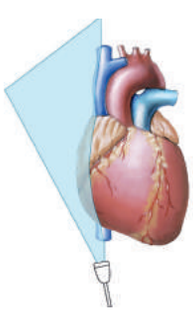
Subcostal four-chamber view

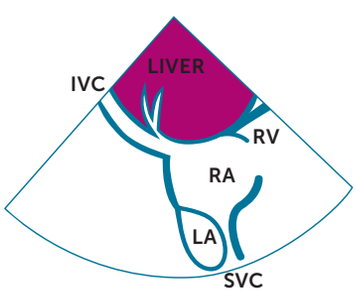


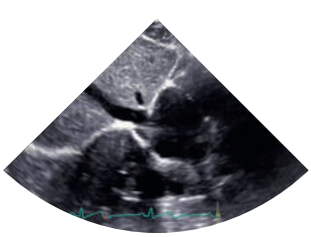




Inferior vena cava view (rotate counterclockwise)







NOTES

The five-chamber view shows the anterior portions of the interventricular septum.

Avoid foreshortening; place the transducer as lateral and caudal as possible.

Abdominal gas may obscure the apex on the subcostal view.

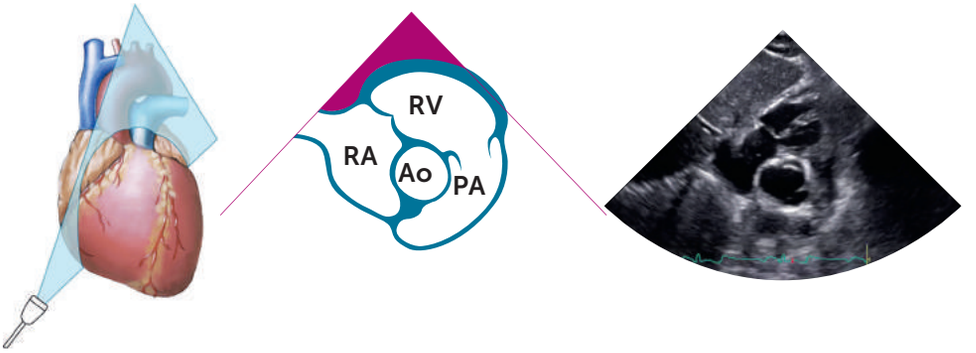
In some patients it may be possible to see the superior vena cava on this view.

NOTES

IMAGE VIEW

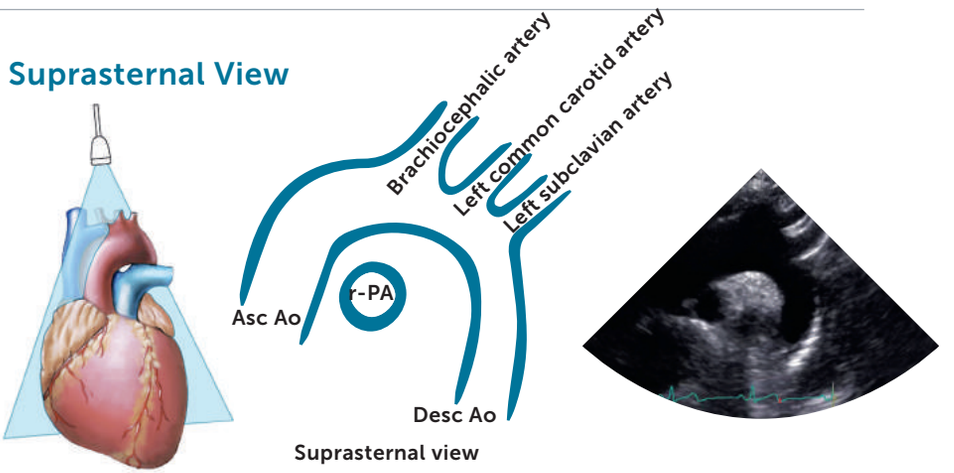
Obtain subcostal views in all patients.

Subcostal short-axis view (rotate clockwise)



The suprasternal view allows you to detect coarctation, a persistent Botalli's duct, or aortic dissection, as well as quantify retrograde flow in the aorta (aortic regurgitation).

Suprasternal View

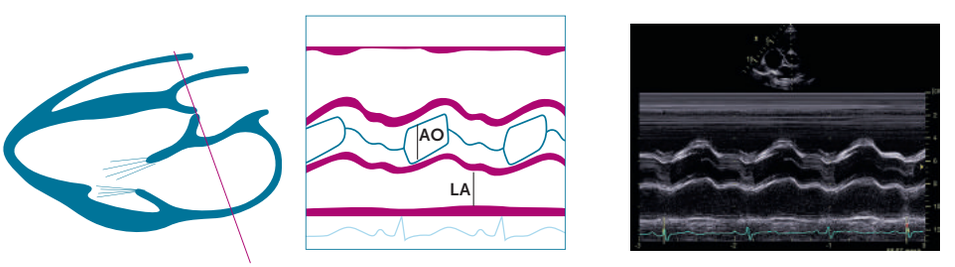


Suprasternal view

MMode – LA is measured in its largest extension at end systole. The dimensions of the aorta are measured at end diastole, shortly before the aortic valve opens.

MMode

MMode aorta/left atrium



Measure the end-diastolic diameter where the LV is largest, shortly before contraction starts (beginning of the QRS complex).

MMode left ventricle

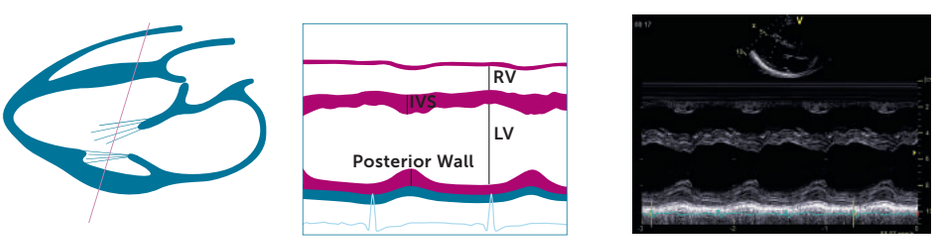


IMAGE VIEW

Reference Values – MMode

Aorta (mm)	< 40	LVEDD (mm)	42 – 59
Left atrium (mm)	30 – 40	Posterior wall (mm)	6 – 10
IVS (mm)	6 – 10	Fractional shortening (%)	> 25
Tricuspid Annular Plane Systolic Excursion (TAPSE)	> 16 mm	MAPSE (longitudinal LV function)	> 12 mm

Reference Values – Doppler

Aortic valve velocity (m/sec)	CW	0.9 – 1.7
LVOT velocity (m/sec)	PW	< 1.3
Pulmonary valve velocity (m/sec)	CW	0.5 – 1.0
Tricuspid valve	PW	0.3 – 0.7
Tricuspid regurgitation (m/sec)	CW	1.7– 2.3
E wave (m/sec)	PW	< 1.3
Mitral annulus e' (cm/sec)	TDI PW	0.8 – 1.3
Right ventricular lateral wall (cm/sec)	TDI PW	12.2 (41 – 60a)/ 10.4 (>60a)

Color Doppler

- Optimize the 2D image before you use color Doppler
- Look for aliasing to detect jets
- Reduce pulse repetition frequency (PRF) to detect low velocity flow (e.g. ASD, PFO)
- Use higher frame rates
- Use multiple views
- Use color flow as a guide for CW/PW sample volume

NOTES

Optimize the 2D image before using color Doppler.

NOTES

ABBREVIATIONS

AC = acoronary cusp
AL = anterolateral papillary muscle
Ao = aorta
Asc Ao = ascending aorta
AV= aortic valve

CS= coronary sinus

Desc Ao = descending aorta

IVC = inferior vena cava
IVS = interventricular septum

LA= left atrium
LC= left-coronary cusp
LL-PV = left-lower pulmonary vein
l-PA= left pulmonary artery
LU-PV= left-upper pulmonary vein
LV = left ventricle
LVOT = left ventricular outflow tract

MV = mitral valve

PA = Pulmonary artery
PM= posteriomedial papillary muscle

RC = right-coronary cusp
RL-PV= right lower pulmonary vein
r-PA = right pulmonary artery
RU - PV= right upper pulmonary vein
RV= right ventricle

SVC = superior vena cava

TV = tricuspid valve

003 // Heart Chambers and Walls

CONTENTS

NOTES

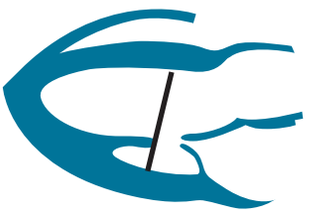
Only use MMode values when your line of interrogation is perpendicular to the LV cavity and walls.

Measure distances between the endocardial borders, not the pericardium (lateral).

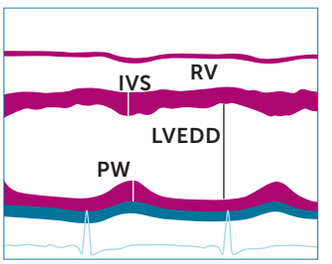
THE LEFT VENTRICLE

Quantification of LV Diameter

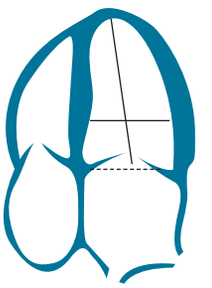
PLAX view



MMODE

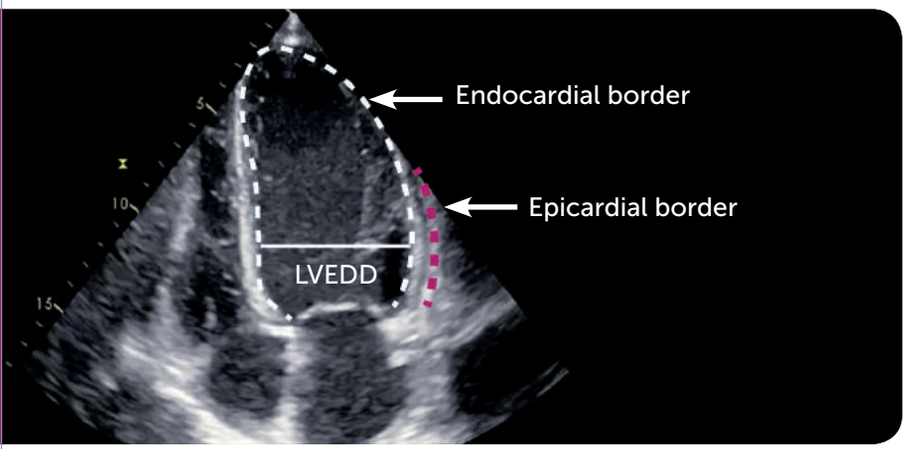


Four-chamber



LEFT VENTRICULAR DIAMETER – apical four chamber view/2D

The endiastolic diameter of the left ventricle (LVEDD) is measured from the lateral to the septal border of the endocardium between the tips of the mitral valve and the papillary muscle at end diastole. If a septal bulge is present, measure more basally.



There must be agreement between M-Mode and 2 D measurements in regard of LV size.

Left Ventricular End-Diastolic (LVED) Diameter – Reference Values

	♂	♀
Normal (mm)	42 – 59	39 – 53
Mild (mm)	60 – 63	54 – 57
Moderate (mm)	64 – 68	58 – 61
Severe (mm)	≥ 69	≥ 62

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Normal chamber size increases with body surface area (and body size).

LVED Diameter/Body Surface Area (BSA) – Reference Values

	♂	♀
Normal (cm/m²)	2.2 – 3.1	2.4 – 3.2
Mild (cm/m²)	3.2 – 3.4	3.3 – 3.4
Moderate (cm/m²)	3.5 – 3.6	3.5 – 3.7
Severe (cm/m²)	≥ 3.7	≥ 3.8

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THE LEFT VENTRICLE

LV End-Diastolic Volume (4-chamber view) – Reference Values

	♂	♀
Normal (mL)	67 – 155	56 – 104
Mild (mL)	156 – 178	105 – 117
Moderate (mL)	179 – 200	118 – 130
Severe (mL)	≥ 201	≥ 131

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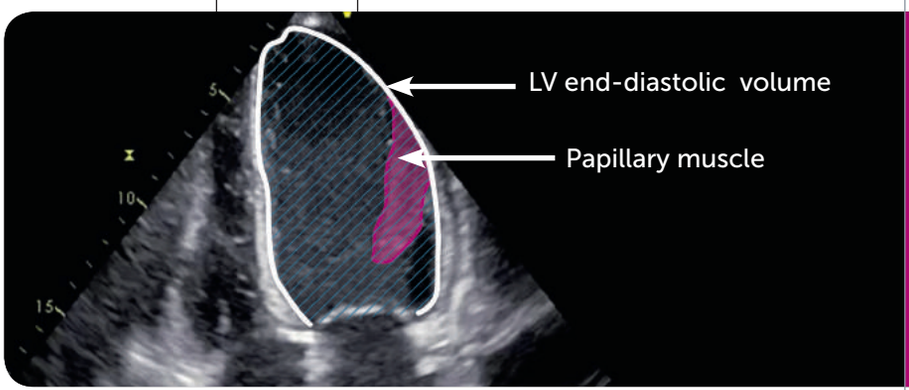

SIMPSON METHOD – apical four-chamber view/2D

Tracing of the endocardial border in end-diastole to quantify end-diastolic volume (LVEDV). For biplane quantification, be sure that the length of the ventricle matches on the four- and two-chamber view.

LV Systolic Volume (4-chamber view) – Reference Values

	♂	♀
Normal (mL)	22 – 58	19 – 49
Mild (mL)	59 – 70	50 – 59
Moderate (mL)	71 – 82	60 – 69
Severe (mL)	≥ 83	≥ 70

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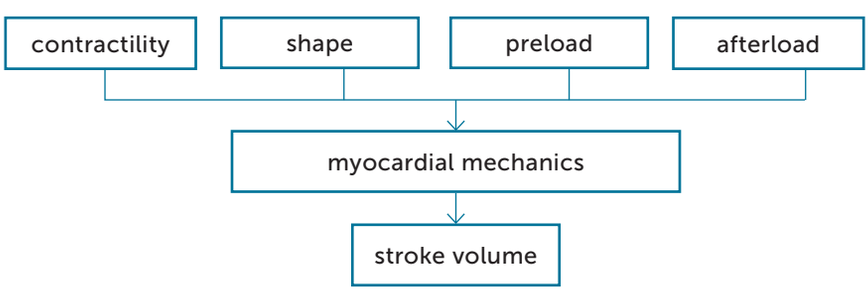
Do not trace the papillary muscles. Their volumes should be included in the calculation.

Pathophysiology

Principles of LV Function:

Factors influencing ejection fraction/stroke volume



A reduction of longitudinal function is an early marker of LV dysfunction.

NOTES

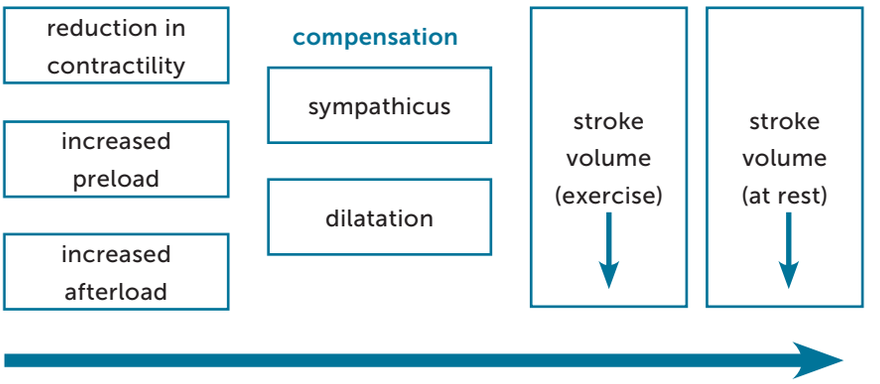
Volume measurements are superior to diameter and area measurements.

NOTES

Contractility, preload and afterload influence myocardial function. A reduction in contractility is initially compensated by activation of the sympathetic nervous system (compensatory increase in heart rate and contractility) as well as dilatation of the left ventricle. Stroke volume is kept adequate at rest, but cannot adapt to exercise (reduced functional reserve). In end-stage heart failure, stroke volume is also reduced at rest (decompensation).

THE LEFT VENTRICLE

Pathophysiology of LV Failure: Cascade and Compensatory Mechanisms



LEFT VENTRICULAR FUNCTION

LV function and (longitudinal) contractility may be reduced despite a "normal" ejection fraction, especially in patients with small ventricles.

Parameters of LV Function

- Fractional shortening
- Cardiac output/index
- "Eyeballing" of LV function
- Deformation parameters (strain, strain rate)
- Ejection fraction (EF) – Simpson method
- Contractility (dp/dt)
- Stroke volume
- Tei index
- TDI velocity of the myocardium
- MAPSE (mitral annular plane systolic excursion)

Fractional shortening is a rough estimate of global left ventricular function. Do not use the Teichhholz formula to derive the ejection fraction from these values.

Fractional Shortening – Reference Values

	♂	♀
Normal	25 – 43%	27 – 45%
Mild	20 – 24%	22 – 26%
Moderate	15 – 19%	17 – 21%
Severe	≤ 14%	≤ 16%

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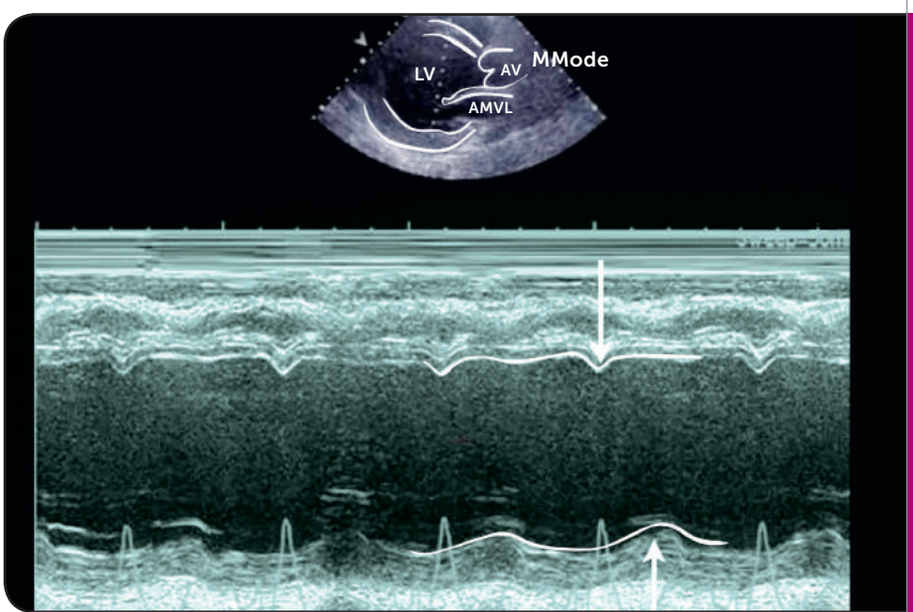
LEFT VENTRICULAR FUNCTION

Fractional Shortening – Contraindications

- LBBB/dyssynchrony/pacemaker
- Abnormal septal motion
- Regional wall motion abnormalities
- Inadequate (oblique) MMode orientation
- Poor image quality
- "Pseudo-shortening" of the LV (very small ventricle)

NOTES

In these settings, fractional shortening cause overestimation or underestimation of left ventricular function.



LEFT BUNDLE BRANCH BLOCK – PLAX/Mmode

Mmode image of the left ventricle displaying dys-synchrony in the left bundle branch block. Early systolic inward motion occurs dissociated from the motion of the posterolateral wall. It is not possible to define end-diastole and end-systole to determine fractional shortening. Increase your sweep speed to best visualize dyssynchrony of the septum. Tissue Doppler imaging may be helpful to delineate the time of contraction.

Ejection Fraction – Simpson Method

Normal	> 55 %
Mild	45 – 54 %
Moderate	30 – 44 %
Severe	< 30%

$$EF = \frac{ED_{vol} - ES_{vol}}{ED_{vol}} \times 100$$

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1) Ejection fractions tend to be higher in small ventricles. 2) Athletes often have ejection fractions in the low normal range.
3) Ejection fraction does not predict exercise capacity or functional reserve.
4) Ejection fraction is super-normal in patients with reduced afterload (e.g. mitral regurgitation).

Stroke Volume, Cardiac Output, Cardiac Index – Reference Values

	Rest	Exercise
Stroke volume	70 – 110mL	80 – 130mL
Cardiac output	5 – 8.5 L/min	10 – 17 L/min
Cardiac index	> 2.5 L/min/m^2	> 5 L/min/m^2

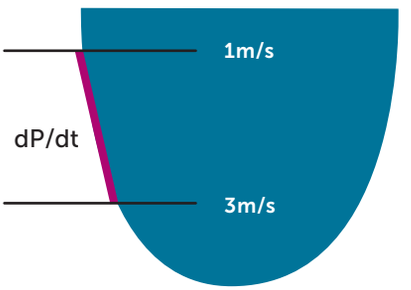
The calculation of these parameters is very highly dependent on correct measurement of LVOT width.

NOTES

A rough estimate of contractility can also be obtained by eyeballing the slope of the MR curve.

LEFT VENTRICULAR FUNCTION

Measuring Contractility – dP/dt

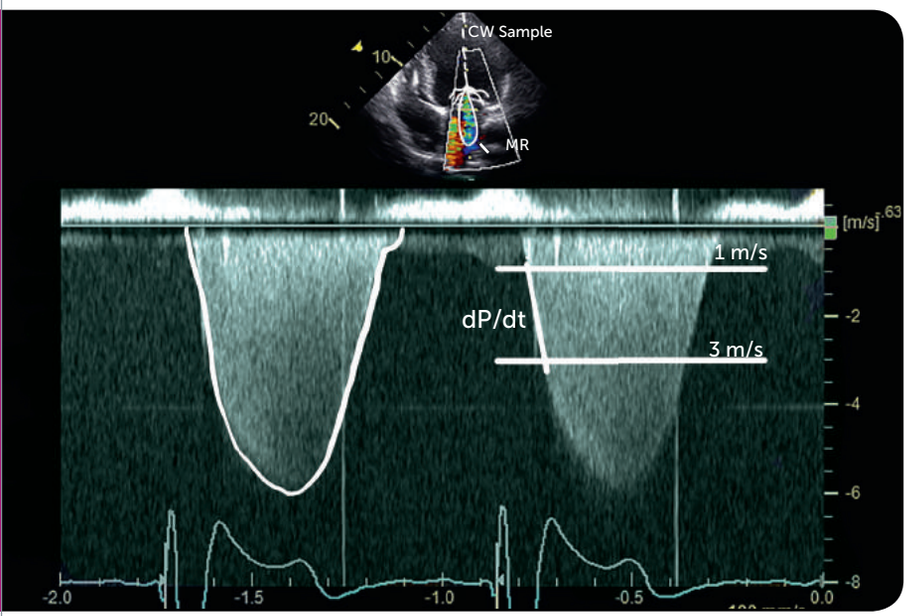


Normal	> 1200 mmHg/sec
Borderline	800 – 1200 mmHg/sec
Reduced	< 800 mmHg/sec
Severely reduced	< 500 mmHg/sec

Limitations: Mitral regurgitation (MR) signal needed, inexact, not completely load independent

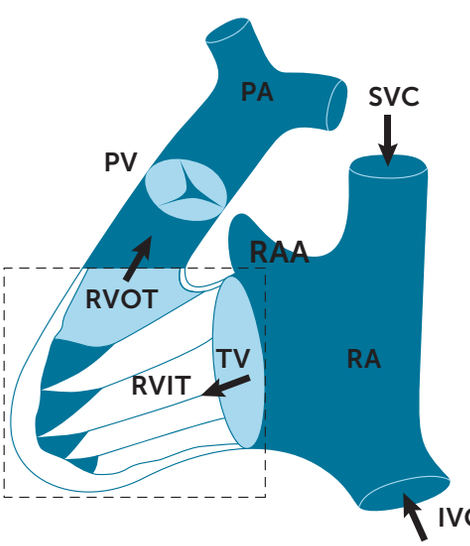
DP/DT – apical four-chamber view/CW Doppler mitral regurgitation

The dP/dt is calculated by measuring the slope of the initial mitral regurgitation signal between 1 m/s and 3 m/s.



THE RIGHT VENTRICLE

The geometry of the right ventricle is more complex than that of the left ventricle: it resembles a bagpipe.



Characteristics of the RV

- The wall is thinner (< 5 mm)
- Moderator band
- Strongly trabeculated
- "Wrapped around" the left ventricle

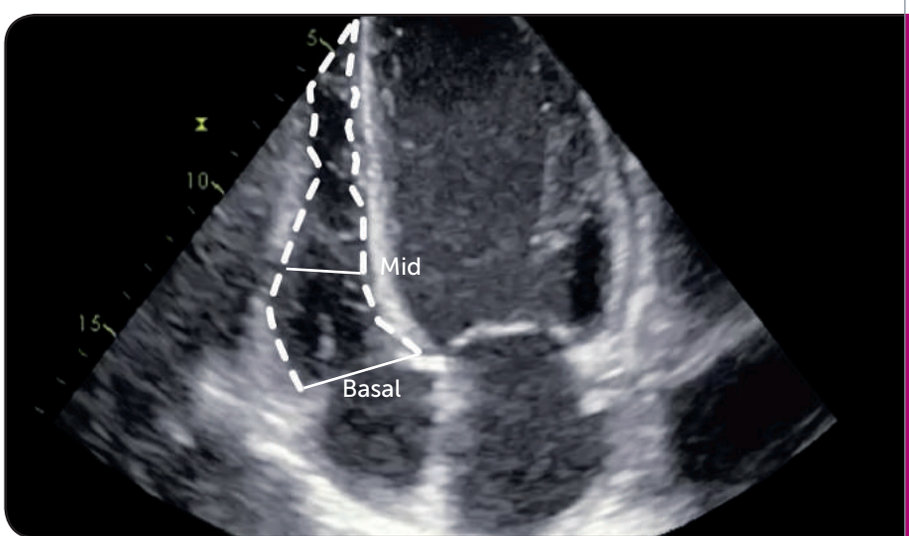
PV = pulmonic valve
RAA = right atrial appendage
RVIT = right ventricular inflow tract
RVOT = right ventricular outflow tract

THE RIGHT VENTRICLE

Measurements of the Right Ventricle

	Reference Range	Slightly Abnormal	Moderately Abnormal	Severely Abnormal
RV dimensions				
Basal RV diameter (mm)	20-28	29-33	34-38	≥ 39
Mid RV diameter (mm)	27-33	34-37	38-41	≥ 42
Base-to-apex length (mm)	71–79	80-85	86-91	≥ 92
RVOT diameter				
Above pulmonary valve (mm)	17-23	24-27	28-31	≥ 32
PA diameter				
Below pulmonary valve (mm)	15-21	22-25	26-29	≥ 30

ESC/ASE 2005



RIGHT VENTRICULAR DIAMETER – apical four-chamber view/2D

Measurement of the basal and mid right ventricular diameter in end-diastole. To enhance accuracy use a four-chamber view that is optimized for the right ventricle. The right ventricular diameter will be overestimated when the ventricle is foreshortened.

Right Ventricular Systolic Function

Tricuspid annular plane systolic excursion (TAPSE)	> 16 – 18 mm
TDI maximum velocity at the basal lateral wall (S')	> 10 cm/s
PW Doppler myocardial performance index	> 0.4
Tissue Doppler myocardial performance index	> 0.55

NOTES

RV diameters appear larger when the transducer is too far cranial.

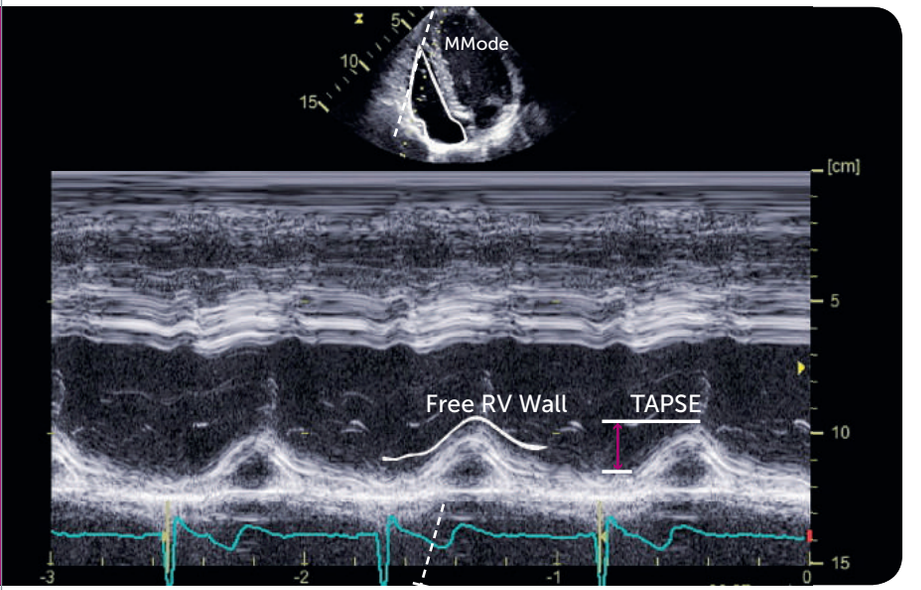
Speckle-trackingderived longitudinal strain of the free right ventricular wall may provide additional information to quantify right ventricular function. It also reflects RV function in the apical segments.

NOTES

TAPSE – apical four-chamber view/Mmode RV wall

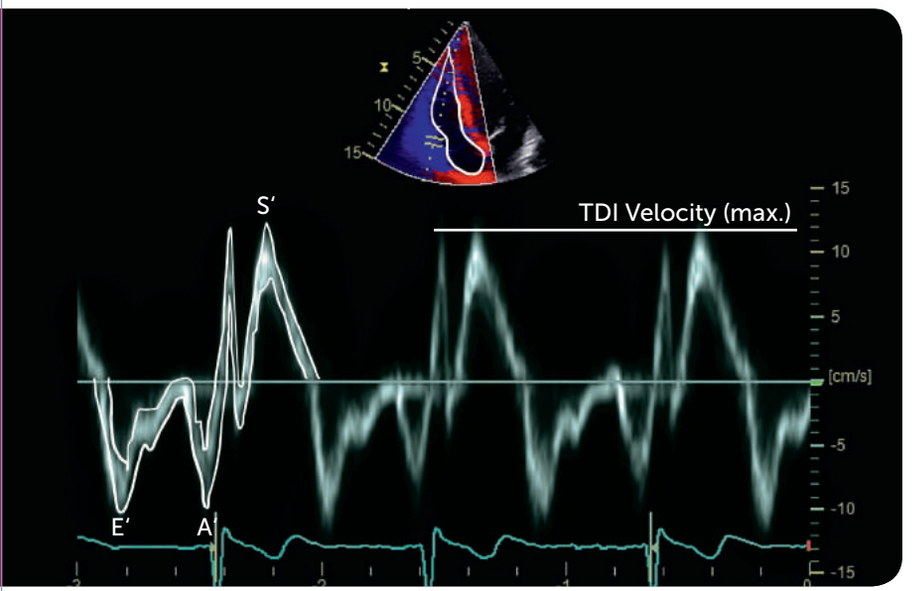
TAPSE is measured by placing the MMode through the tricuspid annulus and measuring the displacement from diastole to systole.

THE RIGHT VENTRICLE



TISSUE DOPPLER IMAGING OF THE RIGHT VENTRICLE – apical four-chamber view/TDI PW RV wall

The sample volume is placed in the basal lateral wall of the right ventricle. S' denotes RV longitudinal function.



Assessment of RV diastolic dysfunction is rarely used in clinical practice.

RV Diastolic Function

E/A ratio	< 0.8 or > 2.1
E/e'	> 6
Deceleration time (ms)	< 120ms

Always look for the cause of RV dilatation.

Causes of RV Dilatation

- Dilated cardiomyopathy
- Right heart infarction
- Myocarditis
- Pulmonary embolism/hypertension
- Right ventricular dysplasia
- RV volume overload (e.g. atrium septal defect, pulmonic/tricuspid regurgitation)
- Athletes (normal reaction to training)

THE RIGHT VENTRICLE

Fractional Area Change (FAC)– Reference Values

Normal	32-60 %
Mild	25 – 31 %
Moderate	18 – 24 %
Severe	≤ 17 %

Trace the RV contour in diastole and systole in an optimized 4-chamber view to obtain the areas. Calculate the percentage of change.
(RV area end-diastole – RV area end-systole)/RV area end-diastole *100

ESC/ASE 2005

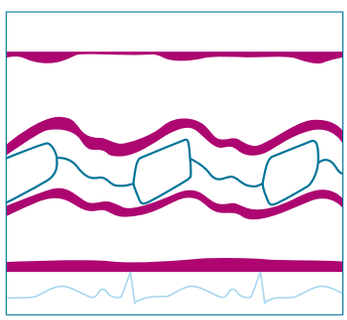
NOTES

Tracing of RV contours may be difficult (trabeculations, thin wall).

THE LEFT ATRIUM

MMode Measurements of LA – Reference Values

	♂	♀
Normal (mm)	30 – 40	27 – 38
Mild (mm)	41 – 46	39 – 42
Moderate (mm)	47 – 52	43 – 46
Severe (mm)	≥ 52	≥ 47



LA size and volume predict adverse events (i.e. afib, stroke) and constitute a marker of disease severity.

LA Length (4-Chamber View)– Reference Values

♀	Reference Range	Slightly Abnormal	Moderately Abnormal	Severely Abnormal
LA diameter (mm)	27–38	39–42	43–46	≥ 47
LA diameter/ BSA (mm/m2)	15–23	24–26	27–29	≥ 30
♂	Reference Range	Slightly Abnormal	Moderately Abnormal	Severely Abnormal
LA diameter (mm)	30–40	41–46	47–52	≥ 52
LA diameter/ BSA (mm/m2)	15–23	24–26	27–29	≥ 32

ESC/ASE 2005

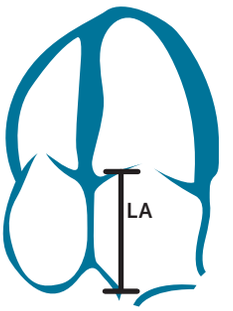
Measure the length of the left atrium parallel to the interatrial septum.

NOTES

THE LEFT ATRIUM

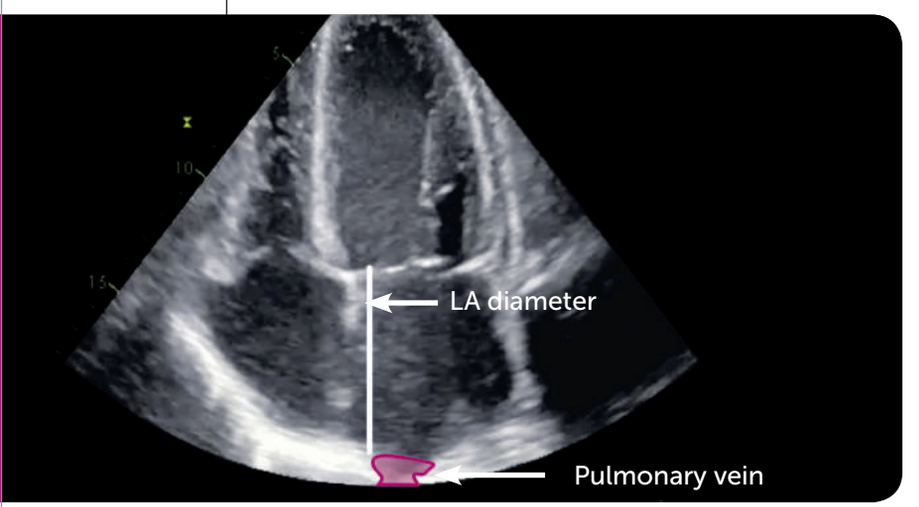
LA Length – A Practical Scale

Normal (mm)	≤ 50
Mild (mm)	51 – 60
Moderate (mm)	61 – 70
Severe (mm)	> 70



LEFT ATRIAL LENGTH –apical four-chamber view/2D

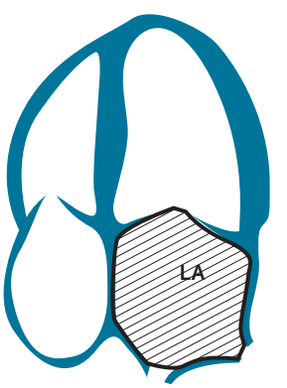
The length of the left atrium is measured from the mitral annular plane to the roof of the left atrium parallel to the interatrial septum in end-systole. Be sure not to measure into the pulmonary vein. This measurement only provides a rough estimate of left atrial size.



LA Area – Reference Values

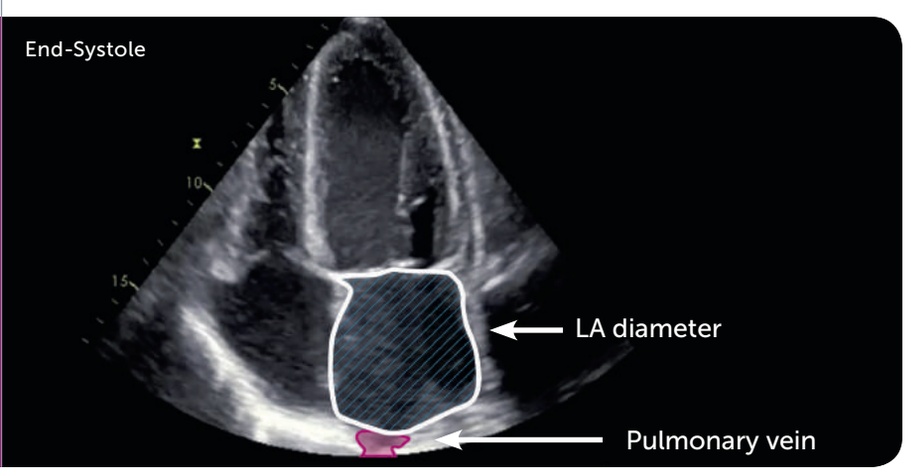
Normal (cm^2)	≤ 20
Mild (cm^2)	20 – 30
Moderate (cm^2)	30 – 40
Severe (cm^2)	> 40

ESC/ASE 2005



LEFT ATRIAL AREA –apical four-chamber view/2D

Tracing of LA area is performed in LA systole. The left atrial appendage (if visible), pulmonary veins, and interatrial aneurysms (if present) are spared.



THE LEFT ATRIUM

LA Volume – Reference Values

$$V = \frac{8\pi}{3} \times \frac{A_{4c} \times A_{2c}}{L}$$

	LA Volume (Area Length Method) – Reference Values		Practical Scale
	♂	♀	
Normal (mL)	18 – 58	22 – 52	<50
Mild (mL)	59 – 68	53 – 62	50 – 70
Moderate (mL)	69 – 78	63 – 72	70 – 90
Severe (mL)	≥ 79	≥ 73	> 90

Pittfalls in Calculating LA Volume

- Inclusion of pulmonary veins
- Tenting area of MV
- Alignment/atrial foreshortening
- Lateral resolution
- Measurement not performed at end systole
- Oblique view of the LA
- Foreshortening of the atrium

Parameters of LA Function

- Doppler (MV inflow)
- Area changes systolic/diastolic
- Pulmonary vein flow
- TDI/2D strain

NOTES

LA volume measurements are superior to MMode and 2D diameter measurements. LA volumes > 200 ml denote very severe atrial dilatation (LA volumes may even exceed 1 liter).

Optimize the 4-chamber view specifically to the left atrium to obtain best results.

In most cases the Doppler (MV inflow) signal is sufficient to estimate LA function. Functional assessment of the LA is still a subject of ongoing research.

The area under the A-wave correlates with the ejection of blood from the left atrium (atrial contraction) into the left ventricle (booster pump function). A small A-wave either means there is poor contraction, high resistance to filling, or the greater part of the blood has already entered the ventricle during the passive filling phase.

NOTES

THE LEFT ATRIUM

Causes of LA Dilatation

- Diastolic dysfunction
- Mitral stenosis/regurgitation
- Aortic stenosis
- *Restrictive/hypertrophic cardiomyopathy*
- Atrial fibrillation
- Impaired LV function

The most frequent cause of LA dilatation in the adult is hypertension.

THE RIGHT ATRIUM

Causes of RA Dilatation

- Pulmonary hypertension
- Tricuspid valve disease
- Right ventricular failure
- Atrial fibrillation

The right atrium can be stretched in length when the left atrium expands. expands ands.

RA Length – Reference Values (4 chamber view)

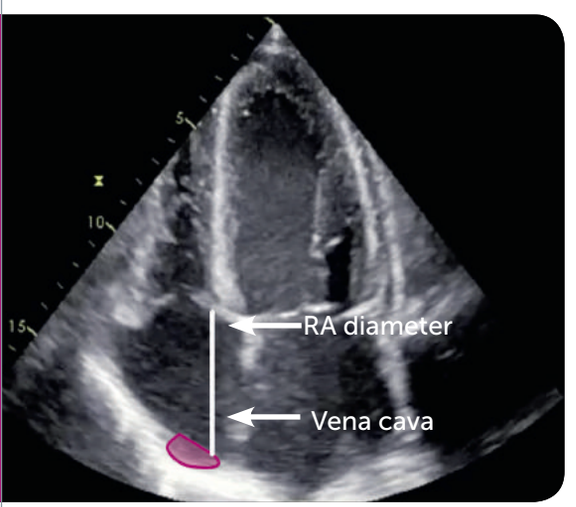
♂ ♀	Reference Range	Slightly Abnormal	Moderately Abnormal	Severely Abnormal
RA minor axis diameter (mm)	29–45	46–49	50–54	≥ 55
RA minor axis diameter/BSA (mm/m²)	17–25	26–28	29–31	≥ 32

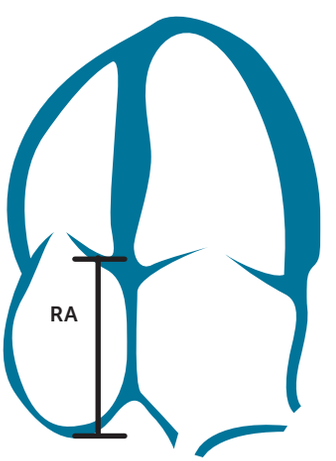
ESC/ASE 2005

The RA is generally smaller than the LA. However, for practical reasons you may also apply the simple grading scale shown for the left atrium.

RIGHT ATRIAL LENGTH – apical four-chamber view/2D

The length of the right atrium is measured from the tricuspid annular plane to the roof of the right atrium, parallel to the interatrial septum, in end-systole. Be sure not to measure into the vena cava.





THE RIGHT ATRIUM

Coronary Sinus

Reference value = 4 – 8 mm (upper limit 15 mm)

Causes of a dilated coronary sinus:

- Elevated RA pressure
- V. cava sin. persistens,
- Malformation (aneurysm/diverticula), – unroofed coronary sinus

Inferior Vena Cava

Size < 17 mm, Inspiratory collapse ≥ 50%
IVC size varies greatly, depending on fluid status and central venous pressure

Causes of IVC dilatation:

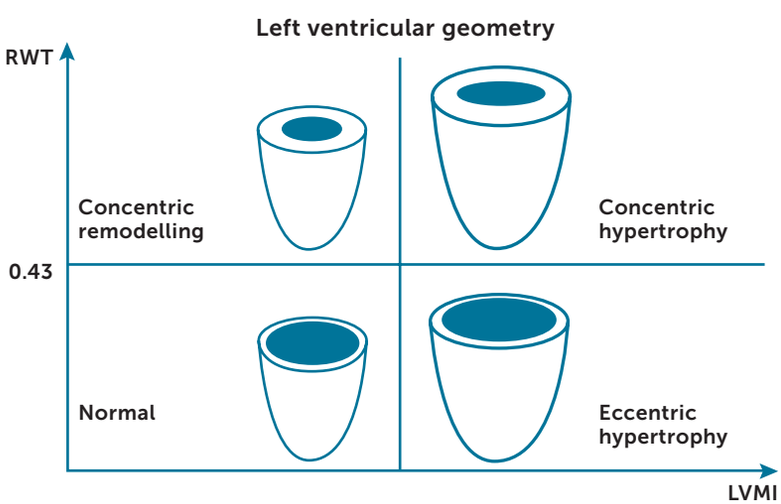
- Tricuspid regurgitation
- Pericardial tamponade constriction
- Restrictive cardiomyopathy
- Right heart failure
- Scimitar syndrome (anomalous pulmonary venous return into the IVC)

IVC allows estimation of RA pressure. Dilated IVC without respiratory changes indicates elevated RA pressure (> 15 mmHg).

A large inferior vena cava does not always indicate a medical condition. Some patients simply have a large inferior vena cava (even in the absence of elevated RA pressure).

LEFT VENTRICULAR HYPERTROPHY

Forms of Left Ventricular Hypertrophy



LVMI (left ventricular mass index) = LV mass/BSA
Reference adapted from Ganau et al. JACC 1992

Relative Wall Thickness (RWT)
Normal values 22 – 42 %

$$RWT = \frac{2 \times PWT}{LVID}$$

Most patients with hypertension have concentric LVH.

NOTES

NOTES

Potential problems: the measurements were not performed at end diastole (2D), RV structures interfere with the measurement, the shape of the IVS (basal septal bulge), incorrect image orientation (non-perpendicular).

LEFT VENTRICULAR HYPERTROPHY

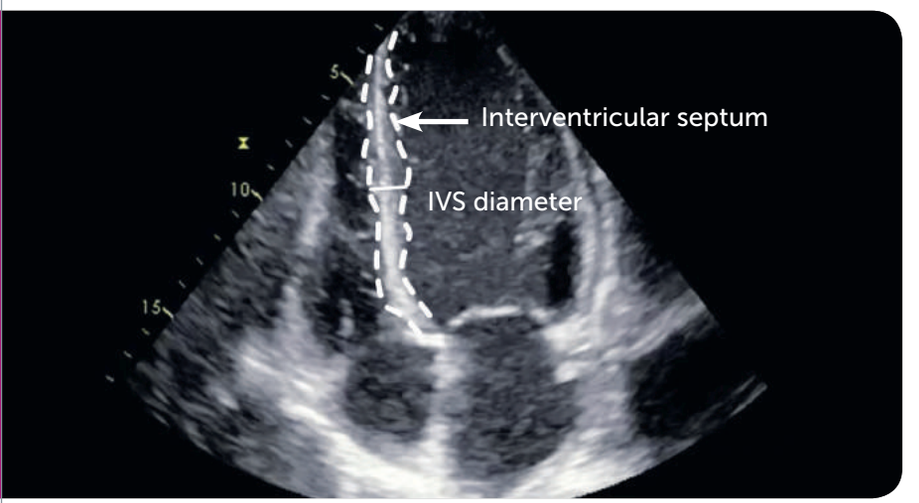
Quantification of LVH – Severity of Septal Thickness

	♂	♀
Normal (mm)	6 – 10	6 – 9
Mild (mm)	11 – 13	10 – 12
Moderate (mm)	14 – 16	13 – 15
Severe (mm)	≥ 17	≥ 16

2D measurements: end-diastole, mid-septum, 4 chamber view ESC/ASE 2005

INTERVENTRICULAR SEPTUM – apical four-chamber view/2D

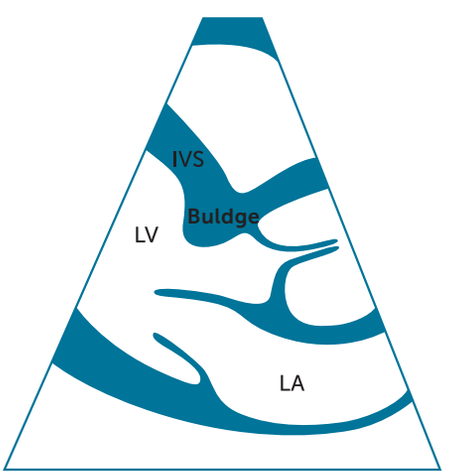
The interventricular septum is a prominent structure. The center of the septum is highly echoge nic. A septal bulge is frequently observed, especially in hyper-tensive patients. The thickness of the bulge should be reported separately.



May cause obstruction and SAM, especially under certain clinical conditions (hypovolemia, hyperkinesia, catecholamines).

Sigmoid Septum

- Septal buldge – less than 3 cm in length
- Associated with hypertension
- Not associated with hypertrophic cardiomyopathy



LEFT VENTRICULAR HYPERTROPHY

Quantification of LV Mass (ESC/ASE 2005)

Measurments obtained from 2D-targeted M-mode or 2D linear LV measurements: LV internal dimensions and wall thicknesses should be measured at the level of the LV minor dimension, at the mitral chordae level.

$$\text{LV mass} = 0.8 \times \left\{1.04\left[\left(\text{LVIDd} + \text{PWTd} + \text{SWTd}\right)^3 - \left(\text{LVIDd}\right)^3\right]\right\} + 0.6\ \text{g}$$

Abbreviations:
LVIDd= left ventricular internal diameter at end diastole
PWTd= posterior wall thickness at end diastole
SWTd= septal wall thickness at end diastole

LV Mass/Body Surface Area – Reference Values

	♂	♀
Normal (g/m²)	50 – 102	44 – 88
Mild (g/m²)	103 – 116	89 – 100
Moderate (g/m²)	117 – 130	101 – 112
Severe (g/m²)	≥ 131	≥ 113

Additional Findings in Hypertensive Patients

- Left atrial enlargement
- Right ventricular hypertrophy
- Diastolic dysfunction
- Dilated aorta
- Aortic valve sclerosis
- Mitral annular calcification

Athlete's Heart

- Left ventricular hypertrophy (RWT ≤ 45 and septum rarely > 13mm)
- Normal or supranormal diastolic function
- Left and right ventricular dilatation
- Supranormal left atrial booster pump function
- Changes occur only after intensive and prolonged training for several years

NOTES

LV mass better reflects the extent of LVH than the measurement of septal thickness. Even small measurement errors are magnified. Therefore, LV mass measurement should only be performed in patients with good image quality.

This formula is appropriate for evaluating patients without major distortions of LV geometry.

In a patient with these findings, left ventricular hypertrophy is likely to be a consequence of hypertension.

Endurance training/ isotonic exercise (such as marathon running) causes an eccentric form of hypertrophy. Isometric exercise (such as weight lifting) causes a more concentric form. Deconditioning reverses left ventricular hypertrophy.

NOTES

004 // Diastolic Function

CONTENTS

NOTES

Any patient with systolic dysfunction also has diastolic dysfunction.

Patients with diastolic dysfunction usually have a dilated left atrium.

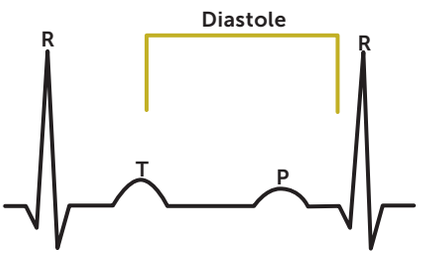
BASICS OF DIASTOLIC DYSFUNCTION

Causes

- Aging
- Sytolic dysfunction
- Heart failure with preserved ejection fraction
- Left ventricular hypertrophy
- Restrictive cardiomyopathy/infiltrative disease
- Coronary artery disease
- Hypertrophic cardiomyopathy
- Heart transplantation

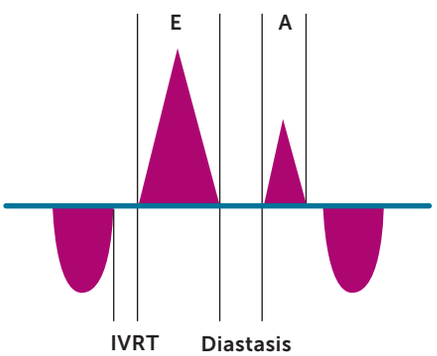
Diastole beginns with aortic valve closure, which can be assessed with PW Doppler sample volume in the LVOT (end of signal).

Diastole Duration



Fusion of the E- and the A- wave may occur in tachycardia. The duration of diastasis decreases with heart rate and PQ duration.

Timing of Diastole

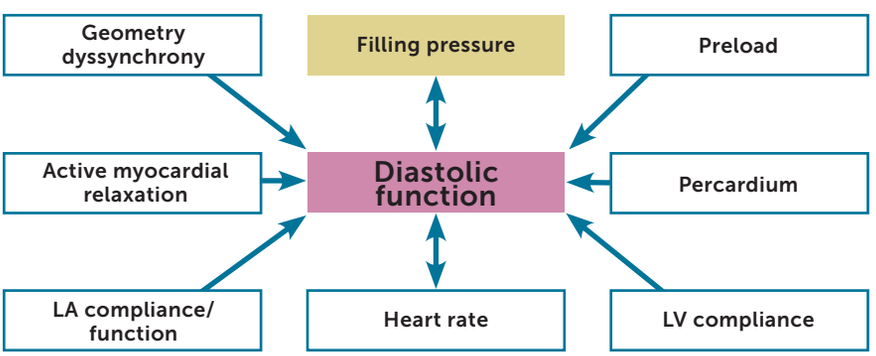


Components

- IVRT – isovolumetric relaxation (AV closure to MV opening)
- E= rapid early (passive) LV filling
- Diastasis
- A= late LV filling – atrial contraction

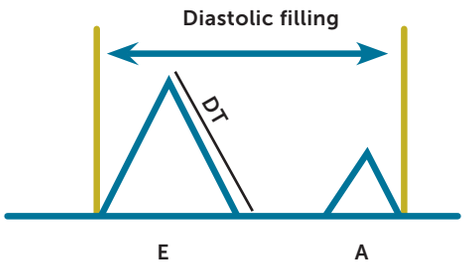
Echo assessment of diastolic function primarily reflects left atrial filling pressure.

Physiology of Diastolic Function

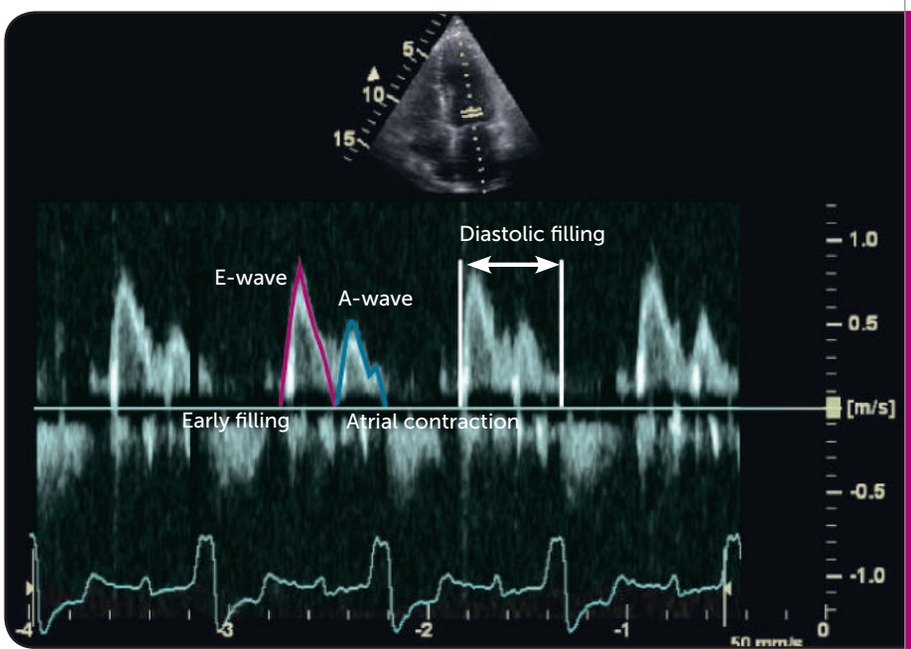


BASICS OF DIASTOLIC DYSFUNCTION

Mitral Inflow Signal



Early filling **Atrial contraction**



MITRAL INFLOW SIGNAL – apical four-chamber view/ PW Doppler MV

The mitral inflow signal allows assessment of diastolic function as well as the timing of events (such as diastolic filling time). The E-wave represents early diastolic filling while the A-wave represents atrial contraction. It is advisible to always use an ECG.

Mitral Inflow – Reference Values

	16–20 years	21–40 years	41–60 years	> 60 years
IVRT (ms)	50 ± 9	67 ± 8	74 ± 7	87 ± 7
DT (ms)	142 ± 19	166 ± 14	181 ± 19	200 ± 29
A duration	113 ± 17	127 ± 13	133 ± 13	138 ± 19
E/A	1.88 ± 0.45	1.53 ± 0.4	1.28 ± 0.25	0.96 ± 0.18

IVRT= isovolumic relaxation time, **DT =** decceleration time

EAE/ASE 2009

NOTES

PW Doppler sample volume should be at the tip of the MV leaflets.

The deceleration time (DT) shows the pressure decay of early filling. In general the shorter the DT, the higher the filling pressure.

In some situations the parameters of diastolic function may be inconsistent and difficult to interpret.

NOTES

An E/e' ratio ≤ 8 (septal or lateral) indicates normal left atrial pressure; a septal E/e' ≥ 15 or a lateral E/e' ≥ 12 indicates elevated left atrial pressure.

BASICS OF DIASTOLIC DYSFUNCTION

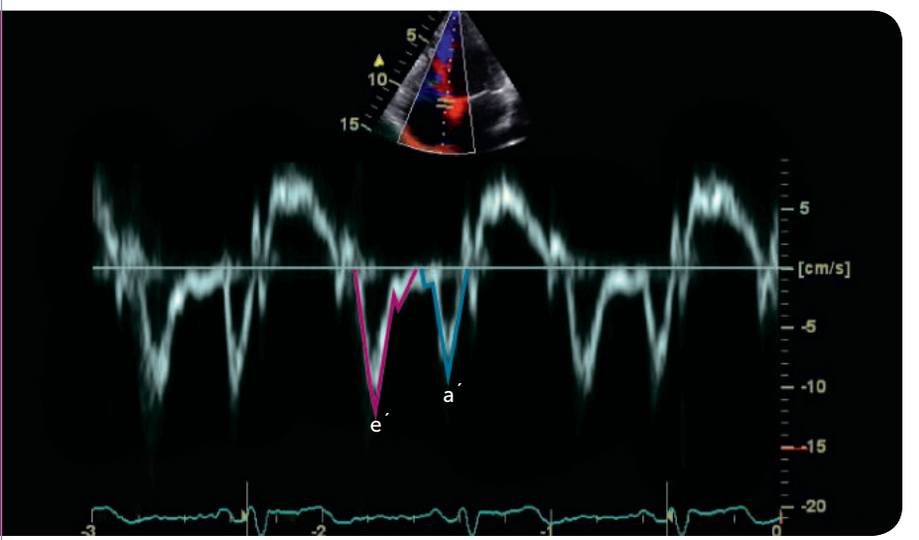
TDI Mitral Annulus – Reference Values

	16–20 years	21–40 years	41–60 years	> 60 years
Septal e' (cm/s)	14.9 ± 2.4	15.5 ± 2.7	12.2 ± 2.3	10.4 ± 2.1
Septal e'/a'	2.4	1.6 ± 0.5	1.1 ± 0.3	0.85 ± 0.2
Lateral e' (cm/s)	20.6 ± 3.8	19.8 ± 2.9	16.1 ± 2.3	12.9 ± 3.5
Lateral e'/a'	3.1	1.9 ± 0.6	1.5 ± 0.5	0.9 ± 0.4

EAE/ASE 2009

TISSUE DOPPLER IMAGING OF THE MITRAL ANNULUS – apical four-chamber view/TDI PW

E' and a' represent the mitral annular velocity towards the base of the heart during early passive (e') and active (a') filling. E/e' is a marker of left atrial filling pressure.



Situations in Which TDI at the Mitral Annulus Should Not Be Used

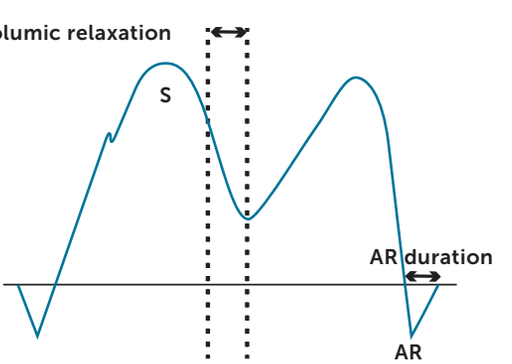
- Annular calcification
- Mitral valve prosthesis
- Myocardial infarction
- Moderate to severe mitral regurgitation

Use right upper PV to record the PW signal. Remember to reduce PRF.

Pulmonary Venous Flow

- Peak systolic PV flow velocity (S)
- Peak diastolic PV flow velocity (D)
- Peak reverse atrial flow velocity (AR)
- AR duration

Signs of impaired diastolic function:
Decrease in systolic component, increase in peak AR, increase in AR duration



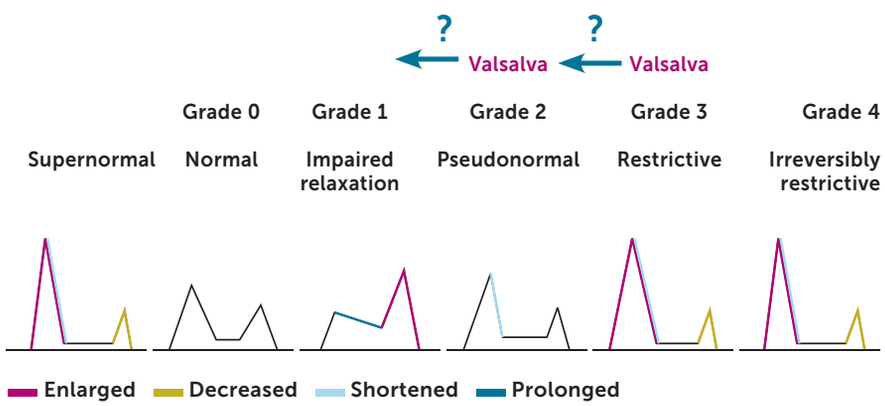
BASICS OF DIASTOLIC DYSFUNCTION

Pulmonary Veins – Reference Values

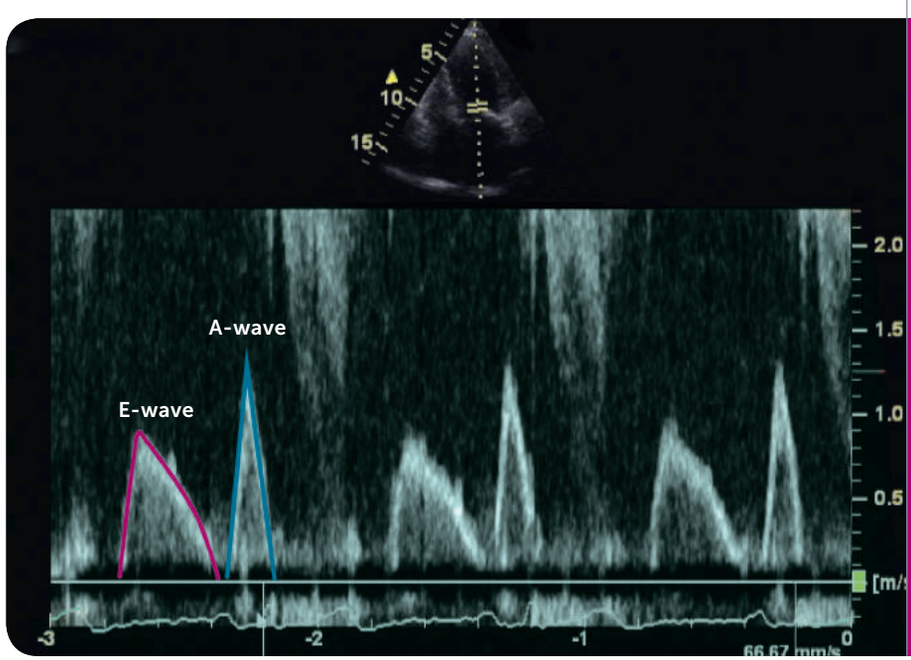
	16 – 20 years	21 – 40 years	41 – 60 years	> 60 years
S/D	0.82 ± 0.18	0.98 ± 0.32	1.21 ± 0.2	1.39 ± 0.47
AR (cm/s)	16 ± 10	21 ± 8	23 ± 3	25 ± 9
AR duration (ms)	66 ± 39	96 ± 33	112 ± 15	113 ± 30

EAE/ASE 2009

Grading of Diastolic Dysfunction



Increasing filling pressures are seen in the patterns from left to right. Provocation maneuvers such as Valsalva that unload the left atrium may cause a reversal of the pattern (pseudonormal -> impaired relaxation and restrictive -> pseudonormal)



IMPAIRED RELAXATION PATTERN – apical four-chamber view/PW Doppler MV

The A-wave is taller than the E-wave. This indicates impaired diastolic relaxation. Large parts of ventricular filling occur during atrial contraction in such patients. In addition, the deceleration of the E-wave is prolonged.

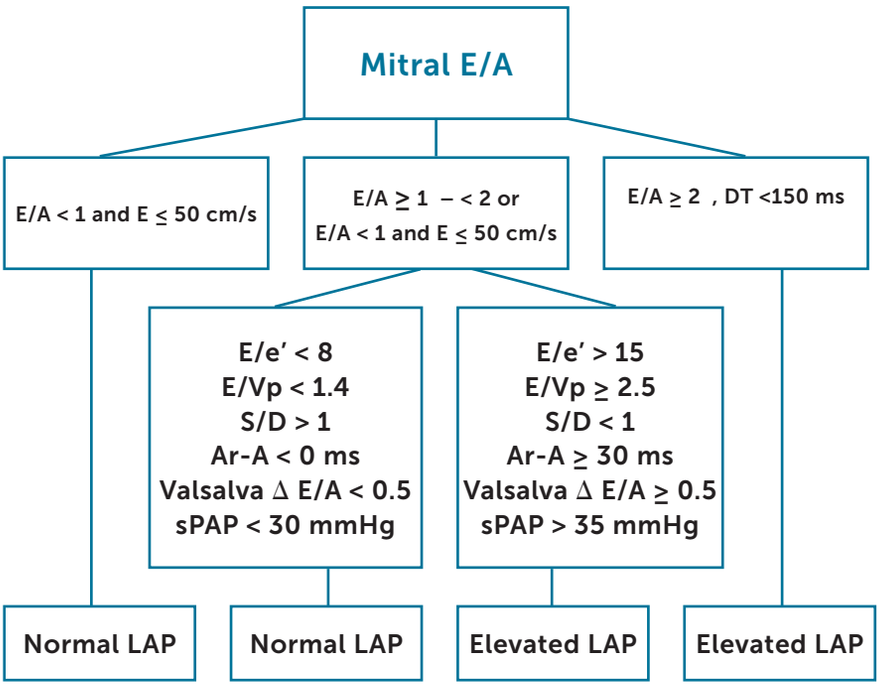
NOTES

Pulmonary vein flow has many limitations and is rarely used in clinical practice.

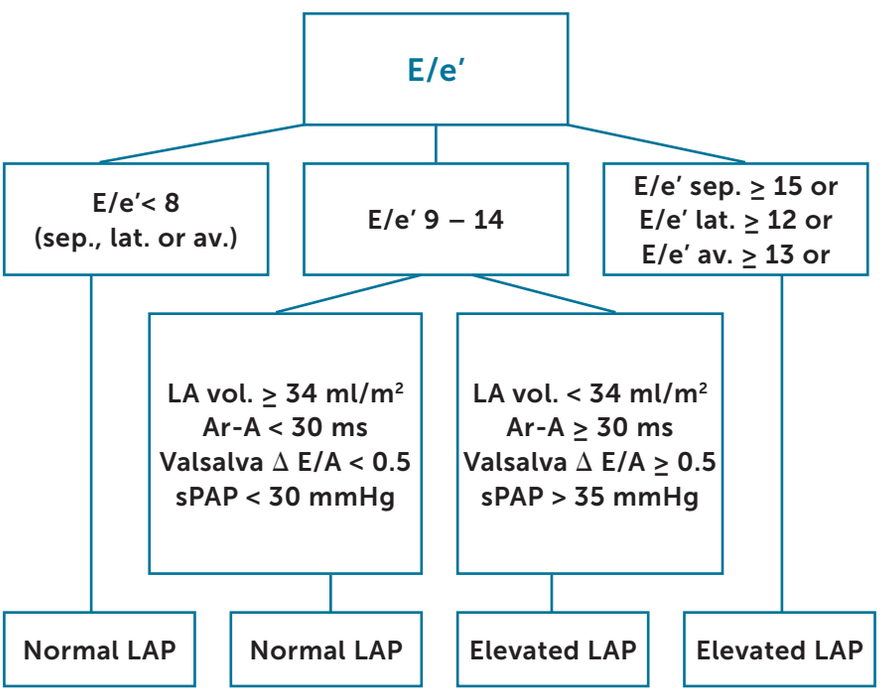
Left atrial filling pressure increases with the degree of diastolic dysfunction.

NOTES

BASICS OF DIASTOLIC DYSFUNCTION



Algorithm for estimating filling pressures in patients with reduced left ventricular function (EF <55%) according to the ASE/EAE guidelines (2009)



LAP = left atrial pressure; **sPAP=** systolic pulmonary artery pressure

Algorithm for the estimation of filling pressures in patients with normal left ventricular function (EF >55%) according to the ASE/EAE guidelines (2009)

BASICS OF DIASTOLIC DYSFUNCTION

A Simple Approach to Diastolic Function/Rules

- **Supernormal diastolic function:** When the echo is normal and the patient is young
- **Normal diastolic function:** When the echo is normal, the patient is < 45 years of age, and E>A
- **Impaired relaxation:** When A is higher than E (E/A ratio is < 1), filling pressure is normal or slightly elevated
- **Pseudonormal diastolic function:** When echo is abnormal (LVH, red LVF, etc) or the patient is > 65 years of age and E is higher than A (E/A ratio > 1)
- **DD normal vs pseudonormal:** Look at deceleration time, LA enlargement, and E/e' (≥ 8 – 12)
- **Restrictive filling:** When E is twice of A (E/A ratio is >2), then filling pressure elevated
- **Perform TDI:** When E/e´ is > 12 – 15 then filling pressure is elevated (PCWP > 12 mmHg)
- **Perform valsalva:** Unloading of the atrium, LA pressure (LAP) drops, unmasking of pseudonormal filling (discrimination between irreversible restrictive vs. reversible restrictive)

NOTES

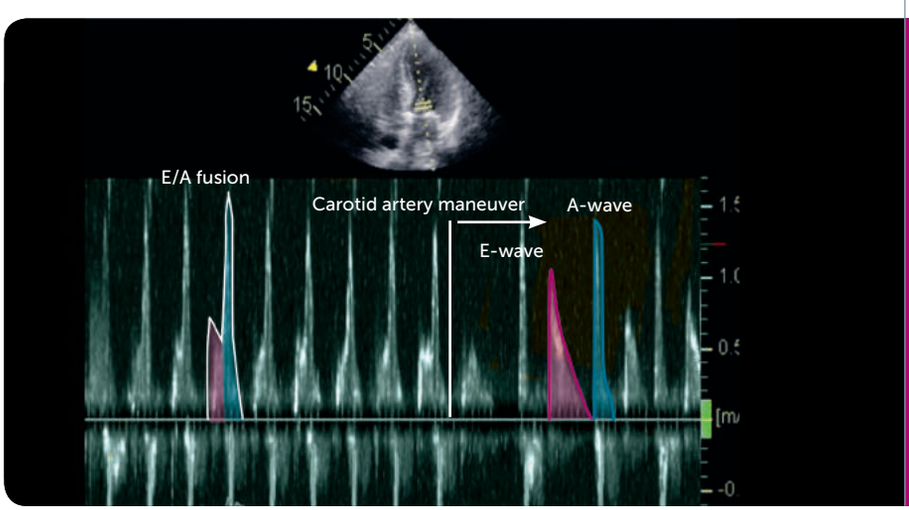
SPECIFIC SITUATIONS

Beat to Beat Variations in E/A Ratio

- Changes in LV filling pressure in relation to respiration?
- COPD patients
- High normal filling pressures (E/e` = 8 – 9)

E/A Fusion

- Tachycardia
- Long systole (left bundle branch block)
- Long AV delay



EA FUSION – apical four-chamber view/PW Doppler MV

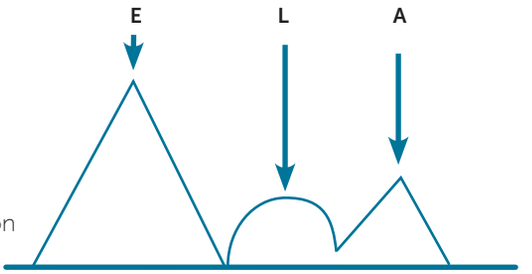
E/A fusion can be abolished by slowing down the heart rate – in this example by performing a carotid artery maneuver.

NOTES

The presence of an L-wave indicates elevated filling pressure.

SPECIFIC SITUATIONS

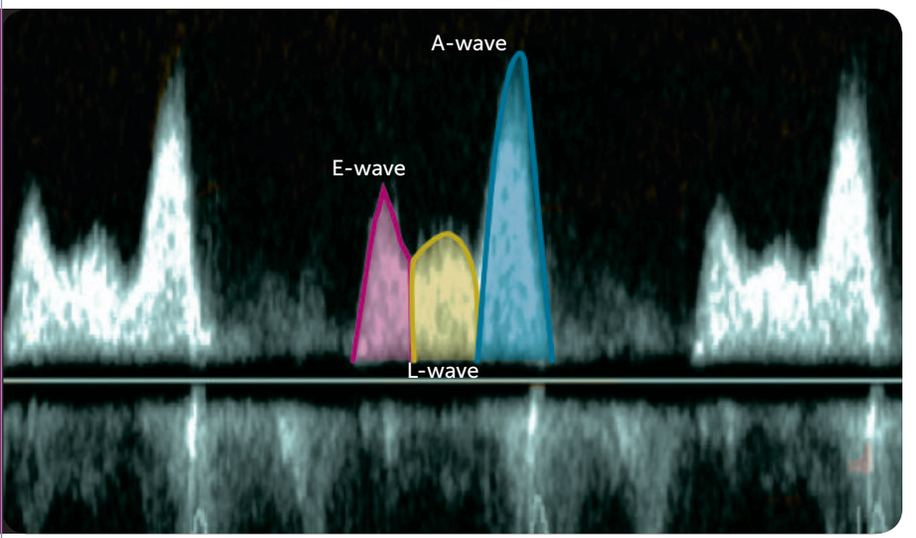
L-Wave



- Mid-diastolic filling of the LV
- Elevated filling pressure?
- Bradycardia
- Can also occur in atrial fibrillation (difficult to detect, no A wave)

L WAVE – apical four-chamber view/PW Doppler MV

The L-wave occurs between the E- and the A-wave, and denotes mid-diastolic filling of the LV. It is indicative of elevated LV filling pressure.



Atrial Fibrillation/Flutter in Diastolic Dysfunction

- Often associated with diastolic dysfunction
- Pulmonary venous flow is difficult to assess
- No A-wave, therefore the E/A ratio cannot be obtained
- Use E/e' and deceleration time (average several beats)

Diastolic dysfunction/LV filling pressure should not be assessed in the setting of mitral regurgitation > grade II.

Estimate filling pressure to determine the severity of disease and how the LV can cope with the problem (e.g. AS, AR, cardiomyopathy).

Left Atrial Pressure in Mitral Valve Disease

- Left atrial size does not necessarily reflect elevated filling pressures
- Left atrial size may also be enlarged due to volume overload + atrial fibrillation
- E-wave velocity also reflects increased stroke volume
- E' is reduced in mitral stenosis and elevated in mitral regurgitation

005 // Dilated Cardiomyopathy

CONTENTS

NOTES

BACKGROUND

Definition

- Myocardial disease (primarily)
- Impaired systolic function
- Left ventricular dilatation
- In the absence of coronary artery disease and significant primary valvular disease

Ischemic cardiomyopathy is similar to dilated cardiomyopathy but is, by definition, NOT a form of dilated cardiomyopathy.

Causes

- Genetic
- Congential
- Infections
- Drug and alcohol abuse
- Certain cancer medications
- Exposure to toxins

The etiology remains unidentified in many cases because a biopsy is not performed.

About 30% of patients with idiopathic cardiomyopathy are estimated to suffer from genetic forms of the disease. In these forms, there is frequently an overlap between dilated and hyptertrophic forms.

Associated Problems

- Left heart failure
- Atrial fibrillation, ventricular arrythmias
- Pulmonary hypertension
- Mitral regurgitation
- Right heart failure
- Tricuspid regurgitation
- Dyssynchrony
- Thromboembolism

ECHO FEATURES

Diagnosis

- Reduced left ventricular function
- Dilated left ventricle
- Reduced right ventricular function
- Exclude other causes (coronary artery disease, valvular)

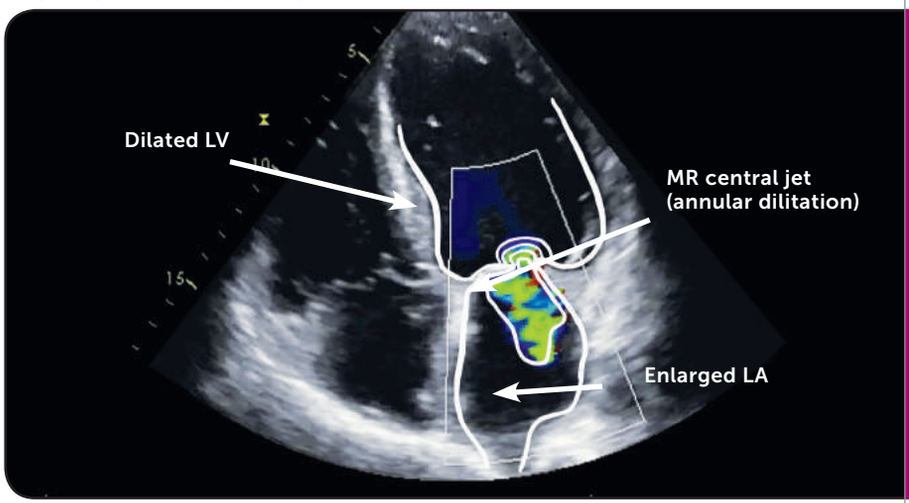
End-stage ischemic cardiomyopathy and dilated cardiomyopathy look very similar.

Signs of Advanced Dilated Cardiomyopathy

- Low cardiac output (LVOT velocity < 0.5 m/sec)
- Very low ejection fraction
- Atrial size (large atria in more advanced forms)
- Significant mitral regurgitation
- Diastolic function/filling pressure (restrictive pattern)
- Severe pulmonary hypertension and tricuspid regurgitation
- Poor right ventricular function
- Pleural effusion

Right ventricular function correlates better with prognosis than LVF (it denotes end-stage heart failure).

ECHO FEATURES



NOTES

ECHOFEATURES OF DILATED CARDIOMYOPATHY – apical four-chamber view/ Color Doppler

Dilated left ventricle with reduced left ventricular function, mitral regurgitation with a central jet caused by annular dilatation,

Mechanisms of Mitral Regurgitation in Cardiomyopathy

- Annular dilatation geometry
- Bileaflet restriction
- Atrial enlargement
- Dyssynchrony

The degree of mitral regurgitation may change rapidly and is related to factors such as increased afterload, preload, and volume status.

MR increases mortality. (additional volume overload of LV).

Rule out a structural cause for mitral regurgitation. It could point to the presence of a primary valvular cause of systolic dysfunction.

SPECIFIC FORMS

Ischemic Cardiomyopathy

- Not really a form of dilated cardiomyopathy but shares several features
- Most common cause of heart failure
- Occurs in large infarctions, leads to ventricular remodeling and global dysfunction
- Thin scarred walls, ventricular distortion and clearly segmental myocardial dysfunction suggests ischemic cardiomyopathy

It may be difficult or even impossible to distinguish between dilated and ischemic cardiomyopathy on echocardiography.

NOTES

Abortive forms of Takotsubo cardiomyopathy with more subtle wall motion abnormalities have been reported.

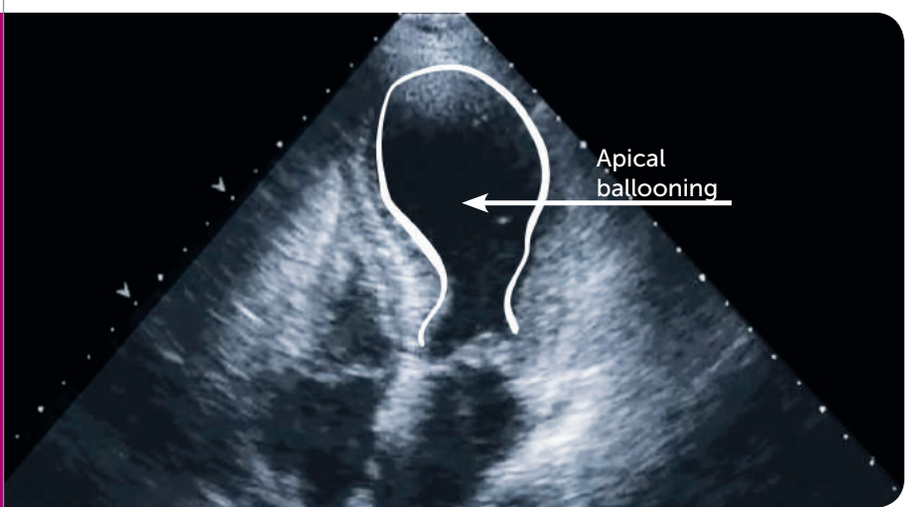
SPECIFIC FORMS

Takotsubo Cardiomyopathy

- Stress–induced cardiomyopathy is more common in women
- Echo features include segmental wall motion abnormalities (in particular apical ballooning), hyperdynamic basal segments which may cause LVOT obstruction, and right ventricular involvement
- Normal coronary angiogram
- Abnormalities are reversible

TAKOTSUBO CARDIOMYOPATHY – apical four-chamber view/2D

A typical feature of Takotsubo cardiomyopathy is apical ballooning. The basal segments tend to be hyperdynamic.



Peripartum Cardiomyopathy

- A non-familial, non-genetic form of dilated cardiomyopathy associated with pregnancy
- Clinical presentation in the last month of pregnancy or 5 months post partal
- Recovery rate > 40%
- Often presents as acute heart failure
- May involve both ventricles
- Has no specific echo features

The duration of, and the heart rate needed for, the induction of tachycardiomyopathy are highly variable and depend on numerous factors.

Tachycardia/Arrythmia-Mediated Cardiomyopathy

- Prolonged periods of tachycardia in atrial fibrillation or ventricular tachycardia
- In arrhythmia-mediated cardiomyopathy, frequent ectopic beats (> 17,000/24h)
- Cardiac function returns in most cases after heart rate control, but may take several weeks or months
- Assessment of left ventricular function is difficult and is underestimated in tachycardia. Always repeat the echocardiogram after heart rate control

SPECIFIC FORMS

HIV-Mediated Cardiomyopathy

- Focal myocarditis
- Most common form of cardiomyopathy in African countries (e.g. Burkina Faso)

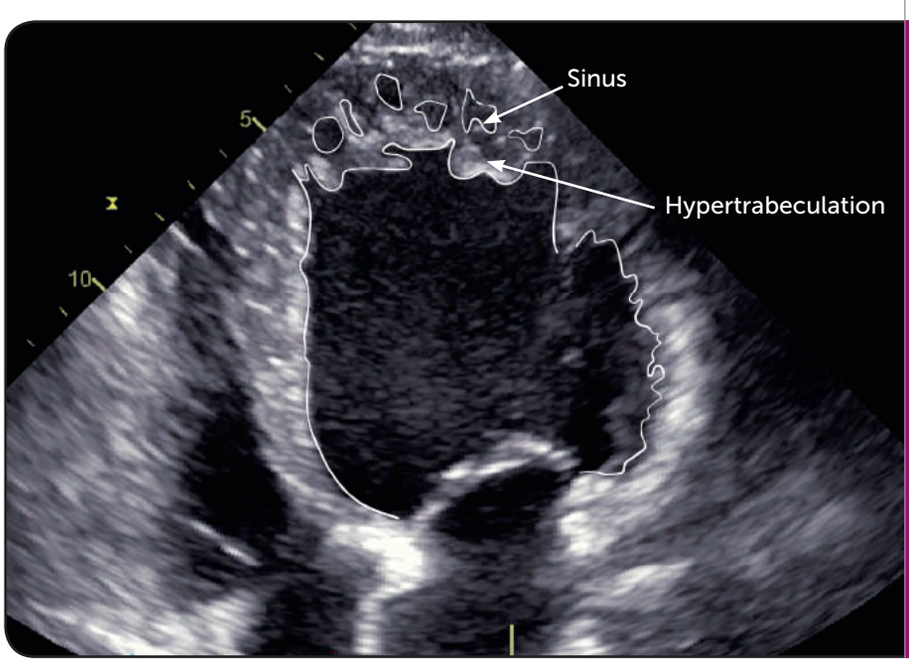
Causes

- Myocarditis
- Autoimmune cardiomyopathy
- Nutritional deficiency
- Drug toxicity (e.g. zidovudine)

The severity and incidence of HIV-mediated cardiomyopathy strongly depends on the treatment regimen (HAART reduced the incidence by 30%).
HIV-mediated cardiomyopathy has no specific echocardiographic features.
One usually finds left ventricular function without regional wall motion abnormalities, and possibly pericardial effusion.

LV Non-Compaction

- Characterized by prominent trabeculae and intertrabecular recesses (sinus)
- Associated with other cardiac abnormalities
- Genetic disease, risk of cardiomyopathy, family screening is important
- Associated with neuromuscular disorders
- Congenital cardiomyopathy characterized by prominent trabeculae and intertrabecular recesses (spongy myocardium)
- May present at any age
- May be associated with normal or reduced left ventricular function
- Echocardiography is the most important diagnostic tool (alternative: MRI)



LV NON-COMPACTION – apical four-chamber view/2D

The apical portion of the left ventricle is strongly trabeculated and appears spongy. Look carefully and visualize all portions of the myocardium to find hypertrabe culated areas. Use contrast and color Doppler when in doubt.

NOTES

There is a genetic link between non–compaction and hypertrophic cardiomyopathy.

NOTES

SPECIFIC FORMS

Echo Evaluation

- The involved segments are mid ventricular (especially inferior and lateral) and apical. Is usually seen best on atypical views
- Right ventricular involvement may be present but is difficult to differentiate from normal trabeculae
- Use color Doppler with low PRF and contrast to visualize blood flow between the trabeculae
- Use deformation imaging to detect myocardial dysfunction (i.e. speckle-tracking echocardiography) at the regions of hypertrabeculation

Chagas Disease

- Trypanosoma cruzi
- Megaesophagus
- Cardiac disease
- Megacolon
- Most common form of cardiomyopathy in Latin America
- Right heart failure is dominant (regional + global dysfunction)
- Caused by infection with Trypanosoma cruzi (present in feces of reducidae e.g. triatoma infestand = kissing bug)
- Most common form of cardiomyopathy in Latin America
- Associated with megaesophagus, megacolon induced by neural degeneration

Echo Features

- Pericardial effusion
- Regional myocardial dysfunction with preserved global left ventricular function
- Often apical aneuryms
- Diastolic dysfunction is present in about 20% of patients

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006 // Hypertrophic Cardiomyopathy

CONTENTS

NOTES

Cardiomyopathy may differ markedly in terms of morphology, clinical presentation and prognosis.

BASICS

Epidemiology

- Prevalence: 1 in 500
- Annual mortality: Adults 2%
  Childhood 4 – 6%
- Most common cause of sudden cardiac death in athletes

The onset of disease may vary: childhood, adolescence, or sometimes late in life.

Perform family screening.

Cause

- Genetic disease (sarcomere)
- Autosomal dominant
- Associated syndromes (Noonan's, Friedreich ataxia, LEOPARD)

Symptoms

- Asymptomatic
- Chest pain
- ECG abnormalities
- Syncope
- Arrhythmias
- Sudden death
- Dyspnea
- Palpitations

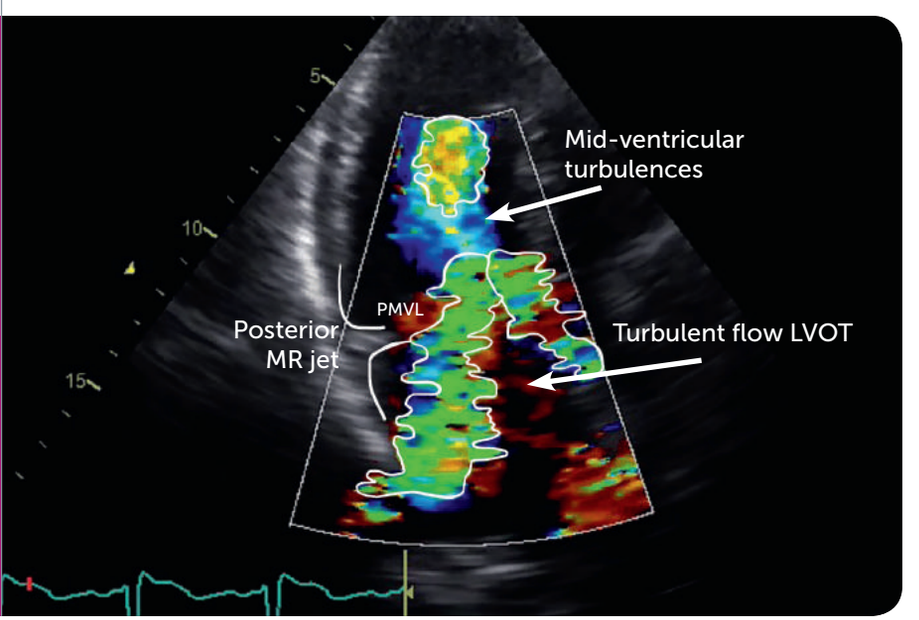
Other causes of left ventricular hypertrophy include hypertension, aortic stenosis, athlete's heart, and infiltrative heart disease.

When to Consider Hypertrophic Cardiomyopathy?

- Unexplained left ventricular hypertrophy (> 15 mm)
- LVOT/LV gradient
- "Spade-shaped" left ventricular cavity
- Speckled appearance of the myocardium
- Asymmetric left ventricular hypertrophy
- Turbulent flow in the LV/LVOT

OBSTRUCTIVE CARDIOMYOPATHY – apical three chamber view

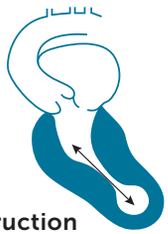
Turbulent flow in the LVOT caused by systolic anterior motion of the MV. Distortion of the MV leads to regurgitation with a posteriorly directed jet. Flow acceleration is also present in the mid-ventricular portion (additional mid-ventricular obstruction).



BASICS

Obstructive Forms

LVOT obstruction



Mid-ventricular obstruction

Non-Obstructive Forms

Asymmetric

Apical

NOTES

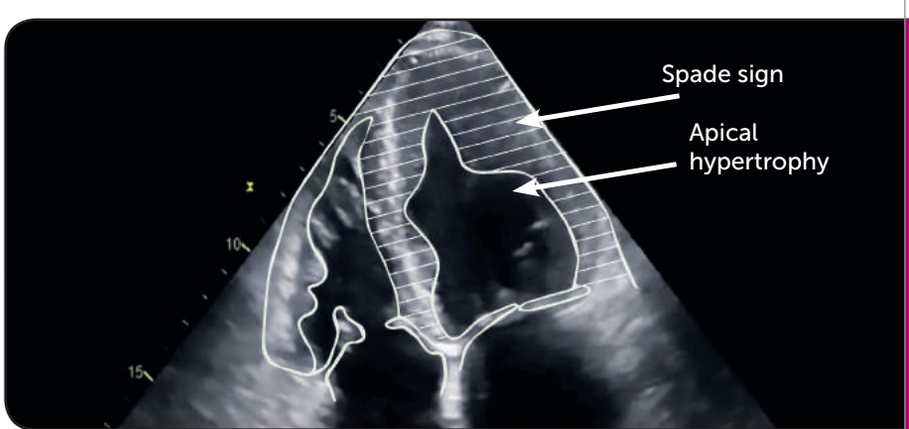
There is an overlap between obstructive and non-obstructive forms; the gradients may be inconsistent.

ECHOCARDIOGRAPHIC EVALUATION

Non-Obstructive Cardiomyopathy (Apical Type)

- More common in the Asian population
- Associated with a favorable prognosis
- ECG tends to show giant negative T-waves
- A typical echocardiographic finding: spade-shaped left ventricle

Apical hypertrophy may be difficult to detect. Use contrast for LV cavity opacification.



APICAL HYPERTROPHIC CARDIOMYOPATHY – apical four-chamber view/2D

Pronounced hypertrophy of the apex with a spade-shaped ventricular cavity. Atrial enlargement is also a common feature of hypertrophic cardiomyopathy.

Views to Display SAM = Systolic Anterior Motion (of the Anterior Mitral Valve Leaflet)

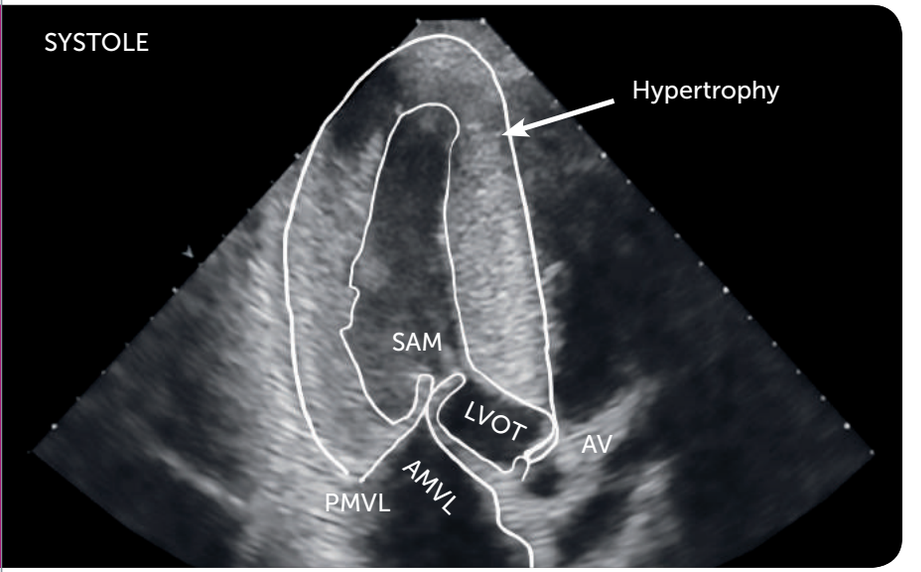
- Parasternal long-axis view
- Parasternal short-axis view at MV
- Apical long-axis view
- Mmode/Color MMode
- Five-chamber view

NOTES

ECHOCARDIOGRAPHIC EVALUATION

SYSTOLIC ANTERIOR MOTION OF THE MV – apical three-chamber view/2D

Dynamic left ventricular outflow tract (LVOT) obstruction is caused by anterior motion of the mitral valve during systole.



Use Valsalva or exercise to provoke a gradient during the exam. It may "unmask" obstructive cardiomyopathy.

SAM (Systolic Anterior Motion) Increases With

- Hypovolemia
- Exercise
- Medication (i.e. nitroglycerin, diuretics)
- Dobutamine
- Valsalva
- Post-extrasystolic

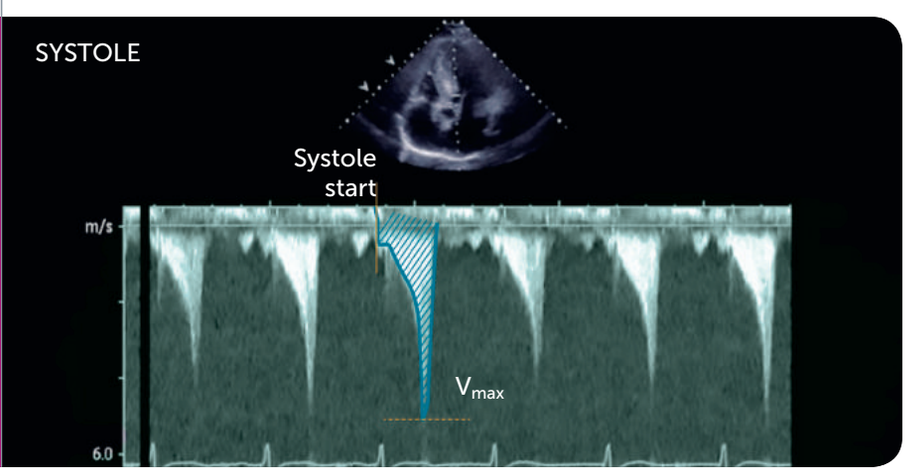
Find the site of obstruction with 2D and color Doppler (SAM), put CW through this site. The CW Doppler focus point should be postioned at the site of obstruction.

Quantification of Obstruction

- Measure maximal LVOT velocity (CW Doppler)
- The Doppler signal is typically dagger-shaped
- A late peak generally indicates obstruction more towards the mid/apical parts of the ventricle
- Early obstruction is hemodynamically more relevant
- It may be difficult to discern the signal of LVOT obstruction from that of aortic stenosis or mitral regrgitation. Use color Doppler for guidance

LVOT FLOW ACCELERATION – apical five-chamber view/CW Doppler

Dagger-shaped spectrum in a patient with obstructive hypertrophic cardiomyopathy. In this example maximum obstruction occurs rather late in systole (late peak).



ECHOCARDIOGRAPHIC EVALUATION

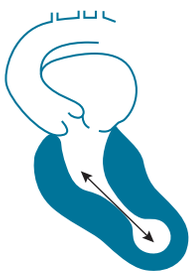
Mitral Regurgitation in Obstructive Cardiomyopathy

- Distortion of mitral valve geometry due to SAM)
- The jet is directed posteriorly
- The severity correlates with the degree of obstruction

Other Causes of LVOT Obstruction

- Hypertensive heart disease caused by a sigmoidal septum
- Following surgery for aortic stenosis due to the presence of left ventricular hypertrophy and a sudden decrease in afterload or increase in contractility
- Post-mitral valve repair when the anterior mitral valve leaflet is left too long
- Hypovolemia
- Hypercontractile state (e.g. hyperthyroidism, fever, catecholamines)

Mid-Ventricular Cardiomyopathy



- Least common type of hypertrophic cardiomyopathy
- Often combined with LVOT obstruction
- Rather late peak of maximum gradient velocity
- Gradients are rarely very high

Echocardiographic Assessment in Hypertropic Cardiomyopathy

- Myocardial thickness and location of hypertrophy
- Systolic/Diastolic function
- Doppler measurement of maximal gradients
- Degree of mitral regurgitation/SAM
- Atrial size
- (Deformation imaging)

NOTES

Mitral regurgitation may also increase with provocation and a rise in gradients.

SAM may also occur in diseases and conditions other than hypertrophic cardiomyopathy.

Mid-ventricular and LVOT obstruction may be combined.

Septal thickness > 30mm = increased risk for sudden cardiac death.

Because the left ventricle cavity is usually small, left ventricular function appears better than it is. In addition, most patients have reduced longitudinal function, especially in those segments which are very hypertrophic or fibrotic.

NOTES

Patient history, distribution of left ventricular hypertrophy, other echo findings and speckle tracking may be helpful in establishing the correct diagnosis.

Also consider surgical myectomy, especially in patients who are candidates for surgery (e.g. aortic stenosis with LVOT obstruction).

ECHOCARDIOGRAPHIC EVALUATION

Differential Diagnosis

- Hypertensive heart disease
- Aortic stenosis
- Amyloid heart disease
- Sarcoid heart disease
- Athlete's heart
- Fabry's disease

Alcohol Septal Ablation – Recommendations

- Severe heart failure symptoms (NYHA classes III or IV) refractory to medication
- Subaortic Doppler gradient > 50 mmHg at rest or with provocation (i.e. exercise)
- Adequate coronary anatomy/echo morphology

ESC 2003

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007 // Restrictive Cardiomyopathy

CONTENTS

NOTES

1) Restrictive cardiomyopathy is NOT the same as a restrictive filling pattern. A restrictive filling pattern may also be present in other forms of cardiomyopathy.

2) Subclinical systolic dysfunction (despite normal ejection fraction) may be present in early stages of disease.

Restrictive cardiomyopathy is the least common form of cardiomyopathy (5% of all cases of primary heart muscle disease).

Patients typically present with signs of right heart failure. Clinical and echocardiographic features may be similar to those of constrictive pericarditis.

Suspect restrictive CMP in patients with normal left ventricular function and unexplained significant bi-atrial enlargement.

BASICS

Definition

- Idiopathic, systemic or infiltrative disorder.
- May involve the left and/or right ventricle.
- Primarily a "diastolic disease" of the ventricles
- Normal or slightly reduced systolic function (in the early stages).

Most Common Causes

- Amyloidosis
- Idiopathic
- Sarcoid heart disease
- Endomyocardial fibrosis
- Radiation
- Chemotherapy
- Carcinoid
- Hemochromatosis

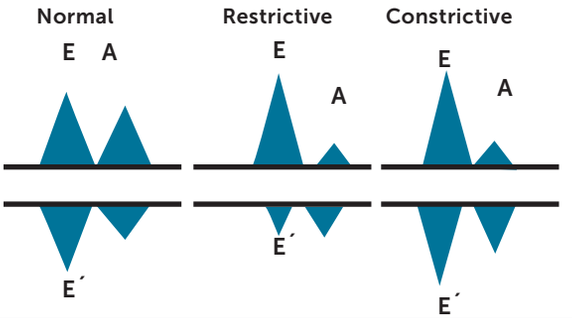
Pathophysiology

- Diastolic dysfunction
- Elevated filling pressure
- Stiff ventricle
- Right heart failure
- Hepatomegaly
- Peripheral edema
- Pericardial effusion
- Pleural effusion

Echo Features

- Left ventricular hypertrophy
- Bi-atrial enlargement
- Normal left ventricular volume (in the early stage)
- Normal left ventricular ejection function (in the early stage)
- Expanded left atrial appendage
- Dilated inferior vena cava and pulmonic veins
- Tricuspid regurgitation

How to Distinguish Restriction from Constriction (Doppler MV Inflow and TDI MV Annulus)



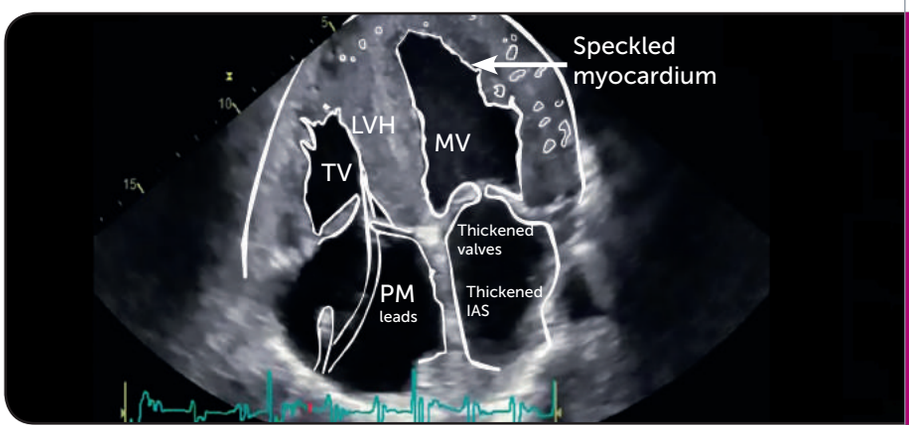
Progressive decline of the E' wave in restrictive CMP

DD: The E' wave is preserved/exaggerated in constrictive pericarditis.

SPECIFIC FORMS

Amyloid Heart Disease – Echo Features

- Ground glass pattern
- Left ventricular hypertrophy
- Atrial enlargement
- Thickened interatrial septum
- Thickened valves frequently with mild regurgitations
- Advanced diastolic dysfunction
- Pericardial/Pleural effusion
- "Apical sparing pattern" of longitudinal strain
- Systolic dysfunction (endstage)
- Right heart involvement



AMYLOIDOSIS – apical four-chamber view/2D

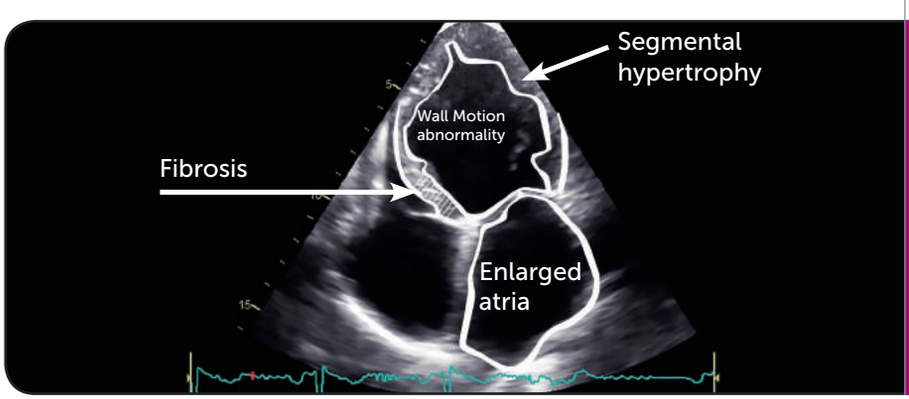
Typical features of amyloidosis, including echogenic/hourglass appearance of the myocardium, thickened valves, and enlarged atria. This patient also received a pacemaker.

Hypereosinophilia/Endomyocardial Fibrosis (EMF) – Echo Features

- Fibrous thickening of the endocardium
- Echogenic eosinophilic infiltrates in the left and right ventricular apex
- Different stages (necrotic/thrombotic/fibrotic)
- Late-stage restrictive filling pattern

Sarcoid Heart Disease – Echo Features

- Cardiac involvement in sarcoidosis is associated with a poor prognosis
- Pericardial effusion
- Left ventricular aneurysms
- Wall motion abnormalities (not related to coronary perfusion territories)
- Hypertrophy (segmental)
- Edema/Fibrosis
- End-stage: left ventricular dilatation, wall thinning and impaired left ventricular function



SARCOIDOSIS – apical four-chamber view/2D

Abnormal cardiac geometry with segmental wall motion abnormalities, thickening, and increased echogenicity in the region of the mid- and distal anterior septum.

NOTES

The echocardiogram is often so typical that it leads to the diagnosis of amyloidosis.

Eosinophilic thombi are found in endomyocardial fibrosis even in the absence of regional wall motion abnormalities or global LV dysfunction.

20 – 30 % of patients with proven sarcoidosis have cardiac involvement. MRI is more sensitive than echo in the detection of sarcoid heart disease.

NOTES

SPECIFIC FORMS

Fabry's Disease: Manifestation

- Rare multisystemic disease
- X-linked genetic disease
- Alpha–galactosidase deficiency
- Renal failure
- Angiokeratoma

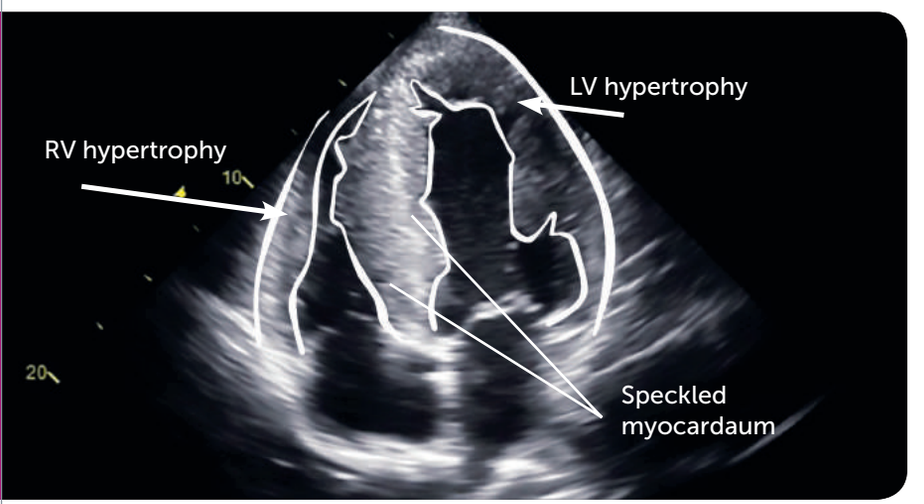
Some authors suggest that the binary sign, defined as binary appearance of the left ventricular endocardial border, aids in the diagnosis of Fabry's disease. However, the sensitivity and specificity of this sign is rather low.

Fabry's Disease: Echo Features

- Left ventricular hypertrophy
- Right ventricular hypertrophy
- Myocardial fibrosis
- Diastolic dysfunction/enlarged left atria

FABRY'S DISEASE – apical four-chamber view/2D

Pronounced bi-ventricular hypertrophy and rather speckled appearance of the myocardium.



008 // Coronary Artery Disease

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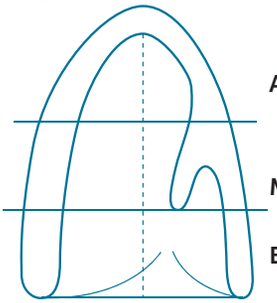
NOTES

SEGMENTAL APPROACH

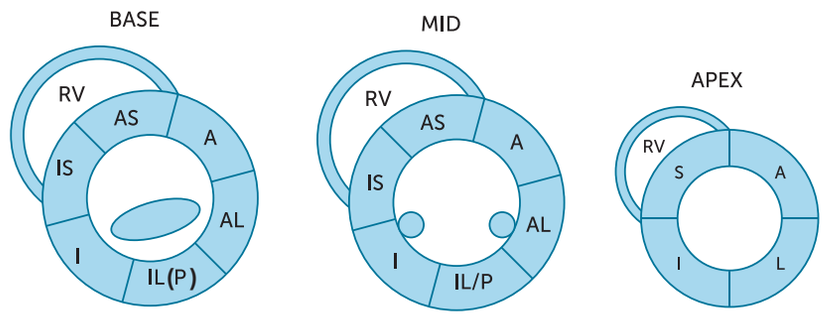
Segmentation (16-Segment Model)



Apical four-chamber view

The left ventricle is divided into basal (6), mid (6) and apical (4) segments.

Subdivision of the corresponding short-axis view (SAX). Note that the basal and mid SAX consist of 6 segments while the apical SAX has only 4 segments (16-segment model).

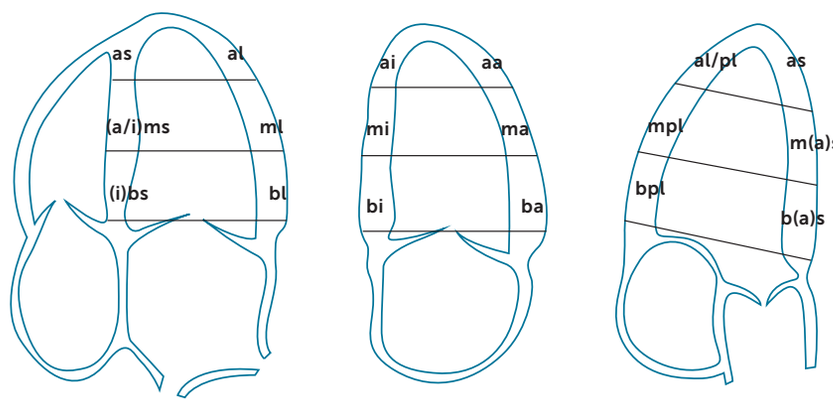


IS= inferoseptal, **AS=**anteroseptal , **A =** anterior,
AL= anterolateral, **IL**=inferolateral, **P=** posterior, **I**=inferior, **S=** septal, **L**=lateral
ESC 2006

The inferolateral segment is also referred to as the posterolateral or posterior segment.

In echocardiographic nomenclature there is no diaphragmatic segment.

Definition of the individual segments on the apical views. Note that the inferior portion of the basal septum is visible on the 4-chamber view.



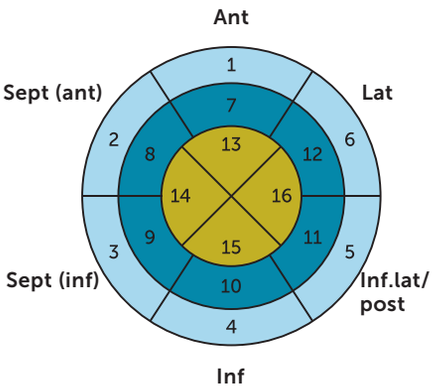
as = apical septum
(a/i)ms= mid inferoseptum
(i)bs = basal inferoseptum
al = apical lateral
ml = mid anterolateral
bl = basal anterolateral

ai = apical inferior
mi= mid inferior
bi = basal inferior
aa = apical anterior
ma = mid anterior
bal = basal anterior

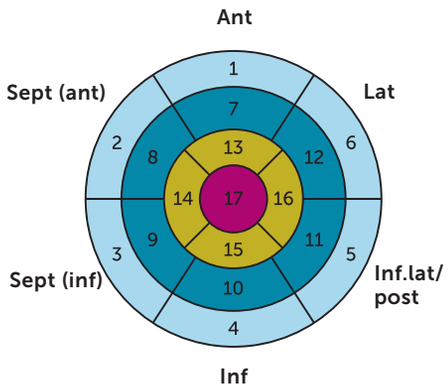
al/pl = apical lateral
mpl= mid inferolateral (posterior)
bpl = basal inferolateral (posterior)
as = apical anteriorl
m(a)s = mid anteroseptum
b(a)s = basal anteroseptum

SEGMENTAL APPROACH

Bull's Eye Representation

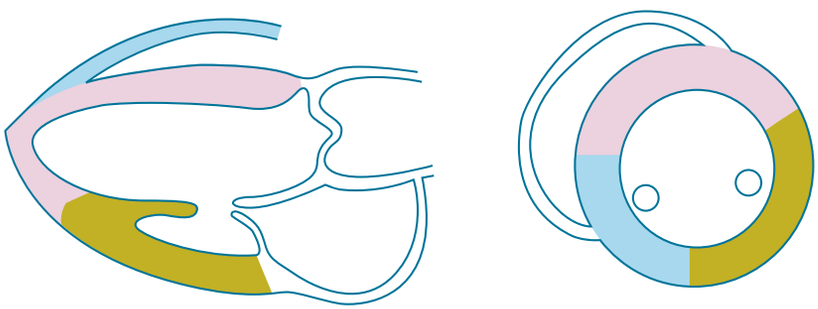


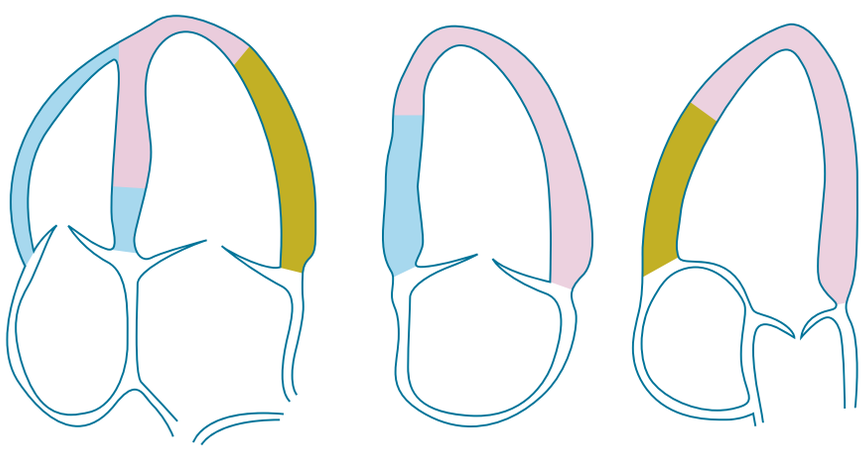
16-Segment model



17-Segment model (supra-apical cap)

Coronary Supply





Left anterior descending (LAD)

Right coronary artery (RCA)

Circumflex artery (Cx)

NOTES

In left dominant perfusion, the posterior (inferolateral) wall and even large portions of the inferior wall are supplied by the LCx. In right dominant perfusion, the RCA supplies the posterior wall in addition to the inferior segments.

NOTES

LV contrast study improves endocardial border detection.

Try your best to obtain the best possible image quality. This is what counts most when you are looking for regional wall motion abnormalities.

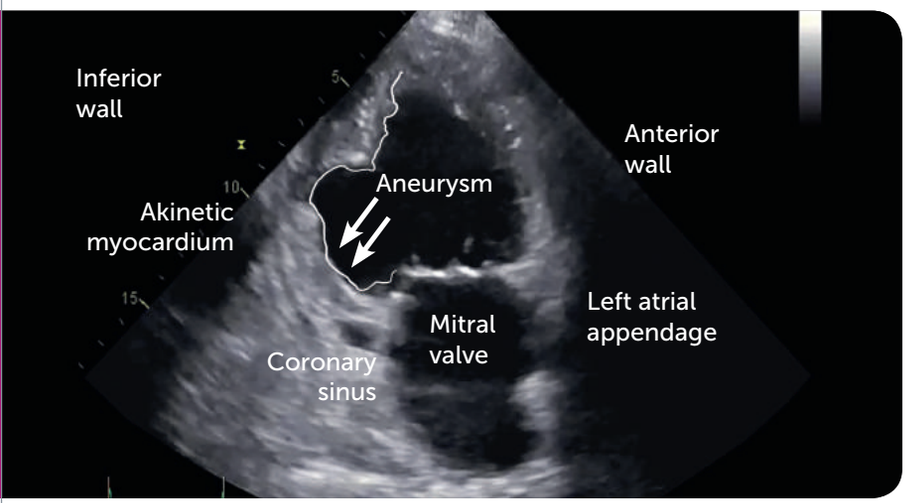
WALL MOTION ABNORMALITIES

What Are We Looking For?

- Lack of wall/myocardial thickening
- Wall motion
- Hinge points
- Ventricular geometry
- Echogenicity/scar

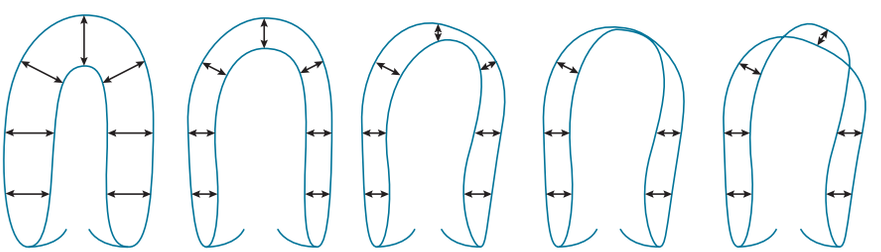
INFERIOR WALL ANEURYSM – apical two-chamber view/2D

Inferior myocardial infarction leading to distortion of ventricular geometry (aneurysm) and regional wall thinning in the basal and mid inferior segments.



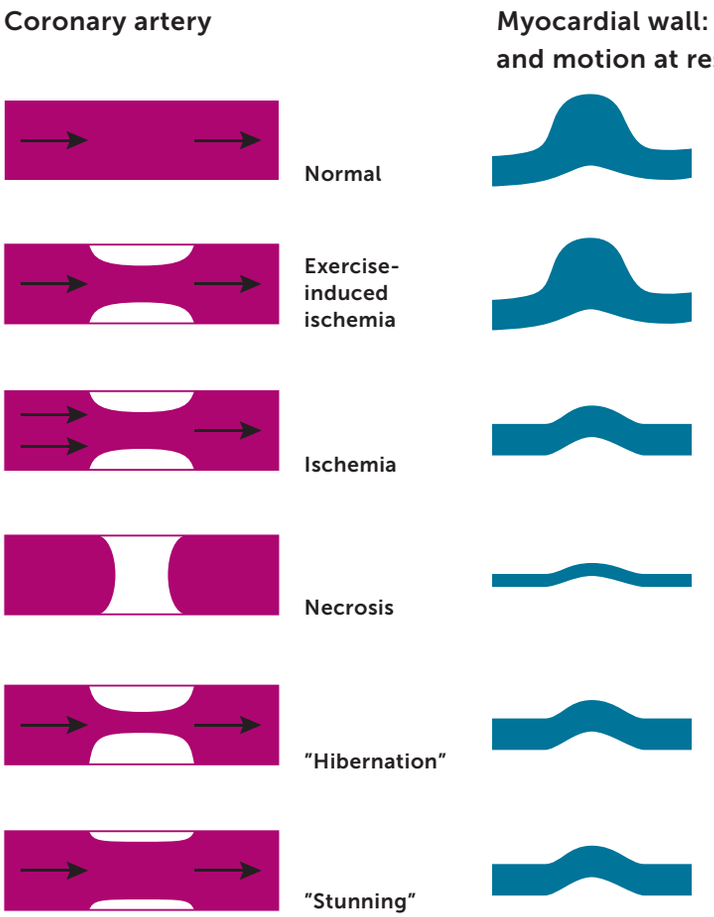
If possible, compare wall motion with a reference segment.

Wall Motion Abnormalties



WALL MOTION ABNORMALITIES

Wall Motion in Ischemic Conditions



Remodeling

- Progressive LV dilatation
- Eccentric LV hypertrophy
- Distortion of geometry
- Hypokinesia of normally perfused segments
- further increase of mitral regurgitation

NOTES

Ischemia, hibernation and stunning are all marked by hypo/akinesia AND preserved wall thickness.

Predisposing factors for remodeling are large infarctions (anterior > inferior), mitral regurgitation, and elevated afterload (hypertension, AS).

NOTES

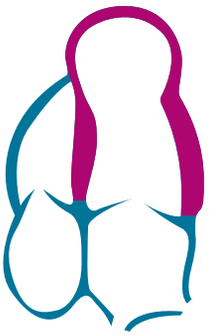
There is no risk of rupture in chronic aneurysms.

WALL MOTION ABNORMALITIES

Aneurysm

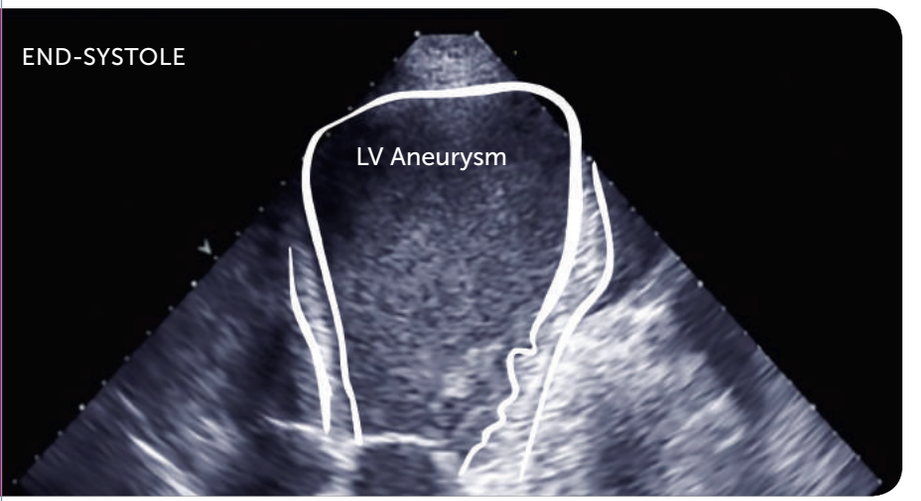
Definition: Abnormal widening of all myocardial layers during diastole

- High risk of thrombi
- Increased risk of heart failure
- Apical aneurysms are best seen on two-chamber and atypical views (avoid "foreshortening")
- The slow flow phenomenon is seen within the aneurysm



APICAL ANEURYSM – apical four-chamber view/2D

Very large apical aneurysm after anterior myocardial infarction. The apical region is dilated and dys-/akinetic.



The degree of wall motion abnormalities depends on the transmurality of the infarction. Various different wall motion abnormalities may exist simultaneously (akinesia, hypokinesia, aneurysm, scars).

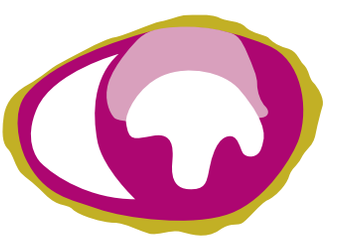
Look for edema (myocardial thickening, bright echoes) in patients with myocardial infarction after reperfusion.

Myocardial Tissue After Acute Coronary Syndrome

Transmural scar: akinesia, dyskinesia, aneurysm, thinning, bright echo

Subendocardial scar: hypokinesia, thickness is normal/mildly thinned

Transmural scar + viability: akinesia + hypokinesia of neighboring segments



Viable myocardium (Acute ischemia/ hibernation/stunning): hypokinesia, akinesia, wall thickness preserved

Normal **Viable ischemia/stunning/hibernation** **Scar/fibrosis**

WALL MOTION ABNORMALITIES

Quantification of Left Ventricular Function in Coronary Artery Disease

- Simpson method
- Visual assessment
- Wall motion scoring
- Center line
- 3D methods (e.g. regional ejection fractions)
- Endocardial contour enhancement (contrast)

Problem Zones (Regions Difficult to Image/Interpret)

Region	Solution
Supraapical	• Avoid foreshortening • Move transducer more laterally + image towards the apex • Use two-chamber view
Lateral	• Rotate four-chamber view clockwise • Move transducer more medially
Basal inferior	• Passive or active motion? • Hinge points? • Wall thickness

Wall Motion Abnormalities – Other Causes

- Dyssynchrony (e.g. left bundle branch block)
- Pacemaker
- Abnormal septal motion (e.g. postoperative, right ventricle pressure/volume load)
- Myocarditis
- Cardiomyopathy (e.g. Takotsubo)
- Sarcoid heart disease

NOTES

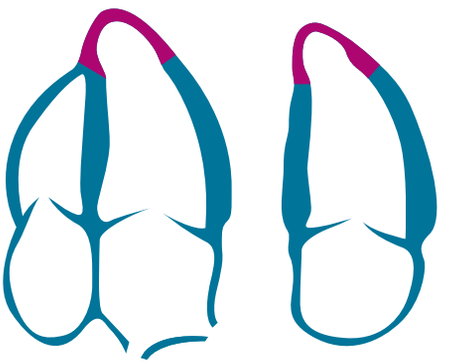
The Simpson method DOES NOT account for regional wall motion abnormalities in the posterior and all anterior septal segments (segments seen on the apical long-axis view).

NOTES

PATTERNS OF MYOCARDIAL INFARCTION

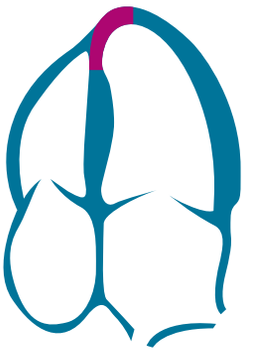
Supra-apical and distal septal infarctions may also occur in proximal LAD occlusion when rapid reperfusion is achieved and only the distal portions of the ventricle are damaged.

Supra-Apical Infarction



LAD (distal, mid., prox.), small supra-apical aneurysm, low remodeling risk

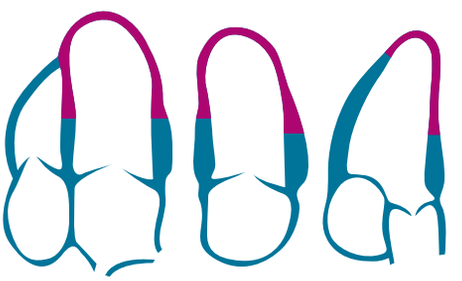
Distal Septum Infarction



LAD (distal,mid., prox.), low remodeling risk

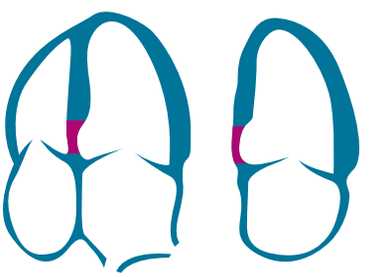
Patients with left main myocardial infarction rarely survive.

Proximal LAD Type Infarction



LAD (before 1st septal branch, left main), always remodeling, poor prognosis RCA

Small Basal Inferior Infarction



Difficult region to interpret, low remodeling risk

Inferior/posterior/postero-lateral infarctions pose an elevated risk for restrictive mitral regurgitation (tethering of the posterior leaflet) .

Inferior Infarction

RCA, low-moderate remodeling risk

Infero-Posterior Infarction

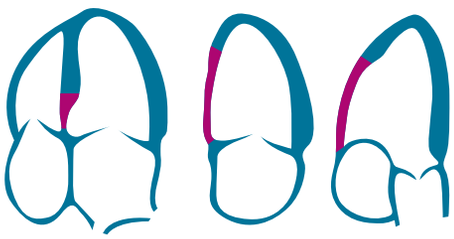
RCA (dominant) or Cx (large, prox.), moderate remodeling risk

PATTERNS OF MYOCARDIAL INFARCTION

Posterolateral Infarction

CX, RCA, moderate remodeling risk

Infero-Posterior-Lateral Infarction



Dominant RCA, CX (large, prox.), high remodeling risk

Lateral Infarction

CX, LAD (diagonal branch), difficult to interpret, low remodeling risk

NOTES

When assessing the patterns of myocardial infarction, always consider the possibility of multiple/sequential infarcts!

COMPLICATIONS

Overview

Acute/subacute

- Cardiogenic shock
- Thrombus formation (acute)
- Myocardial rupture
- Right ventricular infarction
- Papillary muscle rupture
- Ischemic ventricular septal defect

Chronic

- "Remodeling" chronic heart failure
- Right heart failure
- Thrombus formation (late)
- Mitral regurgitation

Perform serial echo exams after infarction. It will help you to detect potential complications earlier and assess the patient's prognosis and risk of further complications.

Pseudoaneurysm

- Short, narrow neck (diameter < 50% of the fundus diameter)
- Hematoma
- Outer walls formed by pericardium and mural thrombus
- Often pericardial effusion

High risk of secondary perforation/rupture.

NOTES

COMPLICATIONS

Myocardial Rupture

- Mortality 95%
- Also small infarctions
- Hematopericardium
- True incidence unknown
- Tamponade
- Urgent surgery required

Ischemic Ventricular Septal Defect

- Incidence 0.5 – 1%
- Within 4–5 days
- 50% Mortality
- Risk factors (hypertension, 1st MCI)

Echo Features

- Left ventricular volume overload
- Disrupted/spliced interventricular septum
- Turbulent flow/jet on color Doppler
- CW Doppler jet velocity depends on the size of the VSD and pressure relation between the left and right ventricle
- Elevated flow velocity across the pulmonic valve
- Acute pulmonary hypertension

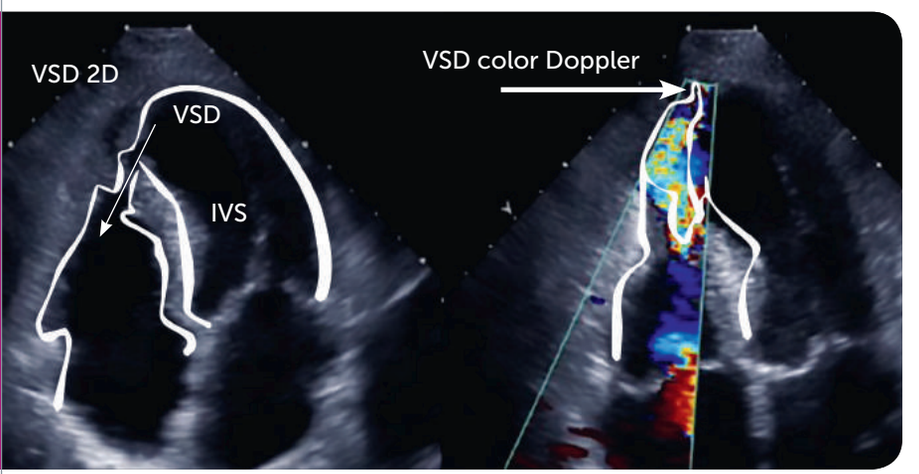
The most common site of rupture is the distal anterior septum (anterior myocardial infarction), followed by the basal inferior septum (inferior myocardial infarction).

Basal VSD jets may be difficult to discern from a tricuspid regurgitation signal in the Color Doppler.

Ischemic VSDs are rarely a simple hole in the septum, but rather the result of splicing of the interventricular septum.

ISCHEMIC VENTRICULAR SEPUTM DEFECT (VSD) – apical four-chamber view

Rupture of the interventricular septum is visible on the 2D image (left). Turbulent flow across the defect is seen with color Doppler (right).



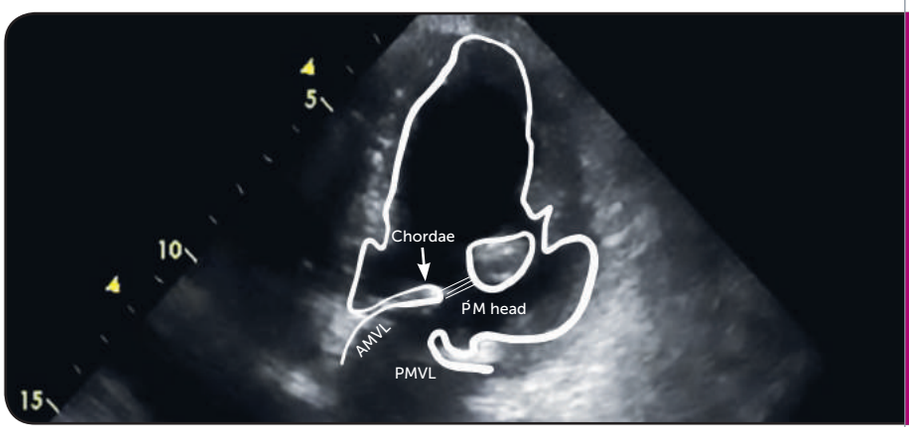
Papillary Muscle Rupture

- Incidence 1%
- Rupture of the posteromedial papillary muscle is more common than the anterolateral one (which has dual blood supply)
- 5% of deaths due to myocardial infarction
- Mortality 70%
- Also in small infarctions

COMPLICATIONS

Echo Features

- Severe mitral regurgitation
- Flail papillary muscle
- Left ventricular volume overload (LV dilatation/hyperdynamic function)
- Low-velocity mitral regurgitation signal
- Triangular shape of the mitral regurgitation spectrum (low systolic blood pressure in shock and pressure equilibration between the left ventricle and the left atrium)
- Pulmonary hypertension
- Dilated pulmonary veins



PAPILLARY MUSCLE RUPTURE – apical four-chamber view/2D

The head of the papillary muscle is detached from its body and swings freely between the left ventricle and the atrium attached to the mitral valve.

Right Ventricular Infarction

- 30 – 50% of inferior myocardial infarction
- Posterior wall, posterior septum affected
- Recovery of right ventricular function is common after acute myocardial infarction
- Poorer prognosis
- Usually in proximal RCA (Cx possible)

Echo Features

- Dilated right ventricle
- Wall motion abnormalities (inferior)
- Global/regional reduced right ventricular function
- Tricuspid regurgitation (common)
- Dilated inferior vena cava

Look at regional and global RV function in EVERY patient with inferior myocardial infarction. When asssessing the right ventricle, rotate around its axis to visualize the entire right ventricular myocardium.

Mural Thrombus

- Thrombogenicity of the infarct tissue
- Low flow state in the infarcted area
- More common in large anterior myocardial infarction
- Usually apex (aneurysm)
- Systemic embolism 2%
- Small thrombi are difficult to detect

Thrombi may be difficult to distinguish from prominent apical trabecula. Use LV contrast.

Echo Evaluation

- Visible in > 1 plane.
- Assess mobility to estimate the risk of embolism.
- Assess echogenicily (fresh/old thrombus).
- Measure size to monitor treatment effects.

Move the focus zone to the apex (near field) to increase your sensitivity.

NOTES

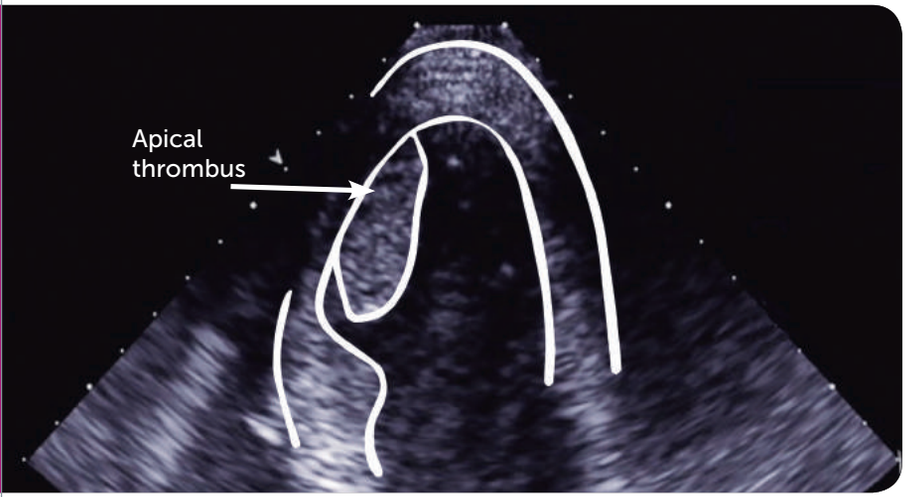
Transthoracic echo assessment may be difficult (due to tachycardia, pulmonary edema, lack of a distinct mitral regurgitation jet due to a large regurgitant orifice and low flow velocity, mitral regurgitation) – perform a transesophageal exam.

NOTES

APICAL THROMBUS – zoomed apical four-chamber view/2D

The thrombus has a slightly different echogenicity than the myocardium. Older thrombi appear more echodense.

COMPLICATIONS



Restriction of the posterior leaflets is a frequent finding in patients with inferior infarctions (regional remodeling of the inferior wall). Restriction of both leaflets is a consequence of global remodeling (and usually combined with annular dilatation).

Mitral Regurgitation in CAD – Mechanism

- Annular dilatation
- Leaflet restriction
- Rupture of papillary muscle (acute)
- Aggravation of mitral regurgitation in pre-existing MR caused by ventricular distortion (combined mechanisms)

Diagnosis of Posterior Leaflet Restriction

- Increase in tenting area
- "Y" position of anterior to posterior leaflet
- Jet origin further within the ventricle
- Immobility of the posterior leaflet (tethering)
- Posterior jet direction
- Increase in tenting area (increase of coaptation depth)

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009 // Aortic Stenosis

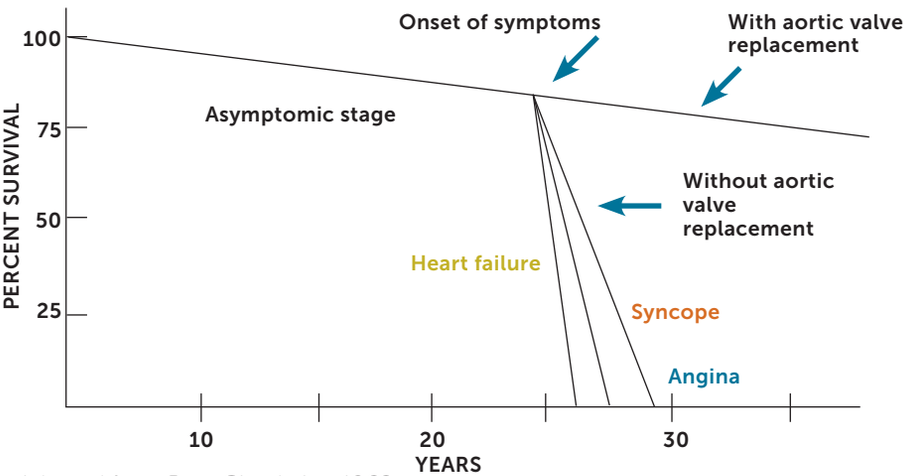
CONTENTS

NOTES

Severe asymptomatic aortic stenosis is generally associated with a favorable prognosis. The risk increases dramatically once symptoms occur.

BASICS

Natural History of Aortic Stenosis



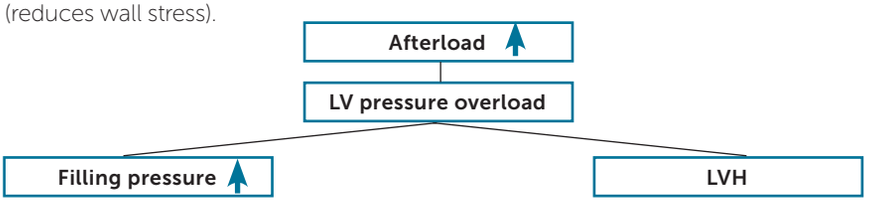
Adapted from Ross Circulation 1968

Epidemiology

- 3rd most common form of heart disease
- Increasing prevalence with older age (2–6% in the elderly)
- AV sclerosis is a precursor of AS

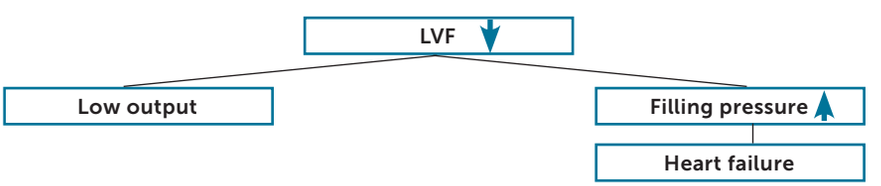
Hemodynamics in Aortic Stenosis

Patients with aortic stenosis have an increased afterload, which results in LV pressure overload. Left ventricular hypertrophy is a compensatory mechanism (reduces wall stress).



Left Ventricular Failure in Aortic Stenosis

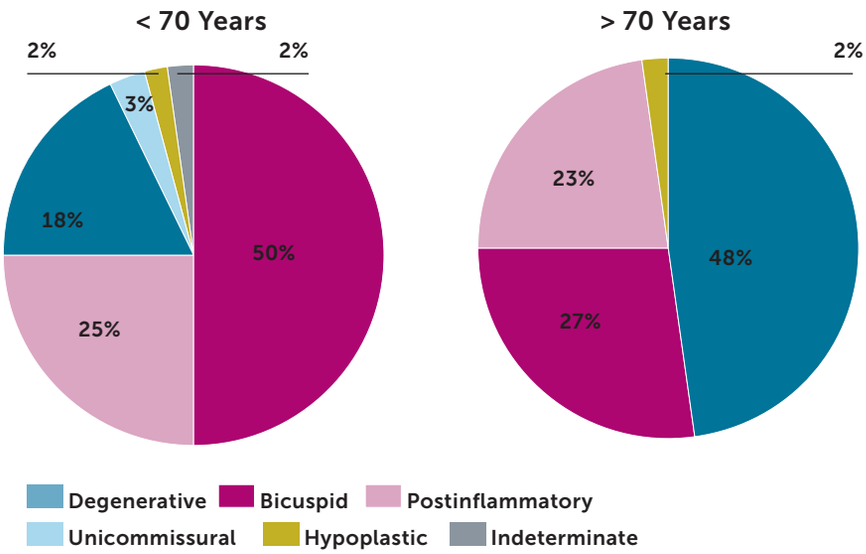
Persistent pressure overload leads to deterioration of left ventricular function and eventually heart failure.



BASICS

Causes of Aortic Stenosis

Congenital abnormalities of the aortic valve are a frequent cause of aortic stenosis. In some patients a stenosis is present at birth; in others congenital abnormal valves predispose the individual to aortic stenosis later in life (accelerated aging/calcification of the valve).



Adapted from Passik et al. Mayo Clinic Proc 1987

Rheumatic Aortic Stenosis

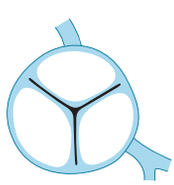
- Usually mild to moderate stenosis
- May progress to severe aortic stensos (accelerated valve aging)
- Often combined with aortic regurgitation
- Thickened leaflets/focal calcification
- Often multivalvular disease

Congenital Abnormalities of the Aortic Valve

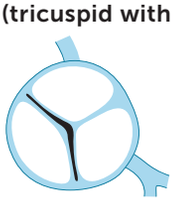
- Unicuspid, bicuspid, quadricuspid
- Syndromes (e.g. Down's, Heyde's)
- May be associated with genetic syndromes (such as Down's, Heyde's)

Morphology of the Aortic Valve

Normal valve (tricuspid)



Functional bicuspid (tricuspid with raphe) – congenital



NOTES

In the Western world, the cause of severe aortic stenosis in patients <50 years is almost always congenital.

The aortic valve is the second most common valve involved in rheumatic heart disease.

To establish the diagnosis of a bicuspid valve, use the short-axis view and observe the opening motion of the valve.

A raphe may be small and subtle. In this setting the valve may appear tricuspid, especially on a still frame.

NOTES

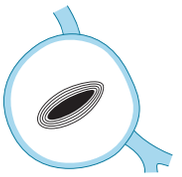
A dilated ascending aorta in a young patient may point to a congenital aortic valve abnormality.

Coronary artery disease is frequent in calcified aortic stenosis.

BASICS

Bicuspid – congenital

Unicuspid – congenital



Echocardiographic Assessment of Aortic Valve

2D

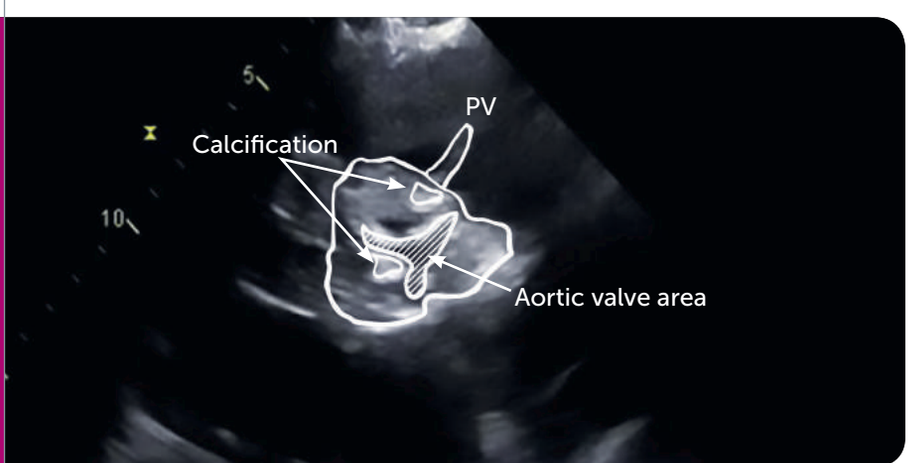
- Valve morphology (cusps)
- Visual assessment of aortic valve opening and motion
- Degree of calcification
- Left ventricular function
- Atrial enlargement
- Exclude subvalvular membrane
- Left ventricular hypertrophy
- Measurement of the aortic annulus (for valve sizing in TAVR)

MMode

- Eccentric AV closure
- "Box" seperation of cusps

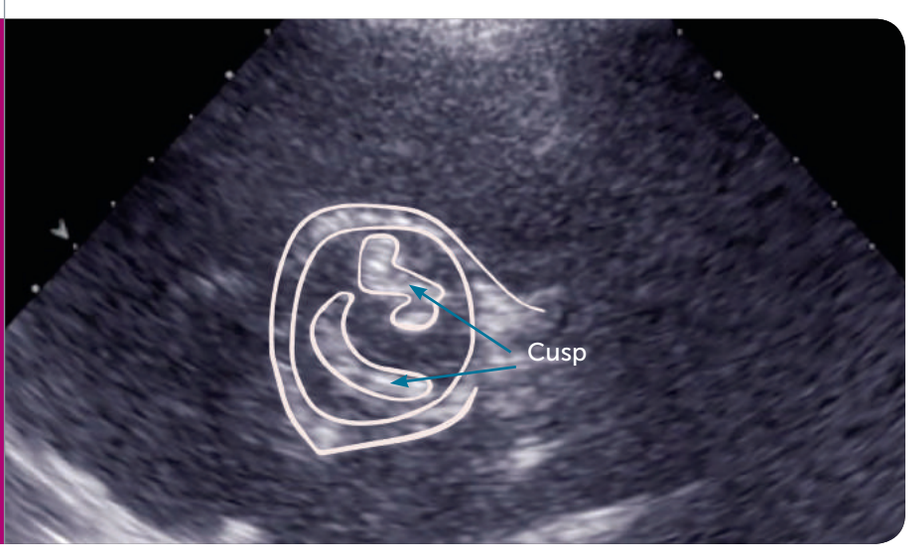
TRICUSPID AORTIC VALVE – zoomed PSAX AV

Calcified aortic valve with reduced opening (aortic valve area= AVA) in a patient with severe aortic stenose.



BICUSPUD AORTIC VALVE – zoomed PSAX AV

Calcified bicuspid aortic valve with severe stenosis. Only 2 cusps are visible. It may be difficult to determine whether a valve is bicuspid when it is heavily calcified.



BASICS

Doppler Assessment of the Aortic Valve

Color Doppler

- Color Doppler aliasing caused by high velocity jet (stenotic turbulences)
- Look for the origin of aortic stenosis jet to exclude LVOT obstruction (SAM/ membrane)?

CW/PW Doppler

- Measurement of maximum and mean velocity gradient across the aortic valve (CW Doppler)
- Measurment of LVOT velocity (PW Doppler)
- Diastolic dysfunction (filling pressure, indirect sign of severity, correlation with symptoms (PW Doppler)
- Elevated pulmonary pressure is a sign of left heart failure (CW Doppler)

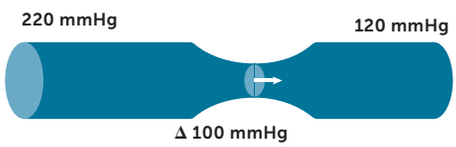
NOTES

Check were aliasing (flow acceleration) occurs: at the valve (valvular AS), below the valve (subvalvular stenosis) or above the valve (supravalvular aortic stenosis).

QUANTIFICATION OF AORTIC STENOSIS

Methods

- Planimetry (TEE)
- Pressure gradients
- Aortic valve area using continuity equation

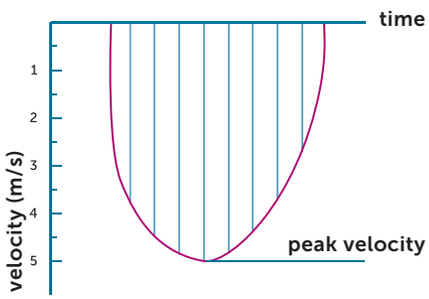


Stenosis results in a pressure gradient. The pressure gradient is high before the obstruction and low behind the stenosis.

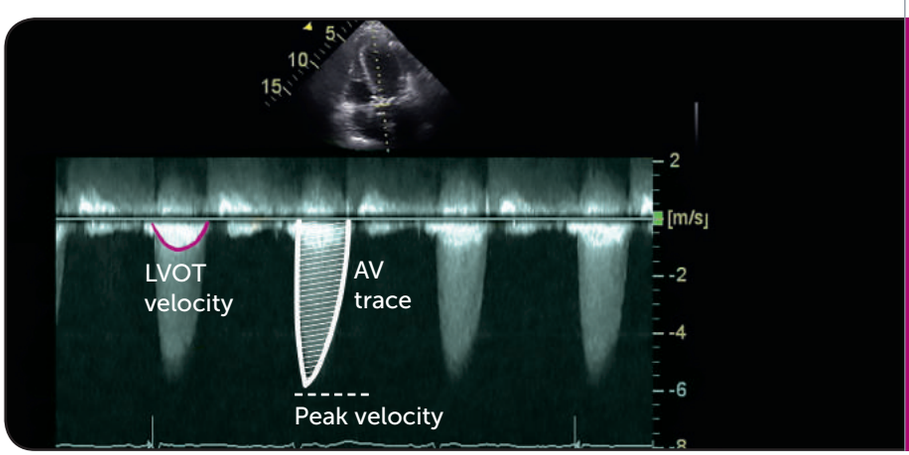
Planimetry (TTE) is usually not possible because the valves in AS are too heavily calcified (tracing the aortic valve orifice will be difficult).

Evaluation of Gradients

- Gradient = 4 x $Vmax^2$ (simplified Bernoulli equation)
- Gradients are influenced by heart rate and stroke volume
- Jet velocity is elevated (> 2m/s) when AVA < 2 – 2.5 cm^2



A late peak of the Doppler signal indicates severe aortic stenosis.



AORTIC STENOSIS SPECTRUM – apical five-chamber view/CW Doppler

Severe aortic stenosis with a peak velocity > 5.9 m/s during systole. The baseline is shifted upward and the velocity range adapted (8 m/s). Additionally, the LVOT velocity can be seen within the AS spectrum, indicating good Doppler alignment.

NOTES

Patients with bicuspid stenosis and those with severe AS generally have eccentric AS jets. In these patients you will usually obtain the highest gradient from a right parasternal approach.

High cardiac output (young or anxious patients, hyperthyroidism, fever, dialysis shunts, etc.) may cause flow velocities >2 m/s and thus mimic AS.

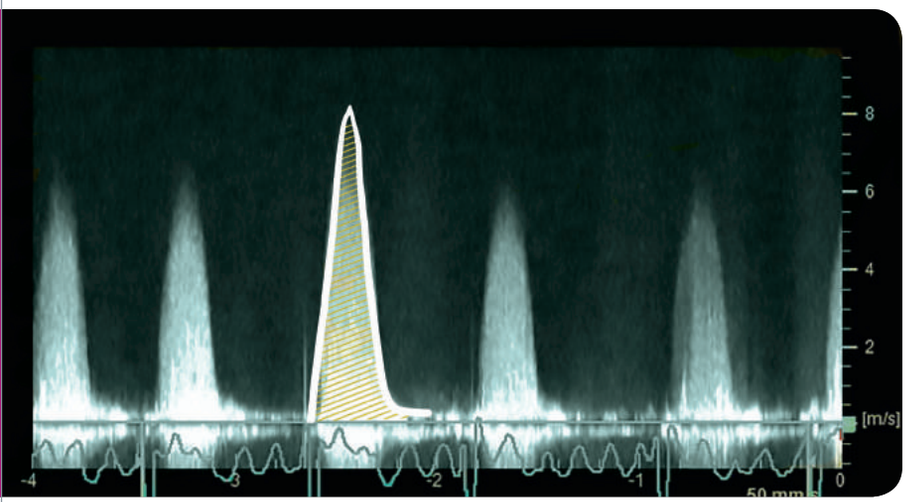
QUANTIFICATION OF AORTIC STENOSIS

Practical Considerations

- Try to be parallel to the stenotic jet and optimize the angle.
- Evaluate gradients from multiple windows (apical, suprasternal and right parasternal).
- Set the focus point of the CW Doppler in the aortic valve.
- Use the pencil probe.
- In the setting of atrial fibrillation, average the gradients of several beats and the PW-LVOT velocity.

RIGHT PARASTERNAL SPECTRUM – right parasternal view/CW Doppler CW

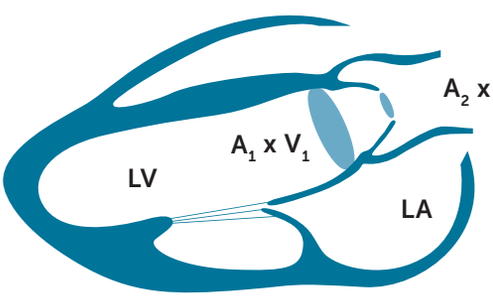
Doppler spectrum of severe aortic stenosis from a right parasternal view. The spectrum is directed towards the transducer and is therefore positive.



Measurement of LVOT width is most critical for the calculation of the aortic valve area. Small measurement errors result in large differences.

Calculation of Aortic Valve Area (Continuity Equation)

LVOT width is measured in the PLAX, slightly proximal to the aortic valve, exactly where you should also place the PW Doppler sample (5-chamber view).



$A_2 = V_1 \times A_1 / V_2$

$LVOT_{diam} = A_1$

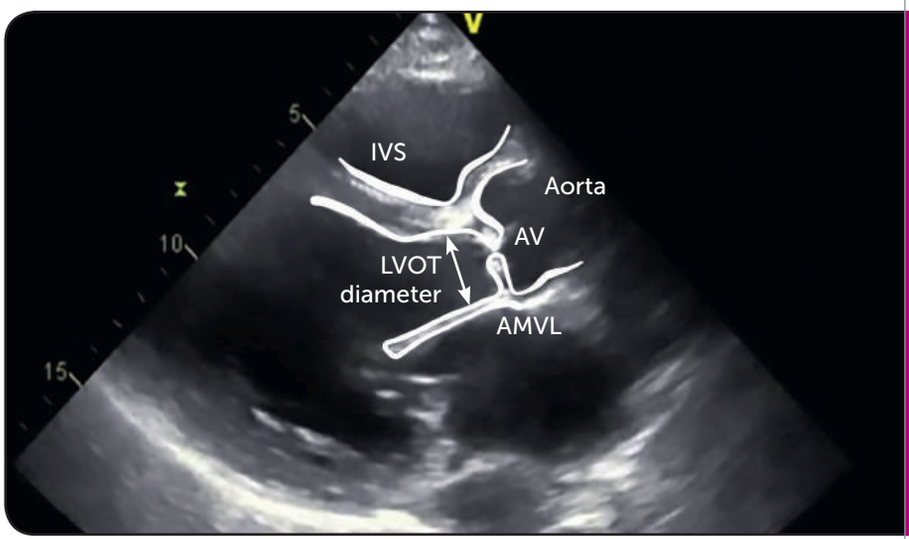
$LV{=}T_{vel} = V_1$

$AV_{vel} = V_2$

QUANTIFICATION OF AORTIC STENOSIS

Limitations of Continuity Equation

- Measurement of LV may be difficult.
- The true geometry of LVOT (round, oval) is not appreciated by the measurement of distances
- PW sample volume position plays an important role
- Underestimation of AV peak velocity in suboptimal Doppler alignment



LVOT DIAMETER – PLAX/2D

The LVOT diameter is measured on a parasternal long-axis view, closely below the aortic valve. It is advisable to slightly over-measure the LVOT diameter and thus compensate the oval shape of the LVOT.

Reference Values for Aortic Stenosis

	Mild	Moderate	Severe
Mean gradient	< 25 mmHg	25 – 40 mmHg	> 40 mmHg
Aortic valve area	> 1.5 cm^2	1.0–1.5 cm^2	< 1.0 cm^2
Jet velocity	< 3 m/s	3–4 m/s	> 4 m/s

ESC 2012

Valvulo-Arterial Impedance

Zva = (SAP + MG)/SVI

- Z(va) = measure of global LV load
- SAP = systolic arterial pressure
- MG = mean transvalvular pressure gradient
- SVI = stroke volume index.

Valvuloarterial impedance <3.5 increases the mortality risk 2.3 to 3 fold.

NOTES

To find the optimal location of the PW Doppler sample volume, place it first in the AS jet and slowly move the sample volume proximally until there is a sudden velocity drop.

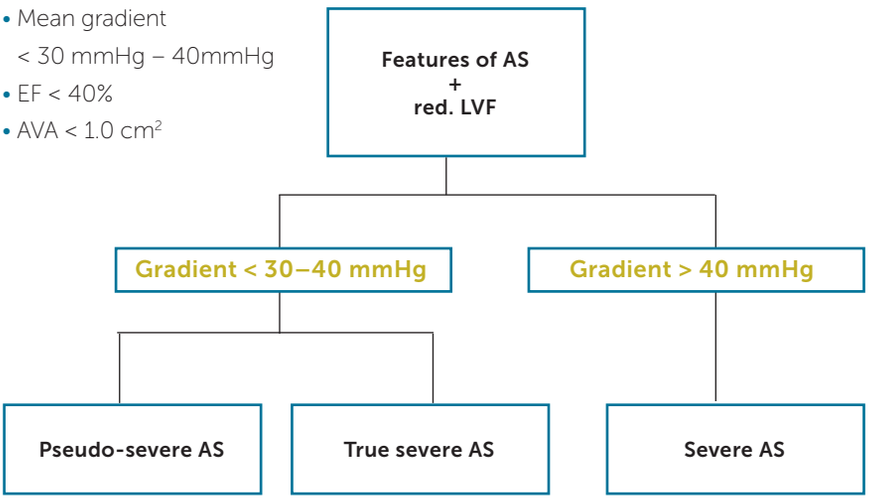
NOTES

SPECIAL CIRCUMSTANCES

Low Gradient Aortic Stenosis

To differentiate between true severe and pseudo-severe AS, you should perform a dobutamine stress echo.

- Mean gradient < 30 mmHg – 40mmHg
- EF < 40%
- AVA < 1.0 cm^2



Factors in Favor of True Severe "Low-Flow Low-Gradient" Aortic Stenosis

Correct classification makes a difference. Patients with true aortic stenosis are potential candidates for valve replacement.

- Heavily calcified valve
- Late peak of AS signal
- LVH (in the absence of hypertension)
- Previous exams with higher gradients

"Paradoxical" Low-Flow Low-Gradient Aortic Stenosis

Patients with paradoxical low-flow low-gradient AS tend to have a higher level of LV global afterload, which is reflected by a higher valvulo-arterial impedance.

Patients with aortic stenosis and very small ventricles/cardiac output may also have low gradients in the setting of severe aortic stenosis.

Low gradients in severe AS/ normal EF

- AVA < 1.0 cm^2
- EF > 50 %
- Mean gradient < 40mmHg

Low stroke volume (<35ml/m^2)

- Concentric LVH ?
- Small, restrictive LV
- Calcified valve
- (Hypertension)

Aortic Stenosis and Aortic Regurgitation

The gradients overestimate AS severity only when aortic regurgitation is moderate or in excess of moderate.

- Tend to occur simultaneously
- Common in bicuspid valves
- Significant aortic regurgitation leads to higher gradients (overestimation of the severity of aortic stenosis)

SPECIAL CIRCUMSTANCES

Pressure Recovery

Increase of pressure downstream from the stenosis caused by reconversion of kinetic energy to potential energy

Where is it relevant?

- Small aorta < 30mm
- Moderate aortic stenosis
- High flow rate
- Bileaflet prosthesis
- Funnular obstruction

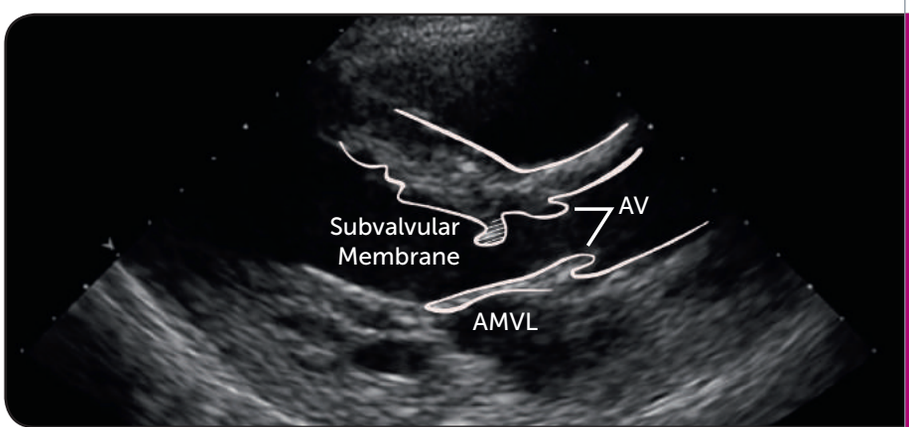
NOTES

Pressure recovery may lead to overestimation of gradients.

SUB- AND SUPRAVALVULAR AORTIC STENOSIS

Subvalvular Aortic Stenosis (Membranous)

- 2nd most common LV outflow obstruction
- Variable morphology (i.e. muscular ridge)
- A transesophageal study is often required



SUBVALVULAR AORTIC STENOSIS – PLAX/2D

A muscular ridge with a membrane causing obstruction is seen in the LVOT. In some patients you will need to scan through the entire LVOT to detect the membrane.

Other Findings in Subvalvular Aortic Stenosis

- Abnormal mitral valve chords
- Associated defects (50%) (e.g. PDA, VSD, bicuspid AV, pulmonic stenosis)

Echo Features

- Color flow aliasing at the site of obstruction
- Elevated CW velocity despite normal AV morphology
- Membrane of varying thickness within the LVOT, often with a small muscular ridge. Best visualized on atypical PLAX views

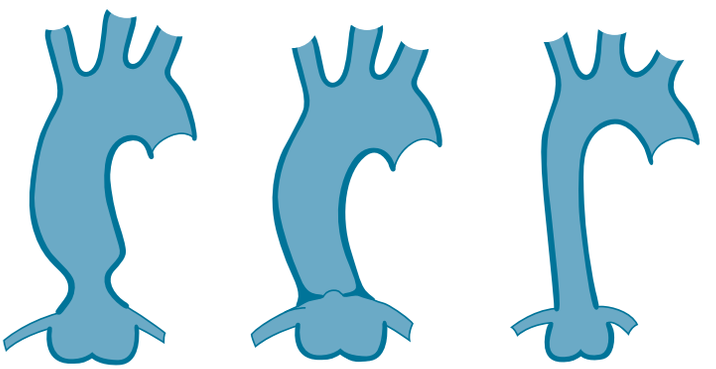
Subvalvular obstruction leads to aortic valve destruction (jet lesion) and aortic regurgitation.

NOTES

Use other imaging modalities (CT/MRI) and look for other congenital abnormalities (Williams syndrome).

SUB- AND SUPRAVALVULAR AORTIC STENOSIS

Types of Supravalvular Aortic Stenosis



Hourglass type (most common) | **Membranous type** | **Tubular type**

INDICATIONS FOR AORTIC STENOSIS SURGERY/INTERVENTION

When the patient does not fulfill the criteria/indications for surgery, annual follow-up should be performed. Shorter intervals are necessary when AS is severe, heavily calcified or when symptoms are uncertain.

Indications for Surgery in Severe AS (Class I/ESC 2012)

- Symptomatic patients with severe AS (dyspnea, syncope, angina)
- Symptomatic patients with severe AS and reduced LV function (<50% EF)
- Asymptomatic patients with severe AS and abnormal exercise test
- When other cardiac surgery is being performed (e.g. CABG; ascending aorta)

The indication for aortic valve surgery must be established individually. Consider age, co-morbidities, the risk of myocardial fibrosis in LVH, longitudinal dysfunction, the degree of calcification, the patient's preference and expectations, the rate of progression, etc.

Other Things to Consider in Asymptomatic Severe AS

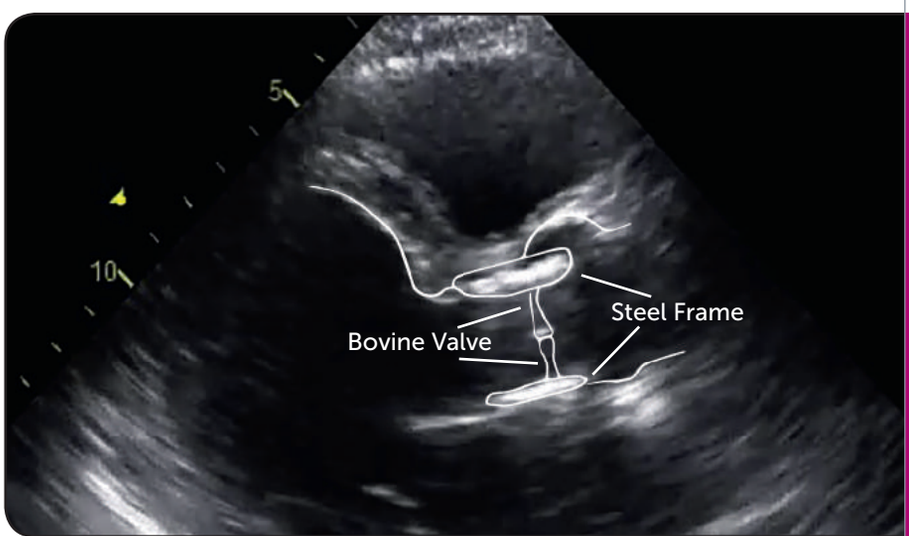
- Valve morphology (bicuspid)
- Severity of AS (very severe AS)
- Degree of calcification
- Subclinical myocardial dysfunction (longitudinal function)
- Rapid progression

INDICATIONS FOR AORTIC STENOSIS SURGERY/INTERVENTION

Transcatheter Aortic Valve Replacement (TAVR)

Consider interventional valve replacement in:

- Symptomatic/severe aortic stenosis
- High-risk patients
- Suitable anatomy (AV annulus diameter)
- Appropriate anatomical access for valve implantation (transfemoral/transapical)



TRANSCATHETER AORTIC VALVE – PLAX/2D

The steel frame and the bovine pericardial tissue leaflets of an Edwards-Sapien valve are visible in the aortic annulus.

Echo Assessment for TAVR

- Establish the presence of severe aortic stenosi.
- Assess annular dimension during systole in a zoomed PLAX for valve sizing Undersizing may lead to device migration or significant paravalvular aortic regurgitation. Oversizing increases the risk of underexpansion, reduces durability, and increases vascular access complications
- Assess the extent and distribution of calcification
- Exclude patients with bicuspid valves (an ellipitical orifice may predispose to incomplete valve deployment)
- Exclude patients with basal septal hypertrophy and dynamic LVOT obstruction

NOTES

The indications for TAVR may change with improvements in methodology.

Consider alternatives for the measurment of the aortic valve annulus (2D/3D TEE, CT), as these methods are more accurate than 2D echocardiography.

NOTES

010 // Aortic Regurgitation

CONTENTS

NOTES

BASICS

Study the morphology of the aortic valve on a PSAX view at the base.

Cause of Chronic Aortic Regurgitation

- Degenerative/Sclerosis/Aging
- Aortic dilatation
- Congenital
- Postendocarditis
- Rheumatic
- Aortic valve prolapse/rupture

Elevated left ventricular filling pressure (diastolic dysfunction) usually denotes LV deterioration (and symptoms).

Hemodynamics in Aortic Regurgitation

- Left ventricle volume overload
- Dilated left ventricle
- Filling pressure elevated
- Afterload increased

Quantification of Aortic Regurgitation Should be Based on

- Aortic regurgitation jet (Vena contracta, width, flow convergence)
- Deceleration time or aortic regurgitation spectrum (PHT)
- Retrograde flow in the aorta
- Indirect findings

LV dilatation is usually less when AS and LVH are additionally present.
In our experience the ventricle compensates more by dilatation than with an increase in ejection fraction.

Indirect Findings in Aortic Regurgitation

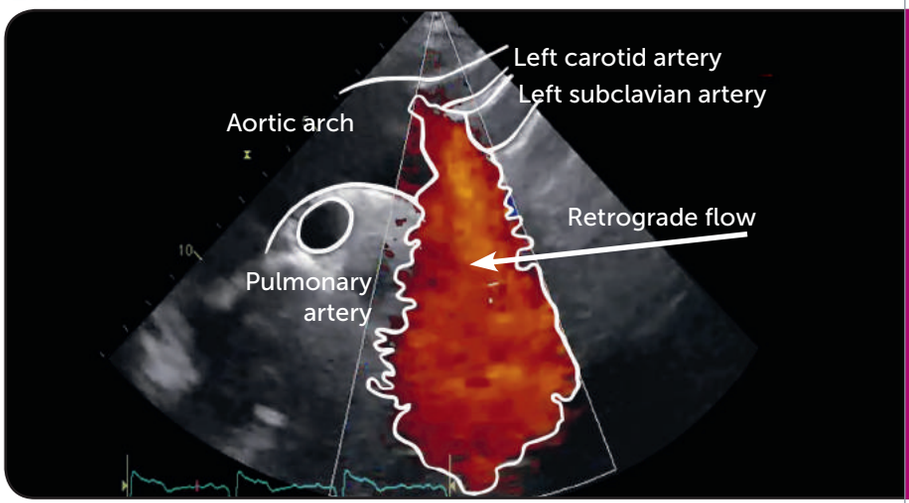
- Dilated left ventricle
- Hyperdynamic function
- Eccentric left ventricular hypertrophy
- Slightly enlarged left atrium
- Mitral regurgitation (annular dilatation)
- Diastolic dysfunction

Look at the vena contracta and PISA. Use an integrative approach for quantification.

Imaging of Aortic Regurgitation Jet

- PLAX
- PSAX (visualize origin of jet)
- Suprasternal (to determine retrograde flow)
- Five-chamber view/ three-chamber view

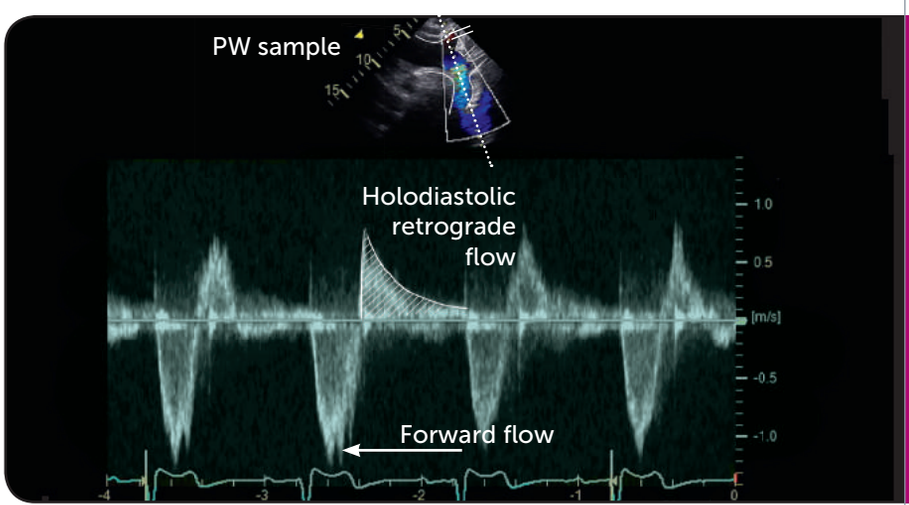
BASICS



NOTES

RETROGRADE FLOW IN AR – suprasternal view/Color Doppler

Severe retrograde flow during diastole. The red color Doppler signal denotes flow towards the transducer from the descending aorta towards the the arch. Color Doppler may be used to guide positioning of the PW Doppler spectrum.



RETROGRADE FLOW IN AR – Suprasternal view/PW Doppler

Holodiastolic flow with a maximum velocity of 0.7 m/s, indicating severe aortic regurgitation.

Aortic Regurgitation – Reference Values

	Mild	Moderate	Severe
Vena contracta	< 3mm	3 – 6mm	> 6mm
Jet width (% of LVOT)	< 25	25 – 65	> 65
Flow convergence	not visible	small	large
Pressure half-time (PHT) aortic regurgitation (msec)	> 500	200 – 500	< 200

ESC 2013

1) AR may be difficult to quantify in tachycardia and higher heart rates. 2) Retrograde flow is very important. 3) Use both color Doppler and PW Doppler to study retrograde flow.

To detect retrograde flow in the descending aorta, place the sample volume (PW-Doppler) at the inner curvature of the cranial portion of the descending aorta.

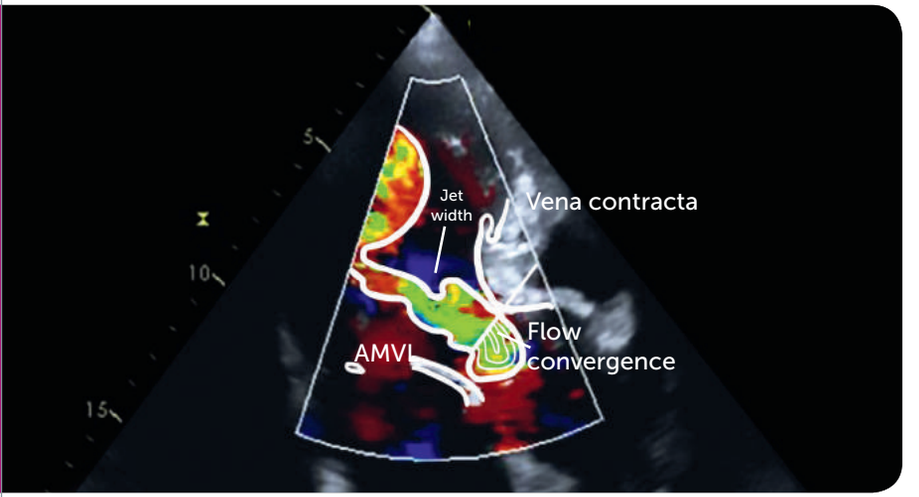
Holodiastolic retrograde flow in the aorta = severe AR.

NOTES

VENA CONTRACTA – apical three-chamber view

Severe aortic regurgitation with a large flow convergence zone, a vena contracta >6 mm, and a jet width of 70% of the LVOT.

BASICS



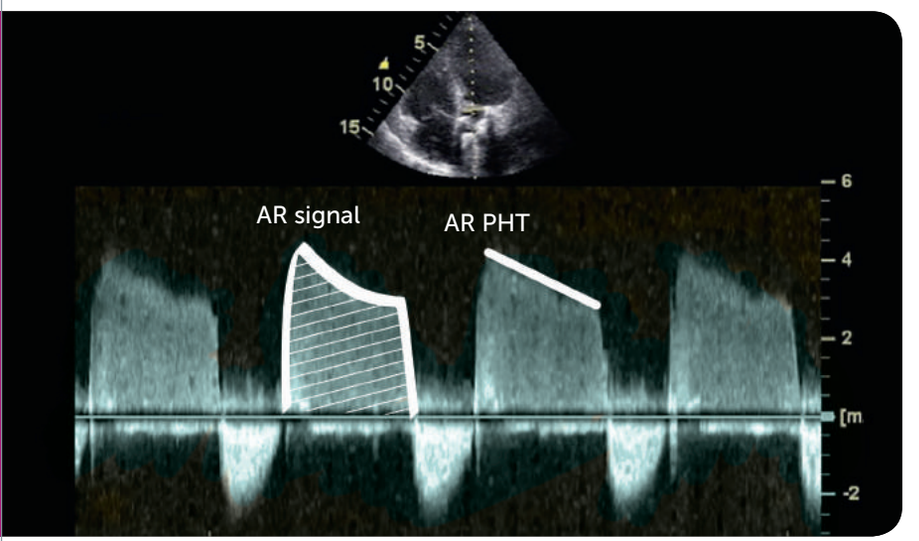
The AR signal should have a velocity above 4.5 m/second. Otherwise the signal quality will be inadequate for assessment of pressure half time (non-parallel jet alignment) .

Pitfalls

- Complex, eccentric, or multiple jets.
- Poor alignment of CW Doppler with the aortic regurgitation jet
- Calcified valves (it will be difficult to see the proximal flow convergence zone)
- Machine settings (PRF)

AR SPECTRUM – apical five-chamber view/CW Doppler AR

Pressure half-time is determined by measuring the slope of the AR signal. Severe AR is characterized by a very steep slope.



Aortic Regurgitation and Other Forms of Valvular Heart Disease

- Aortic regurgitation increases gradients in aortic stenosis.
- Aortic regurgitation shortens the PHT of mitral inflow in mitral stenosis.
- Volume overload of aortic regurgitation and mitral regurgitation add up (two halves make a whole).

HEMODYNAMIC CALCULATION OF REGURGITANT VOLUME AND FRACTION

$$RF\ (\%) = \frac{SV_{LVOT} - SV_{MV}}{SV_{LVOT}} = \frac{AR_{vol}}{SV_{LVOT}}$$

$$SV_{MV} = CSA_{MV} \times VTI_{MV} \qquad SV_{LVOT} = CSA_{LVOT} \times VTI_{LVOT}$$

$$CSA = d^2 \times 0.785$$

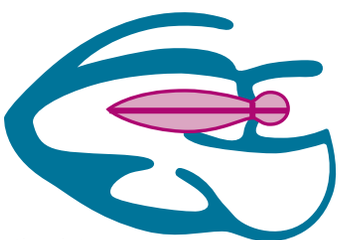
CSA = cross-sectional area **SV =** stroke volume **d=**diameter (MV/LVOT)

Reference Values

	Mild	Moderate	Severe
Regurgitant volume (ml/beat)	< 30	30 – 59	≥ 60
Regurgitant fraction (%)	< 30	30 – 49	≥ 50

PROXIMAL ISOVELOCITY SURFACE AREA (PISA) METHOD

$$ERO\ (PISA) = \frac{AR_{Flow} - SV_{MV}}{AR_{vel}} =$$



Aortic regurgitation$_{flow}$ = $2\pi \times r^2 \times Vr$
r = radius of flow convergence,
Vr = corresponding aliasing velocity,
Rvel = maximum velocity of the aortic regurgitation jet,
ERO = effective regurgitant orifice

Reference Values

	Mild	Moderate	Severe
Effective regurgitant orifice (cm^2)	< 0.1	0.1 – 0.29	≥ 0.3
Regurgitant volume (ml)	< 30	30 – 59	≥ 60

ESC 2013

NOTES

Hemodynamic calculations of AR are rarely used. Their main limitation is the inaccuracy of calculating the MV cross-sectional area.

No one ever uses this calculation, but you can impress your friends with it!

The PISA method for AR quantification is rarely used, but you can use flow convergence (PISA zone) for semiquantitative assessment.

NOTES

LV size = normal or slightly dilated and hyperdynamic (the ventricle has not had time to dilate/adapt).

ACUTE AORTIC REGURGITATION

Causes

- Endocarditis
- Cusp rupture
- Aortic dissection
- Iatrogenic (trauma)

Echo Features of Acute Aortic Regurgitation

- Small/slightly dilated left ventricle
- Tachycardia
- "Initially" hyperdynamic left ventricle
- Holodiastolic retrograde flow in the descending aorta
- Short pressure half-time
- Premature mitral valve closure

INDICATIONS FOR SURGERY IN SEVERE AORTIC REGURGITATION (ESC 2012)

Surgery is indicated

- In symptomatic patients
- In asymptomatic patients with reduced resting LVF (LVEF < 50%)
- In patietnts undergoing CABG or surgery of the ascending aorta, or another valve.
- In asymptomatic patients with severe LV dilatation: (left venricular enddiastolic diameter=LVEDD > 70 mm, LV endsystolic diameter=LVESD > 50 mm or LVESD/BSA >25 mm/m^2))
- If EF is too poor (< 30 – 35%) → Candidates for heart transplantation

011 // Mitral Stenosis

CONTENTS

NOTES

Vavular involvement is present in 2/3 of patients with rheumatic fever.

Rheumatic heart disease is very common in developing countries.

INTRODUCTION

Causes

- Rheumatic (most common)
- Stenotic annular calcification
- Congenital

The Shone complex is characterized by a combination of congenital mitral stenosis and other forms of left-sided inflow and outflow obstructions (coarctation, valvular/subvalvular aortic stenosis).

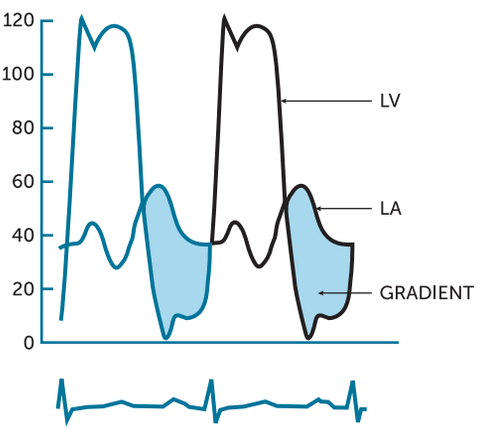
Congenital Mitral Stenosis

- Rare (0.6% of CHD)
- Combined with other congenital defects
- Forms: MV annulus hypoplasia, parachute MV, double-orifice MV

In mitral stenosis there is no "burden" on the left ventricle (no pressure or volume overload).

Effects of Mitral Stenosis

- LA-LV gradient
- Elevated pressure in LA
- Elevated pressure pulm. capillaries
- Pulmonary congestion/edema
- Pulmonary hypertension
- Right ventricular dilatation
- Tricuspid regurgitation
- Right heart failure
- Atrial fibrillation



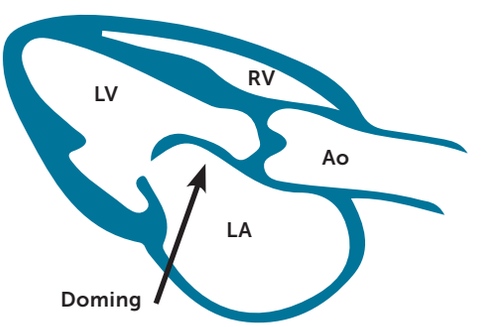
The pressure difference between the left atrium and the left ventricle as recorded with invasive measurements. The area between the curves corresponds to the mean gradient.

The MMode is no longer used to diagnose or quantify mitral stenosis.

Echo Characteristics of Mitral Stenosis

Valve features:

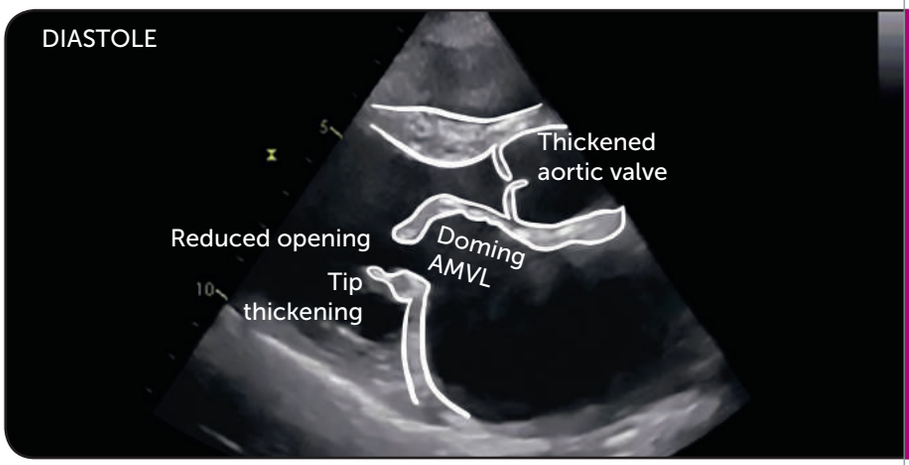
- Doming (diastolic bulging) of the anterior mitral valve leaflet
- Reduced valve opening
- Commissural fusion
- Leaflet tip thickening
- Subvalvular involvement (thickened and fused tendinae)
- Secondary calcification



Doppler Features

- Color Doppler is indicative of mitral stenosis (candle flame appearance)
- CW Doppler is used to quantify mitral stenosis (gradients/pressure half-time)

INTRODUCTION



NOTES

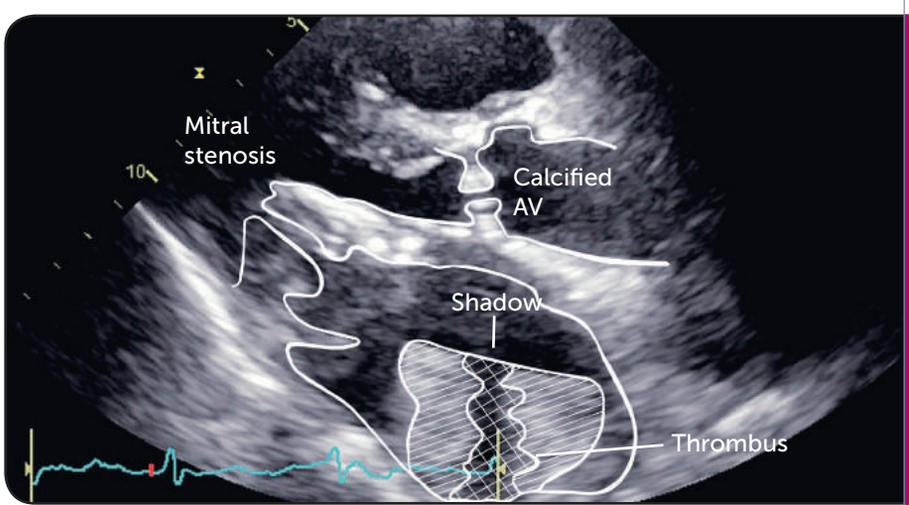
MITRAL STENOSIS – PLAX/2D

Typical features of mitral stenosis: Doming of the anterior leaflet, thickening of leaflet tips, thickened aortic valve (aortic valve involvement), and enlarged left atrium.

Other features of mitral stenosis/rheumatic heart disease

- Thickened aortic valve
- Reduced left ventricular function (high risk of atrial fibrillation)
- Enlarged left atrium, atrial fibrillation
- Pulmonary hypertension
- Aortic regurgitation
- Tricuspid stenosis
- Left atrial thrombuss

Many of these features develop and progress over time. Also consider these problems in your management strategy.



THROMBUS IN MITRAL STENOSIS – PLAX/2D

Severe mitral stenosis with large left atrial thrombus (partly shadowed by the calcified aortic valve).

Risk of Thrombus Formation

- Systemic embolism in 20% of all MS patients
- 80% of patients with severe MS are in atrial fibrillation
- 45% have left atrial spontaneous echo contrast

Most thrombi are seen in the left atrial appendage. Thus, you will miss them on transthoracic echo.

NOTES

QUANTIFICATION

MV Area – Reference Values

Normal (cm^2)	4 – 6 cm^2
Mild (cm^2)	> 1.5 cm^2
Moderate (cm^2)	1 – 1.5 cm^2
Severe (cm^2)	< 1 cm^2

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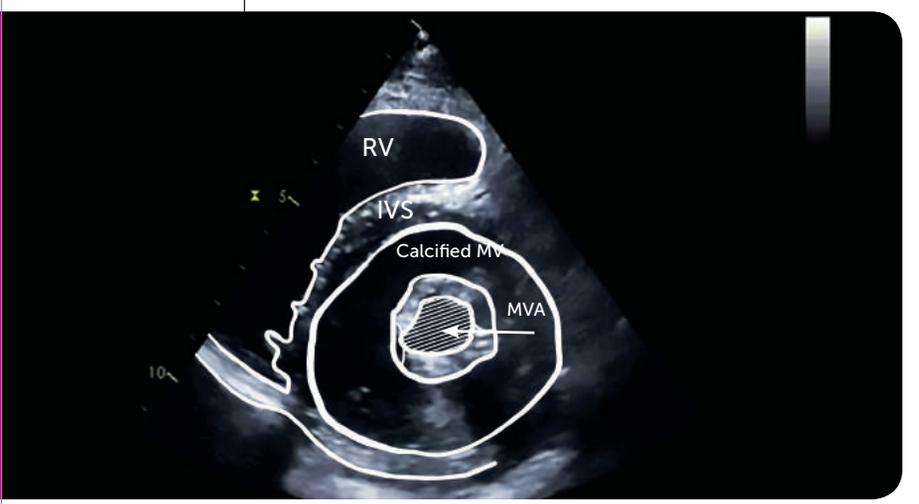
MITRAL VALVE PLANIMETRY – PSAX MV/2D

The mitral valve was investigated at the tip of the leaflets, where the mitral valve opening is smallest. The image is frozen in diastole at the time when mitral valve opening is largest. Tracing may be difficult when the valve is calcified.



Planimetry is the most direct method to quantify MS. It does not rely on hemodynamic assumptions. However, it is also technically the most challenging method.

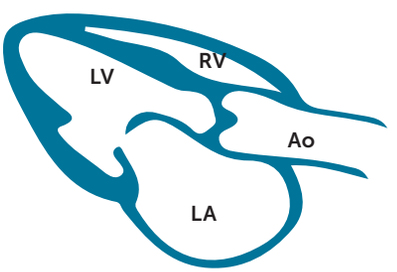
Problems of Mitral Valve Planimetry

Mitral valve area is measured on an optimized parasternal short-axis view at the smallest mitral valve orifice.

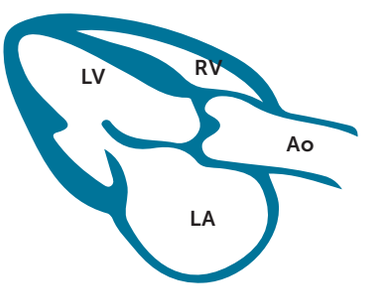
- Image quality
- Alignment
- Timing
- Calcification
- Atrial fibrillation
- Incomplete commissural fusion
- Operator experience

The funnular form is usually seen when there is strong involvement of the subvalvular apparatus.

Forms of Mitral Stenosis



Classic form

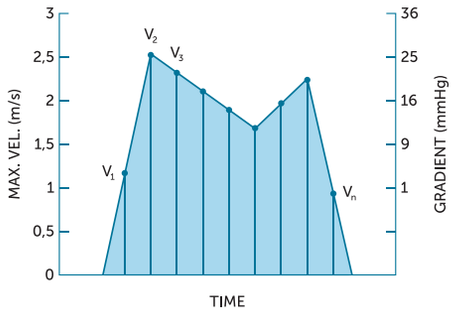


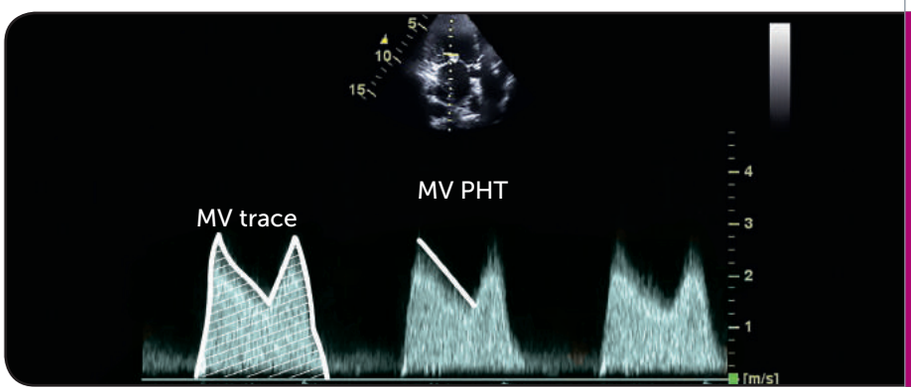
Funnular form

QUANTIFICATION

Mitral Stenosis Mean Gradient – Reference Value

Mild (mmHg)	< 5
Moderate (mmHg)	5 – 10
Severe (mmHg)	> 10





MITRAL STENOSIS SPECTRUM – apical view/CW Doppler

Mean gradients are obtained by tracing of the CW Doppler mitral valve inflow spectrum. The deceleration time (pressure half-time) is used to calculate mitral valve area.

NOTES

Transvalvular gradients are higher in the setting of additional mitral regurgitation.

MITRAL VALVE PRESSURE HALF-TIME

$$\text{MV Area} = \frac{220}{\text{PHT}}$$

The rate at which the gradient between the left atrium and the left ventricle diminishes corresponds to the size of the mitral valve orifice. The smaller the orifice, the longer is the pressure half-time.

PHT – pitfalls

- Diastolic dysfunction leads to overestimation of mitral stenosis
- Aortic regurgitation leads to underestimation of mitral stenosis
- PHT is unreliable after valvuloplasty.
- Heavily calcified valves make PHT unreliable
- Concave shape of tracing

The pressure half-time method is based on hemodynamic assumptions and was initially tested in young patients with rheumatic heart disease. It works less well in elderly and multimorbid patients with additional valvular lesions, left ventricular dysfunction and left ventricular hypertrophy.

Color Doppler, PISA and Continuity Equation

- Candle flame appearance of mitral valve inflow with color Doppler
- PISA for quantification (rarely used)
- **MVA = Mitral volume flow/peak velocity of diastolic mitral flow**
- Continuity equation (does not work when aortic regurgitation and mitral regurgitation are both present)

$$\text{MVA} = \frac{D^2_{\text{LVOT}}}{4} \times \frac{\text{VTI}_{\text{Aortic}}}{\text{VTI}_{\text{Mitral}}}$$

NOTES

MITRAL VALVE PRESSURE HALF TIME

Quantification of Mitral Stenosis in Atrial Fibrillation

Planimetry	Several different measurements (use average)
Mean gradients	Average 5 cycles with small variations of R-R intervals close to normal heart rate
Pressure half-time	Avoid mitral flow from short diastoles/ average different cardiac cycles

VALVULOPLASTY

Indication and Results

Indication

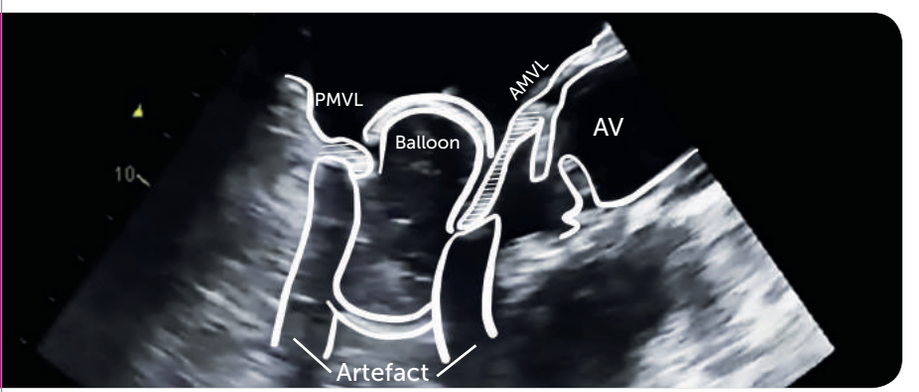
Clinically significant MS (valve area less than 1..5 cm^2 (1.8 cm^2 in unusually large patients)

Results

Good immediate results (valve area > 1.5 cm^2 without regurgitation) can be obtained in over 80%

BALLOONVALVULOPLASTY IN MITRAL STENOSIS – TEE long-axis view

The balloon is positioned within the mitral valve and expanded to enlarge the mitral valve orifice.



Suitability of Valve Morphology

- Mobility
- Valve thickening
- Thrombus
- Tricuspid regurgitation
- Subvalvular thickening
- Valve calcification
- Mitral regurgitation

VALVULOPLASTY

Wilkins Score

Patients with a Wilkins score > 8 – 10 are not ideal for mitral valve valvuloplasty.

Grade	Mobility	Thickening	Calcification	Subvalvular thickening
1	Highly mobile valve with only leaflet tips restricted	Leaflets near normal in thickness (4-5 mm)	A single area of increased echo brightness	Minimal thickening just below the mitral leaflets
2	Leaflet mid and base portions have normal mobility.	Mid-leaflets normal, considerable thickening of margins (5-8 mm)	Scattered areas of brightness confined to leaflet margins	Thickening of chordal structures extending to one third of the chordal length
3	Valve continues to move forward in diastole, mainly from the base.	Thickening extending through the entire leaflet (5-8 mm)	Brightness extending into the mid portions of the leaflets	Thickening extended to distal third of the chords
4	No or minimal forward movement of the leaflets occurs in diastole.	Considerable thickening of all leaflet tissue (>8–10 mm)	Extensive brightness throughout much of leaflet tissue	Extensive thickening and shortening of all chordal structures extending down to papillary muscles

Adapted from Wilkins et al. Br Heart J 1988

Complications of Mitral Valve Valvuloplasty

- Acute mitral regurgitation
- Iatrogenic atrium septal defect
- Embolism
- Tamponade (perforation following transseptal puncture)
- Vascular access complications/ bleeding

NOTES

For the suitability of mitral valve valvuloplasty also look at the commissural region. Patients with calcification of the commissures are not ideal candidates.

NOTES

012 // Mitral Regurgitation

CONTENT

NOTES

BASICS

Natural History of Severe Mitral Regurgitation

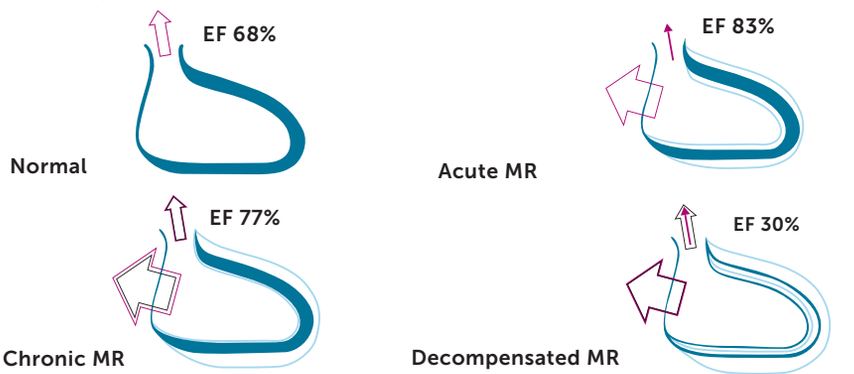
Severe mitral regurgitation is no benign condition.

- 10-year survival rate of 57%
- The 5-year all-cause mortality in patients with asymptomatic mitral regurgitation patients is 22%
- The 5-year risk for cardiac events in asymptomatic mitral regurgitation patients is 33%

Hemodynamics of Mitral Regurgitation

In the setting of significant mitral regurgitation, an ejection fraction of 55% to 60% (which is otherwise considered normal) already denotes left ventricular failure.

In acute mitral regurgitation (MR), the ejection fraction is high and the size of the left ventricle is normal or slightly enlarged (unadapted). In chronic mitral regurgitation the ejection fraction is "supranormal" and the left ventricle is dilated (adapted). In decompensated mitral regurgitation the left ventricle is significantly enlarged and the ejection fraction starts to drop.



Consequences of Mitral Regurgitation

Even when mitral regurgitation is severe, the patient may remain asymptomatic for a long period of time.

- Left ventricular volume overload
- Elevated left ventricular filling pressure
- Pulmonary hypertension
- Tricuspid regurgitation
- Reduced systolic wall stress
- Reduced afterload

Causes

Echocardiography provides important clues as to the cause of mitral regurgitation. Combinations of several etiologies are not uncommon (e.g. annular dilatation and restrictive leaflets).

Primary (structural) causes

- Mitral valve prolapse, myxomatous mitral valve disease
- Flail leaflet
- Valve fibrosis and calcification
- Rheumatic heart disease
- Congenital
- Papillary muscle rupture
- Endocarditis
- Drugs
- Systemic diseases

Secondary (functional) causes

- Annular dilatation
- Restrictive leaflets
- Systolic anterior motion
- Atrial enlargement

QUANTIFICATION OF MITRAL REGURGITATION

Integrative Approach

Color Doppler	Jet (flow convergence, vena contracta)
2D Imaging	Indirect signs

Quantification Based on Color Doppler

	Mild	Moderate	Severe
Vena contracta (mm)	< 3	3 – 6.9	≥ 7
Jet area (%)	Small, central jet (<20% of LA area)	Variable	Large, central jet (> 40% of LA area)

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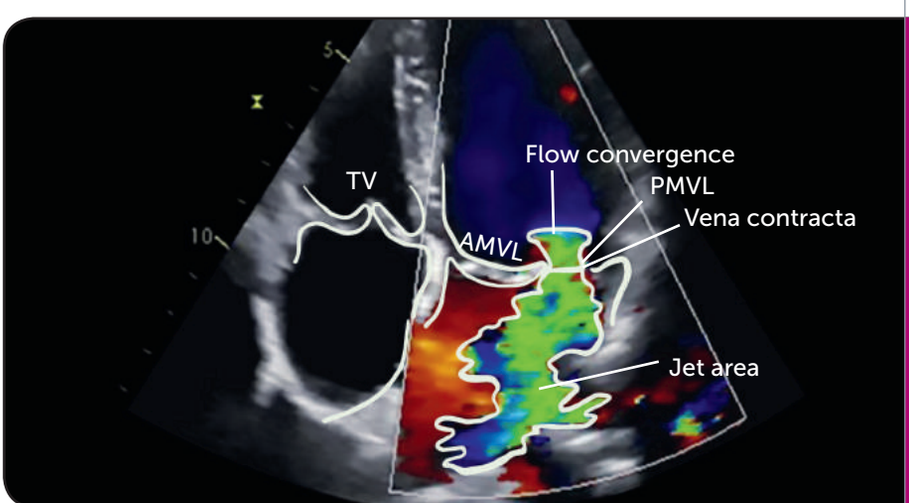


QUANTIFICATION OF MITRAL REGURGITATION – apical four-chamber view/Color Doppler

Typical color Doppler features of mitral regurgitation with a prominent flow convergence zone (PISA), a vena contracta ≥ 7mm, and a jet area > 40% of LA area.

Color Doppler Confounders

- Geometry of regurgitant orifice
- Multiple jets
- Coanda effect ("wall hugging" jets)
- Driving force (systolic pressure)
- LA compliance

NOTES

Your ability to image jets is more important than quantitative parameters. Use multiple views.

The proximal portions of the jet (the vena contracta and the flow convergence zone) are more important for the quantification of mitral regurgitation than jet area, length or width.

Do not base the quantification of mitral regurgitation on a single parameter.

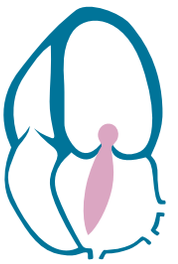
The PRF setting greatly influences the size of the jet. Always use the same PRF. If not, you will be unable to make comparisons.

The maximal mitral regurgitation velocity (CW Doppler) represents systolic blood pressure and does not correlate with the severity of mitral regurgitation.

NOTES

The size of the left atrium does not permit quantification of mitral regurgitation.

QUANTIFICATION OF MITRAL REGURGITATION

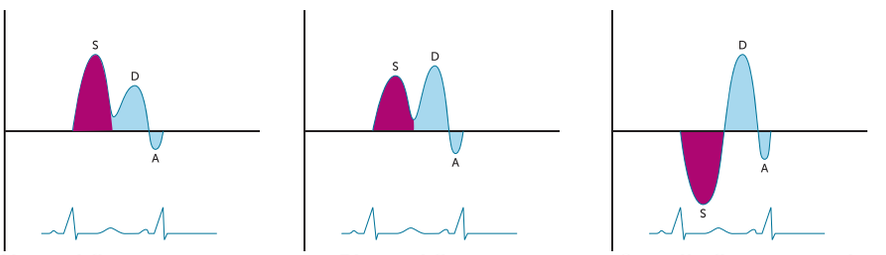


Indirect Signs

- Dilated left ventricle
- Hyperdynamic left ventricular function
- Left atrial enlargement
- Interatrial septum bulging (towards RA)

In most instances you will not need pulmonic vein Doppler to quantify mitral regurgitation. In addition, a good signal can only be obtained in 50–75 % of patients. Interpretation is difficult in atrial fibrillation.

Retrograde Flow in Pulmonic Veins



Normal flow **Blunted flow** **Systolic flow reversal**

With increasing degrees of mitral regurgitation, you will first note blunted flow of the systolic component of pulmonary venous inflow. Very severe forms of mitral regurgitation are accompanied by flow reversal of the systolic component.

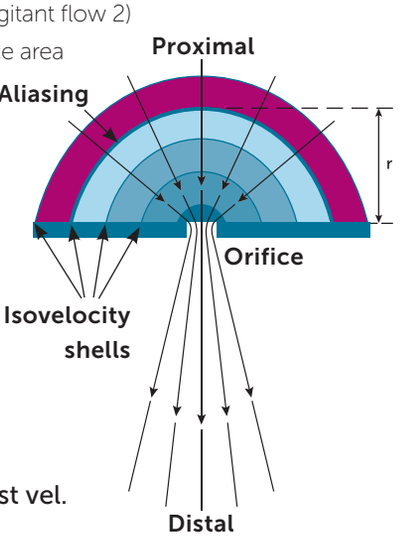
Use magnifications (zoom/RES) to enhance the accuracy of your measurement.

To calculate the regurgitant volume, you need to trace the mitral regurgitation spectrum obtained with CW Doppler.

Proximal Isovelocity Surface Area (PISA) Method

The PISA method allows calculation of: 1) regurgitant flow 2) regurgitant fraction 3) effective regurgitant orifice area

- Flow through hemispheric surface = flow through the orifice
- Shift aliasing limit to lower velocity 20 – 40cm/s (larger hemisphere)
- Effective regurgitant orifice area (EROA) = $[(2r^2 \times V_{pisa})/V_{mr}]$
- r= PISA radius, V_{pisa}= aliasing velocity, V_{mr}= peak MR velocity



Regurgitant volume= EROA x MR VTI

Regurgitant flow = $Q = 2 \times r^2 \times \pi \times$ Nyquist vel.

There is much controversy as to whether PISA should be used. New 3D echo techniques are likely to make PISA more reliable (better approximation of PISA geometry).

Limitations of PISA

- The geometry of orifice is not truly hemispheric.
- Multiple or excentric jets
- Difficulties in delineation of PISA
- Dynamic mitral regurgitation (flow changes throughout the cardiac cylce

QUANTIFICATION OF MITRAL REGURGITATION

Reference Values for Parameters of Mitral Regurgitation

	Mild	Moderate	Severe
Regurgitant volume (ml/beat)	< 30	31 – 59	≥ 60
Regurgitant fraction (%)	< 30	30 – 49	≥ 50
Effective regurgitant orifice area (mm^2)	< 20	20 – 40	≥ 40
Volumetric methods MR volume = MR inflow – aortic outflow (in the absence of AR)			

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Features that Affect the Severity of Mitral Regurgitation

- Blood pressure (afterload)
- Volume status
- Atrial fibrillation
- Dyssynchrony
- Anesthesia
- Exercise

The severity of mitral regurgitation may differ markedly in one and the same patient, especially in cases of functional mitral regurgitation.

Echo Signs of Acute Mitral Regurgitation

- Hyperdynamic left ventricle with a normal size
- Tachycardia
- Abnormal valve morphology (e.g. papillary muscle rupture, flail leaflet)
- Low velocity of the MR signal (shock)
- Triangular shaped MR spectrum
- Elevated MV inflow velocity

Patients with acute MR are difficult to image and interpret. These patients usually have low MR velocity jets (shock), tachycardia, and tachypnea.

MECHANISMS OF MITRAL REGURGITATION

Why Is the Mechanism Important?

- Etiology
- Prognosis (reversible)
- Management
- Repair?

Usually transthoracic echo is sufficient to determine the mechanism. If not, use transesophageal echo.

What Should Be Examined?

- Valve morphology (thickened, myxomatous)
- Extent of involvement (which parts of the valve are involved?)
- Origin of regurgitant defect
- Mechanism of mitral regurgitation

The extent of morphologic abnormalities of the mitral valve does not necessarily correlate with the severity of mitral regurgitation.

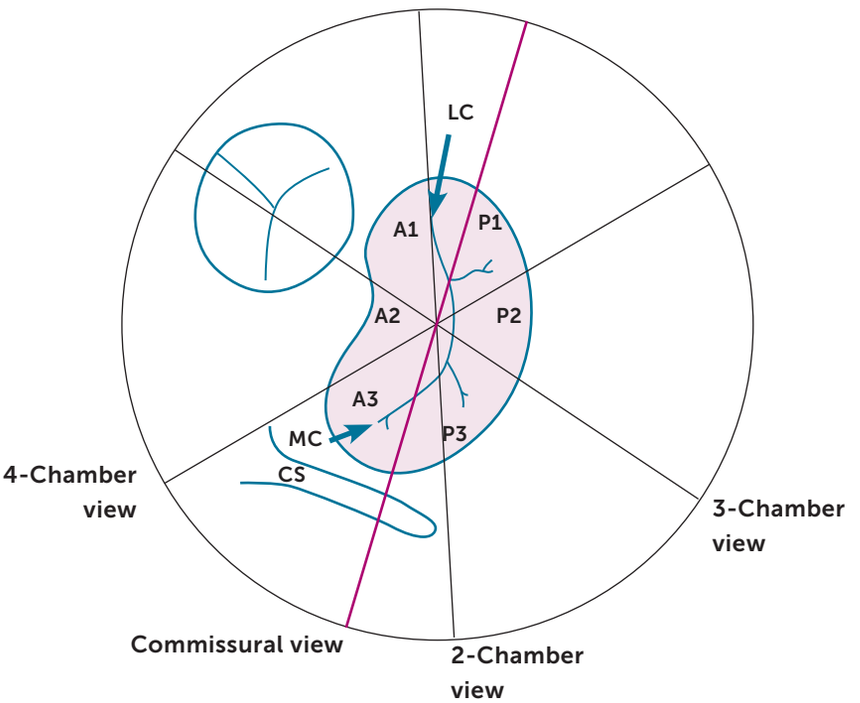
NOTES

NOTES

MECHANISMS OF MITRAL REGURGITATION

Do not forget to image the commissural regions. It is easy to miss mitral regurgitation.

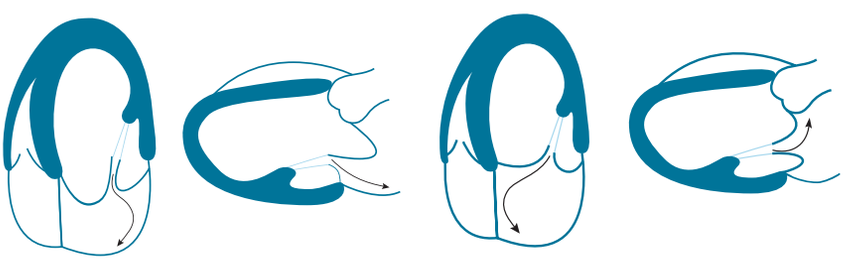
How to Visualize Mitral Valve Segments



CS = coronary sinus **LC =** lateral commissure **MC =** medial commissure

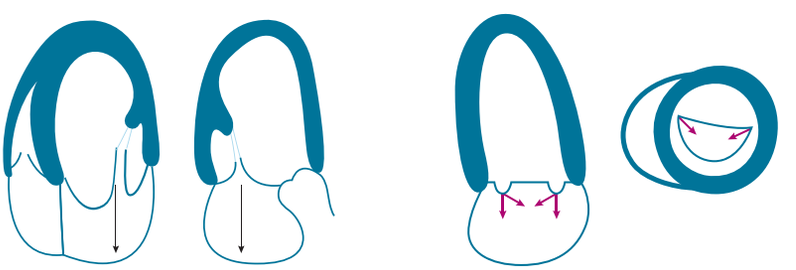
As a general rule in MV prolapse/flail leaflet, the jet direction is always opposite to the location of the defect (i.e. anterior jet direction in a posterior leaflet defect).

Mitral Valve Prolapse



Anterior leaflet prolapse (jet direction posterior + lateral)

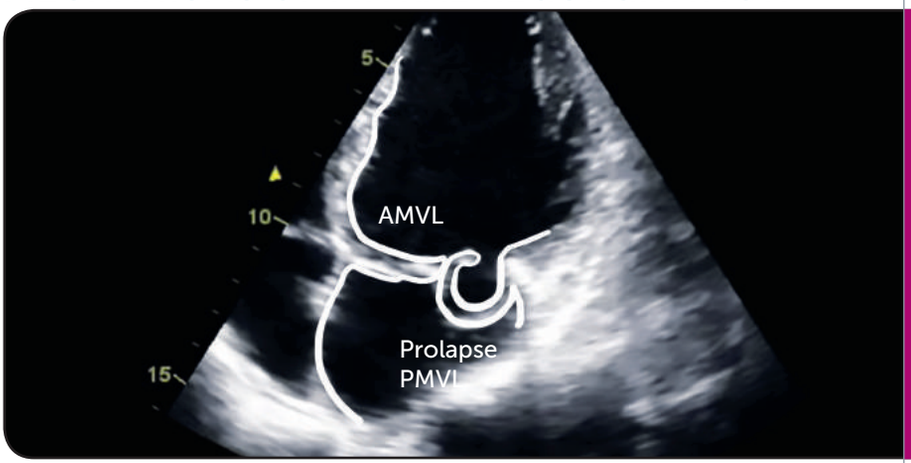
Posterior leaflet prolapse (jet direction anterior + medial)



Bileaflet prolapse (central jet)

Commissural prolapse/defect (jet at the origin of the commissure)

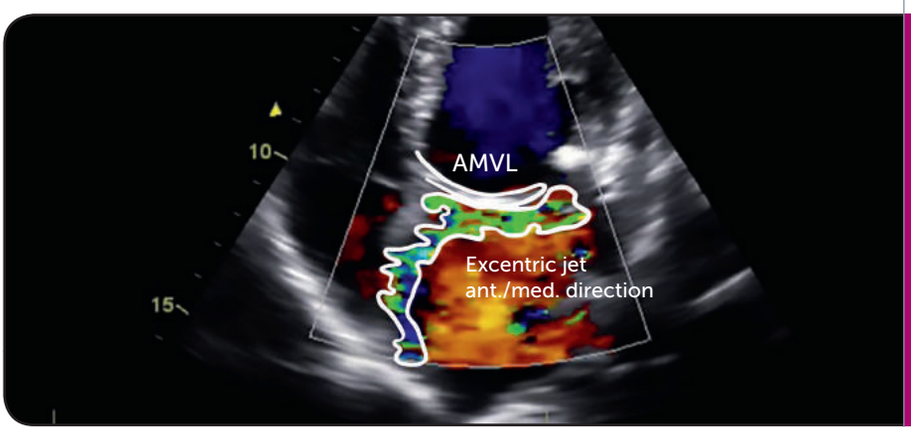
MECHANISMS OF MITRAL REGURGITATION



NOTES

PMVL PROLAPS – apical four-chamber view/2D

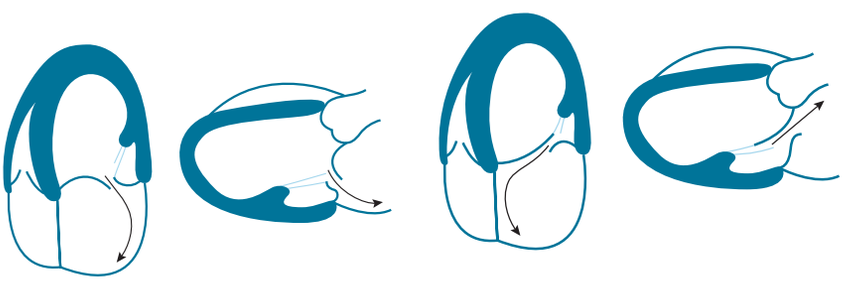
Severe prolapse of the posterior mitral valve leaflet (medal scallop – P2). The valve is thickened (myxomatous) and the left atrium/ventricle are enlarged.



PMVL PROLAPSE – apical four-chamber view/Color Doppler

The jet direction is typically anterior and medial (towards the interatrial septum).

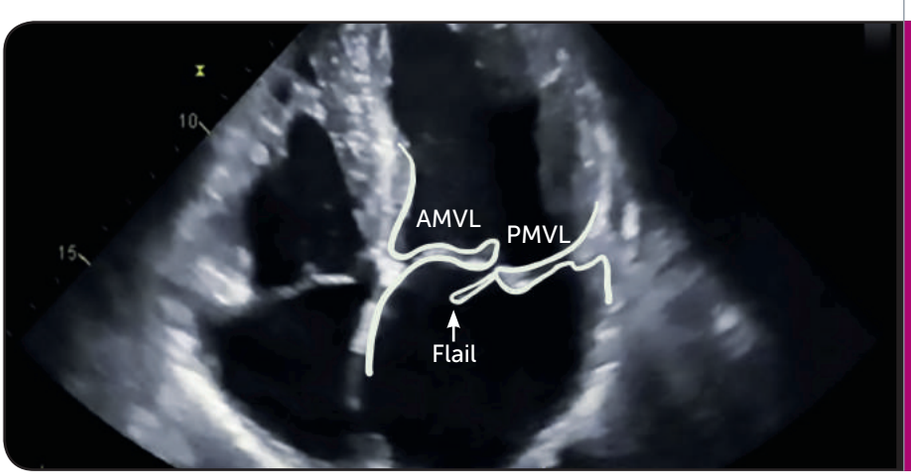
Flail Mitral Leaflet



Anterior flail leaflet
(jet direction posterior + lateral)

posterior flail leaflet
(jet direction anterior + medial)

The direction of the jet may vary throughout systole (like a loose garden hose).



PMVL FLAIL – apical four-chamber view/2D

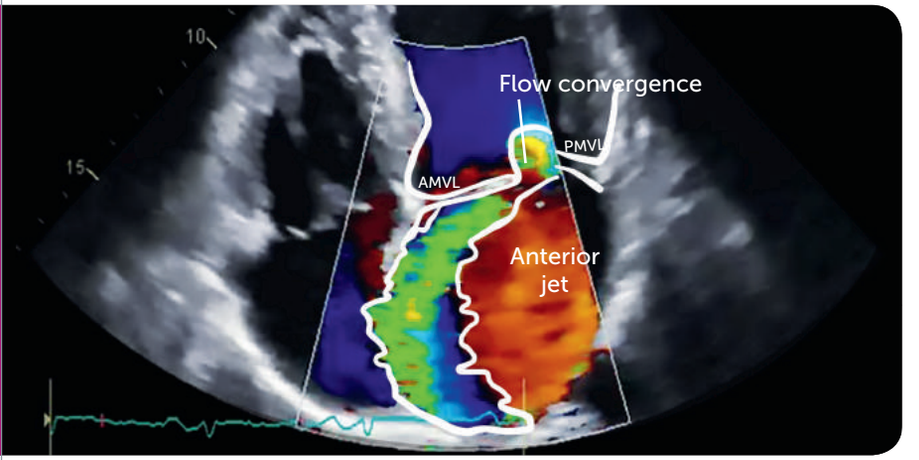
Flail posterior leaflet; the posterior leaflet protrudes behind the anterior leaflet into the left atrium. Small chordal structures are seen attached to the tip of the posterior leaflet.

NOTES

MECHANISMS OF MITRAL REGURGITATION

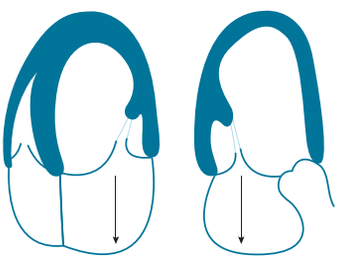
PMVL FLAIL – apical four-chamber view/Color Doppler

Chordal ruputure of the posterior leaflet directs the jet towards the interatrial septal and anterior (seen best on an apical long-axis view).

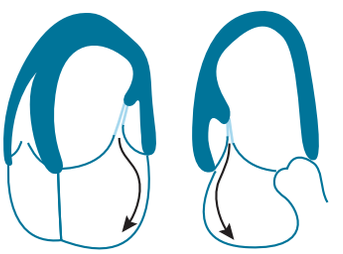


It is not uncommon to see a combination of mechanisms (e.g. annular dilatation and leaflet restriction)

Mitral Valve Leaflet Restriction



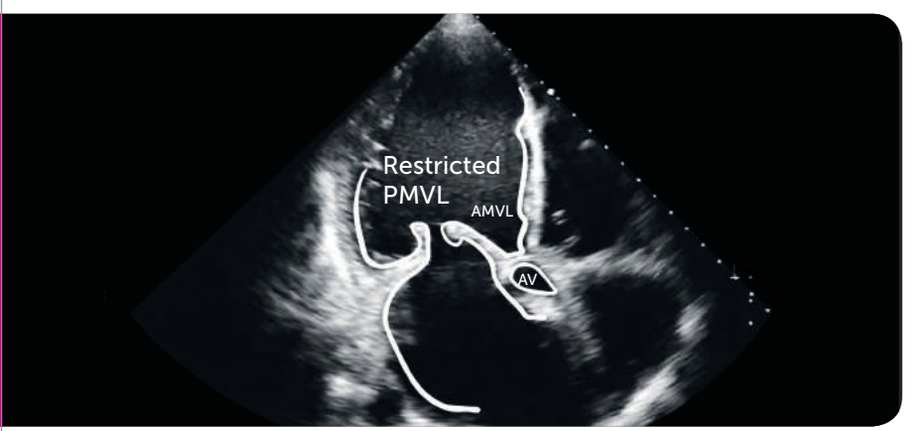
Restriction of both leaflets (central jet direction)



Posterior leaflet restriction ((jet direction lateral, posterior)

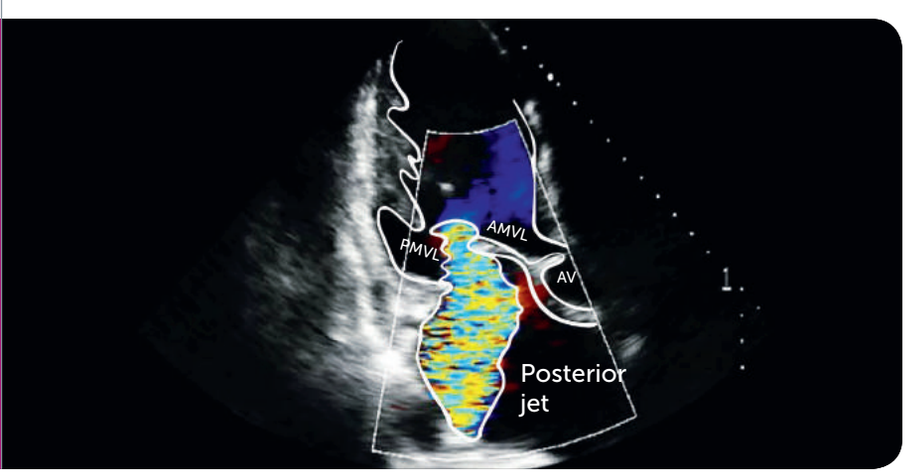
RESTRICTED PMVL – apical three-chamber view/2D

Inferior infarction and change of LV geometry restricts the motion of the PMVL. The leaflet is drawn towards the apex. This results in incomplete closure of the mitral valve.



RESTRICTED PMVL – apical three-chamber view/Color Doppler

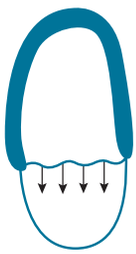
The jet in restricted posterior leaflet motion is typically direc ted posteriorly. It aligns with the position of the posterior leaflet.



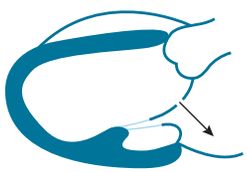
MECHANISMS OF MITRAL REGURGITATION

Other Causes

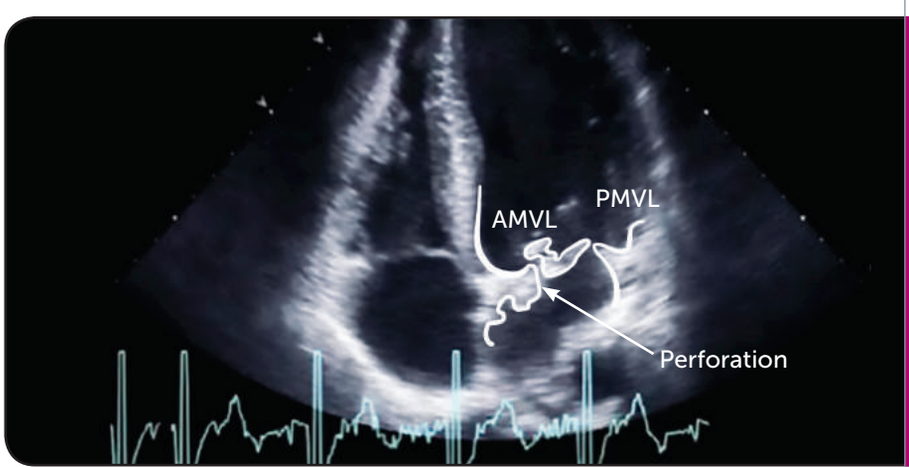
Annular dilatation
(central jet direction)



MR in hypertrophic CMP
(posterior jet direction)

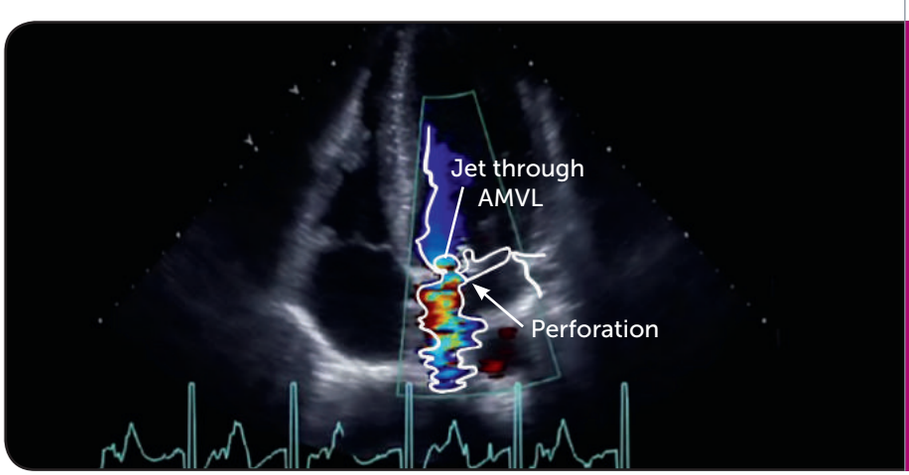


Valve perforation
(jet through leaflet)



AMVL Perforation – apical four-chamber view/2D

The anterior leaflet is thickened and destroyed. A small gap can be seen in the anterior leaflet. This patient has a perforated mitral valve after endocarditis.



AMVL PERFORATION – apical four-chamber view/ color Doppler.

The color jet clearly traverses the basal anterior leaflet through the perforation. The most frequent site of perforation is the anterior leaflet.

NOTES

In annular dilatation the jet direction may be slightly off the axis when other conditions such as mitral valve prolapse, asymmetric restriction, or other abnormalities of the valve are present.

Other mechanisms of mitral regurgitation include: annular calcification, leaflet retraction, and leaflet shrinkage (drugs/toxins).

NOTES

The success of mitral valve repair strongly depends on the surgeon's experience.

Repair techniques include quadrangular resection with sliding plasty, chordal transfer, and the use of artificial chords. Mitral valve repair usually includes implantation of an annuloplasty ring.

MECHANISMS OF MITRAL REGURGITATION

Unfavorable Factors for Repair

- Extensive involvement (more than two segments)
- Repair of the anterior leaflet is more difficult than the posterior one
- Commissural defects
- Calcification

MITRAL VALVE PROLAPSE

The normal mitral valve plane is shaped like a saddle. Do not base your diagnosis solely on the four-chamber view since the non-planer shape of the MV mimics a prolapse in this view.

Forms of Mitral Valve Prolapse

- Barlow's syndrome (classic mitral valve prolapse, myxomatous)
- Fibroelastic deficiency
- Pseudoprolapse (small ventricles, MV enlargement)
- Connective tissue disease (e.g. Marfan, Ehlers-Danlos)

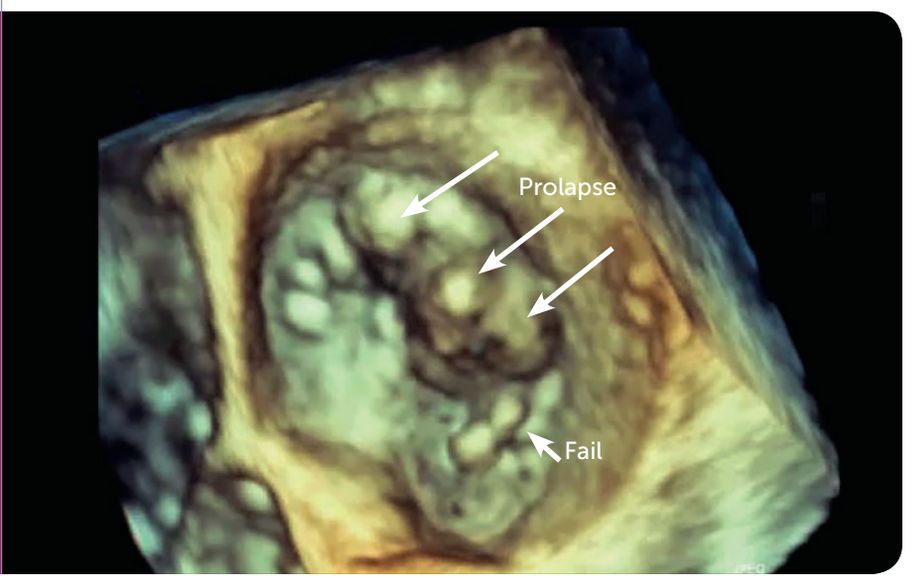
Barlow's syndrome is a structural disease of the mitral valve. It has many features. Do not base your diagnosis on the presence of a prolapsing valve alone.

Myxomatous Mitral Valve (Floppy Valve, Barlow's Syndrome)

- Prevalence = 2 – 3%
- Rapid multiplication of cells
- Rocking motion of the annulus
- Involvement of the entire subvalvular apparatus
- Billowing
- Excessive tissue
- Segmental involvement
- Elongated chords

MITRAL VALVE PROLAPSE – TEE 3D surgical view

A myxomatous mitral valve with a prolapse of the posterior leaflet (P3/P2). Chordal rupture is also present. 3D may be helpfu l in localizing a prolapse or defect.



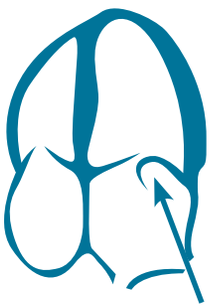
FLAIL LEAFLET

Etiology of the Flail Leaflet

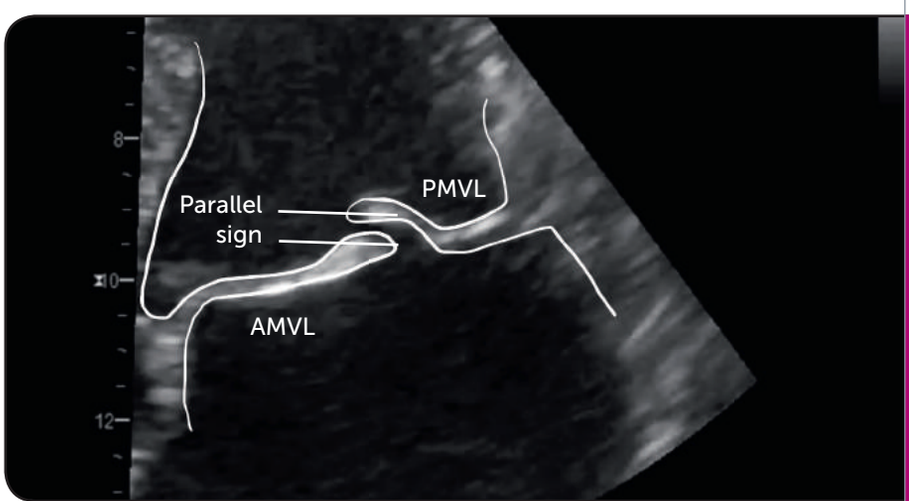
- Myxomatous mitral valve
- Endocarditis
- Degenerative
- Rheumatic

Echo Criteria – Flail Leaflet

- Chordal structures in the LA
- Concave position of leaflet
- Double contour (parallel sign)



concave



NOTES

Ruptured chordae may be found in more than 50% of myxomatous valves.

A flail leaflet can be very subtle, especially when secondary chords are involved.

The degree of mitral regurgitation depends on the location and type of chord that is ruptured. A flail leaflet does not always imply severe MR.

PARALLEL SIGN – zoomed apical four-chamber view/2D

The ruptured leaflet always extends behind the non-ruptured leaflet to which it frequently lies parallel (as seen in the example with a ruptured AMVL). This sign may be helpful in cases of subtle chordal rupture.

OTHER CAUSES OF MITRAL REGURGITATION

Degenerative/Aging	Rheumatic	Endocarditis
Common	Doming of AMVL	Valve destruction
Thickened, fibrotic MV	Other features of rheumatic heart disease are present	Perforation
Annular calcification	Combined MS + MR	Leaflet rupture
Papillary muscle fibrosis	Often leaflet restriction and thickened chords	Leaflet shrinkage/ calcification
Usually mild to moderate mitral regurgitation	Calcification of the subvalvular apparat	

NOTES

Cleft mitral valve is almost always present in primum septal defects (ASD I).

OTHER CAUSES OF MITRAL REGURGITATION

Congenital Abnormalities of the Mitral Valve

- Chordal abnormalities
- Papillary muscle abnormalities
- Cleft MV, parachute MV
- Abnormal leaflet shape/length

INDICATIONS

Repair is better than replacement. Chordae should be preserved whenever possible.

Indications for Mitral Valve Surgery (ESC Class I)

- Surgery is indicated in symptomatic patients with LVEF > 30% and LVESD <55 mm
- Surgery is indicated in asymptomatic patients with left ventricular dysfuntion (left ventricular end systolic diameter [LVESD]≥ 45 mm and/or left ventricular ejection fraction ≤ 60%)
- Mitral valve repair should be the preferred technique when it is intended to last for a long time

LVF < 30%: no surgery (conservative, HTX or MitraClip procedure)

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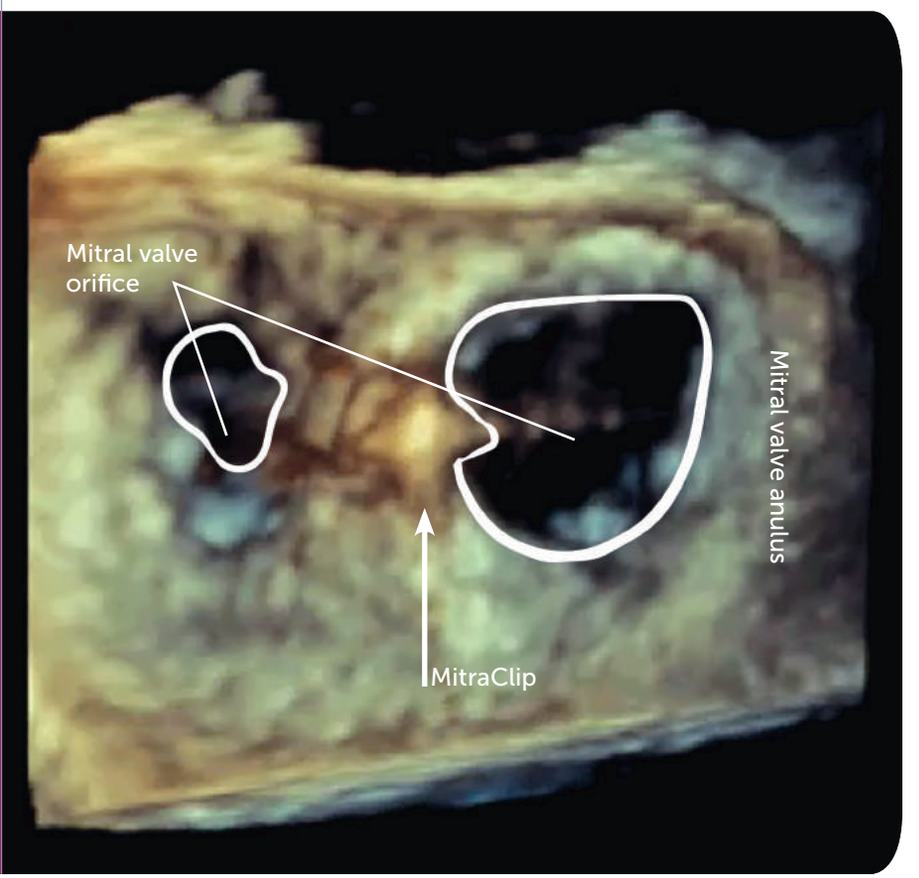
MitraClip Procedure

MITRACLIP – TEE 3D surgical view

3D echo is used to monitor the MitraClip procedure. A central clip was placed, resulting in two incongruent mitral valve orifices.



INDICATIONS

Suitability for the MitraClip procedure (german society of cardiology)

OPTIMAL	POSSIBLE
• Central pathology (segment 2),	• Pathology in segment 1 or 3,
• No calcification	• Calcification (mild) outside the clip zone,
	• Post annulopasty/ring
• MVA > 4 cm^2	• MVA > 3cm^2, good mobility of leaflets,
• Mobile length of post leaflet > 10 mm ,	• Mobile length of the posterior leaflet 7-10 mm
• Coaptation depth <11 mm,	• Coaptation defect > 11 mm
• Normal leaflet thickness + mobility,	• Leaflet constriction during systole, flail leaflet >15 mm (only with large MV annulus and multiple clips)
• Flail leaflet width <15 mm, gap <10 mm	

Unsuitable valve morphology for MitraClip:

- Perforated mitral leaflet/ cleft mitral valve
- Severe calcification in the clip zone
- Significant mV stenosis (mean gradient ≥ 5 mmHg)
- Mobile length of the posterior leaflet < 7 mm
- Rheumatic thickening of the leaflets and restriction in systole and diastole,
- Barlow's syndrome with extensive involvement

Echocardiographic Approach in Asymptomatic Patients

- Monitor left ventricular function and size.
- Check for pulmonary hypertension.
- Atrial size correlates with the risk of atrial fibrillation.
- Consider stress tests.
- Early surgery when repair is likely.

NOTES

The MitraClip procedure is an interventional therapy by which a clip is used to attach the anterior leaflet to the posterior one. It is similar to the surgical procedure know as the "Alfieri" stitch. Studies have shown that this technique is able to reduce mitral regurgitation and improve symptoms in both functional and structural MR.

The indication and suitability for the MitraClip procedure are still evolving. They depend on operator/center experience and the improvments of the technique.

The prognosis depends on preoperative LVF.

NOTES

013 // Tricuspid Valve Disease

CONTENTS

NOTES

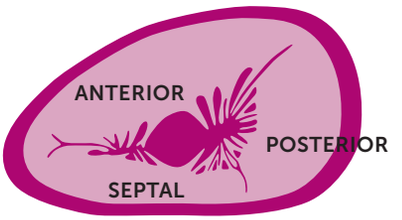
The posterior leaflet is usually rather small!

The location and size of the papillary muscles is highly variable.

BASICS

Morphology

- Three leaflets
- Larger than mitral valve (3.2 – 6.4 cm^2)
- More apical and thinner leaflets than mitral valve

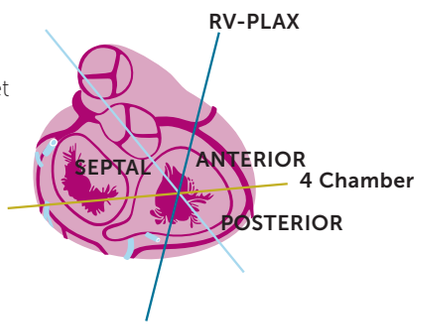


The tricuspid valve is more difficult to image than the mitral valve. Use a more cranial four-chamber view (1 intercostal space higher).

How to Image the Tricuspid Valve

RV PLAX	ant. + post. leaflet
RV inflow-outflow view	ant./sept. +post leaflet
RV optimized 4-chamber view	sept. + ant. leaflet

RV inflow E/A wave lower than MV inflow, velocity varies with respiration



CAUSES OF TRICUSPID REGURGITATION

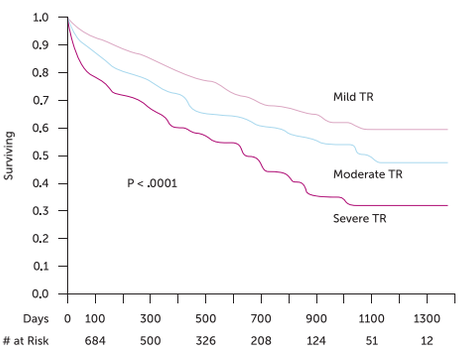
Trivial (physiologic) TR is common! (70% of adults).

TR severity is a good marker of disease progression. This is true for many conditions (cardiomyopathy, valvular heart disease, pulmonary hypertension etc.)

Prognosis of TR

Survival depends on:

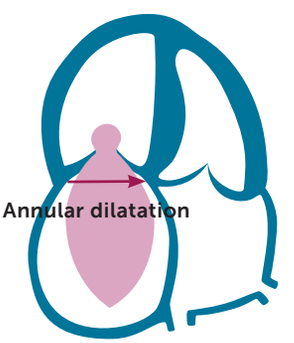
- Severity of tricuspid regurgitation
- Presence and degree of pulmonary hypertension
- Reduced left/right ventricular function



Functional (secondary) tricuspid regurgitation is much more common than structural (primary) TR!

Causes of Functional Tricuspid Regurgitation

- Left heart disease
- Mitral valve disease
- Pulmonary hypertension
- RV dilatation (e.g. atrium septal defect/left-right shunt)



CAUSES OF TRICUSPID REGURGITATION

Causes of Primary Tricuspid Regurgitation

- Rheumatic (TR combined with TS)
- Trauma (blunt trauma, flail/rupture)
- Pacemaker lead associated
- Endocarditis
- Congenital (e.g. dysplasia, Ebstein's anomaly)

Heart Disease and Carcinoid Tricuspid Regurgitation

Release of vasoactive substances (such as serotonin) leads to:

- Endocardial fibrosis
- Tricuspid leaflet restriction
- Wide coaptation defect
- May be associated with pulmonary valve stenosis/regurgitation

Left heart/valve involvement may be found in the presence of ASD or PFO.

Morbus Ebstein

- Variable morphology
- Large anterior leaflet
- Leaflet tethering
- Apical displacement (atrialized RV)

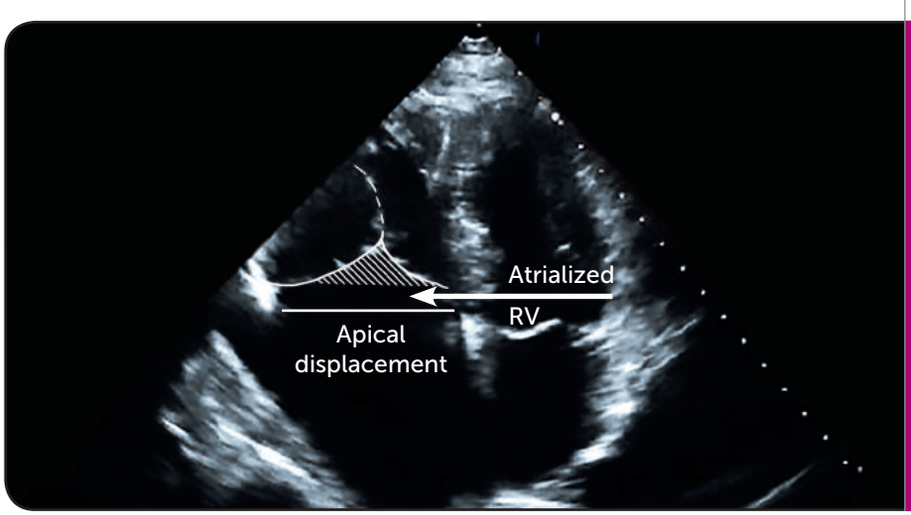
Associated with

- Atrium septal defect (> 1/3 of patients)
- Ventricular septal defect
- Patent ductus arteriosus
- Aortic coarctation
- RVOT obstruction,
- Arrhythmia (e.g. WPW syndrome)

The origin of the tricuspid regurgitation jet is far in the right ventricle, caused by apical displacement of the tricuspid valve.

Consider a rudimentary form of Ebstein's anomaly or tricuspid valve dysplasia. Look for apical displacement of the valve in the setting of unexplained tricuspid regurgitation.

Tricuspid dysplasia is common in dogs (Labrador retrievers).



EBSTEIN'S ANOMALY –
apical four-chamber view/2D

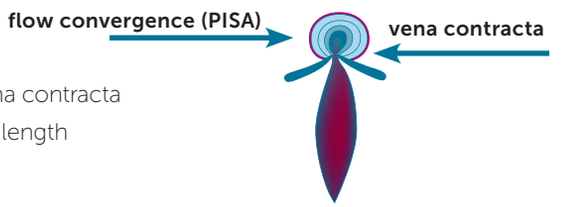
Ebstein's anomaly is characterized by elongated leaflets and displacement of the tricuspid valve. This leads to partial atrialization of the right ventricle.

NOTES

NOTES

QUANTIFICATION OF TRICUSPID REGURGITATION

Quantification

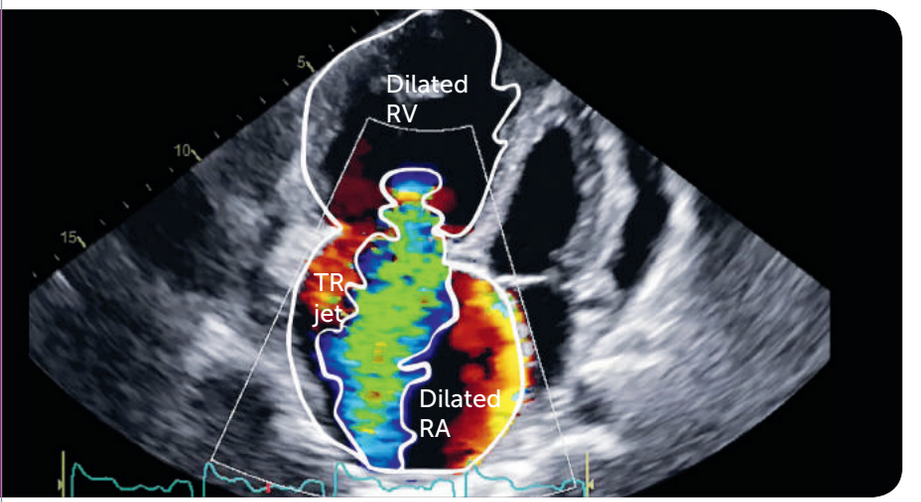


- Flow convergence
- Jet area
- Eye-balling
- Vena contracta
- Jet length

The degree of tricuspid regurgitation may increase with inspiration. Therefore, observe several beats with echo.

SEVERE TRICUSPID REGURGITATION – apical four-chamber view RV optimized/color Doppler

Tricuspid regurgitation with a large flow convergence zone and a wide vena contracta. The right ventricle and atrium are severely dilated (volume overload).



One overestimates right ventricular function in the presence of tricuspid regurgitation (reduced afterload).

Tricuspid Regurgitation – Reference Values

	Mild	Moderate	Severe
PISA radius (mm) Nyquist limit 28 cm/s	5 mm	6 – 9 mm	> 9 mm
Vena contracta Nyquist limit 50 – 60 cm/s		<7 mm	>7 mm

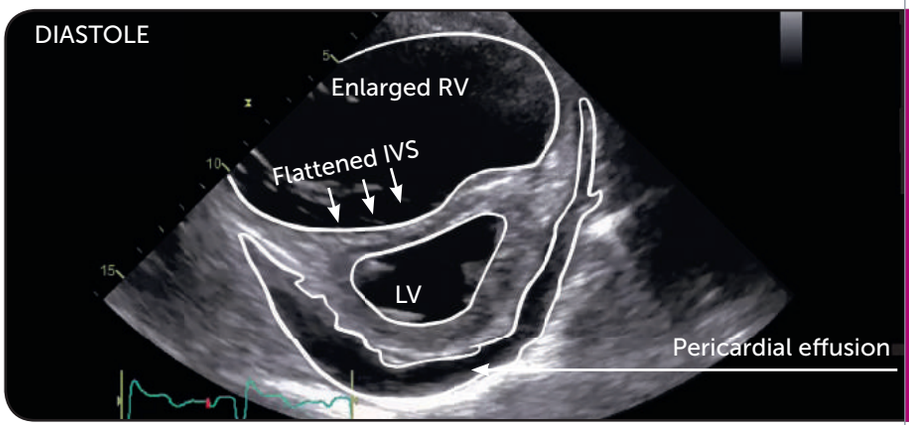
ESC 2013

Right ventricular function is hyperdynamic in the initial phase, but may deteriorate in later stages.

Echo Findings in Severe Tricuspid Regurgitation

- Dilated right ventricle/atrium
- Dilated inferior vena cava without respiratory variations
- Systolic flow reversal in hepatic veins
- Flattened interventricular septum in diastole
- Visible coaptation defect

QUANTIFICATION OF TRICUSPID REGURGITATION



NOTES

FEATURES OF SEVERE TR – PSAX/2D

D-shaped ventricle with a flattened interventricular septum, both in systole and diastole – in severe TR and pulmonary hypertension.

Indications for Tricuspid Valve Surgery (ESC Class I)

- In patients with severe primary or secondary TR undergoing left-sided valve surgery
- In symptomatic patients with severe isolated primary TR without severe right ventricular dysfunction

ESC 2012

When patients with severe TR develop signs of right heart failure (pleural effusion, peripheral edema, ascites), it may be too late for surgery (irreversible RV dysfunction).

Adding tricuspid repair, if indicated, during left-sided surgery does not increase the risk of surgery.

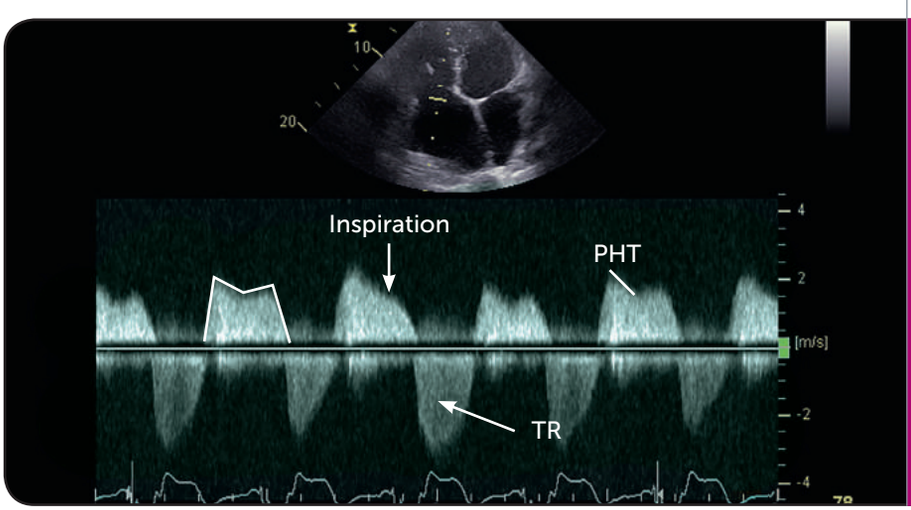
TRICUSPID STENOSIS

Overview

- In 9 % of rheumatic heart disease
- Congenital tricuspid stenosis (very rare)
- Functional tricuspid stenosis due to intracardiac (obstruction) or extracardiac (compression) masses
- Endocarditis (very rare)
- After repair/replacement.

Look for doming of the tricuspid valve in 2D and turbulent flow on color Doppler.

Tricuspid stenosis may also occur after tricuspid valve repair (undersizing of the annuloplasty ring).



TRICUSPID VALVE STENOSIS – apical four-chamber view/CW Doppler

Elevated flow velocity across the tricuspid valve with a mean gradient >5 mmHg. Fluctuations in inflow velocity, which increase during inspiration.

NOTES

Symptoms of tricuspid valve may mimic those of right heart failure.

You will find a significant increase in gradients during inspiration. Therefore, average several beats.

Look for turbulent flow on color Doppler across the tricuspid valve in all patients with rheumatic mitral stenosis. Doming of the tricuspid valve may be difficult to visualize. Thus, you will not miss associated tricuspid stenosis.

TRICUSPID STENOSIS

Hemodynamics

- Diastolic RA-RV gradient
- Dilatation and elevated pressure in the right atrium
- Dilated inferior vena cava

Quantification of Tricuspid Stenosis

- **Pressure half time:** Tricuspid valve area (TVA)= 190/PHT – A TVA < 1 cm2 indicates severe TS (not validated).
- **Mean gradient:** Mean gradient > 5 mmHg indicates significant tricuspid regurgitation.

014 // Prosthetic Valves

CONTENTS

NOTES

Consider mechanical valves in younger patients.

The risk of mechanical failure of a prosthesis is very low.

Newer models include Open Pivot (Medtronic) and the OnX mechanical valve (OnX).

Biological valves for the elderly (but not exclusively).

Biological valves also include prosthetic material (struts, sewing ring). These can be seen on the echo.

TYPE OF VALVES

Mechanical Valves

- Metal case/occluders
- Types: ball cage, tilting disc, bileaflet
- Anticoagulation necessary
- High durability
- Composite graft (prosthesis + aortic tube graft – Bentall procedure)

Types of Mechanical Valves – Few Examples

	Manufacturer	Model	Year
Ball	Baxter	Starr-Edwards	1965
Disk	Medtronic Medical Alliance	Medtronic Hall Omniscience Monostrut	1977 1978 1982
Bileaflet	St. Jude Baxter Edwards Carbomedics Sorin Biomedica	St. Jude Duromedics Carbomedics Sorin Bicarbon	1977 1982 1986 1990

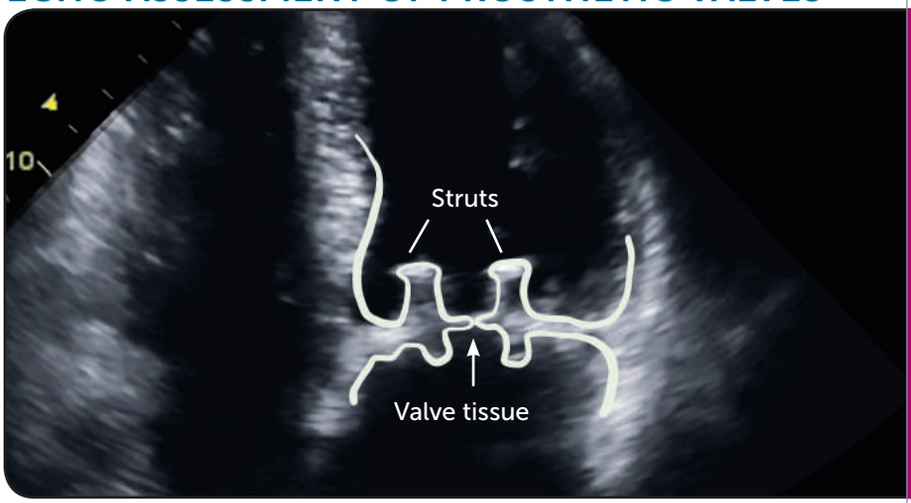
Biological Valves

- Ring (struts)/stentless valves
- No anticoagulation
- Less durable than mechanical valves
- Homograft (cadaver)
- Autograft (pulmonic valve) – Ross operation
- New implantation systems for rapid deployment (e.g. Edwards Intuity)

Types of Biological Valves (examples)

Manufacturer	Model
Carpentier- Edwards	Perimount
Carpentier- Edwards	Magna
Medtronic	Hancock
Medtronic	Mosaic
Sorin Group	Mitroflow

ECHO ASSESSMENT OF PROSTHETIC VALVES



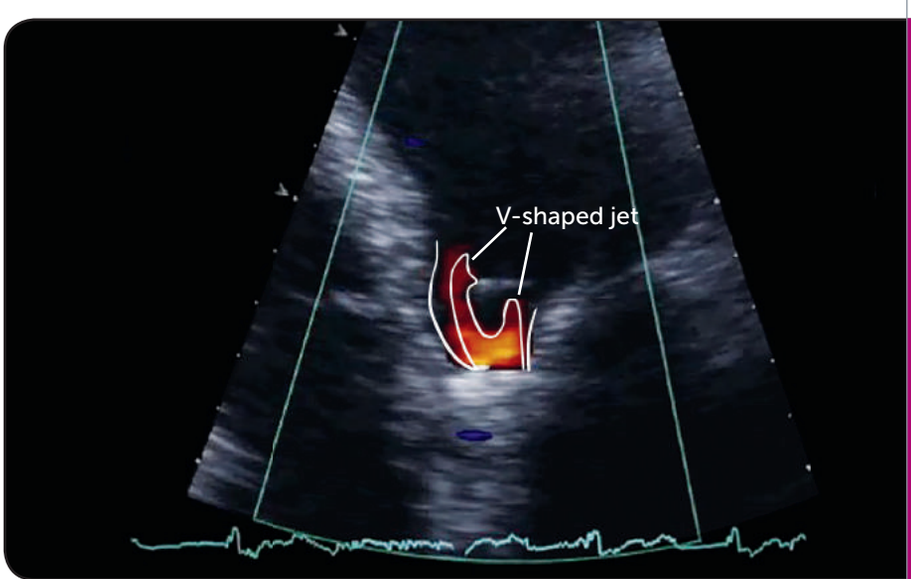
Assessment of Valve Prosthesis

2D Assessment

- Occluder/cusp motion,
- Rocking motion of the prosthesis
- Cusp thickening/calcification (biological valve)
- Annulus (cavities, pseudoaneurysms, thrombi/vegetation)

Doppler Assessment

- Maximum and mean gradients across the valve using CW Doppler
- Valvular and paravalvular regurgitation using Color Doppler



NOTES

BIOLOGICAL MITRAL VALVE – apical four-chamber view/2D

The struts (2 of 3 visible) protrude into the left ventricle. The tissue component of the valve cusps are seen between the struts.

Do not forget to look at the ventricle and systolic pulmonary artery pressure in mitral valve prosthesis.

Obtain an early postoperative baseline study for comparison later on.

FLOW PATTERN IN MECHANICAL VALVE PROSTHESIS – zoomed apical five-chamber view

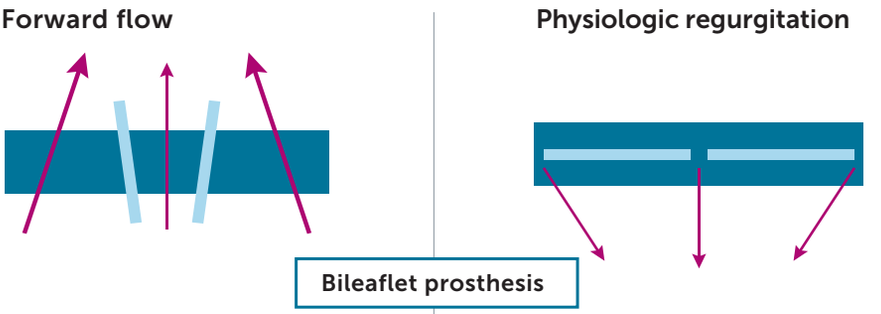
Typical flow pattern of a mecha nical bileaflet aortic prosthesis. The regurtitant jets originate within the frame of the prosthesis (central) and the jet direction is "V-shaped".

NOTES

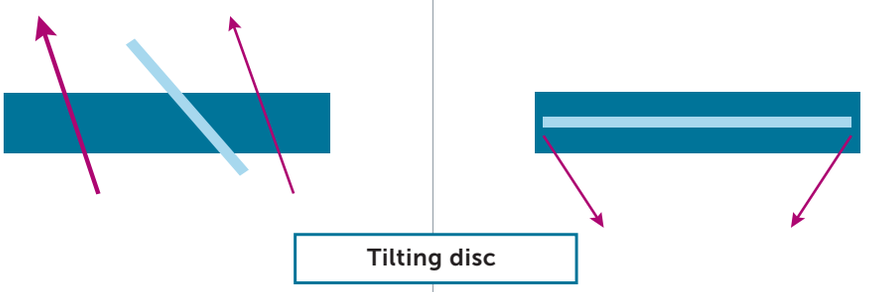
ECHO ASSESSMENT OF PROSTHETIC VALVES

Flow Patterns in Mechanical Valve Prosthesis

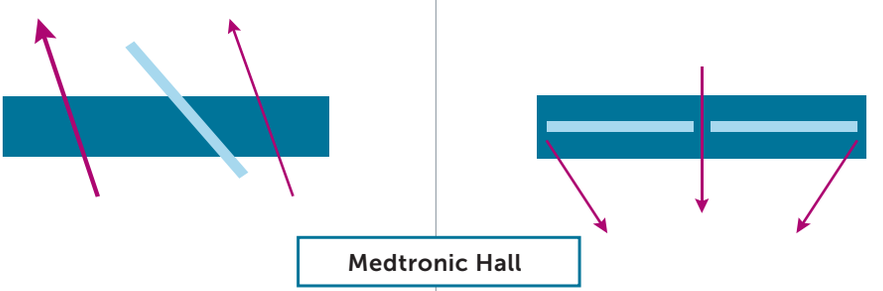
Search for a view that displays the opening/closing motion of the occluders (mitral valve prosthesis).



The inflow and regurgitation pattern varies, depending on the type of prosthesis.



The motion of mechanical valves in the aortic position is difficult to assess.



Common Findings

- Residues of the subvalvular apparatus
- Cavitations
- Abnormal septal motion
- Suture material + normal regurgitations

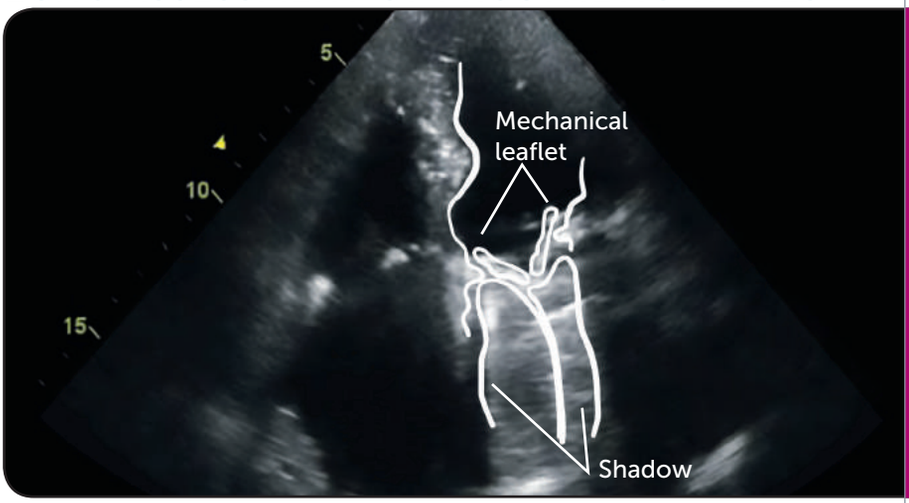
Use atypical views.

TEE allows visualization of the atrial side of the prosthesis. TTE shows the ventricular side. Combine TTE and TEE if you are in doubt.

Imaging Problems in Patients With Mechanical Valves

- Artefacts
- Shadowing
- Limited visibility of LA
- Limited visibility of the left atrium in patients with mitral valve prosthesis
- Limited visibility of the regurgitant jet
- Endocarditis is difficult to diagnose
- Visualization of a thrombus is difficult
- Difficult to see leaflet motion
- Difficult to assess flow convergence

ECHO ASSESSMENT OF PROSTHETIC VALVES



NOTES

MECHANICAL MITRAL VALVE – apical four-chamber view/2D

The two mechanical leaflets are almost parallel during diastole. The prosthesis causes shadowing of the left atrium.

Reference Values for Prosthetic Aortic Valves

Bioprosthesis	**Vmax (m/s)**	**Max. gradient (mmHg)**	**Mean gradient (mmHg)**
Carpentier Edwards	2.37 ± 0.46	23.18 ± 8.72	14.4 ± 5.7
Hancock	2.38 ± 0.35	23.0 ± 6.71	11.0 ± 2.29
Mitroflow	2.0 ± 0.71	17.0 ± 11.31	10.8 ± 6.51
Stentless biopros-thesis (25 mm)	**Vmax (m/s)**	**Max. gradient (mmHg)**	**Mean gradient (mmHg)**
Biocor Stentless	2.8 ± 0.5	28.65 ± 6.6	17.72 ± 6.35
Medtronic Freestyle	–	–	5.35 ± 1.5
Toronto Porcine	1.74 ± 1.19	38.6 ± 11.7	24 ± 4
Mechanical prosthesis	**Vmax (m/s)**	**Max. gradient (mmHg)**	**Mean gradient (mmHg)**
St. Jude Medical	2.37 ± 0.27	25.5 ± 5.12	12.5 ± 6.35
Björk-Shiley	2.62 ± 0.42	23.8 ± 8.8	14.3 ± 5.25
Starr-Edwards	3.1 ± 0.47	38.6 ± 11.7	24.0 ± 4.0

Consider prosthetic aortic valve dysfunction when the maximal velocity is > 3 m/s and the mean gradient > 20 mmHg.

NOTES

ECHO ASSESSMENT OF PROSTHETIC VALVES

Consider prosthetic mitral valve dysfunction if the maximal velocity is ≥ 2 m/s and the mean gradient is ≥ 8 mmHg.

Reference Values for Prosthetic Mitral Valves

Bioprosthesis	Vmax (m/s)	Max. gradient (mmHg)	Mean gradient (mmHg)	PHT (ms)
Hancock	1.54 ± 0.26	9.7 ± 3.2	4.29 ± 2.14	128.6 ± 30.9
Carpentier-Edwards	1.76 ± 0.24	12.49 ± 3.64	6.48 ± 2.12	89.8 ± 25.4
Ionescu-Shiley	1.46 ± 0.27	8.53 ± 2.91	3.28 ± 1.19	93.3 ± 25.0
Mechanical prosthesis	**Vmax (m/s)**	**Grad.max (mmHg)**	**Grad. mean (mmHg)**	**PHT (ms)**
St. Jude Medical	1.56 ± 0.29	9.98 ± 3.62	3.49 ± 1.34	76.5 ± 17.1
Björk-Shiley	1.61 ± 0.3	10.72 ± 2.74	2.9 ± 1.61	90.2 ± 22.4
Starr-Edwards	1.88 ± 0.4	14.56 ± 5.5	4.55 ± 2.4	109.5 ± 26.6

Nobody understands pressure recovery anyway! Just remember these key issues.

Pressure Recovery

- Leads to overestimation of gradients by Doppler
- Relevant in a small aortic root (< 30 mm)
- Common in small bileaflet valves
- Especially when high flow present

Prosthesis–patient mismatch leads to high transvalvular gradients through normal functioning valves. This influences the resolution of left ventricular hypertrophy and may also influence prognosis and exercise capacity.

Prosthesis Patient Mismatch (Aortic Valve)

- A calcified aortic annulus can make it difficult to implant adequately large valves
- Associated with increased late mortality
- Think of mismatch in the setting of left ventricular dysfunction

The geometric orifice area is not the effective orifice area.

Prosthetic Effective Orifice Area (EOA) in Aortic Valve Prosthesis

$$EOA = \frac{\text{Stroke volume}}{\text{VTI}}$$

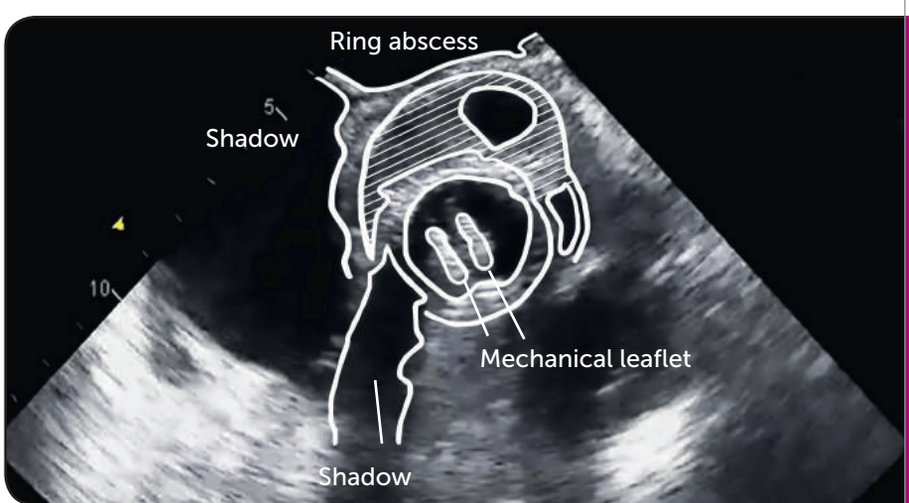
VTI of AV velocity Stroke volume LVOT

Consider prosthesis-patient mismatch when the indexed prosthetic effective orfice area < 0.85 cm2/m2

COMPLICATIONS

Prosthetic Valve Complications

- Paravalvular leaks
- Valve obstruction (thrombus/pannus)
- Endocarditis
- Mechanical failure (mechanical valves)
- Degenerative changes (biological valves)
- Pseudoaneurysm/fistula



PROSTHETIC VALVE ENDOCARDITIS – TEE short-axis view/2D

Staphylococcal infection of the valve, resulting in paravalvular abscess. Infectious material and echo-free cavities suround the prosthesis. Always look for partial dehiscence and paravalvular regurgitation.

Predisposing Factors for Structural Failure in Bioprosthesis

- Renal failure
- Hemodialysis
- Hypercalcemia
- Adolescence (growing)
- Porcine > pericardia
- Autoimmune disease

Bioprosthesis Obstruction – Echo Findings

- Thickened calcified leaflets
- Reduced mobility
- Elevated gradients
- Prolonged pressure half-time (mitral prosthesis)
- Turbulent flow
- Dilated left atrium with spontaneous contrast (mitral prosthesis)
- LV dysfunction (eventually)

NOTES

Left ventricular dysfunction may occur after valve surgey due to intraoperative ischemia, residual valvular defects, or ventricular dysfunction at the time of surgery (too late). It may occur several years after surgery.

Look for pseudoaneurysms of the intervalvular fibrosa, especially in patients with suspected endocarditis or in patients who have received a prosthetic valve because of endocarditis.

Compare with previous studies and initial postoperative gradients.

Structural failure (obstruction) is unlikely when the prosthesis is < 2 years old and the patient does not have endocarditis.

NOTES

Use fluoroscopy to detect mechanical valve obstruction.

Quite often only the surgeon can give the answer if a thrombus or a pannus is present

THROMBUS OF MITRAL PROSTHESIS – TEE/2D

Mechanical obstruction of a bileaflet prosthesis caused by thrombus. Thrombi are difficult to see with transthoracic echo. They are usually located at the atrial side of the prosthesis, which is shadowed in the transthoracic exam.

Use color Doppler to guide the position of the CW Doppler (mitral valve). Use several windows to quantify prosthetic aortic valve obstruction.

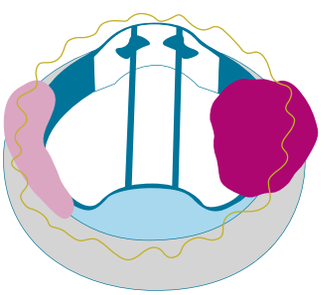
COMPLICATIONS

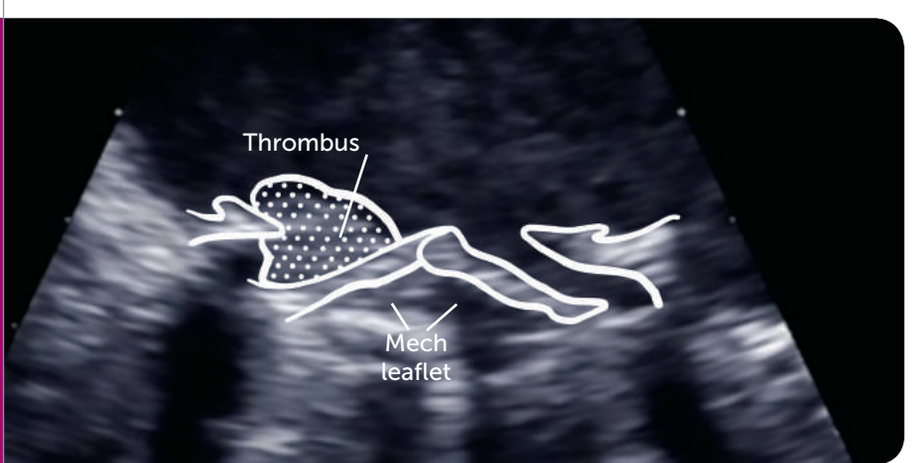
Mechanical Valve Obstruction – Echo Findings

- Impaired/stuck leaflet
- Echogenicity in valve region (thrombus?)
- Pathologic flow pattern on color Doppler
- Elevated gradients
- Pressure half time (MV)

Mechanical Valve Obstruction – Pannus vs. Thrombus

Pannus	Thrombus
INR in the therapeutic range	INR too low
Slow onset of symptoms	Sudden symptom onset
Higher age of prosthesis	Stroke/embolism
Stable gradients	Variable gradients





Quantification of Obstruction

Aortic Valve Prosthesis

Morphologic findings
Symptoms
Velocity > 3.0 m/sec
Doppler Vel. Index < 0.3
(Doppler Velocity Index = $V_{LVOT}/V_{Prosth\ valve}$)

Mitral Valve Prosthesis

Morphologic findings
Symptoms
Mean gradients (>6–8 mmHg)
PHT > 130 ms

COMPLICATIONS

Regurgitation in Valve Prosthesis

- Normal/physiologic
- Pathologic (paravalvular)
- Valvular/structural failure (bio)
- Valvular/mechanical failure (mech)

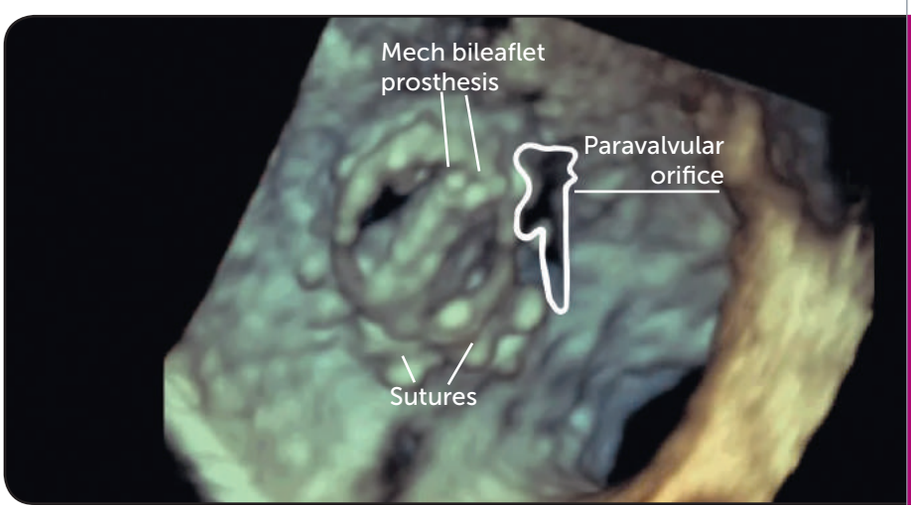
Mitral Regurgitation and Type of Prosthesis

Type	Valvular	Paravalvular	Normal/physiologic
Mechanical	X (mech. failure)	X	X
Biological	X	X	----
Composite	X (mech. failure)	----	X
Homograft	X	----	X

Table showing possible forms of regurgitation in the individual types of prostheses.

Paravalvular Regurgitation

- Prevalence: 6–32% early, 7–10% late
- More common in aortic than in mitral valve prosthesis
- Predisposing factors: calcified annulus, endocarditis, suture technique
- Small atria



PARAVAVULAR LEAK – TEE/3D surgical view

Paravavular leak in a patient with a bileaflet mechanical mitral valve.

Echo Evaluation of Regurgitation

- Multiple/atypical views
- Eccentric jets
- Parasternal short axis (aortic valve)
- CW Doppler + gradients

NOTES

Some degree of paravalvular regurgitation is always present.

Patients with relevant paravalvular regurgitation often have hemolysis.

Paravalvular regurgitation of the aortic valve is best seen on the parasternal short-axis view (color Doppler).

NOTES

COMPLICATIONS

Elevated Gradients – Considerations

In the setting of elevated gradients in mitral valve prosthesis, measure the pressure half-time. If the pressure half-time is high, prosthesis obstruction is likely. If the pressure half-time is normal, consider significant mitral regurgitation or high flow states.

- Compare with baseline/ reference values
- Likelihood of obstruction (anticoagulation within the therapeutic range/ symptoms)
- Presence of regurgitation (increase gradients per se or as a secondary sign of prosthetic dysfunction)
- Prosthesis mismatch?
- Presence of mobile structures (thrombi/vegetations)
- High flow state (dialysis shunt, high cardiac output, heart rate)

Other Complications

Tricuspid regurgitation tends to increase after left heart valve surgery.

Valve dehiscence	Look for rocking valve motion
Iatrogenic ventricular septal defect	Rare complication
Tricuspid regurgitation following mitral valve surgery	Pulmonary hypertension, tricuspid annular dilatation, atrial fibrillation, prior degree of tricuspid regurgitation

Pseudoaneurysm

If you suspect an aortic valve pseudoaneurysm, look for a pulsatile cavity with oscillating flow in (systole) and out (diastole) of the cavity.

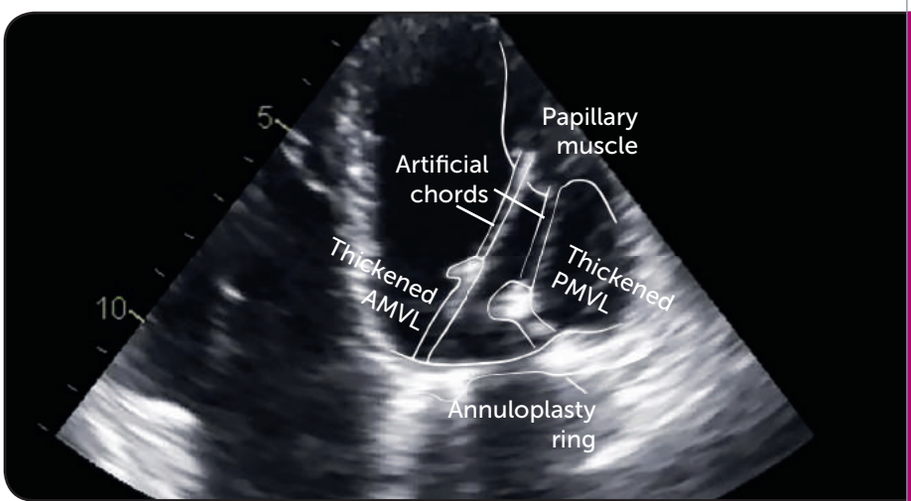
- Often caused by endocarditis (before and after surgery).
- Occurs in native and prosthetic valves.
- May lead to the formation of fistulas.

Prosthetic Valve Endocarditis (see Chapter 15)

MITRAL VALVE REPAIR

Mitral Valve Repair – Ring Implantation (Annuloplasty)

- Different types of rings (flexible, open, closed)
- Prevents annular dilatation
- May resemble annular calcification on echo
- The posterior leaflet may appear rather short after ring implantation



MITRAL VALVE REPAIR – apical four-chamber view/2D

Artifical chords and annuloplasty ring after mitral valve repair.

Common Techniques of Mitral Valve Repair

- Annuloplasty (see above)
- Quadrangular/triangular resection (with/without sliding plasty)
- Chordal transfer
- Artificial chords

Complications of Mitral Valve Repair

- Residual regurgitation
- Obstructed left ventricular inflow (undersizing of the ring)
- Ring dehiscence (partial dehiscence, the origin and path of regurgitation are outside the ring)
- LVOT obstruction/SAM caused by redundant leaflets in the setting of small hyperdynamic left ventricles

NOTES

Mitral valve repair is always combined with ring implantation.

Measure the mean gradient and the pressure half-time across the mitral valve in patients after mitral valve repair. Undersizing of the ring may lead to mitral valve stenosis.

Patients with unsuccessful repair (if not corrected) have a poor prognosis.

NOTES

015 // Endocarditis

CONTENTS

NOTES

The prevalence of endocarditis associated with prothetic valves and pacemaker leads is on the increase.

PRINCIPLES OF ENDOCARDITIS

Definition

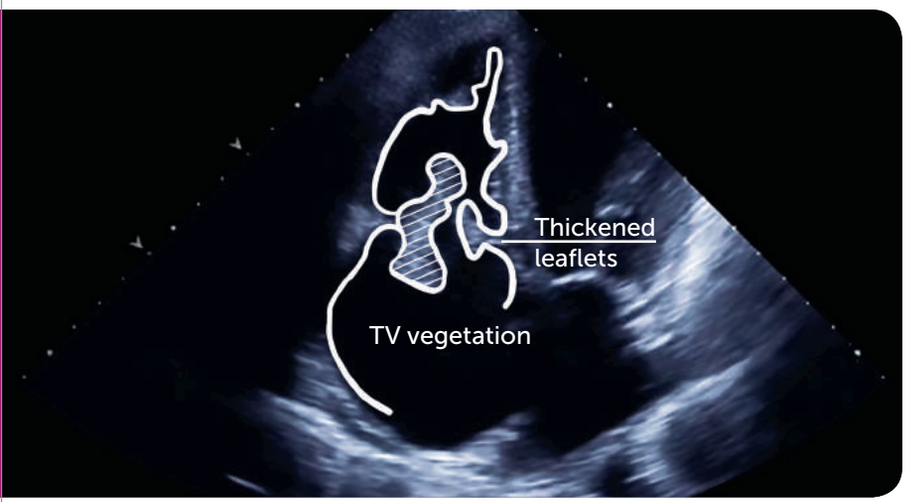
Endovascular microbial infection of cardiovascular structures

Location

- Valves
- Large intrathoracic vessels
- Ventricular and atrial endocardium
- Prosthetic material
- Polymere associated structures (lines)
- Eustachian valve

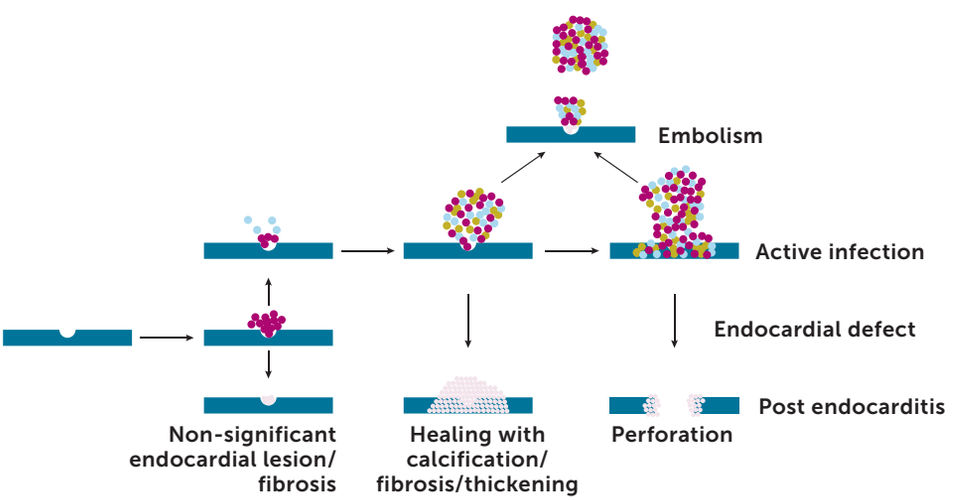
TRICUSPID VALVE ENDOCARDITIS – apical four-chamber view RV optimized/2D

Endocarditis with a large vegetation attached to the native tricuspid valve.



Vegetation is an infected mass attached to endocardial structures, such as valves or implanted intracardiac material. On 2D echo they frequently appear as oscillating structures of variable size and morphology.

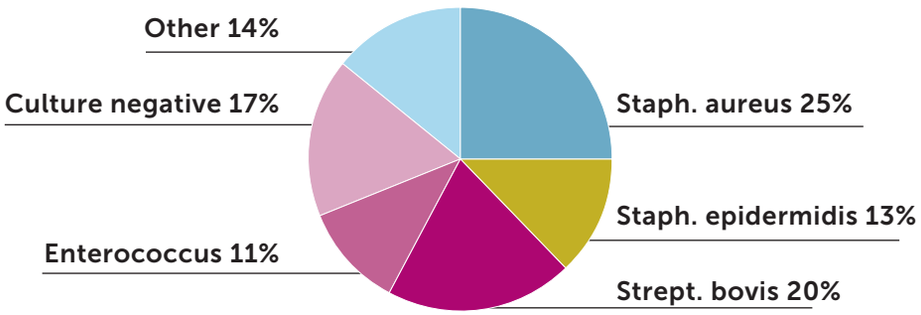
Pathophysiology of Endocarditis

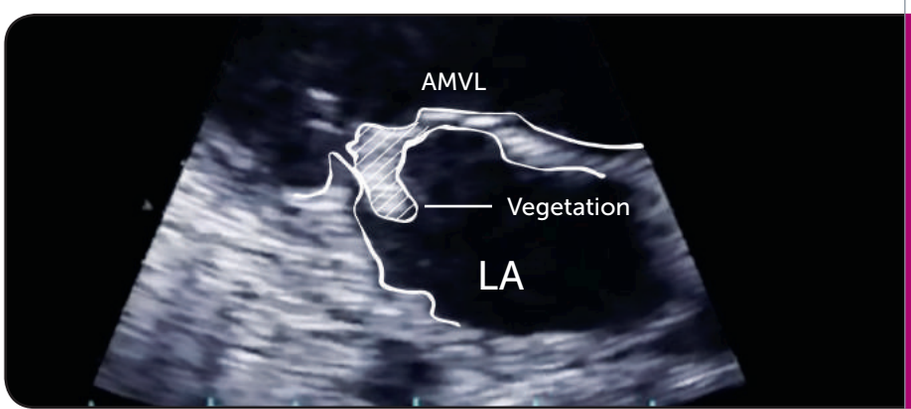


Principle of a "super-infected" thrombus: The endothelial lesion initiates a repair process which involves thrombus formation. In the presence of bacteremia this thrombus may be super-infected. Further consequences include repair ad integrum, tissue destruction, embolism, and defect healing.

PRINCIPLES OF ENDOCARDITIS

Microbiology





MITRAL VALVE ENDOCARDITIS – PLAX zoomed/2D

A vegetation is attached to the tip of the anterior mitral valve leaflet.

Epidemiologic Facts on Endocarditis

- Large geographical variations in the incidence of endocarditis (3–10 episodes/100.,000 person-years)
- Increase in the elderly population
- Sclerosis and aging also predispose to endocarditis

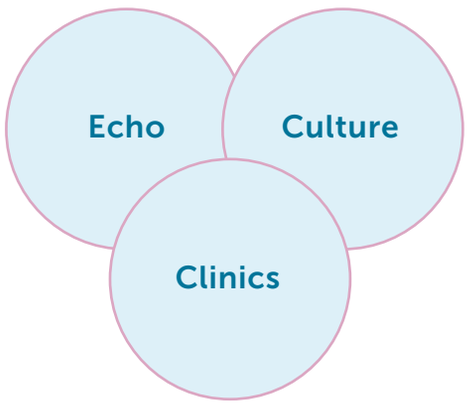
NOTES

Staph. aureus infection predisposes to abscess formation and complications of endocarditis!

NATIVE VALVE ENDOCARDITIS

Diagnosis, Symptoms and Findings

- Fever/night sweat
- Predisposing factors
- Conjunctival petechiae
- Janeway lesions
- Roth spots
- Splinter hemorrhages
- Vegetations
- Regurgitations
- Complications of endocarditis (abscessive destruction)
- Pericardial effusion



Endocarditis may be manifested in many ways, many of which may be atypical
In the setting of infection, heart murmur or atypical symptoms, think of endocarditis. Early diagnosis saves lives.

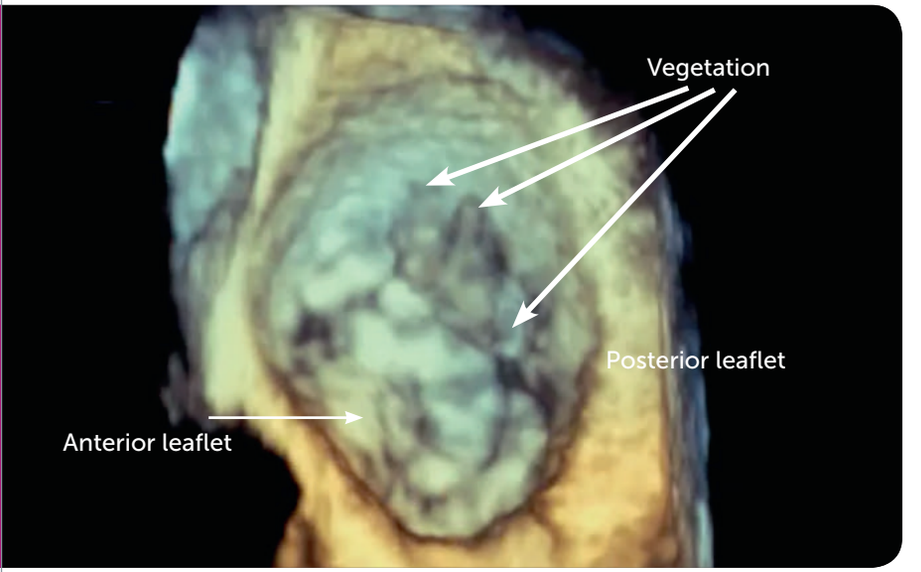
Blood culture and other signs of infection (CRP, leukocytes, etc.) are equally important. A negative blood culture does NOT rule out endocarditis.

NOTES

MITRAL VALVE ENDOCARDITIS – TEE surgical view/3D

Large vegetation on the posterior leaflet prolapsing into the left atrium

NATIVE VALVE ENDOCARDITIS



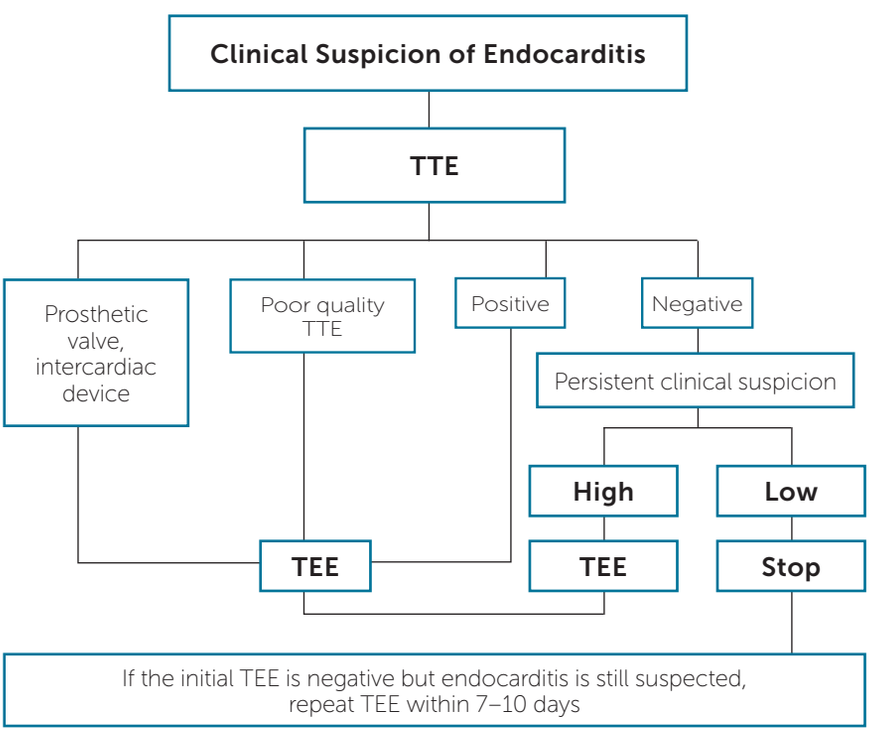
Follow-up studies help to make an accurate diagnosis (progression?).

Differential Diagnosis

- Fibrosis/calcification
- Myxomatous degeneration (e.g. mitral valve prolapse)
- Lambl's excrescence/strands
- Tangential imaging of structures
- Old vegetations
- Tumors/thrombi

Transesophageal echocardiography is not mandatory in isolated right-sided native valve endocarditis with good transthoracic quality.

Indication for Transthoracic Echo in Suspected Endocarditis



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NATIVE VALVE ENDOCARDITIS

What Else to Look For?

- Involvment of other valves
- Regurgitations and resulting volume overload
- Myocardial function (right + left)
- Pericardial/pleural effusion
- Valve obstruction (large vegetations, rare)
- Coronary embolization of vegetation leading to wall motion abnormalites (rare)

NOTES

"Healing" usually leads to some degree of fibrosis or calcification of the affected valve.

COMPLICATIONS OF NATIVE VALVE ENDOCARDITIS

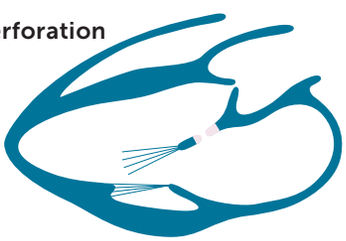
Complications

- Embolism
- Valve destruction
- Regurgitation/heart failure
- Abscess
- Pseudoaneurysm
- Perforation
- Fistula
- Mycotic aneurysm

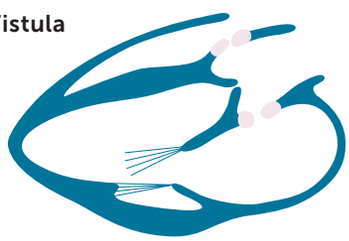
Embolization is the primary manifestation of endocarditis in 28–47% of all patients. The risk of embolization depends on the size (>10 mm) and mobility of the vegetation.
Exclude endocarditis in the setting of stroke and fever.

Types of Valve Destruction

MV perforation

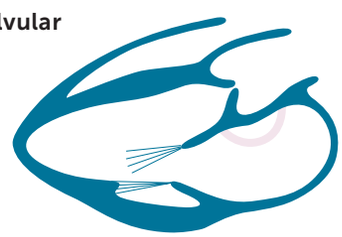


Fistula

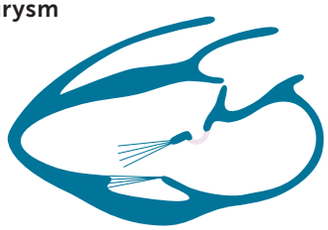


Valve perforation is a hole in the cusp or leaflet which appears as an interruption in endocardial tissue continuity, best seen with color Doppler. In contrast, a fistula is a communication with neighbouring cavities that does not directly involve the valve (for instance, between the aorta and the left atrium).

Pseudoaneurysm – intervalvular fibrosa



MV pseudo-aneurysm



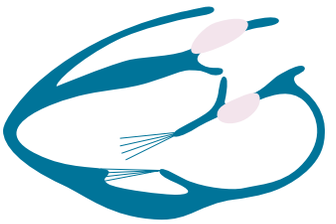
Pulsatile perivalvular (echo-free) cavity communicating with the cardiovascular lumen.

NOTES

COMPLICATIONS OF NATIVE VALVE ENDOCARDITIS

Types of Valve Destruction

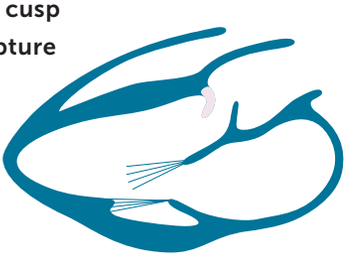
AV ring abscess



MV annular abscess

Perivalvular cavity filled with infectious material which has a non-homogeneous (echodense/echolucent) appearance

AV cusp rupture

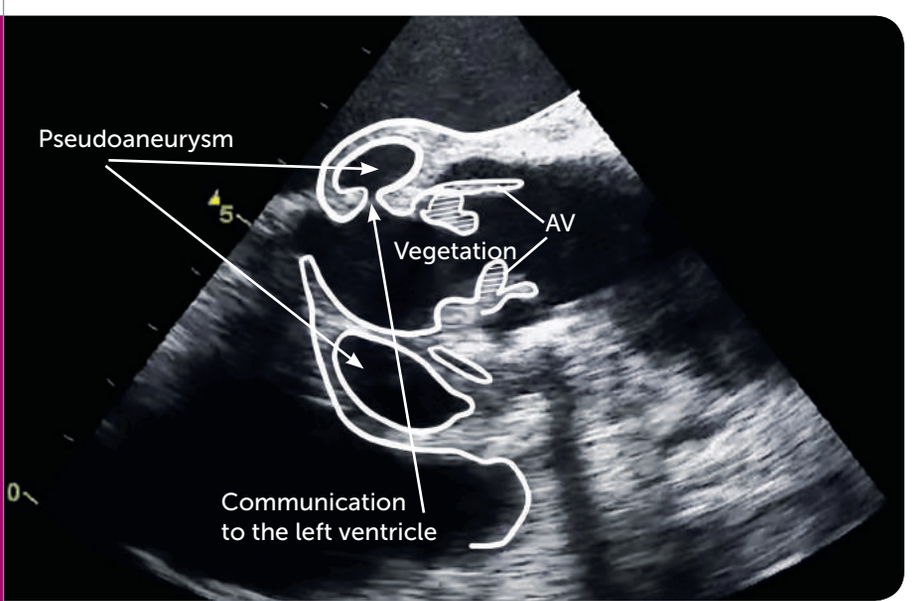


MV flail leaflet

Tear in the aortic cusp or chordal rupture, which usually leads to excentric regurgitation jets.

PSEUDOANEURYSM IN AV ENDOCARDITIS – TEE long-axis view/2D

A pulsating cavity surounds the aortic valve (pseudoaneurysm). Numerous vegetations are present at the aortic cusps.



RIGHT HEART ENDOCARDITIS

Causes of TV Endocarditis

- Intravenous drug abuse
- Immunocompromised
- Indwelling catheters
- Pacemaker

Tricuspid Valve Endocarditis – Facts

- The most common organisms are Staphylococcus aureus (60–80%) and Pseudomonas.
- Pulmonary hypertension, pulmonary embolism or tricuspid regurgitation may result in right heart failure.
- The prognosis is relatively good (10% inhospital mortality), but is poor in fungal infection.
- High recurrence rates.
- Endocarditis frequently causes a flail tricuspid valve leaflet..
- Tricuspid valve endocarditis may also occur in patients without predisposing factors.

Complications

- Valve destruction
- Involvement of neighbouring cardiac structures
- Septic pulmonary embolism
- Lung abscess

PROSTHETIC VALVE ENDOCARDITIS

Risk Factors

- Heart failure
- Wound complications
- Direct contamination during cardiac surgery
- Valve degeneration
- Prior history of endocarditis
- Prosthesis thrombi (super-infection)

Differential Diagnosis

- Artefacts
- Subvalvular residuals
- Surgical materials
- Strands
- Thrombus
- Hematoma

Compare your findings with previous studies.

NOTES

Tricuspid valve endocardits is very likely in patients with pulmonic abscess + drug abuse + new heart murmur.

Use atypical views to image tricuspid valve endocarditis and also look for pleural effusion (secondary to pulmonary infection).

Tricuspid valve vegetations may become very large.

Prosthetic valve endocarditis is difficult to detect.
Transesophageal echo is recommended in case of suspicion.
Find out which operation was performed, talk to the surgeon.
Surgical material such as suture material or patches may mimic endocarditis.

NOTES

PROSTHETIC VALVE ENDOCARDITIS

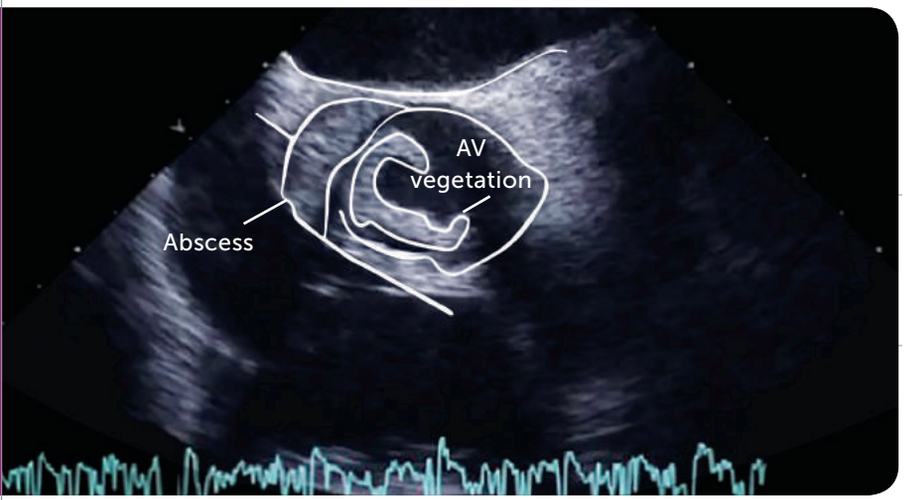
Prosthetic valve endocarditis is a life-threatening condition and is associated with a poor prognosis.

Complications

- Periannular abscess
- Pseudoaneurysms
- Paravalvular leaks
- Valve dehiscence
- Valve obstruction
- Fistula

PERIANNULAR PROSTHETIC VALVE ABSCESS – TEE short-axis/2D

The echodense area surounding the prosthesis corresponds to a periannular abscess. Additionally, a large vegetation is seen on the rim of the cusps.



PACEMAKER/POLYMER-ASSOCIATED ENDOCARDITIS

Pacemaker lead infection is difficult to diagnose. A negative study does not rule out endocarditis. Combine transthoracic and transesophageal echo to visualize as many portions of the leads as possible.

Predisposing Factors

- Pouch/Pocket infection
- Impaired immunity
- Systemic infection
- Temporary pacing before implantation
- Diabetes
- The surgeon's experience
- Advanced age

Clinical Presentation

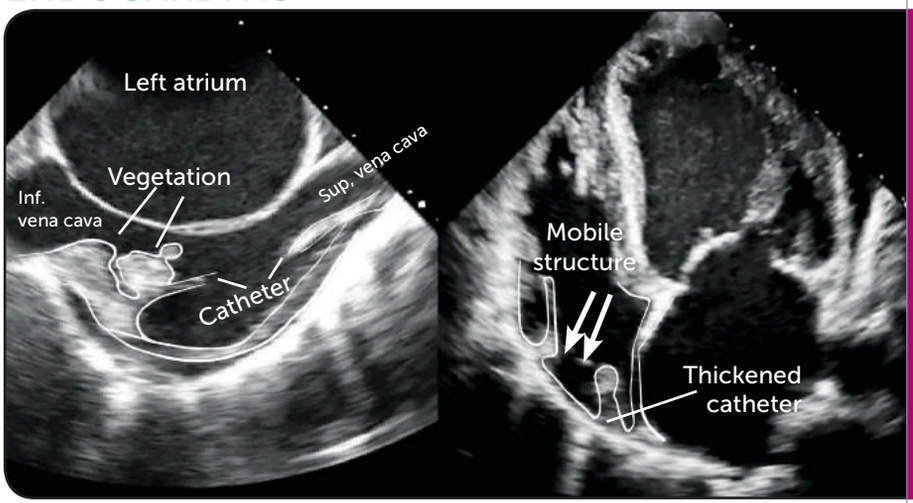
- Fever, subfebrile (recurrent)
- Pulmonary embolism
- Local complications
- Septic shock (acute)
- Poor general condition

Lead infection usually occurs at sites where the leads are in contact with the endothelium.

Typical Sites of Infection

- Vena cava superior
- Right atrium
- Tricuspid valve
- Tricuspid annulus

PACEMAKER/POLYMER-ASSOCIATED ENDOCARDITIS



NOTES

CENTRAL LINE ENDOCARDITIS – apical four-chamber view/2D &TEE bicaval view/2D

Central line with its tip in the right atrium. Mobile vegetation attached to the catheter (thickened tip) on transthoracic echo (left) and the adjacent wall (right) seen in TEE.

NON-INFECTIVE/ABACTERIAL ENDOCARDITIS

Types

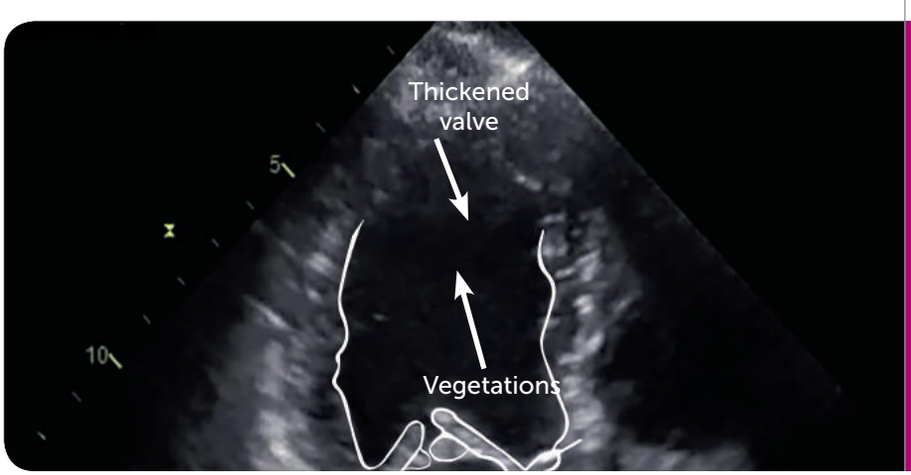
- Marantic endocarditis
- Hypercoagulable state
- Libman-Sacks endocarditis
- Antiphospholipid syndrome

Echo Characteristics

- Valve thickening
- Mild or moderate regurgitation
- Small vegetations
- Pericardial effusion

Cardiac Manifestations of Libman-Sacks Endocarditis

- Valve thickening and vegetations
- Mural thrombus
- Spontaneous contrast
- Left + right ventricular dysfunction
- Pericardial effusion



LIBMAN-SACKS ENDOCARDITIS – apical three-chamber view/2D

Patient with lupus and antiphospholipid syndrome. Several small vegetations are seen on the mitral valve.

NOTES

INDICATIONS FOR SURGERY

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Recommendations for Surgery in Infective Endocarditis (IE)

Heart Failure	Timing	Class	Level
Aortic or mitral IE with severe acute regurgitation or valve obstruction, causing refractory pulmonary edema or cardiogenic shock	Emergency	I	B
Aortic or mitral IE with fistula into a cardiac chamber or pericardium causing refractory pulmonary edema or shock	Emergency	I	B
Aortic or mitral IE with severe acute regurgitation or valve obstruction and persistent heart failure or echocardiographic signs of poor hemodynamic tolerance (early mitral closure or pulmonary hypertension)	Urgent	I	B
Aortic or mitral IE with severe regurgitation and no HF	Elective	IIa	B
Uncontrolled Infection			
Locally uncontrolled infection (abscess, false aneurysm, fistula, enlarging vegetation)	Urgent	I	B
Persistent fever and positive blood cultures > 7 – 10 days	Urgent	I	B
Infection caused by fungi or multiresistant organisms	Urgent elective	I	B
Prevention of Embolism			
Aortic or mitral IE with large vegetations and one or more embolic episodes despite appropriate antibiotic therapy	Urgent	I	B
Aortic or mitral IE with large vegetations (>10 mm) and other predictors of complicated course of disease (heart failure, persistent infection, abscess)	Urgent	I	B
Isolated very large vegetations (>15 mm)	Urgent	IIb	B

016 // Right Heart Disease

CONTENTS

NOTES

BASICS OF PULMONARY HYPERTENSION

Definition and Classification of Pulmonary Hypertension

By definition, the diagnosis of pulmonary hypertension can only be made by introducing a right heart catheter.

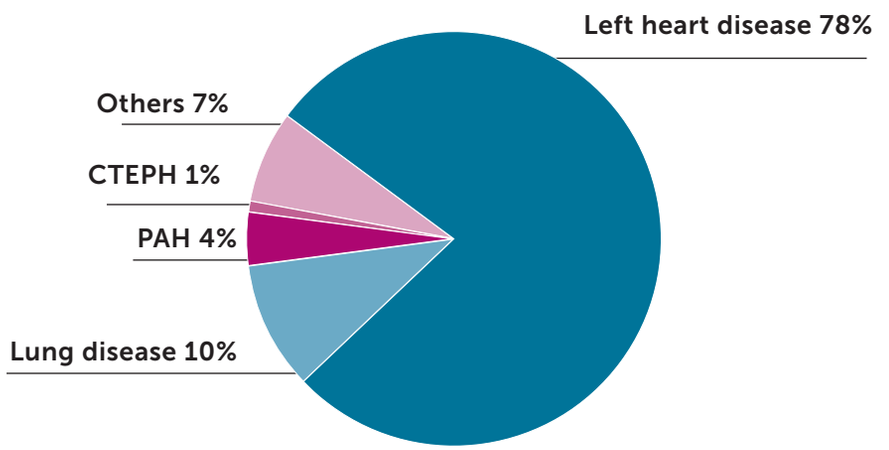
Definition: mPAP ≥ 25 mmHg at rest

- Pulmonary arterial hypertension (PAH)
- Pulmonary hypertension owing to left heart disease (CTEPH)
- Pulmonary hypertension owing to lung disease and/or hypoxia
- Chronic thromboembolic pulmonary hypertension
- Pulmonary hypertension with unclear multifactorial mechanisms

Left heart disease (postcapillary) is the most common cause of pulmonary hypertension.

Causes of Pulmonary Hypertension

Patients with chronic obstructive pulmonary disease rarely develop severe forms of pulmonary hypertension.



Hemodynamic Definition of Pulmonary Hypertension

Look at the left heart. Does it explain pulmonary hypertension? Is LV filling pressure elevated? The echo can provide clues as to whether pre- or post-capillary pulmonary hypertension is present.

Definition	Characteristics	Clinical groups
Pulmonary hypertension	Mean PAP ≥ 25 mmHg	All
Pre-capillary pulmonary hypertension	Mean PAP ≥ 25 mmHg PCWP ≤ 15 mmHG	PAH Lung disease CTEPH Unclear/multifactorial
Post-capillary PH	Mean PAP ≥ 25 mmHg PCWP > 15 mmHG	PH due to left heart disease
Passive	TPG ≤ 12 mmHg	

Reactive (out of proportion) TPG > 12 mmHg

The transpulmonary gradient is the difference between mean PAP and PCWP

PAP = pulmonary artery pressure **TPG=** transpulmonary gradient

BASICS OF PULMONARY HYPERTENSION

Prognosis of Pulmonary Hypertension

NOTES

Pulmonary hypertension is a disease with a poor prognosis, especially in advanced stages. Early diagnosis is important.

Echocardiographic Screening for Pulmonary Hypertension

Exercise Doppler echocardiography is currently not recommended for screening patients for pulmonary hypertension.

		Class	Level
PH unlikely	Tricuspid regurgitation velocity ≤ 2.8 m/s, sPAP ≤ 36 mmHg and no additional echocardiographic variables suggestive of PH	I	B
PH possible	Tricuspid regurgitation velocity ≤ 2.8 m/s, sPAP ≤ 36 mmHg, but the presence of additional echocardiographic variables suggest PH	IIa	C
	Tricuspid regurgitation velocity 2.9–3.4 m/s, sPAP 37–50 mmHg with/without additional echocardiographic variables suggestive of PH	IIa	C
PH likely	Tricuspid regurgitation velocity > 3.4 m/s, sPAP > 50 mmHg, with/without additional echocardiographic variables suggestive of PH	I	B

Additional echo variables suggestive of pulmonary hypertension = IVS flattening, short PVAT, PA- dilatation

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NOTES

ECHO ASSESSMENT OF PULMONARY HYPERTENSION

systolic PAP (sPAP) = = 4 TR Vmax² + Right Atrial Pressure (RAP)

Normal tricuspid regurgitation velocity is age dependent. The severity of TR tends to increase with age.

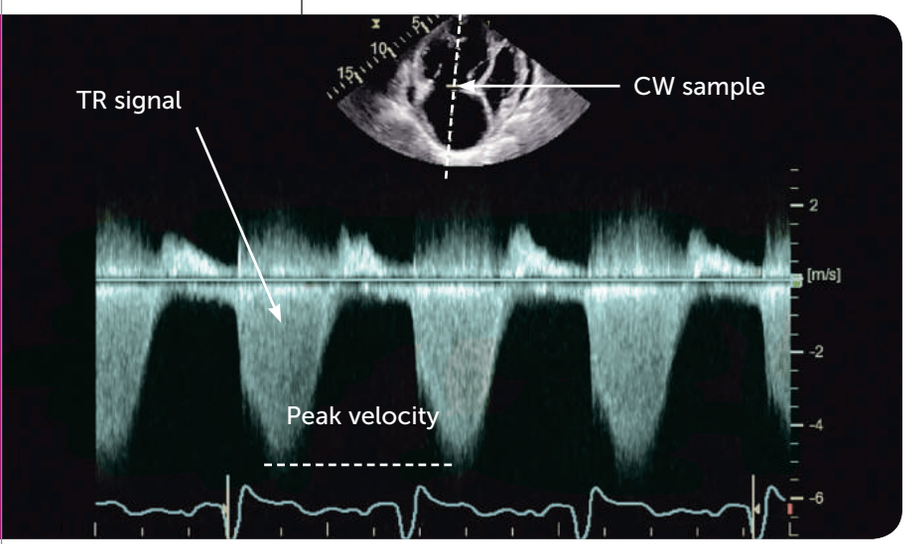
Quantification of sPAP and Pulmonary Hypertension

- Normal TR velocity is 1.7– 2.3 m/s
- Elevated when TR velocity > 2.8–3.0 m/s
- sPAP = TR velocity-derived RV/RA gradient + RA pressure

Mild PHT	sPAP > 40 (35) mmHg
Moderate PHT	sPAP > 50 mmHg
Severe PHT	sPAP > 60 mmHg

MEASUREMENT OF SYSTOLIC PULMONARY ARTERIAL PRESSURE – apical four-chamber view/CW Doppler TR

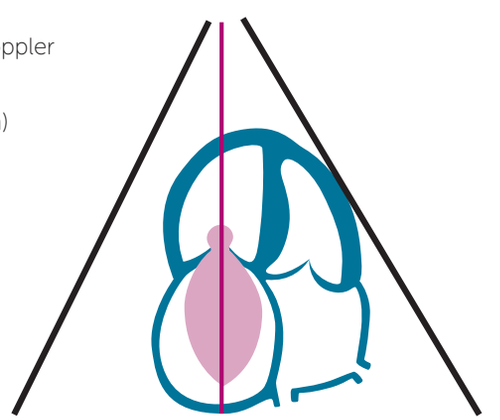
The RV/RA gradient is derived from the peak tricuspid regurgitation velocity using CW Doppler. Be sure to measure the true maximim velocity (good signal quality).



Pulmonary hypertension does not imply severe tricuspid regurgitation and severe TR does not imply severe pulmonary hypertension.

Factors That Influence TR velocity/sPAP

- Severity of tricuspid regurgitation
- Pulmonary hypertension
- Doppler/image quality
- Alignment of the TR jet to CW Doppler
- Right ventricular function
- Inspiration (higher with inspiration)

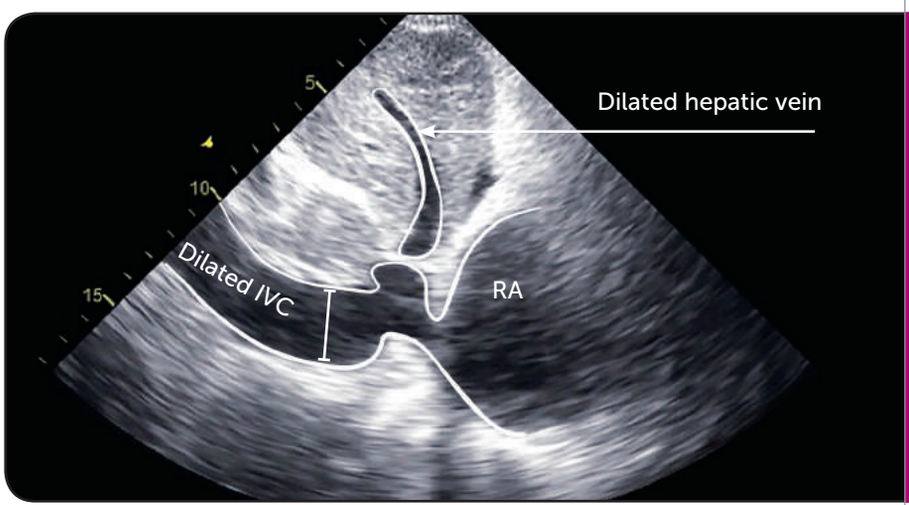


ECHO ASSESSMENT OF PULMONARY HYPERTENSION

Estimation of Right Atrial Pressure

RA pressure	IVC (diameter)	Inspiration
0 – 5 mmHg	small (< 1.5 cm)	collapsing
5 – 10 mmHg	normal (1.5 – 2.5 cm)	> 50% diameter reduction
10 – 15 mmHg	dilated (>2.5 cm)	< 50% diameter reduction
> 20 mmHG	IVC + liver veins dilated	no diameter change

RA pressure estimation based on this scale is not always reliable.



DILATED INFERIOR VENA CAVA – subcostal IVC view/2D

Severely dilated inferior vena cava without respiratory fluctuations in diameter and dilated hepatic veins in a patient with pulmonary hypertension. These findings suggest right atrial pressures > 20 mmHg.

Quantification of mPAP

mPAP = 4 x maximum pulmonary regurgitation velocity

mPAP =79–0.45 x (pulmonary acceleration time) (Mahan's regression equation)

Pulmonary Acceleration Time (PVAT)

- Shortened in elevated pulmonary artery pressure
- May be normal in elevated right-sided cardiac output

Should only be applied for heart rates between 60 – 100

Normal	> 130 ms	**Mild**	80 – 100 ms
Borderline	100 – 130 ms	**Severe**	< 80 ms

NOTES

In very severe tricuspid regurgitation, the TR spectrum is triangular. In this case RAP and thus pulmonary artery pressure cannot be estimated (no gradient between RA and RV).

Elevated RA pressure may lead to significant shunts across a patent foramen ovale, or ASD causing undersaturation.

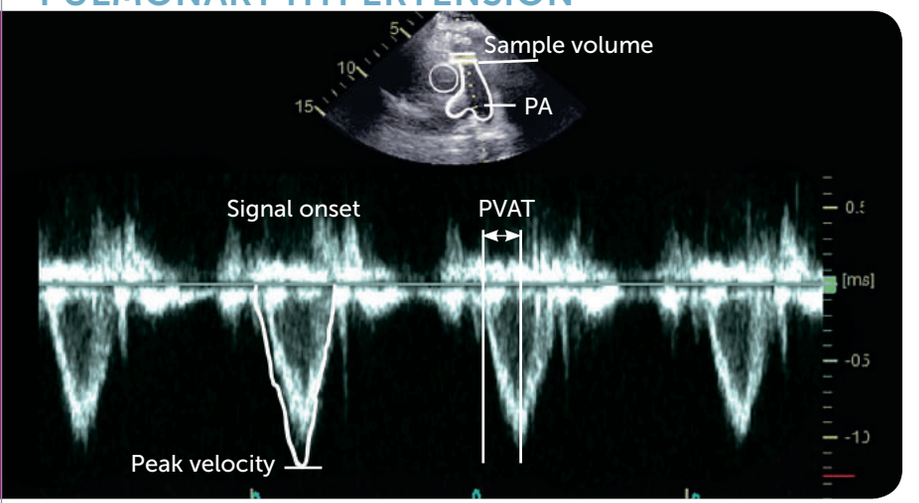
PVAT can be very valuable in situations where sPAP cannot be measured due to insufficient TR signal.

NOTES

ECHO ASSESSMENT OF PULMONARY HYPERTENSION

PULMONARY ACCELERATION TIME (PVAT) – PSAX/PW PV

PVAT is measured from the onset to the peak of the RVOT/PV outflow signal. In the abscence of pulmonary hypertension, the peak is rather late and the curve symmetrical.



The normal pulmonary artery is a) smaller than the ascending aorta b) <27 mm in women and <29 mm in men.

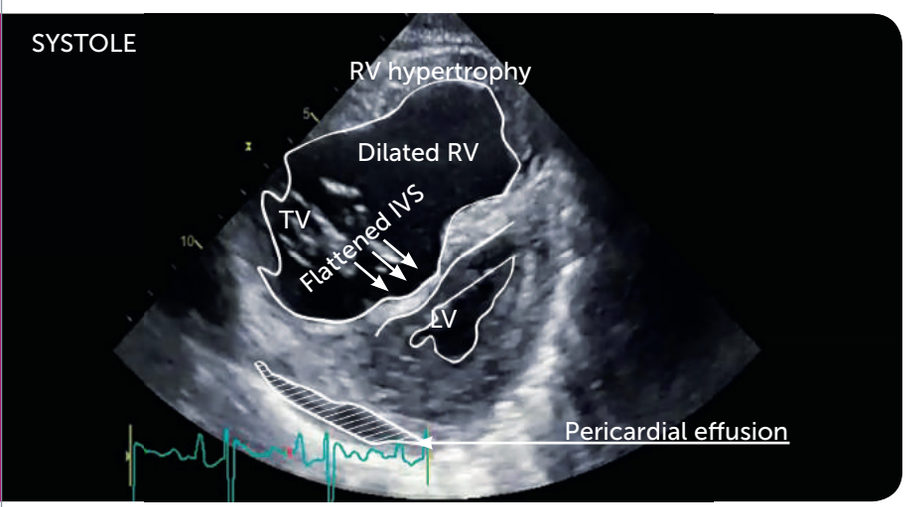
Patients with pericardial effusion have a poor prognosis. Septal flattening can be very subtle, especially when systolic pressure is high.

Echo Findings in Pulmonary Hypertension

- Dilated right ventricle
- Reduced right ventricular function
- Right ventricular hypertrophy
- Septal flattening (systolic) = D-shaped ventricle
- Dilated pulmonary artery
- Pulmonary regurgitation
- Enlarged right atrium
- Pericardial effusion
- Pleura effusion
- Low cardiac output

ECHO FINDINGS IN PULMONARY HYPERTENSION – PSAX/2D

Echo features of severe pulmonary hypertension: D-shaped left ventricle with a flattened interventricular septum in systole, a dilated right ventricle, right ventricular hypertrophy, and pericardial effusion.



DISEASE OF THE RIGHT VENTRICLE

Echocardiographic Signs of Acute Pulmonary Embolism

- The sensitivity of echo for the detection of pulmonary embolism is low. In cases of typical echo findings (especially dilated RV with reduced RV function), the patients are usually very symptomatic (large PE)
- McConnel sign: Characterized by akinesia of the mid-free wall but normal motion in the apex (poor positive predictive value)
- 60/60 sign: Characterized by a PVAT below 6 0ms in the presence of a tricuspid regurgitation maximum gradient above 30 mmHg but below 60mmHg
- Right ventricular pressure overload: Characterized by a D-shaped right ventricle

DD: Pulmonary Embolism and RV Infarction

- Similar symptoms
- Similar ECG
- Similar echo findings
- Look for regional wall motion abnormalities (inferior infarction)

RIGHT VENTRICULAR INFARCTION

Right Ventricular Infarction

- Associated with inferior myocardial infarction (30–50%)
- Poor prognosis
- Hypotension/shock
- Arrhythmia

Echo Findings

- Global and regional reduction in right ventricular function
- Low cardiac output
- Low annular velocity (Tissue Doppler) and decreased longitudinal strain (speckle-tracking)
- Tricuspid regurgitation
- Dilated inferior vena cava

NOTES

The McConnell sign is marked by akinesia of the mid-free wall but normal motion of the apex. It is also present in right ventricular infarction. The 60/60 sign is a PVAT below 60 ms in the presence of a TR maximum gradient above 30 but below 60 mmHg.

The untrained right ventricle is unable to cope with acute pressure overload. Therefore, very high sPAP measurements are uncommon in acute pulmonary embolism (exceptions are patients with recurrent pulmonary embolism/CTEPH with preexisting pulmonary hypertension).

The majority of patients with RV infarction recover in a period of weeks or months.

Look at the right ventricular wall motion in all patients with inferior infarcts.

NOTES

RIGHT VENTRICULAR HYPERTROPHY

Use atypical views of the RV (2-chamber RV view, inflow/outflow RV view).

- Right ventricle free wall ≥ 6mm
- Use a subcostal 4-chamber view to image the free right ventricle wall
- Consequence of pressure overload on the right ventricle
- Concentric right ventricular hypertrophy in pulmonary stenosis
- Measurement may be difficult; also use visual assessment
- Right ventricle hypertrophy may also lead to right ventricular outflow tract obstruction (narrow right ventricular outflow tract)

Causes of Right Ventricular Hypertrophy

- Chronic pulmonary hypertension
- Pulmonic valve stenosis (including congenital abnormalities, e.g. tetralogy of Fallot)
- Tetralogy of Fallot
- High altitude
- Athlete's heart syndrome
- Hypertrophic cardiomyopathy (with right heart involvement)

ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA (ARVD)

ARVD may affect both ventricles. Echo has rather low sensitivity and specificity in subtle forms of ARVD -> MRI will be needed.

- Usually autosomal dominant
- Fatty and fibrous replacement of myocardium, especially in the right ventricular outflow tract
- 5–10% of sudden cardiac deaths (<65 years)
- Its prevalence is 3-fold higher in males

Echo Findings in ARVD

Echocardiographic assessment should always include the RVOT (aneurysm?). Use atypical views.

- Aneurysmal dilatation, usually in the diaphragmatic, apical and infundibular regions (triangle of dysplasia)
- Reduced right ventricular function
- Regional wall motion abnormalities + thin wall
- Right ventricular dyssynchrony

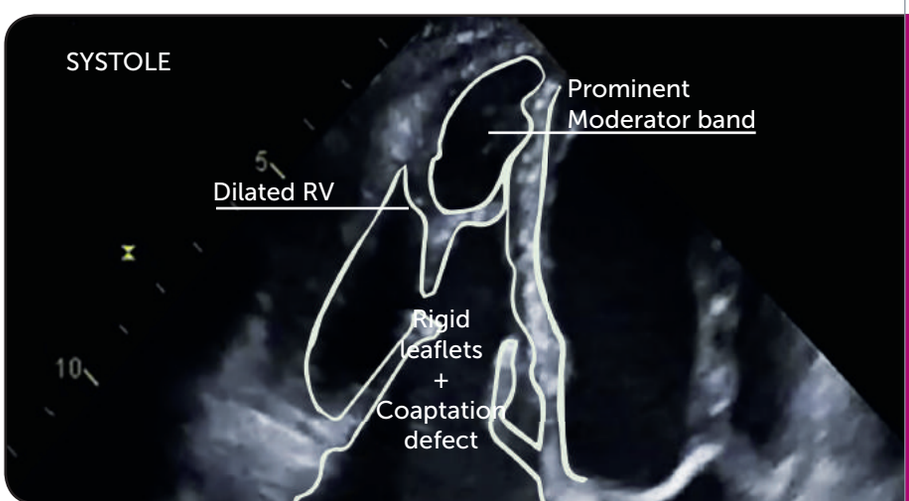
Carcinoid Heart Disease

- Characterized by plaque-like deposits of fibrous tissue, which most commonly occur on the endocardium of valvular cusps and the leaflet.
- Occurs in 50% of patients with carcinoid syndrome
- High circulating concentrations of serotonin in the heart is the underlying substrate of carconoid heart disease.
- The right heart is most commonly affected because serotonin is inactivated by the lung and therefore protects the left heart

ARRYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA (ARVD)

Echo Findings in Carcinoid Heart Disease

- Right ventricular enlargement
- Tricuspid valve, pulmonic valve leaflets and the subvalvular apparatus are thickened and rigid
- Usually significant tricuspid regurgitation with restricted motion of the leaflets, causing a wide coaptation defect.
- Abnormal motion of the interventricular septum (volume overload caused by tricuspid regurgitation).
- Triangular CW spectrum indicative of severe tricuspid regurgitation.
- Associated with pulmonic stenosis (and regurgitation).



CARCINOID HEART DISEASE – apical four-chamber view RV optimized/2D

Restricted motion/position of the tricuspid leaflets, leaving a wide coaptation defect. The leaflets are thickened (from the base) and rigid. The endocardium is bright. These findings are highly indicative of carcinoid heart disease.

NOTES

If you suspect carcinoid heart disease, tilt the transducer to the abdomen and image the liver. The majority of patients with carcinoid heart disease have hepatic metastases.

NOTES

017 // Aortic Disease

CONTENTS

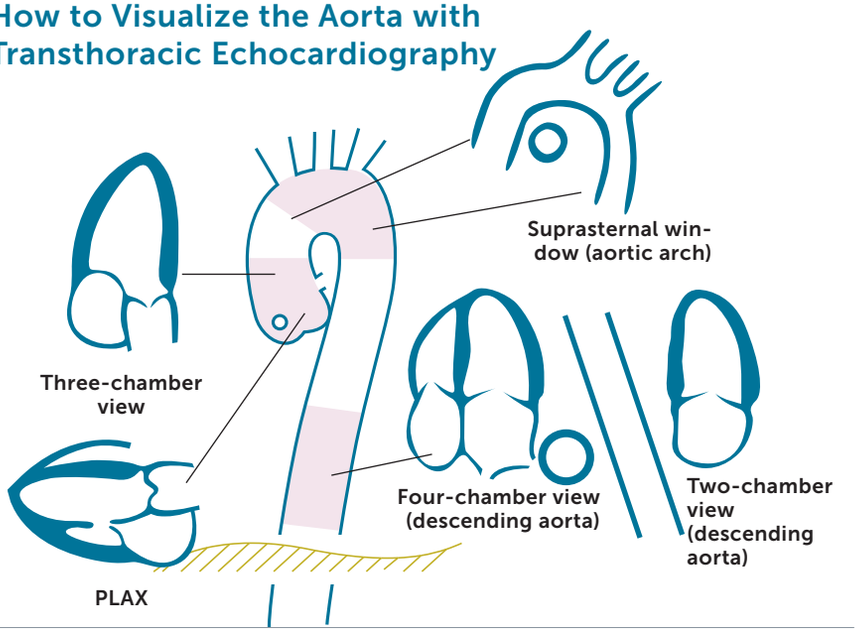
NOTES

IMAGING OF THE AORTA

Use a modified parasternal long-axis view (one intercostal space cranial) to see more of the ascending aorta.

Every echo report should include a description of the ascending aorta (normal/dilated) with corresponding measurements.

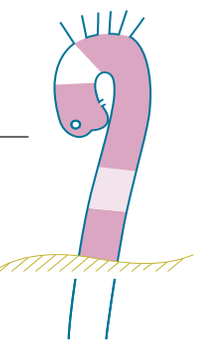
How to Visualize the Aorta with Transthoracic Echocardiography



Even with TEE it may be difficult to see cranial segments of the ascending aorta.

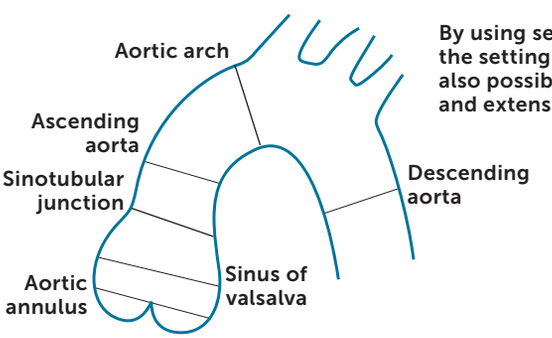
Transoesophageal Echo (TEE)

BETTER RESOLUTION	MORE SEGMENTS
The esophagus is close to the aorta. We may therefore use higher transducer frequencies, which translate into better resolution.	TEE is much better for the assessment of the descending thoracic aorta



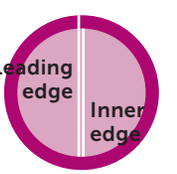
The aortic diameter is slightly larger in systole than in diastole.

Where and How to Measure

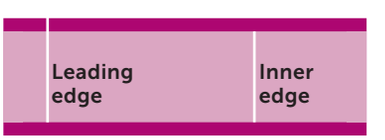


By using several measurements (in the setting of aortic dilatation), it is also possible to determine the shape and extension of aortic aneurysms.

The aorta can be measured on a long- and/or short-axis view. Most reference values were obtained with the leading edge method. However, to correlate measurments better with other imaging modalities (CT, MRI), measurements of the inner diameters (inner edge to inner edge) are applied to an increasing extent. The difference between these measurements methods is minimal and insignificant, thanks to improved image resolution.

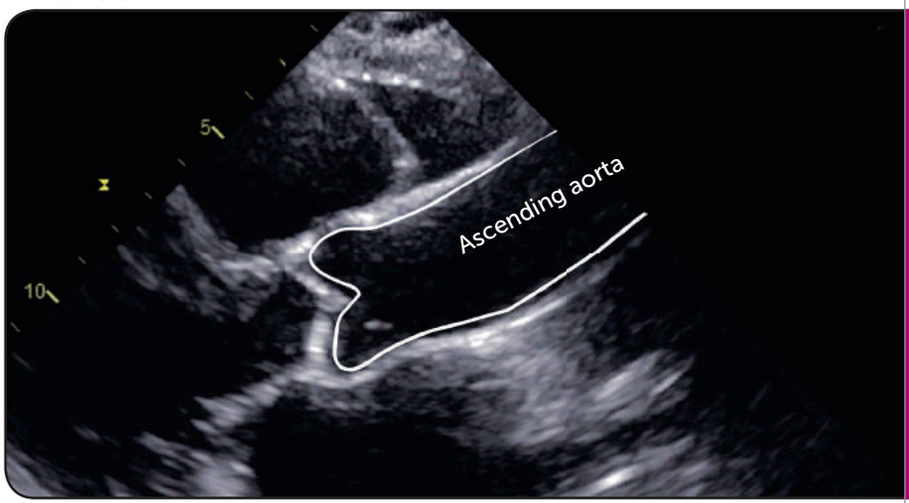


Axial view



Longitudinal view

BASICS



NOTES

VISUALIZATION OF THE ASCENDING AORTA – modified PLAX/2D

The more cranial portions of the ascending aorta can be better visualized by moving the transducer up one intercostal space and more laterally.

Size of the Aorta

	Diameter	Diameter/BSA
Aortic annulus	20-31mm	13 mm/m2
Sinus of valsalva	29- 45mm	19 mm/m2
Sinotubular junction	22-36mm	15 mm/m2
Ascending aorta	22-36mm	15 mm/m2
Aortic arch	22-36mm	
Descending aorta	20- 30mm	
Abdominal aorta	18- 28mm	

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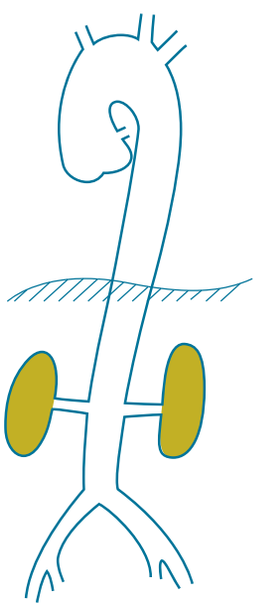


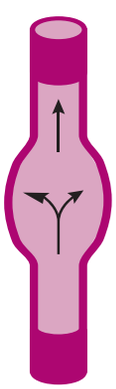
The size of the aortic is strongly related to body surface area (in particular hight) and age.

AORTIC ANEURYMS

Definitions

True aneurysm
Localized dilatation > 50% of the reference segment (circumscribed or diffuse aneurysms)

Aortic ectasia
Arterial dilatation of less than 150% of the normal arterial diameter



NOTES

AORTIC ANEURYMS

Incidence – Facts

- Death – aneurysm = 0.7/100,000 per year
- Death – dissection = 1.5/100,000 per year
- No difference between prevalence in men and women
- Thoracic aneurysms >6 cm are subject to a rupture and dissection risk of 6.9% per year.

Any increase in the diameter of the aorta is related to (blood) pressure, the size of the aorta, and the thickness of the wall (law of Laplace).

Forms of Aneurysms

Pure ascendens type **"Sausage" type** **Bulbus type (Marfan)**

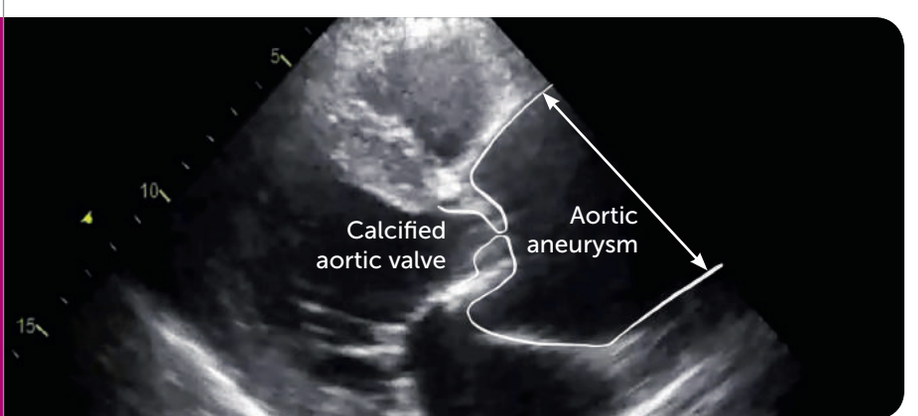
In the setting of aneurysms the aorta changes its orientation (to the right); it may even be elongated.

To quantify aneurysms of the ascending aorta, always use a parasternal long- and short-axis view. In the presence of an aneurysm of the ascending aorta, also image from a suprasternal window to determine whether the aortic root is involved. Ascending aortic aneurysms are sometimes visualized best from a right parasternal approach.

Look at the shape of the ascending aorta: something is wrong when there is no narrowing at the sinotubular junction.

ANEURYSM OF THE ASCENDING AORTA – PLAX/2D

Patient with bicuspid valve, aortic stenosis and aneurysm of the aortic root and the ascending aorta. There is no narrowing at the sinotubular junction.



Bicuspid Aortic Valve and Aneurysm

- Dilatation of the aorta may be present in patients with congenital abnormal valves (e.g. bicuspid).
- 9-fold higher risk of dissection in the presence of bicuspid valves.
- 6–10% of all dissections occur in the setting of bicuspid valves.

Progressive dilatation of the aorta continues even after aortic valve replacement in patients with bicuspid valves. Follow such patients closely.

AORTIC ANEURYMS

Inherited Disorders Affecting the Aorta

- Marfan
- Ehlers Danlos (type IV)
- Familial forms of connective tissue disorders
- Annulo-aortic ectasia
- Loeys-Dietz syndrome

Marfan Syndrome – Cardiac Manifestations

- Aortic dilatation
- Aortic dissection
- Aortic regurgitation (annular dilatation)
- Mitral valve prolapse
- Pulmonary artery dilatation
- Large aortic valve cusps

Inflammatory Diseases of the Aorta

- Syphilis
- Staph. aureus infection
- Kawasaki disease
- Giant cell arteritis
- Takayasu arteritis

Risk of Rupture – Stratification Based on Aortic Size

Low risk	≤ 2.75 cm/m^2	4%/year
Moderate risk	2.75 – 4.25 cm/m^2	8%/year
High risk	≥ 4.25 cm/m^2	20%/year

Indications for Aortic Surgery (ACC Class I)

- Asymptomatic patients with an ascending aortic diameter or an aortic sinus diameter ≥ 55mm
- Patients with Marfan syndrome with an aortic diameter between 40-50 mm
- Patients with a growth rate of more than 0.5 cm/year in an aorta less than 5.5 cm in size
- Patients undergoing aortic valve repair, with an aortic aneurysm ≥ 4.5 cm in size

ACC 2010

NOTES

Inherited disorders also include so called "overlap syndromes".

Aortic disease/dissection is the main cause of morbidity and mortality in Marfan syndrome.

Infections may trigger non-infectious vasculitis by generating immune complexes or by cross-reactivity. Inflammation may result in aortic dilatation and ostial stenosis of major branches.

Use other imaging modalities (mitral regurgitationl and CT) for precise measurements and for decision-making. Use the technique you are most familiar with.

NOTES

The false lumen is usually larger than the true lumen, with slower flow, and often with thrombi.

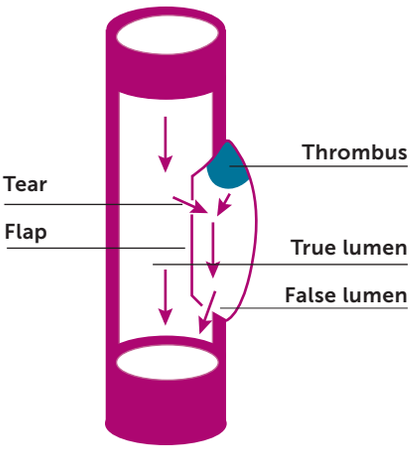
Intimal flaps may prolapse through the aortic valve. Also look for intimal flaps in the aortic arch (using a suprasternal window).

AORTIC DISSECTION

Aortic Dissection

Characteristics:

- Intima (media) disruption/ intimal flap – true + false lumen
- Spiral-shaped dissections may occur, sometimes involving branches (coronaries!!, supraortic branches)
- 2.6–3.5 cases per 100,000 persons/year
- 2/3 males



Classifications of Aortic Dissection

Stanford classification

A Ascending | A | B Descending

Type A involves the ascending aorta, type B only the descending aorta

DeBakey classification

I Ascending Descending | II Ascending | III Descending

Type I involves the ascending and the descending aorta, type II only the ascending aorta and type III only the descending aorta.

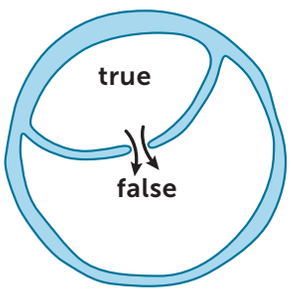
AORTIC DISSECTION

Risk Factors for Dissection

- Aortic aneurysm
- Marfan + other connective tissue disorders
- Atherosclerosis
- Iatrogenic (e.g. left heart catheter, heart surgery cannulation)

Aortic Dissection

Classic dissection



Complications of dissection

- Aortic rupture
- Branch vessel dissection (coronaries)
- Expansion
- Intramural hematoma
- Aortic regurgitation
- Rupture with pericardial tamponade
- Leriche syndrome

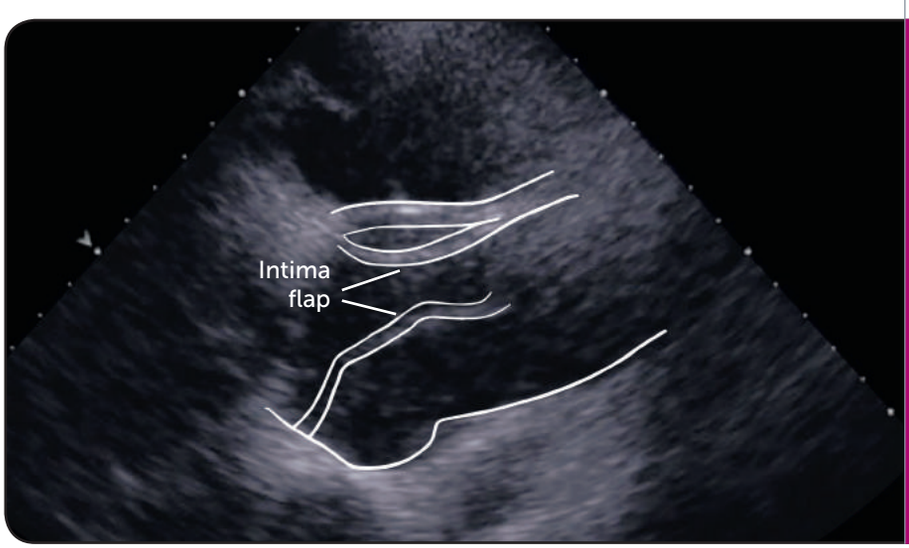
TTE in Aortic Dissection

- Sensitivity = 77–80%
- Specificity = 93–96%

Always confirm dissection by using other imaging modalities.

Aortic regurgitation in dissection

- Dilatation of the root
- Bicuspid valves
- Prolapse of the intimal flap



DISSECTION OF THE ASCENDING AORTA – PLAX/2D

Highly mobile intimal flap in the ascending aorta, denoting aortic dissection. This flap is almost circumferential and thus visualized both anteriorly and posteriorly.

NOTES

Untreated dissection of the ascending aorta is associated with a mortality rate of 90% within 1 year (rupture into the pericardium, mediastinum, or left pleural cavity).

The intima/media is detached (flap), and divides the aorta into a true and a false lumen.

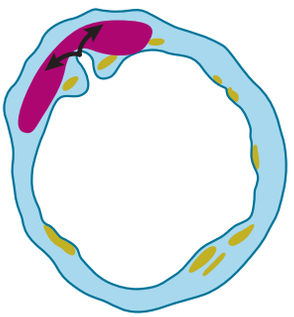
Beware of reverberations of the aortic wall or adjacent structures. They may mimic an intimal flap. A true intimal flap is marked by motion independent of the aortic wall.

NOTES

AORTIC DISSECTION

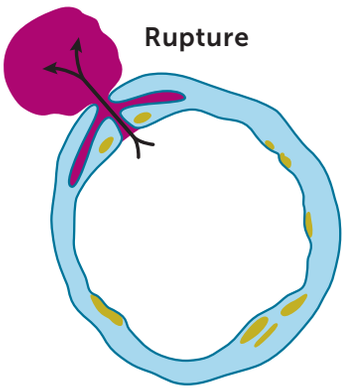
Aortic Syndromes

Intramural hematoma



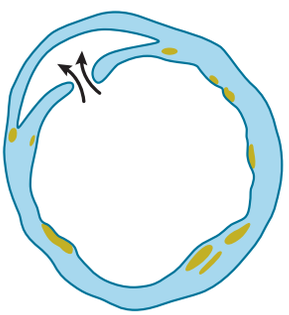
Bleeding into the aortic wall (such as after plaque rupture) causes an intramural hematoma.

Rupture



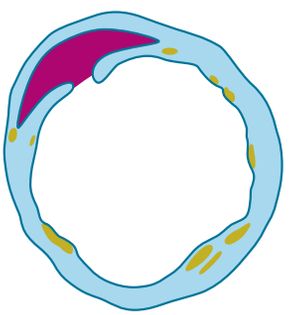
Plaque rupture, penetrating ulcers, and intramural hematoma may lead to aortic rupture.

Localized dissection



Localized dissection is usually a result of atherosclerosis. Dissection is limited to a circumscript region.

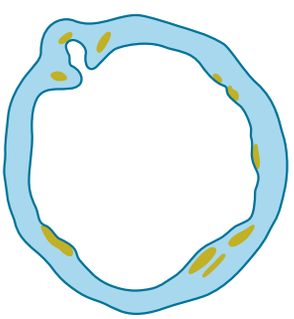
"Healed" dissection



The false lumen of dissection may thrombose and eventually heal.

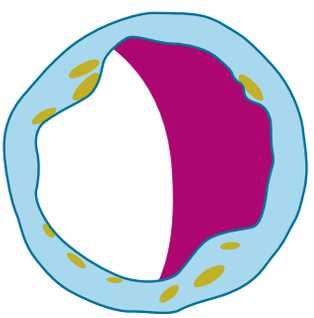
Aortic syndromes are no benign condition. The bear a high risk of rupture. Further evaluation with CT/mitral regurgitation is mandatory.

Penetrating ulcer



Rupture of an atherosclerotic plaque results in a penetrating ulcer.

Intraluminal thrombus



Regional thickening of the aorta > 7 mm (circular shape) (DD: thrombus in false lumen, intramural hematoma)

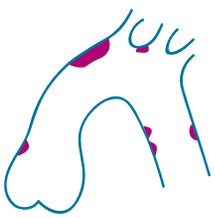
AORTIC DISSECTION

Aortic Plaque

- Patients with artherosclerotic plaques in the aorta are subject to a high risk of coronary artery disease and myocardial infarction.
- Increased risk of embolism/stroke (plaque in the ascending aorta/aortic arch).
- Increased risk of aortic dissection.
- Increased risk of aortic syndromes.

Typical Locations of Plaques in the Aorta

- Aortic arch
- Cranial segments of the descending aorta



NOTES

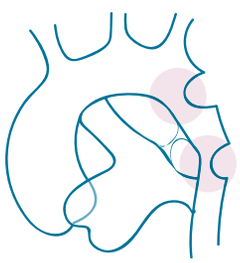
Plaque size is important for risk stratification. When the plaque size is > 4 mm, the risk of stroke is significantly increased. (OR=9.1)

TTE is also Capable of demonstrating plaques /especially in the ascending aorta). Capable of demonstrating plaques/especially in the ascending aorta).

AORTIC COARCTATION (COA)

Facts

- 5–10% of all congenital defects
- Predominantly men
- Higher blood pressure at the upper extremities compared to the lower extremities
- Located distal to the subclavian artery
- Increased risk of intracranial hemorrhage



Kinking may lead to flow turbulence (seen in color Doppler), thereby mimicking CoA = pseudocoarctation

Echo Features

- Left ventricular hypertrophy
- Narrowing of the aorta
- Turbulent flow is visible on color Doppler
- Elevated CW Doppler gradient in the aorta
- The presence of a systolic and an additional diastolic gradient denotes hemodynamic significance of obstruction

The suprasternal view is the most valuable window to identify coarctation. Quantification is based on the maximal velocity/gradients (measured with CW Doppler) and the presence of a systolic AND diastolic gradient.

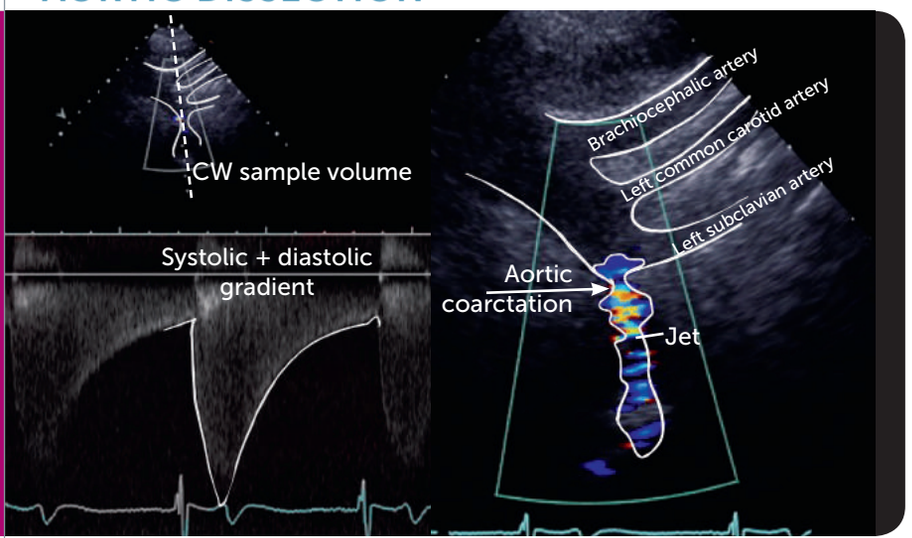
Doppler measurments usually overestimate gradients in comparison to hemodynamic assessment.

NOTES

AORTIC COARCTATION – suprasternal view/Color and CW Doppler

Turbulent flow in the descending aorta (left) denotes the location of coarctation. The Doppler spectrum (right) shows a systolic and diastolic gradient (>4 m/s), suggesting severe coarctation.

AORTIC DISSECTION



Patients with hemodynamically relevant forms of CoA also have left ventricular hypertrophy.

Coarctation – Associated Abnormalities

- Bicuspid aortic valve
- Persistent ductus arteriosus/ventricular septal defect
- Hypoplasia of the aortic arch
- Left ventricular outflow tract obstruction

018 // Pericardial Disease

CONTENTS

NOTES

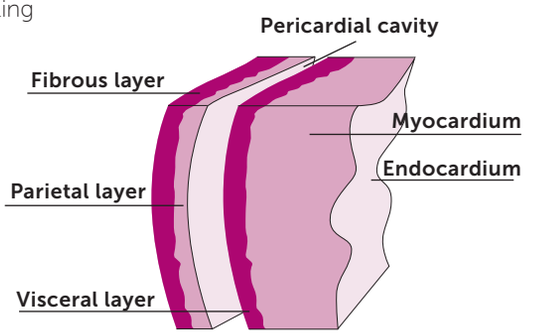
THE PERICARDIUM

The Pericardium – Importance

The pericardium consists of a visceral and a parietal layer.

Patients with an open pericardium or chest (cardiac surgery) have an abnormal contractile pattern.

- Limits distension
- Facilitates interaction and coupling of the ventricles/atria
- Facilitates twist and torsion
- Normal quantity of pericardial fluid < 50ml



PERICARDIAL EFFUSION

Forms of Pericardial Effusion

Bacterial infection (especially tuberculosis) predisposes to constriction.

Exudative effusion is characterized by fibrous strands.

Transudative
Congestive heart failure, myxedema, nephrotic syndrome

Exudative
Tuberculosis, spread from empyema

Hemorrhagic
Trauma, rupture of aneurysms, malignant effusion, iatrogenic

Malignant
Often hemorrhagic

Causes of Pericardial Effusion

The cause of pericardial effusion depends on the setting of your lab and the part of the world you practice in (e.g. tuberculosis in developing countries, iatrogenic when interventions and cardiovascular surgery are performed at your center).

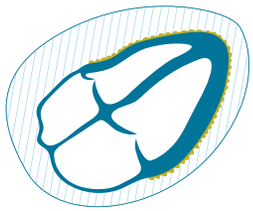
The cause of effusion may remain unclear because the diagnosis would require peri-and/or myocardial biopsy as well as cytological, histoimmunological, and microbiological analysis of the fluid.

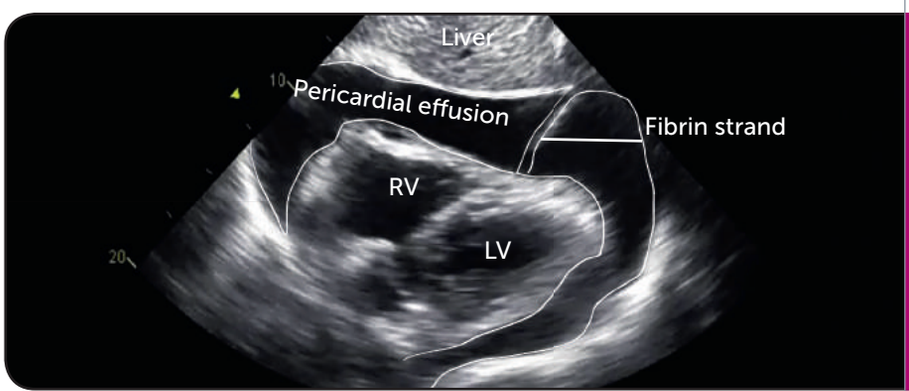
- **Idiopathic:** no cause is found despite full diagnostic investigation
- **Infectious:** common in viral infection (direct + immune response)
- **Iatrogenic:** pacemaker, catheter procedures, biopsy, cardiac surgery
- **Neoplastic:** often hemorrhagic, denotes poor prognosis
- **Myocardial infarction:** myocardial rupture, epistenocardic (early) + Dressler syndrome (late)
- **Renal failure:** uremia- or dialysis-associated
- **Autoimmune disease:** particularly: systemic lupus erythematodes, rheumatoid. arthritis., systemic sclerosis
- **Radiation:** 20% develop constriction
- **Rheumatic:** usually small pericardial effusion
- **Traumatic:** contusio cordis or heart/aortic rupture
- **Endocrine disorder:** e.g. myxedema
- **Pulmonary hypertension:** the mechanism is unclear (poor prognosis)
- **Post cardiac surgery:** usually hematoma, often localized
- **Aortic rupture:** hemorrhagic effusion, pericardial effusion in 45% of dissections.

PERICARDIAL EFFUSION

Echo Diagnosos of Pericardial Effusion

- Echo-free space measured in end-diastole.
- Use multiple views, especially subcostal views.
- Use atypical views; specifically visualize the surroundings of the right ventricle.





PERICARDIAL EFFUSION – subcostal four-chamber view/2D

Large circumferential pericardial effusion with fibrin strands. The image loop shows swinging heart motion.

Facts

Large effusion	Regional effusion
Neoplastic	Postoperative
Uremic	Trauma
Tuberculosis	Purulent
Myxedema	

Talk to the patient. Thorough history-taking often helps to clarify the cause of effusion.

Differential Diagnosis

- Pleural effusion
- Epicardial fat
- Pericardial cyst
- Ascites

Pericardial effusions are anterior to the descending aorta while pleural effusions are posterior to it.

If you are still not sure, make the patient sit up and image the pleura (from the back). Here you will see whether a pleural effusion is present or not.

Epicardial Fat

- Follows the normal motion of the pericardium
- Is related to the presence of abdominal fat
- Is not completely echo-free (low-intensity echos)
- Absent above the right atrium and usually very prominent in the atrioventricular groove as well as around the atrial appendages

Epicardial fat is common in obese patients, diabetes, atrial fibrillation and coronary artery disease. Epicardial fat is seen better in the presence of a pericardial effusion.

NOTES

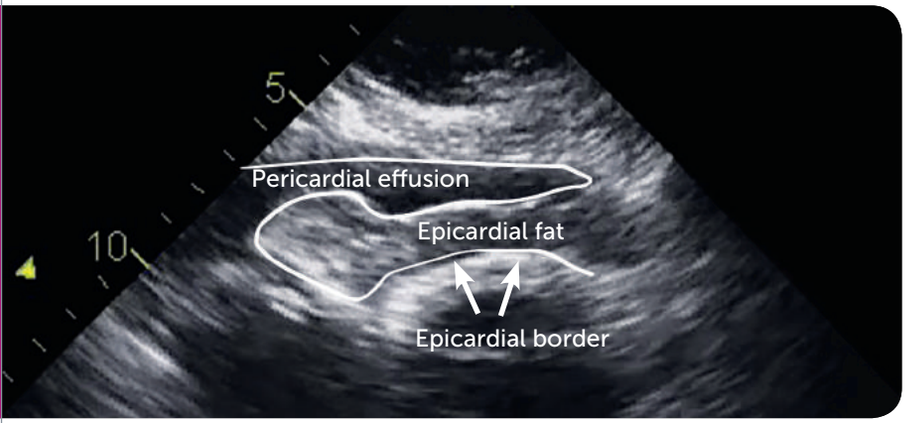
The pericardium is highly reflective in echocardiography.

NOTES

EPICARDIAL FAT – subcostal four-chamber view/2D

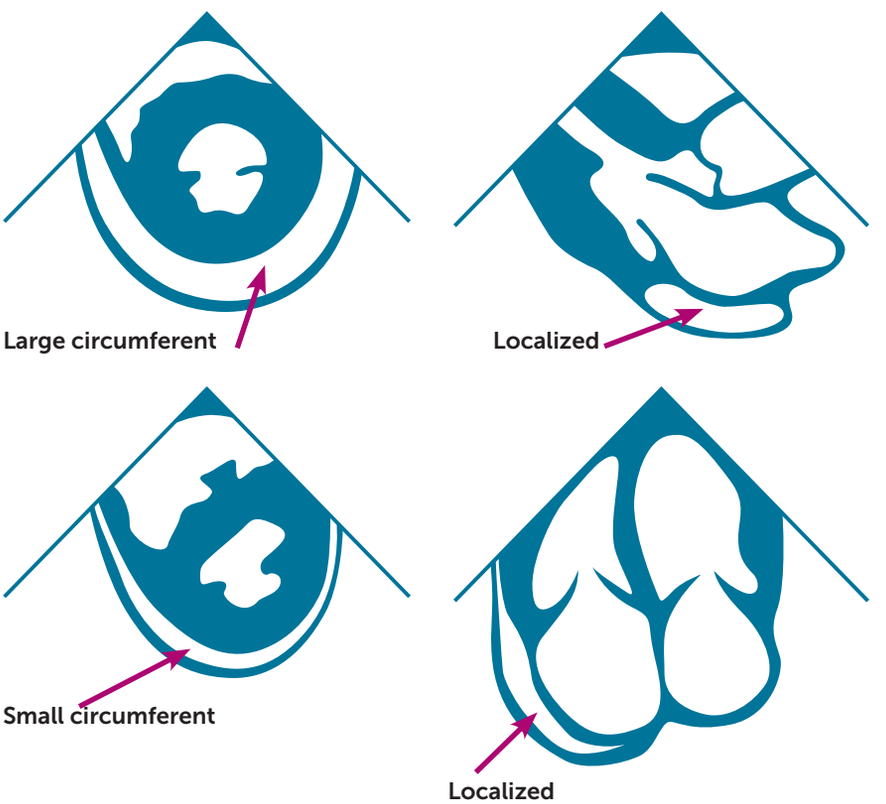
A patient with a small pericardial effusion and pronounced epicardial fat. Epicardial fat is prominent in the AV groove and absent in the region of the right atrium.

PERICARDIAL EFFUSION



Localized effusions occur in the setting of fibrinous and iatrogenic (hemorrhagic) pericardial effusion.

Location of Pericardial Effusion



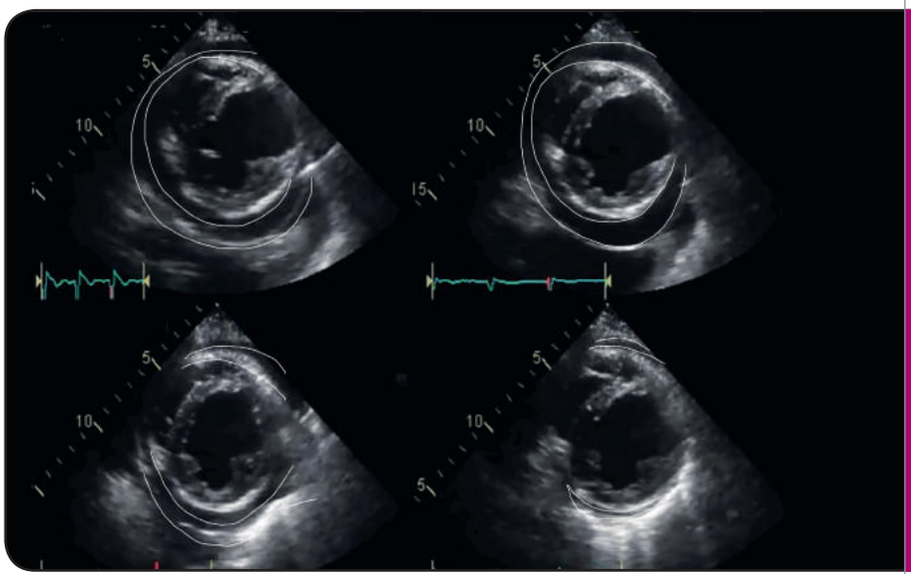
The separation of pericardial layers can be detected on echocardiography, when pericardial fluid exceeds 15–35 ml.

Follow-up of pericardial effusion requires using the same views. Always measure in the same region and also assess pericardial efussion visually.

Quantification of Circumferential Pericardial Effusion

Small	< 1 mm	300 ml
Moderate	10–20 mm	500–700 ml
Large	> 20 mm	> 700 ml
Very large	> 30 mm + compression	

PERICARDIAL EFFUSION



NOTES

SEQUENTIAL IMAGES OF PERICARDIAL EFFUSION – PSAX/2D

Changes in the size of a pericardial effusion can be best appreciated by recording similar images and displaying them in split-screen format. The effusion in this patient clearly diminishes over time.

Quantification of Volume

Subtract the volume derived by tracing the cardiac contour from the volume derived by tracing the epicardial contour (+ pericardial effusion). The difference is the volume of the pericardial effusion.

Volume quantification is best performed from a subcostal view.

Importance of Echo in Pericardial Effusion

- Establish the diagnosis
- Help to find its cause?
- Hemodynamic importance
- Direct pericardiocentesis

Always look for other echo features which may reveal the cause of effusion (e.g. myocardial infarction, pulmonary hypertension, endo-myocarditis).

PERICARDIAL TAMPONADE

Definitions

Tamponade: Intrapericardial fluid
Constriction: "Stiff" pericardial sac
Effusive constricitive: "Stiff" pericardial sac + fluid

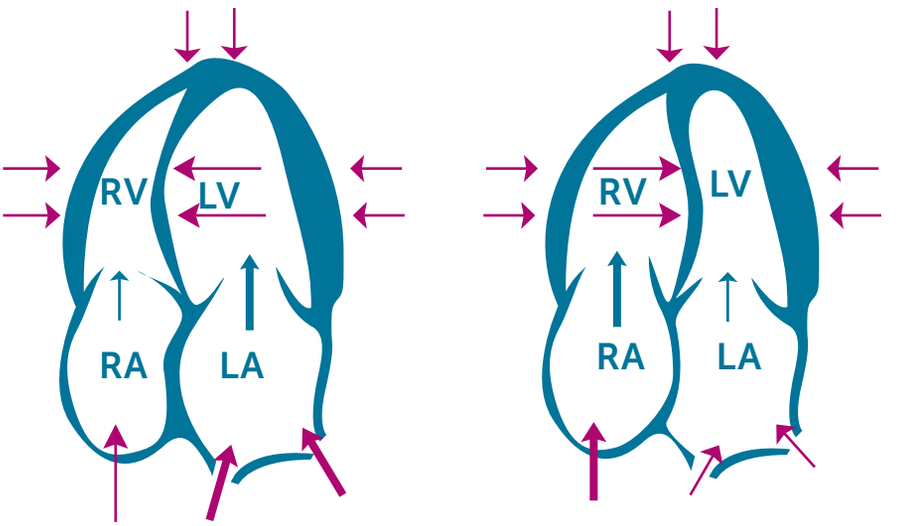
Tamponade, constriction and effusive constriction share many common features.
Tamponade is a medical emergency, and occurs when fluid accumulates rapidly.

NOTES

PERICARDIAL TAMPONADE

Use a respiratory curve while imaging the patient to determine the phase of inspiration and expiration.

Pathophysiology of Tamponade – Interventricular Interdependence



Tamponade – expiration **Tamponade – inspiration**

In tamponade, systemic venous return is shifted towards inspiration. The heart is unable to adapt to the increase in volume of the right heart during diastole, especially during inspiration. To accomodate the volume, the septum shifts to the left (septal shift) during inspiration.

Echocardiography is important for the diagnosis of tamponade, but a tamponade is also a clinical diagnosis.

Hallmarks of Tamponade

- Systemic venous return shifted to inspiration
- Impaired filling of the left ventricle during inspiration
- Interventricular interdependence

Symptoms	Signs
Pain	Tachycardia
Dyspnea	Edema
Shock	Low blood pressure

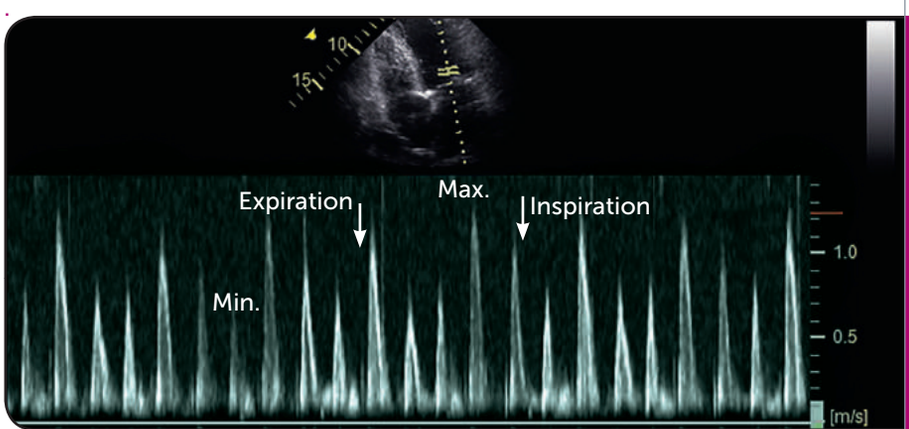
Triggers of Tamponade in Chronic Pericardial Effusion

- Hypovolemia – low pressure tamponade
- Paroxysmal tachyarrhythmia
- Intercurrent pericarditis

PERICARDIAL TAMPONADE

Echo Signs of Tamponade

- Right atrial collapse (early sign, alone usually does not denote relevant tamponade)
- Dilated inferior vena cava and hepatic veins
- Right ventricular collapse (difficult to assess in swinging heart due to out of plane motion of the right ventricle, but if present usually associated with symptoms)
- Left ventricular collapse (severe tamponade, emergent pericardiocentisis required)
- Swinging heart phenomenon (usually associated with some degree of hemodynamic relevance of effusion)
- Septal shift towards the left ventricle during inspiration (indicator of hemodynamic significance)
- Respiratory changes in PW Doppler mitral valve inflow (Changes > 30% are indicative for hemodynamic significance), Apply with caution in atrial fibrillation
- Exaggerated respiratory changes in tricuspid valve inflow (PW Doppler)
- PW Doppler flow reversal in hepatic veins



VARIATIONS OF MITRAL VALVE INFLOW– apical four-chamber view/PW Doppler

Respiratory variations (>25%) of the mitral inflow in pericardial tamponade. Inflow velocities are less during the first beat following inspiration.

PERICARDIAL CONSTRICTION

Pericardial Constriction – Characteristics

- Pericardial calcification/fibrosis/scarring
- Subacute/chronic disease
- Normal systolic function
- Impaired filling
- Venous distention
- Edema
- Hepatomegaly
- Ascites

Patients with radiation-associated constriction have a poor prognosis.

Causes of Pericardial Constriction

- Inflammation (bacterial/tuberculosis)
- Radiation
- After cardiac surgery
- Connective tissue disease
- Idiopathic

Constriction may be local, but in most cases it causes impairment of biventricular filling.

NOTES

Use multiple views to assess septal shift and use respiratory curves.

Tamponade is often a "stagewise" process. It may occur gradually.

NOTES

PERICARDIAL CONSTRICTION

Types of Constriction

- Annular form
- Left-sided form
- Right-sided form
- Global form + myocardial atrophy
- Global form + perimyocardial fibrosis
- Restrictive cardiomyopathy

To confirm constriction, it is sometimes necessary to use hemodynamic catheter studies (dip and plateau pressure drop between the left ventricle and right ventricle during inspiration).

The size of the right ventricle increases in the phase of septal shift.
In our experience, the easiest and best way to diagnose constriction is by displaying inspiratory septal shift and septal bounce. This can be done in any view that depicts the interventricular septum.

Echo Features of Pericardial Constriction

- Dilated inferior vena cava and hepatic veins
- Predominant forward flow in early diastole (pronounced E-wave) (PW Doppler)
- Exaggerated trans-tricuspid flow during inspiration (PW Doppler)
- Expiratory flow reversal in hepatic veins (PW Doppler)
- Septal bounce (oscillating septum)
- Septal shift (pronounced shift of the intervenricular septum towards the left ventricle during inspiration)
- Distorted heart contour, especially in regional forms of constriction
- Poor image quality
- Echogenic pericardium
- Rather small ventricle/atria
- Pleural effusion

OTHER DISEASES OF THE PERICARDIUM

Pericardial cysts may be quite large and are often first suspected on a chest X-ray.

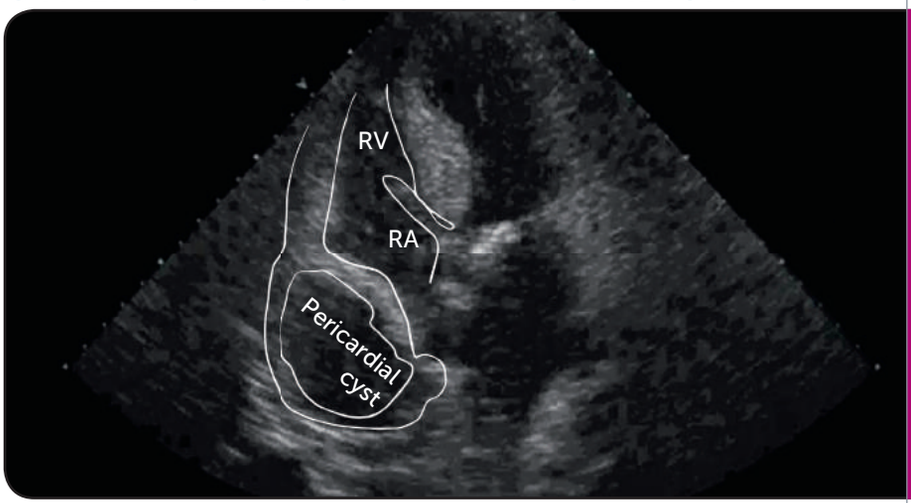
Pericardial Cyst

Benign tumor: 6% of mediastinal masses and 33% of mediastinal cysts

Failure of fusion of mesenchymal lacunae that form the pericardial sac

- Usually asymptomatic
- Unilocular/multilocular
- Typically located at the right cardiophrenic angle

OTHER DISEASES OF THE PERICARDIUM



NOTES

PERICARDIAL CYST –
apical four- chamber view/2D

Incidental finding of a large pericardial cyst located in the right cardiophrenic angle.

Differential Diagnosis: Pericardial Cyst

- Localized pericardial effusion
- Hepatic/renal/mediastinal cyst
- Echinococcal cyst
- Diaphragmatic hernia
- Atrial diverticula
- Aneurysmatic vessels

Malignant Disease of the Pericardium

- Primary malignancy
- Metastasis
- Pericardial carcinosis
- Pericardial involvment is associated with pericardial effusion (hallmark)

Symptomatic pericardial effusion in malignancy has a poor prognosis (median survival, 4 months).
Even in patients with a malignancy and pericardial effusion, the former is not always related to the latter.

Congenital Absence of the Pericardium

- 1/10.000 autopsies
- Various forms (total/left/right absence of the pericardium)
- Often asymptomatic or chest pain
- Higher risk of traumatic dissection
- Potential complications include herniation or entrapment of cardiac chambers (e.g. left atrial appendages)

Consider the absence of the pericardium in patients with unusually shaped ventricles with abnormal contractile motion.

Echo Features of Congenital Absence of the Pericardium

- Displacement of the heart
- Excessive cardiac motion
- Abnormal septal motion
- Enlargement of the left atrial appendage

Use MRI or CT to confirm the diagnosis.

NOTES

019 // Tumors and Masses

CONTENTS

NOTES

If you have the opportunity, attend an autopsy and see what these structures really look like.

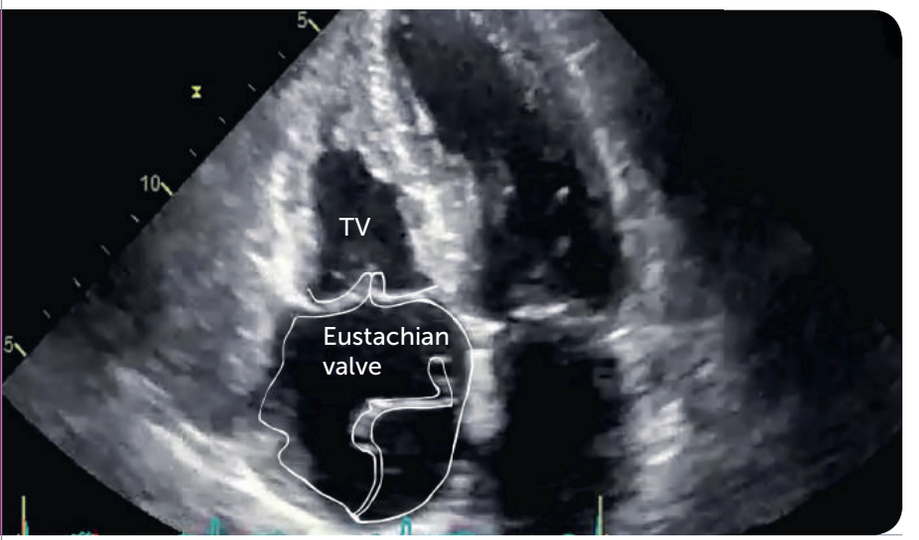
PSEUDOTUMOURS (STRUCTURES THAT MIMIC A MASS)

Pseudotumors of the Right Atrium

- Pectinate muscles
- Eustachian valve
- Chiari network
- Crista terminalis
- Lipomatous hypertrophy of interatrial septum (dumbbell sign)
- Prominent (lipomatous) tricuspid valve annulus
- Catheters/pacemakers
- PFO/ASD occluders

EUSTACHIAN VALVE – zoomed apical four-chamber view/2D

Very prominent and long Eustachiian valve in the right atrium. The Eustachian valve typically arises from the inferior vena cava.

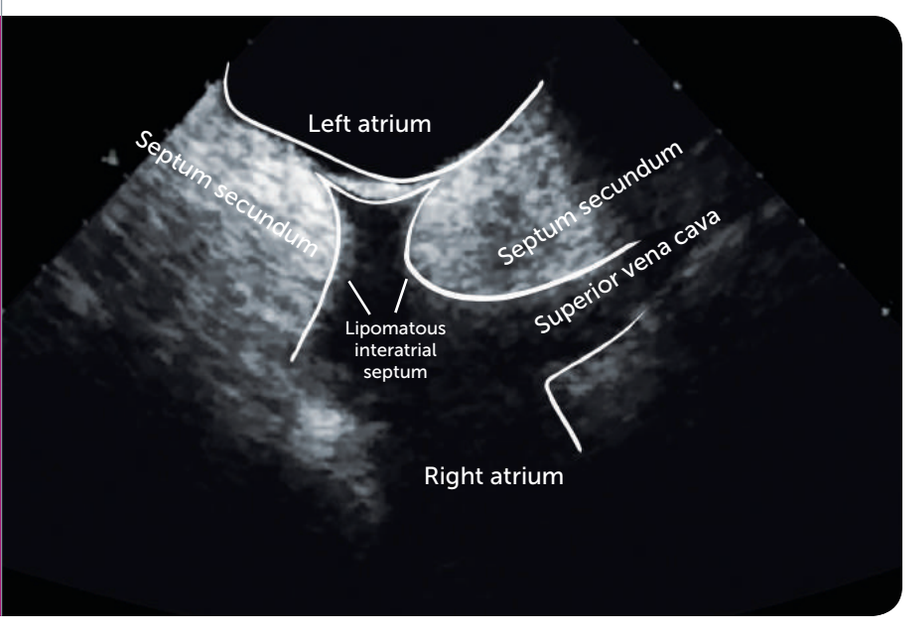


Structures of the Left Atrium

- Pectinate muscles
- Lipomatous hypertrophy of interatrial septum
- PFO/ASD occluders
- Calcified mitral annulus
- Ridge between the left superior pulmonary vein and the left atrial appendage

LIPOMATOUS INTERATRIAL SEPTUM – TEE bicaval view/2D

A lipomatous interatrial septum is best seen with TEE. The fossa ovalis is typically spared, resulting in a "dumbbell".



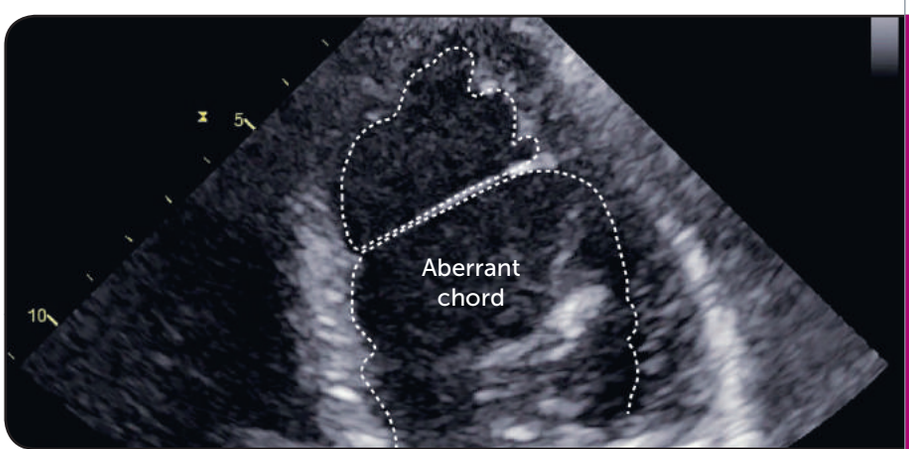
PSEUDOTUMOURS

Pseudotumors of the Right Ventricle

- Catheters (ICU)
- Pacemakers
- Muscle bundles
- Trabeculations
- Moderator band

Pseudotumors of the Left Ventricle

- Abberant/artifical chords
- Trabeculations
- Papillary muscles



MASSES

Distinguish between

	Thrombi	Tumors	Endocarditis
Fever/infection			X
Located on native valves		X	X
Embolism	X	(X)	X
Expansive growth located in > 1 chamber		X	
Spontaneous contrast	x		

Combine clinical and morphological clues to determine the etiology of the mass.

NOTES

These structures can also be visualized from subcostal views – use them.

Elongation of chords may be mistaken for vegetations. They may also mimic systolic anterior motion and falsely suggest the presence of hypertrophic obstructive cardiomyopyopathy.

ABBERANT CHORD –
apical four- chamber view/2D

Abberant chord that traverses the left ventricle from the septum to the lateral wall.

NOTES

MASSES

Mural thrombi have an overall incidence of 20%. In large infarctions with aneurysms the incidence is as high as 60%. The risk of systemic embolization is 2%.

Risk Factors for Thrombus Formation

Atria

- Atrial fibrillation
- Mitral valve replacement
- Mitral stenosis
- Reduced left ventricular function

Left ventricle

- Reduced left ventricular function
- Aneurysm (apex)
- Acute myocardial infarction
- First week after STEMI

The appearance of thrombi may vary greatly, ranging from fibrotic/solid/high echogenicity to soft/jelly-like/low echogenicity.

Echocardiographic Aspects of Thrombus

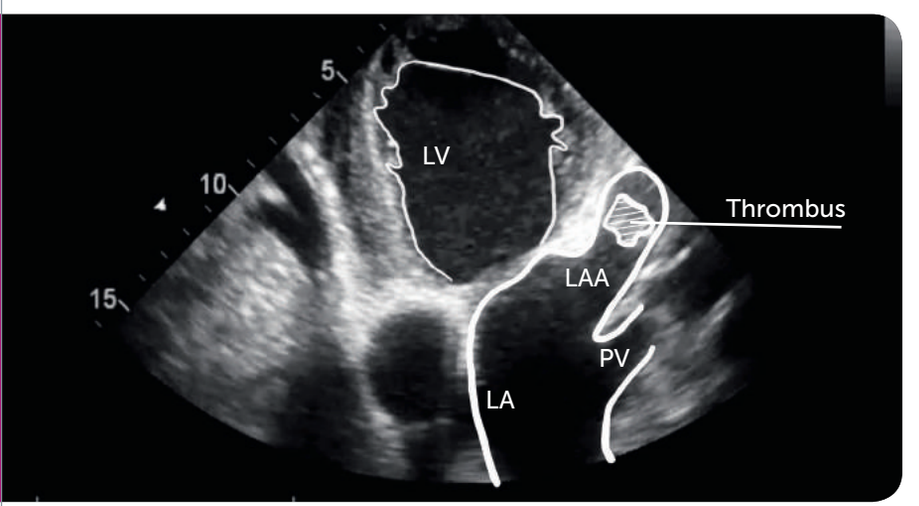
- Size
- Echogenicity (fresh vs. old)
- Mobility
- Location

Always describe these aspects of a thrombus for better comparison over time.

Tumors of the Heart

THROMBUS IN LEFT ATRIAL APPENDAGE/atypical apical four-/two-chamber view/2D

This rare example shows that it may be possible to detect left atrial appendage thrombi with transthoracic echo, especially when using atypical views.



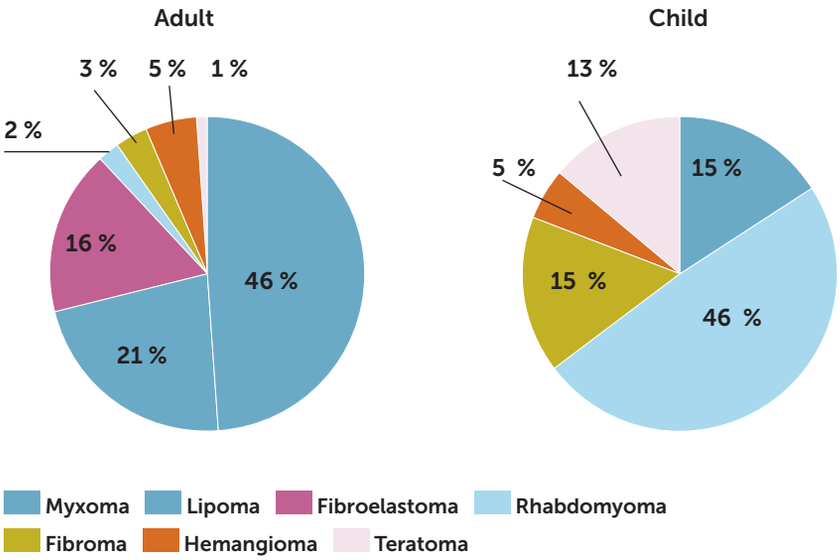
Metastatic lesions of the heart are almost 20 times more common than primary cardiac tumors.

Common Sources of Metastatic Lesions

- Melanoma
- Soft tissue sarcoma
- Thyroid cancer
- Lung cancer
- Breast cancer
- Esophageal cancer
- Renal carcinoma
- Hepatocellular carcinoma
- Secondary involvement with leukemia and lymphoma

MASSES

Benign Primary Cardiac Tumors



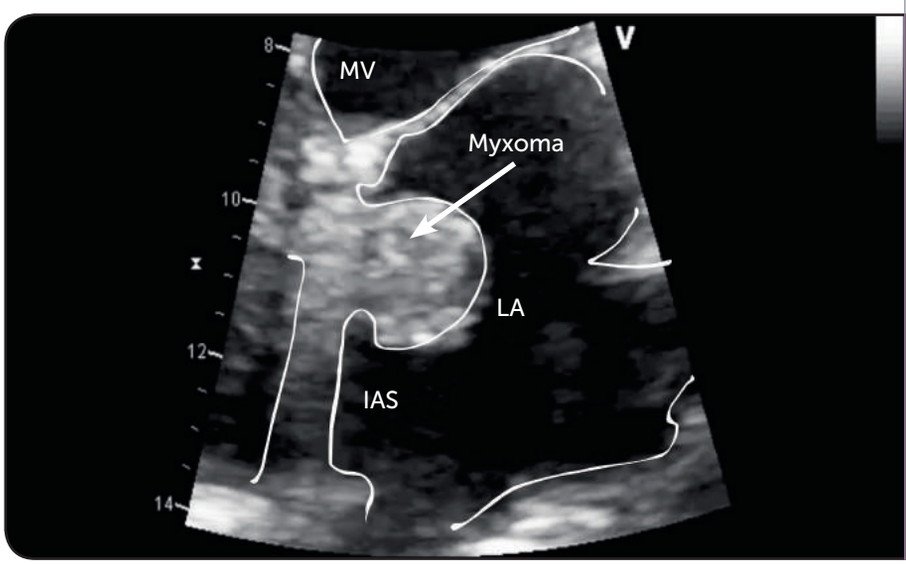
NOTES

About 75% of all primary cardiac tumors are benign.

Myxoma – Echo Facts

- More common in the left atrium than the right atrium (typically located at the fossa ovalis of the interatrial septum)
- Less common in other heart chambers or on valves
- Myxomas are typically pedunculated (often short stalk), either round/oval with a smooth surface, or villous in appearance
- Large myoxomas may cause valvular obstruction
- Systemic embolism or microembolism may occur

Given a typical presentation, the echo study is virtually diagnostic. If uncertain perform TEE or MRI.



MYXOMA – zoomed apical four- chamber view/2D

A typical myxoma originating from the interatrial septum. Its surface is rather smooth, it has a very short stalk and is homogeneous. Myxomas may be much larger, filiform, and more inhomogeneous.

NOTES

MASSES

Lipoma

Do not confuse a lipoma with lipomatous hypertrophy of the interatrial septum.

- Second most common benign cardiac tumor
- Common locations: LV, RA, IAS
- May be found in the intramyocardial region
- CT & MRI: high specificity for fat

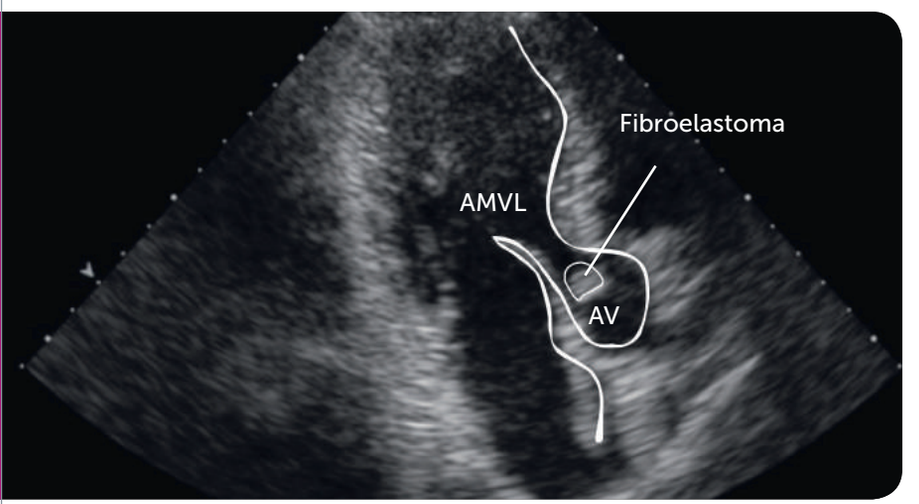
Papillary Fibroelastoma

When valvular dysfunction is present, think of endocarditis rather than papillary fibroelastoma.

- Most frequently located on the aortic valve, followed by the mitral valve
- Its mobility predicts the risk of embolism
- May cause coronary occlusion (rare)
- Rarely causes valvular dysfunction (DD: endocarditis)
- Usually located on the downstream side of the valve

FIBROELASTOMA (AORTIC VALVE) – apical three-chamber view/2D

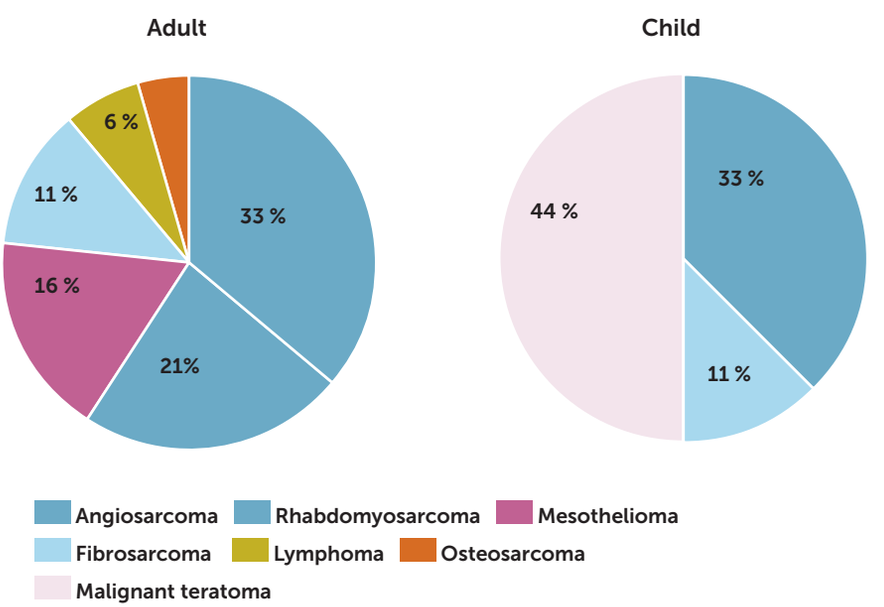
Small mass on the ventricular aspect of the aortic valve, which was histologically proven to be a fibroelastoma. Fibroelastomas may also appear as pedunculated or berry-like structures.



Malignant Cardiac Tumors

Various percentages have been reported. Some authors claim that up to 95% of malignant primary cardiac tumors are sarcomas. Irrespective of the true underlying number, sarcomas are certainly the most common malignant primary neoplasms in adults.

If a tumor involves the wall of more than one chamber, it is usually malignant (invasive growth). Malignant tumors are frequently associated with pericardial effusion.



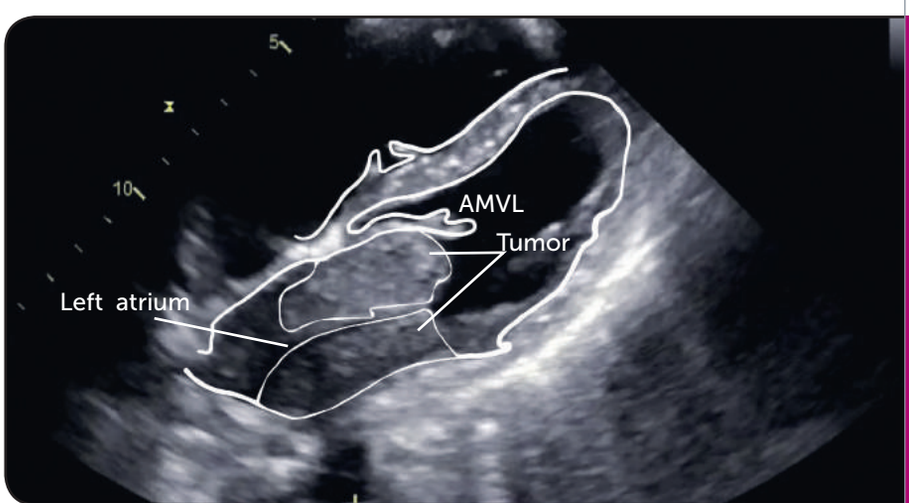
MASSES

Imaging Tips for the Evaluation of Masses

- Use atypical views focusing on the mass
- Do not be too focused on the tumor – perform a complete exam
- Use different gain settings. Intramyocardial tumors are sometimes difficult to see.
- Use color Doppler. It may help to tell whether the tumor is vascularized and whether there is flow within the tumor.
- Use echo contrast. It helps to delineate the tumor and determine whether the tumor is vascularized.
- Do not forget to point the transducer to the liver, the inferior vena cava, and the pleura.

Complications of Malignant Tumors

- Local compression
- Obstruction
- Pericardial effusion with tamponade
- Spread to surrounding structures
- Arrhythmias
- Valvular dysfunction



MALIGNANT MASS (RHABDOMYOSARCOMA) – atypical apical four-chamber view/2D

Tumor masses in the left atrium. The structure of the tumor is inhomogeneous and it is causing inflow obstruction into the left ventricle.

Consequences/Therapeutic Options

- If you are not certain whether it is a tumor, perform other imaging modalities (i.e. TEE, MRI, CT) and perform follow-up exams.
- In benign tumors, consider surgical removal when the tumor is in the left heart. LV tumors are subject to a high risk of embolization (e.g. fibroelastoma).
- If it is a thrombus., anticoagulate and repeat study. It should become smaller.
- If it is a malignant tumor, determine what it is (biopsy of primary tumor, pericardial tap, lab., etc.) Some tumors respond well to treatment with radiation or chemotherapy (such as lymphoma).

NOTES

Malignant tumors of the right atrium tend to grow along the interatrial septum. Look closely at this structure when you see a mass in the right atrium.

To determine changes in size of a tumour/mass or thrombus compare images side by side. This is often more reliable than comparing measurements.

Small and very mobile masses are difficult to see on MRI. Echo is superior because its frame rate is much higher.

NOTES

020 // Congenital Heart Disease

CONTENTS

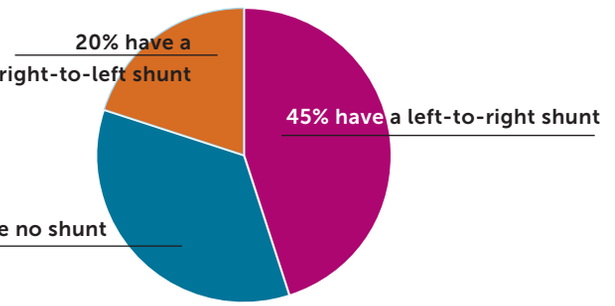
NOTES

20% of all congenital defects are atrial septal defects.

BASICS

Prevalence (Adults)

- Complex jet lesions: 418 per million



ATRIAL SEPTAL DEFECTS (ASD)

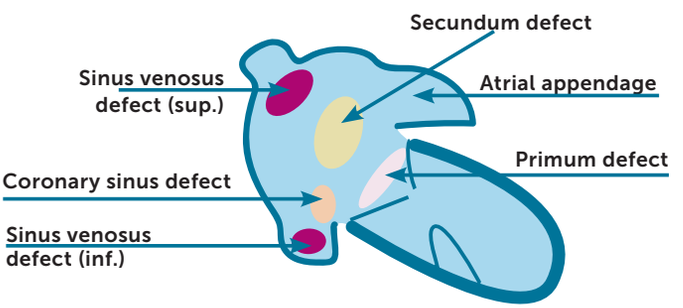
Severe pulmonary hypertension is rare in the setting of isolated atrial septal defects.

Hemodynamics of Atrial Septal Defects

- Right ventricular volume overload
- Pulmonary hypertension
- Potential for paradoxical embolism
- Reduced compliance of the left ventricle

75% of all atrial septal defects are secundum defects.

Types of Atrial Septal Defects



Patients with a primum ASD tend to have left axis deviation and a long PQ interval on the ECG, whereas patients with a secundum ASD have right axis deviation and RBBB.

A patent foramen ovale and a secundum ASD (ASD II) are not the same entitiy. A patent foramen ovale is a shunt across a "channel" (between a septum primum and secundum) while an ASD II is a hole in the septum.

It is possible to have both, an ASD and a PFO.

Associated Lesions

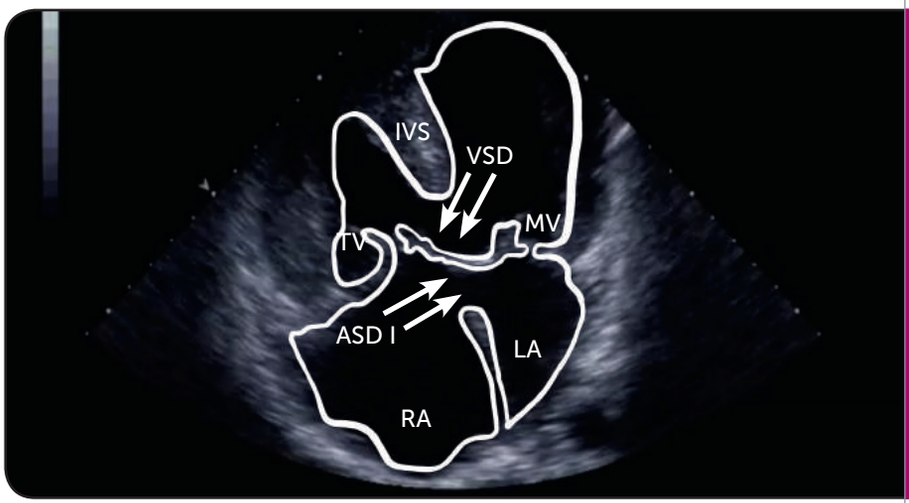
ASD I (primum defect)
- Cleft mitral valve (always)
- Inlet ventricular septal defect
- Septal aneurysms

ASD II (secundum defect)
- Mitral valve prolaps
- Pulmonic stenosis
- Partial anomalous venous return

Sinus venosus defect
- Partial anomalous venous return
- Overriding superior vena cava

Coronary sinus septal defect
- Unroofed coronary sinus
- Left superior vena cava persistence
- Partial/total anomalous venous return

ATRIAL SEPTAL DEFECT (ASD)



NOTES

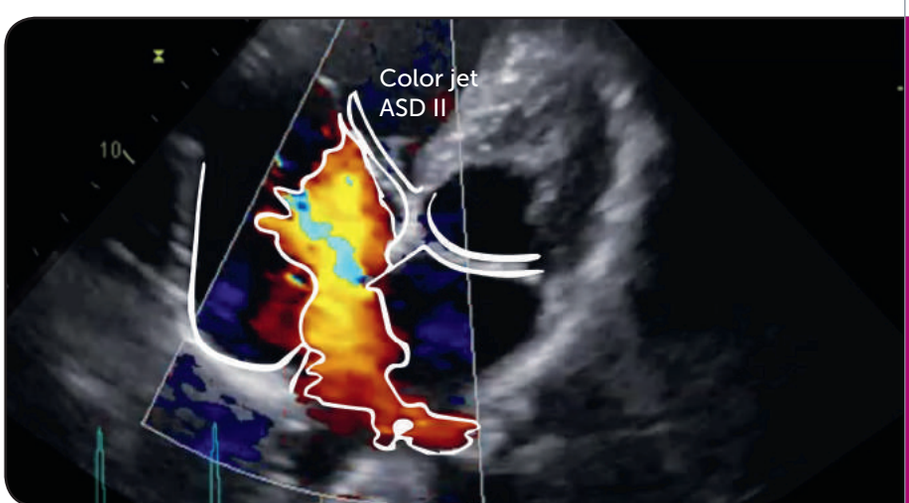
COMPLETE ATRIOVENTRICULAR CANAL DEFECT – apical four-chamber view/2D

Improperly formed atrioventricular valve (shared atrioventricular valve). Both an ASD (primum type) and a VSD are present.

Views to Detect an ASD

- Slanted four-chamber view
- Parasternal SAX view
- Subcostal views
- Not all ASD's can be detected with TTE

Transesophageal echocardiography (TEE) is superior in quantifying the size and morphology of an ASD (two orthogonal planes). TEE is also required to diagnose a sinus venosus defect.



SECUNDUM ATRIAL SEPTAL DEFECT – slanted apical four-chamber view/color Doppler

Moving the transducer medially allows more parallel alignment to the Doppler and therefore better visualization of the ASD jet.

Difficulties in Detecting Shunts

- Poor image quality
- Suboptimal angle to shunt flow
- Low flow velocity
- Inferior vena cava inflow may mimic ASD
- Tricuspid regurgitation may obscure the ASD signal during systole
- Shunt flow depends on left and right ventricular compliance
- Elevated right heart pressure may reduce left-to-right shunt

The intertrial septum may show dropouts that mimic an ASD.

NOTES

ATRIAL SEPTAL DEFECT (ASD)

When to Suspect an ASD:

An ASD must be excluded in every patient with an enlarged RV.

The absence of a color jet across the IAS and even a negative contrast study do not entirely rule out an ASD. It could be a sinus venosus defect and it may be possible that, despite an ASD, there is only a left-right shunt (negative contrast study).

- Enlarged right ventricle
- Dilated pulmonary artery
- Positive contrast study
- Abnormal septal morphology (aneurysm, discontinuity of the interatrial septum, etc.)
- Elevated flow in the pulmonary artery (VTI >25 cm)
- Patient history (arrhythmias, dyspnea, atrial fibrillation + ECG + right ventricle enlargement)

Quantification of Atrial Septal Defects

The size of the ASD is quantified with a balloon during intervention. This "stretched size" of the ASD is relevant for device sizing.

Large	> 10 mm
Small	5–10 mm
No relevant shunt	< 5 mm

A warning note: Even small defects can generate significant left-to-right shunts when the gradient between the left and the right atrium is high.

Quantification of Shunt Flow

The measurement of LVOT/PA diameter is most critical for shunt calculation (measurement errors may have grave consequences).

$$Qp/Qs = \frac{Flow_{pulm} = (PA\ diameter/2)^2 \cdot \pi \cdot VTI_{PA/RVOT}}{Flow_{system} = (LVOT\ diameter/2)^2 \cdot \pi \cdot VTI_{LVOT}}$$

PA = pulmonary artery, **RVOT=** right ventricular outflow tract, **LVOT=** left ventricular outflow tract, **VTI =** velocity time integral

Suitabilty for Interventional Closure

The guidelines recommend interventional closure in patients with a stretched diameter <38 mm and a sufficient rim > 5 mm towards the aorta.

ESC 2010

ASD closure must be avoided in patients with Eisenmenger (right-to-left shunt) syndrome (ESC Class III).

Indications for ASD closure (ESC Class I)

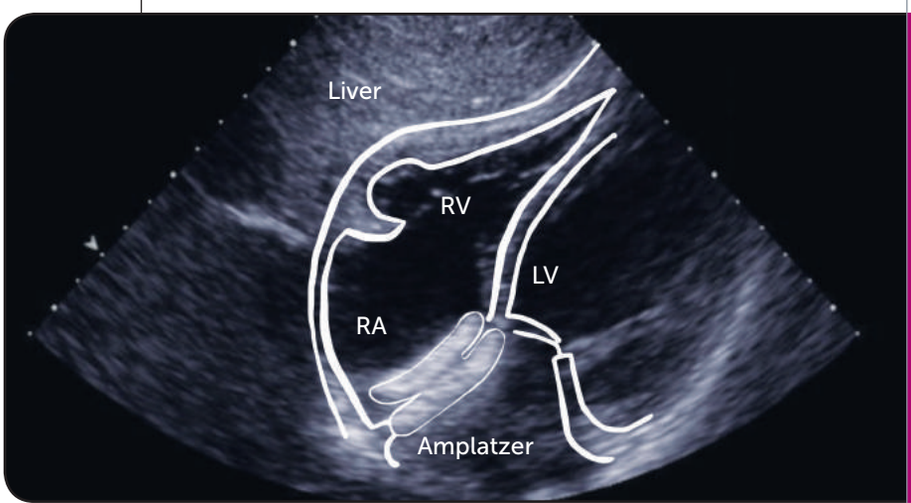
- Patients with significant shunts (signs of RV volume overload) and pulmonary vascular resistance < 5 Wood units, regardless of symptoms.
- Device closure is the method of choice for secundum ASD closure when applicable.

ESC 2010

ATRIAL SEPTAL DEFECT (ASD)

Suitabilty for Interventional Closure

Ideal	< 20 mm
Uncertain	20 – 25 mm
Too large	> 25 mm



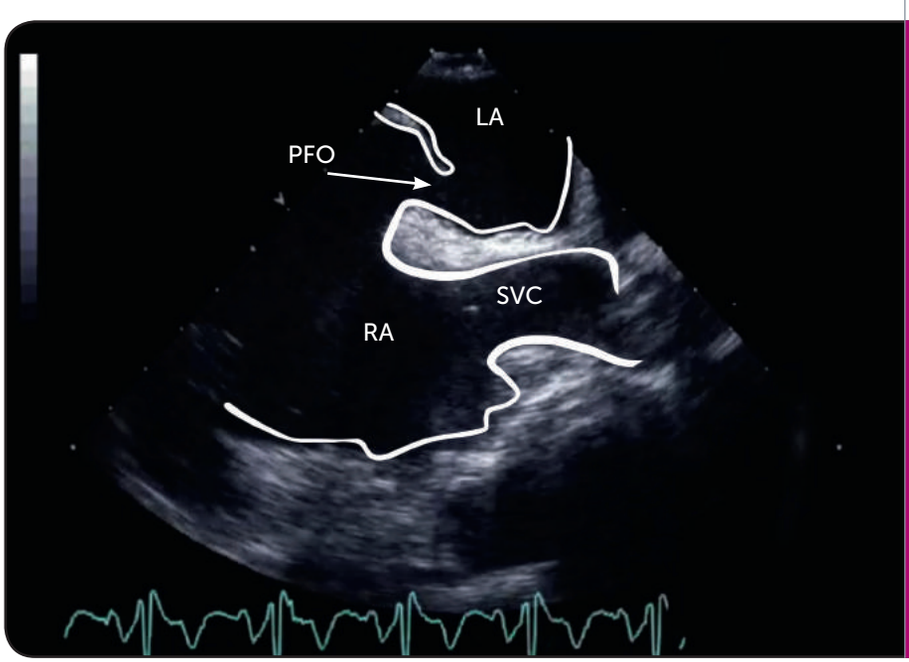
ASD OCCLUDER – subcostal four-chamber view/2D

The left and the right atrial disks of an Amplatzer occluder are visible. The interatrial septum is captured in between.

Echo Assessment following Interventional ASD Closure

- Look for a residual shunt using color Doppler (reduce PRF) and echo contrast
- Location and stability of the device
- Thrombus on the device
- Pericardial effusion

PATENT FORAMEN OVALE (PFO)



PATENT FORAMEN OVALE – TEE bicaval view/2D

Separation between the primum and the secundum septum forming a patent foramen ovale (PFO). The primum septum overlaps the secundum septum and the PFO is a channel rather than a hole.

NOTES

Intervention should be monitored with the help of echo (TEE, intracardiac ultrasound).

NOTES

PATENT FORAMEN OVALE (PFO)

Epidemiologic Facts

- 25% of the general population have a PFO.
- In patients with cryptogenic stroke the prevalence increases to 40%.

Perform a Valsalva maneuver when looking for PFO in the contrast study and reduce PRF for color Doppler assessment.

A negative transthoracic contrast study does not rule out a patent foramen ovale. You need a transesophageal exam.

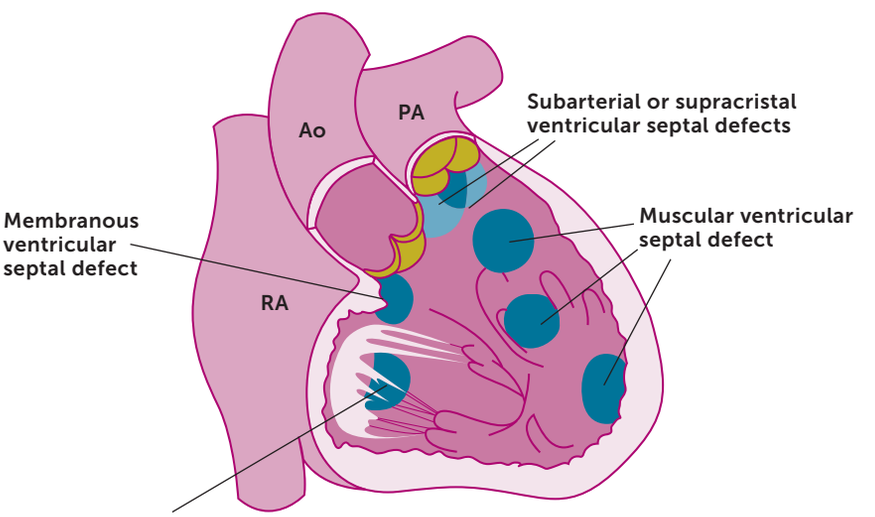
Echo Assessment of Patent Foramen Ovale

- Frequently associated with mobile and aneurysmatic interatrial septum
- Positive contrast study – contrast appearance in the left atrium within 3 heart cycles after opacification of the right atrium
- Small jet into the right atrium seen with color Doppler (usually close to the aortic rim)
- For color Doppler assessment, use a subcostal view or a slanted four-chamber view to improve Doppler alignment
- For contrast study use a four-chamber view

VENTRICULAR SEPTAL DEFECTS (VSD)

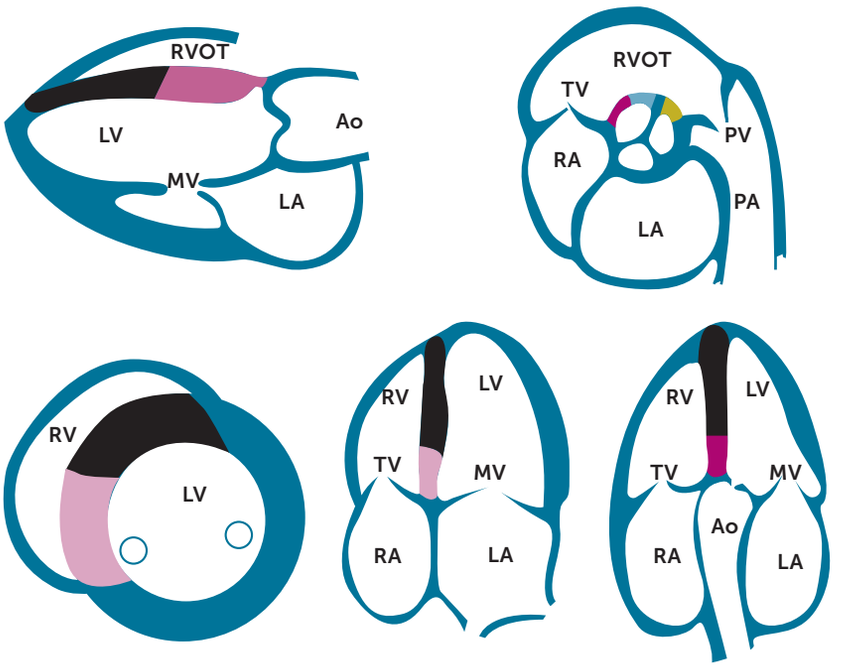
The prevalence of VSD is 10% of all congenital lesions of the heart in the adult population. Perimembranous VSD is the most common type.

Ventricular Septal Defect Types

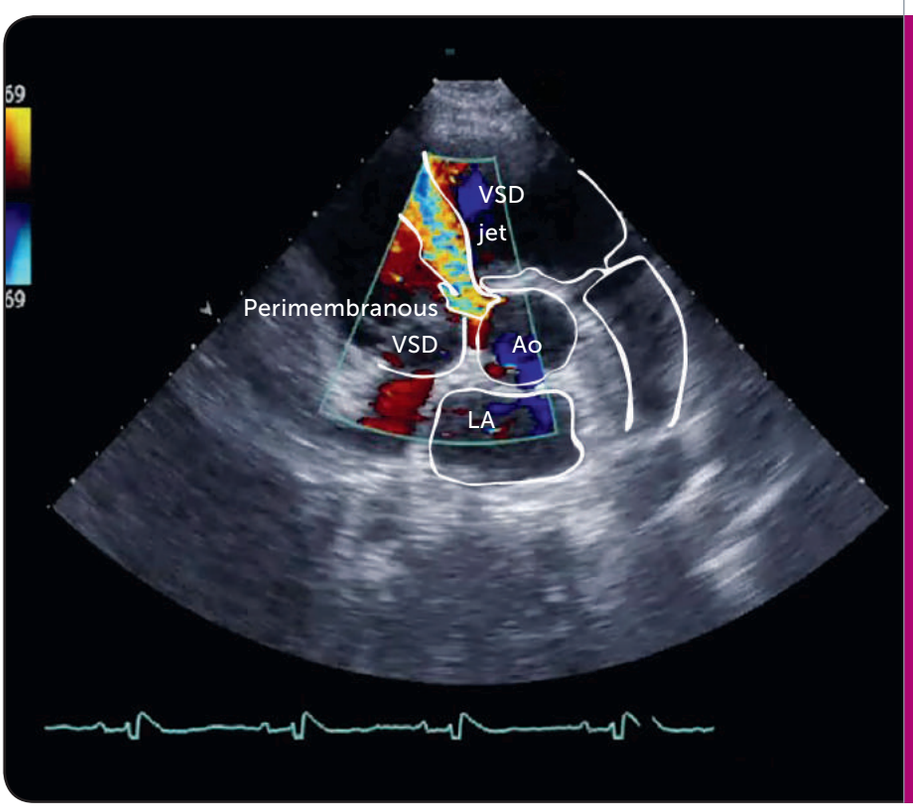


VENTRICULAR SEPTAL DEFECTS (VSD)

Views and Locations of the Various VSD Types



- Perimembranous
- Outlet infracristal
- Outlet supracristal
- Inlet
- Trabecular
- Perimembranous or Outlet



PERIMEMBRANOUS VENTRICULAR SEPTAL DEFECT – PSAX/ color Doppler

Typical jet origin and direction of a perimembranous VSD. The defect is located below the aortic valve. The jet is directed more towards right ventricular inflow.

NOTES

If you are not sure whether a VSD is present use the good old stethoscope!

NOTES

VENTRICULAR SEPTAL DEFECTS (VSD)

Contrast is not helpful in ventricular septal defects.

VSD Quantification

- Left ventricular volume overload
- Use atypical views to visualize the myocardial discontinuation
- Color Doppler detection of flow across the interventricular septum
- Restrictive VSD has a high velocity (> 4.5 m/sec) and occurs in small or medium-sized defects
- Non-restrictive VSDs have a low velocity (< 4.5 m/sec), indicating a large defect

Aneurysmal Transformation in VSD

- Partly or completely sealed VSD by fibrous tissue proliferation of the septal leaflet of the tricuspid valve
- Best visualized on a five-chamber view
- No risk of rupture

Interventional VSD closure is only possible in muscular VSD with a distance > 3mm from the aortic valve.

Associated Lesions

Membranous VSD	Supracristal VSD	Inlet VSD
Septal aneurysms	Aortic valve prolapse	ASD I
Subaortic stenosis		Cleft mitral valve
Double chambered RV		

PATENT DUCTUS ARTERIOSUS (PDA)

PDA is present in 2% of the adult population and is often associated with coarctation and VSD. Always suspect a PDA in the setting of a dilated hyperdynamic left ventricle in the absence of other forms of LV volume overload.

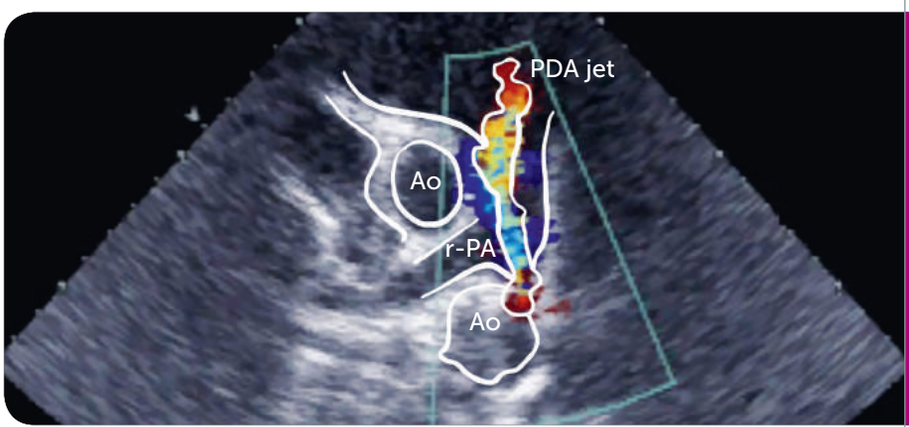
Hemodynamics of PDA – Different Presentations

- Variable, depending on size
- Left-to-right shunt
- Left ventricular volume overload
- Elevation of pulmonary artery pressure
- Eisenmenger reaction
- Hemodynamically insignificant (small)

PATENT DUCTUS ARTERIOSUS (PDA)

Visualization of the Patent Ductus Arteriosus

- Parasternal short axis (pulmonary artery bifurcation)
- Suprasternal view
- Systolic + diastolic flow in spectral and color Doppler
- Dilatation of the pulmonary artery is common
- 2D (suprasternal view) often allows measurement of PDA size



PATENT DUCTUS ARTERIOSUS – PSAX/Color Doppler

Shunt (color jet) between the aorta and the pulmonary artery at its bifurcation. The jet is present during systole as well as diastole.

CORONARY FISTULAS

Coronary Fistulas

- Abnormal communication between coronary artery and heart chamber
- 90% into right ventricle
- RV volume overload
- Coronary steal

Echo Features of Coronary Fistulas

- Dilated coronary artery (> 0.6 cm)
- Enlargement of heart chambers
- Turbulant flow
- Continous flow (shunt) to right heart

NOTES

Patients with high-velocity PDA jets are candidates for closure (exception: small asymptomatic PDA).

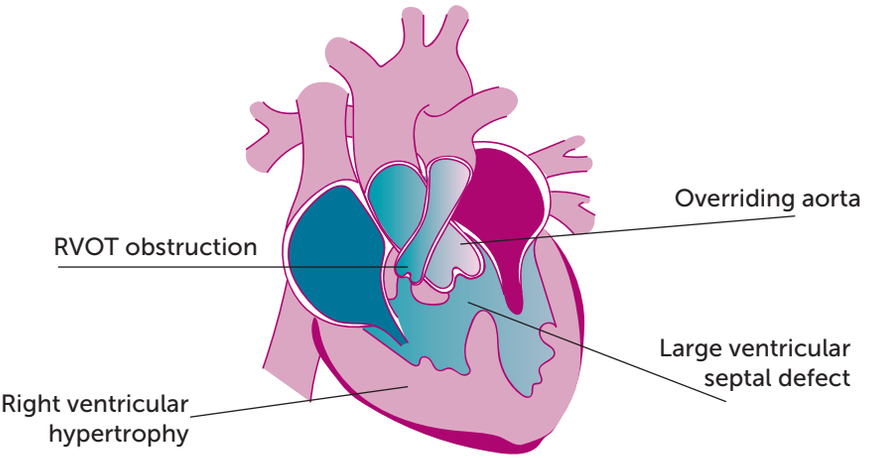
Coronary fistulas are found in 0.2% of coronary angiograms.

The hemodynamic presentation greatly depends on the degree of RV outflow obstruction. In the setting of a VSD with a left-to-right shunt, it may prevent pulmonary hypertension and eventually shunt reversal (right to left) and the Eisenmenger reaction.

NOTES

The hemodynamic presentation greatly depends on the degree of RV outflow obstruction. In the setting of a VSD with a left-to-right shunt, it may prevent pulmonary hypertension and eventually shunt reversal (right to left) and the Eisenmenger reaction.

TETRALOGY OF FALLOT



- Stenosis of the pulmonary artery (right ventricular outflow obstruction)
- Ventricular septal defect
- Deviation of the origin of the aorta to the right (overriding aorta)
- Concentric right ventricular hypertrophy

In patients with a more severe RVOT obstruction, PW and color Doppler will demonstrate a significant right-to-left shunt at the VSD. In patients with a large left-to-right shunt, left atrial and left ventricular dilatation will be present.

Right ventricular outflow obstruction tends to occur at multiple levels - infundibular, RVOT, often hypoplastic annulus valve abnormalities (bicuspid valve).

When assessing patients after Fallot repair, look for residual pulmonary regurgitation.

Echocardiographic Assessment in Fallot

Ventricular septal defect and overriding aorta

- Assess the characteristic and large VSD on multiple views and define the location and number of VSDs
- The degree of aortic override is best assessed on parasternal long-axis and apical views.
- The extension of the defect from the membranous septum is best seen in the parasternal short axis
- Assess the relationship between the defect and the tricuspid and aortic valve.

Right ventricular outflow tract obstruction

- Use parasternal short-axis views.
- Assess the infundibulum and pulmonary vale.
- Infundibular muscle bundles often contribute to the RVOT obstruction
- The pulmonary valve annulus is often hypoplastic (important information in regard of a transannular patch).
- The pulmonary valve tends to look thickened and may be dome-shaped.

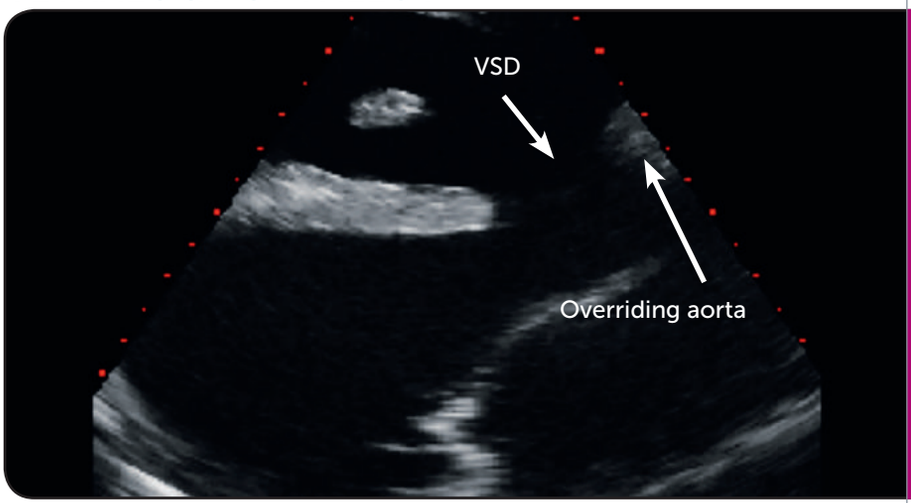
Hemodynamic assessment

- A large and generally unrestricted defect permits equalization of right and left ventricular pressures.
- The direction and degree of shunting strongly depend on the severity of right ventricular outflow tract obstruction.

Aortic arch and coronary arteries

- Use suprasternal views to investigate the aortic arch and exclude the presence of aortopulmonary collaterals and the presence of a patent ductus arteriosus.
- The anatomy of the proximal coronary arteries should be assessed using parasternal short-axis views
- Exclude a right aortic arch (present in 25%)

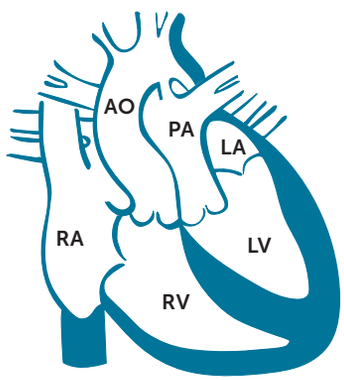
TETRALOGY OF FALLOT



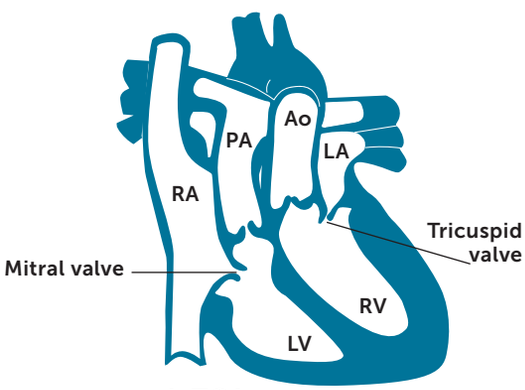
NOTES

TETRALOGY OF FALLOT – PLAX/2D
A patient with a tetralogy of Fallot, a large VSD and an overriding aorta.

TRANSPOSITION OF THE GREAT ARTERIES



D-TGA



L-TGA

In D-TGA a shunt on the atrial/ventricular/great vessels (PDA) is required to live, either present at birth or artificially created (e.g. Rashkind's procedure)

- Lesion in which the aorta arises from the right ventricle and the pulmonary artery from the left ventricle.
- Its prevalence is 4.7 per 10,000 live births.
- It is not associated with any common gene abnormality.
- The most common form is the dextro type (D-TGA), in which the aorta arises from the right ventricle and the pulmonary artery from the left ventricle (ventriculoarterial discordance).
- Levo- or L-looped transposition of the great arteries (L-TGA) is very rare and is commonly referred to as congenitally corrected TGA. Venous blood returns from the correctly located right atrium to the discordant left ventricle via the mitral valve and into the lung via the pulmonary artery. Oxygenated blood flows through the pulmonary veins to the left atrium into the discordant right ventricle, and via the tricuspid valve into the systemic circulation through the aorta (atrioventricular and ventriculoarterial discordance).
- The D-TGA leads to cyanotic heart disease while L-TGA usually does not present with cyanosis (unless the patient has associated cardiac defects).

Patients with L-TGA are at risk for (systemic) heart failure because the morphological right ventricle (which was not formed to sustain a high pressure system) supplies the systemic circulation.

NOTES

TRANSPOSITION OF THE GREAT ARTERIE

Cardiac Lesions Associated With D-TGA

- A ventricular septal defect in any region of the ventricular septum (50% of patients).
- Left ventricular outflow tract obstruction (25%)
- Abnormalities of the mitral and tricuspid valve, e.g. straddling tricuspid valve (septal chordal attachment of the tricuspid valve extending into the left ventricle), overriding valves.
- Coronary abnormalities

Echocardiographic Assessment in D-TGA

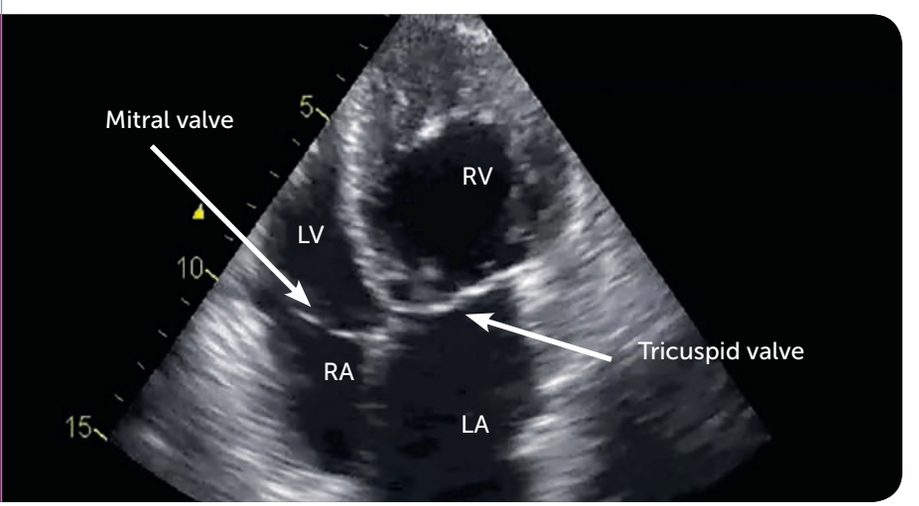
- Subcostal views show the pulmonary artery arising from the posterior left ventricle.
- Parasternal short-axis views show the aorta rising anteriorly from the right ventricle.
- Look for associated cardiac lesions.

Cardiac Lesions Associated With L-TGA

- Ventricular septal defect (70-80% of patients), most commonly perimembranous VSD.
- Pulmonary outflow (i.e. left ventricular) tract obstruction (30- 60% of patients). The obstruction is commonly subvalvular due to an aneurysm of the interventricular septum fibrous tissue tags or a discrete ring of subvalvular tissue.
- Tricuspid valve abnormalities (90% of patients) e.g. tricuspid valve regurgitation, Ebstein-like malformation of the tricuspid valve accompanied by right ventricular dysfunction and failure (20- 50% of patients).
- Mitral valve abnormalities (50% of patients) e.g. abnormal number of cusps, straddling chordal attachments of the subvalvular apparatus resulting in outflow tract obstruction, mitral valve dysplasia.

L-TGA –
Apical four-chamber view/2D

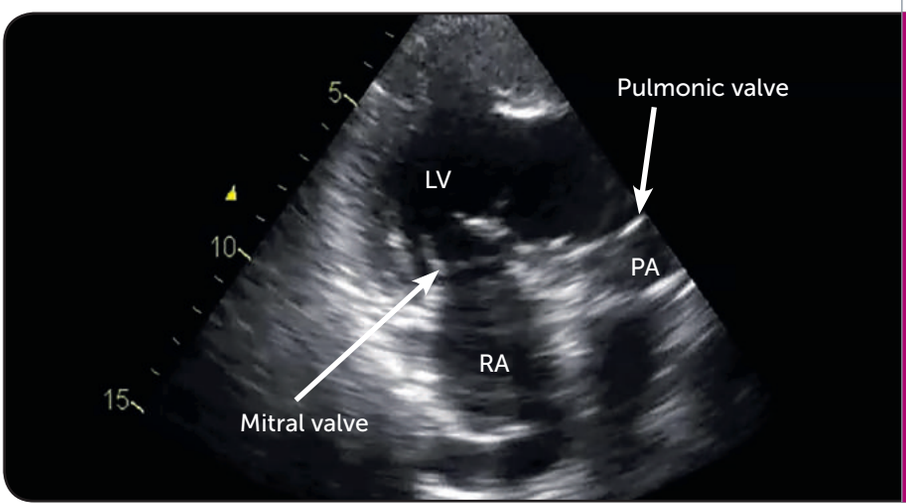
Since the tricuspid valve and the mitral valve are in opposite positions, the valve on the left side of the screen is more apical (lower in the screen) than the valve on the right. This is one of the key features that help to identify L-TGA. The right ventricle is in the position of the left ventricle. It can be identified because it is heavily trabeculated.



TRANSPOSITION OF THE GREAT ARTERIE

Echocardiographic Assessment in L-TGA

- Systemic location of the tricuspid valve and morphologic right ventricle. It is best seen on an apical four-chamber view or parasternal short-axis views.
- Subcostal imaging usually provides the clearest view of the pulmonary artery arising from the morphologic left ventricle.
- Look for associated cardiac lesions.



L-TGA – Atypical long-axis view, subpulmonic ventricle/2D

The subpulmonic ventricle, which is anatomically the left ventricle, ensures pulmonary circulation.

NOTES

The diagnosis of L-TGA is often missed at adult cardiac echo laboratories!

NOTES

021 // Stress Echocardiography

CONTENT

NOTES

Stress echo will help to improve your imaging skills.

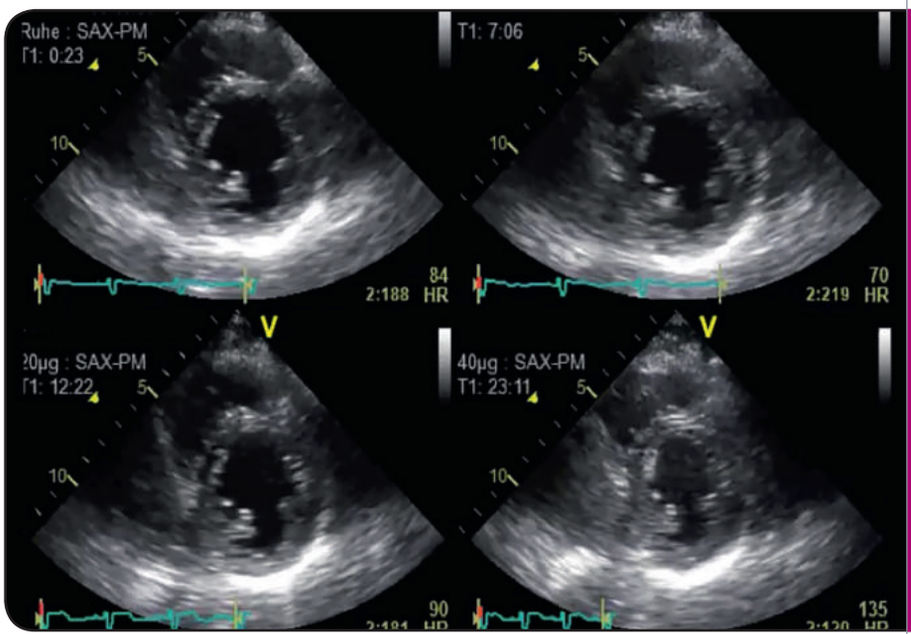
Stress echo for coronary artery disease is operator dependent. Therefore you need a lot of experience in imaging and interpreting wall motion abnormalities to obtain valid results.

INDICATIONS AND ECHOCARDIOGRAPHIC FEATURES

Indication	Clinical question	Echo features of specific interest
Ischemia	Does the patient have CAD?	New wall motion abnormalities
Viability	Are akinetic segments viable (hibernating myocardium)?	Improvement of contractility with low-dose dobutamine
Low-flow low-gradient AS	Is aortic stenosis severe?	An increase in gradients driven by an increase of contractility (cardiac output) under stress
Asymptomatic moderate/severe mitral regurgitation	Should mitral valve surgery be performed?	Increase of ejection fraction with stress or occurrence of symptoms (=adequate functional capacity)
Symptomatic moderate/mild mitral regurgitation	Is dynamic mitral regurgitation present?	Increase of mitral regurgitation severity increases during stress
Aortic regurgitation	Should aortic valve surgery be performed	Increase of ejection fraction with stress or occurrence of symptoms (=adequate functional capacity)
Mitral stenosis with unclear severity/ symptoms	Should valvuloplasty or mitral valve replacement be performed?	Excessive increase in transmitral gradients (>18 mmHg) or systolic pulmonary artery pressure (sPAP) (>60 mmHg) or occurrence of symptoms
Pulmonary arterial hypertension	Detection of early disease	Increase in sPAP with exercise*
Hypertrophic cardiomyopath	Is LVOT obstruction present? Indication for myectomy or septal ablation	Exercise tolerance and an increase of the gradient during stress > 50 mmHg
Dyspnea	Is dyspnea related to the heart (dilated cardiomyopathy, coronary artery disease)	Exercise tolerance, increase of ejection fraction during exercise, wall motion abnormalities suggesting coronary artery disease

*Stress echo is currently no diagnostic criterion for pulmonary hypertension

INDICATIONS AND ECHOCARDIOGRAPHIC FEATURES



NOTES

STRESS REACTION – PSAX Quad view/2D

Quad view comparing four different levels of dobutamine stress from baseline (left upper corner) to 40 mcg/kg/min (right lower corner). The global contractility of the left ventricle is increased (see moving image).

CLINICAL TARGETS OF STRESS ECHOCARDIOGRAPHY AND STRESS OF CHOICE

Clinical condition	Pathophysiologic target	Stress of choice	Echo variable
Coronary artery disease	Myocardial ischemia	Exercise, dobutamine, dipyridamole	Wall motion abnormalities
Dilated cardiomyopathy	Contractile reserve	Dobutamine (exercise, dipyridamole)	Wall motion abnormalities
Diabetes, hypertension, hypertrophic cardiomyopathy	Coronary flow reserve	Dipyridamole (dobutamine, exercise)	PW Doppler Left anterior descending (LAD)
Transmitral gradient	Increase in cardiac output	Exercise, dobutamine	PW/CW Doppler mitral valve
Transaortic gradient	Increase in cardiac output	Exercise, dobutamine	CW Doppler aortic valve
Pulmonary hypertension	Pulmonary congestion/ vasoconstriction	Exercise	CW Doppler tricuspid regurgitation

EAE Guidelines 2008

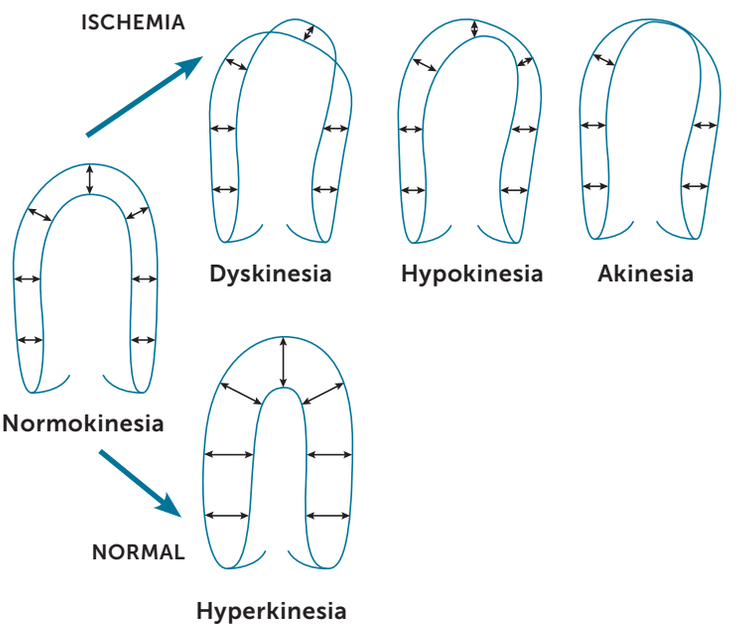
The choice of the stress test depends on the indication/relative contraindication, and the stress form your laboratory is most familiar with. (see cine loop at www.123sonography.com/echofacts)

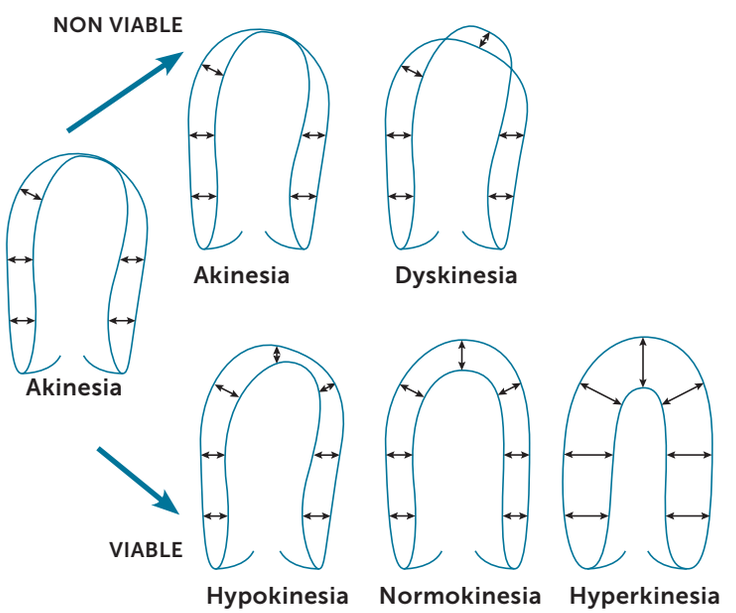
NOTES

STRESS ECHOCARDIOGRAPHY – AN EASY APPROACH

Rest	Stress	Myocardium
Normokinesia	Normo-, hyperkinesia	Normal
Normokinesia	Hypo-, A-, Dyskinesia	Ischemic
Akinesia	Hypo-, Normokinesia	Viable
A-, Dyskinesia	A-, Dyskinesia	Necrotic

EAE Guidelines 2008





STRESS ECHOCARDIOGRAPHY – AN EASY APPROACH

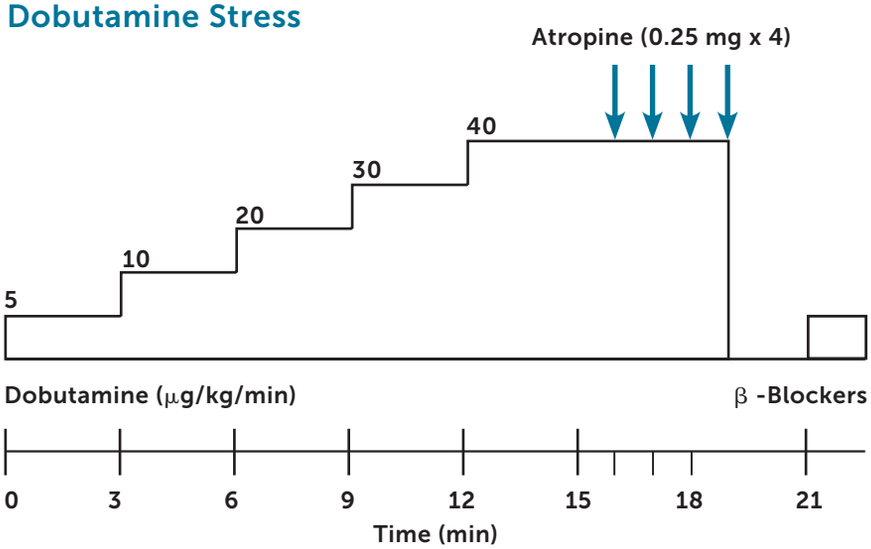
Forms of Stress

- Semisupine exercise
- Dobutamine (antidote: beta-blocker)
- Dipyridamole (antidote: aminophylline)
- Adenosine
- Pacing

Exercise Stress

- The patient pedals against an increasing workload at a constant cadence (60 rpm)
- Workload is escalated in a stepwise manner

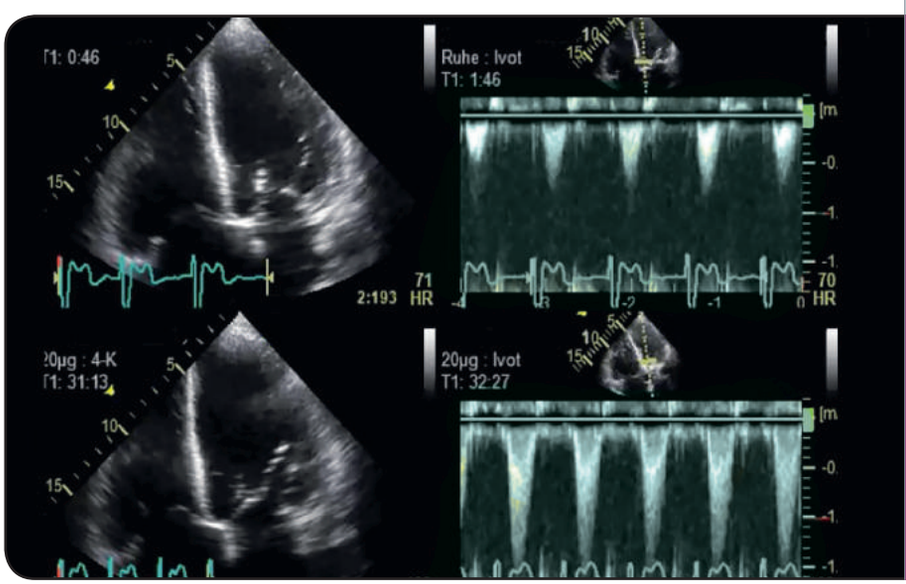
Dobutamine Stress



NOTES

It is easier to image patients when you use a pharmacological stressor. You will have less hyperventilation, tachycardia and chest motion. However, exercise is a more physiological stressor.

An ischemic response may occur late after stress – record images post stress!



VIABILITY TESTING – Apical four-chamber view/2D & PW Doppler

Patient with akinesia of the septum (top left) with low LVOT velocity at rest (top right), There is no change in the contractility of the septum during dobutamine stress (bottom left). The segment is not viable. The residual myocardium increases its contractility (non-ischemic). This is also reflected by the increase in LVOT velocity (bottom right).

NOTES

STRESS ECHOCARDIOGRAPHY – AN EASY APPROACH

Aminophylline (240mg intravenously) should be available for immediate use in case of dipyridamole-related adverse events.

Dipyridamole Stress

Infusion of 0.56 mg/kg dipyridamole over 4 minutes

↓

4 minutes of no dose

↓

0.28 mg/kg over 2 minutes (if the stress test is still negative)

↓

Atropine doses of 0.25 mg to maximum 1 mg (if the stress test is still negative)

Adenosine Stress

- Typically infused at a maximum dose of 140 mcg/kg/min over 6 minutes.

Pacing

- Pacing is started at 100 bpm and increased every 2 minutes by 10 bpm until the target heart rate (85% of age-predicted maximal heart rate) is achieved.

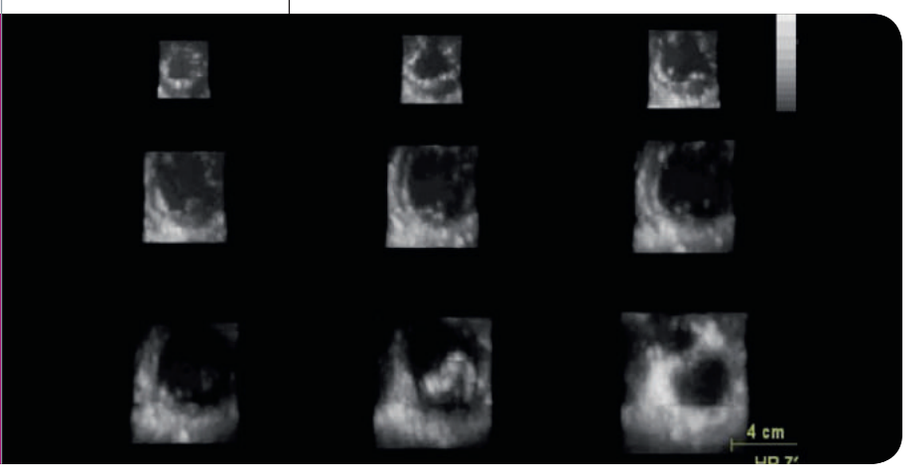
STRESS ECHO AND "OTHER ECHO MODALITIES"

Speckle tracking is a promising technique, especially for the assessment of contractile reserve in patients with valvular heart disease.

Speckle tracking	Assessment of longitudinal function during stress
Contrast	Improves the visibility of endocardial borders, simplifies the assessment of global and regional myocardial function
3D	Quicker data acquisition, multislice approach

3D RECONSTRUCTION–
short-axis views/3D

Live 3D Reconstruction of short-axis views for the analysis of contractility during stress echocardiography (see moving image)



ISCHEMIA TESTING

Facts

- Changes in wall motion (hypokinesia, akinesia) during stress strongly suggest significant coronary artery disease and are more accurate than ECG changes. They are also more specific than perfusion abnormalities.
- Worsening of wall motion in at least two adjacent segments is required for a positive outcome of the test.
- Clear endocardial delineation is crucial – use contrast to enhance the visibility of the endocardium.
- Use parasternal (long and short axis) as well as apical views (four- three-, and two chamber). Use atypical views if the image quality is better there.
- Make sure the images you record during stress testing closely correspond to the ones you recorded at baseline (use specific stress acquisition protocols that allow simultaneous review, such as split/quad screens).
- Blood pressure measurements (each stage) and 12-lead ECG (every minute) should be recorded.

Endpoints in Ischemia Testing

- Maximal dose or exercise level reached
- Achievement of target heart rate
- Obvious positivity of the test (echo and/or ECG)
- Severe chest pain/Severe dyspnea or other symptoms
- Hypertension (systolic hypertension ≥ 220 mmHg, diastolic hypertension ≥ 120 mmHg)
- Hypotension (drop by more than 40 mmHg)
- Occurrence of supraventricular arrhythmias (supraventricular tachycardia, new atrial fibrillation)
- Occurrence of ventricular arrhythmias (ventricular tachycardia, polymorphic premature ventricular beats)

Coronary Flow Reserve

- Transthoracic echo allows imaging of the left anterior descending (LAD) and posterior descending (PD) coronary arteries.
- With the help of coronary PW Doppler one can measure blood flow in the coronary arteries.
- By comparing the velocities with those obtained during pharmacologic stress (infusion of adenosine), it is possible to calculate coronary flow reserve.

NOTES

Acquire several RR intervals and be careful not to record image loops during ectopic beats.

Stress echo is a safe procedure. Nevertheless, you need to have a defibrillator/ advanced life support equipment in your lab!

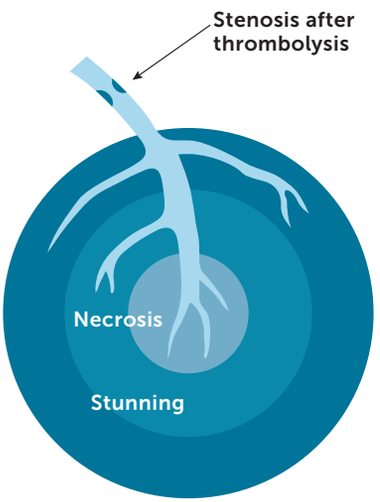
NOTES

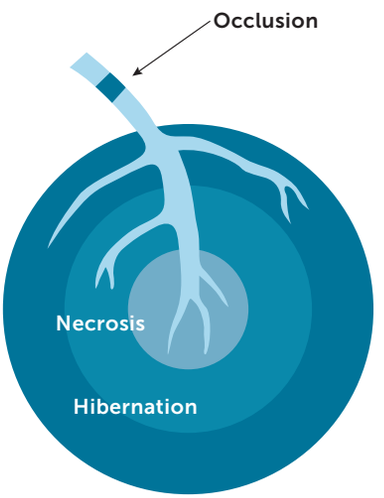
VIABILITY TESTING

Myocardial segments can be viable even when they do not contract (akinesia). This phenomenon occurs in the setting of stunning or hibernation.

Phenomena	Definition/Cause
Stunning	Segmental dysfunction which persists for a variable period of time - about two weeks - even after ischemia has been relieved
Hibernation	Abnormal contractility caused by inadequate blood supply (chronic stable angina, unstable angina, silent ischemia)

The function of viable segments may be restored when revascularization (PCI or CABG) is achieved.





Stunning: Reversible reduction of function of heart contraction after reperfusion

Hibernating: Downregulation of myocardial function to match chronic reduced blood flo

It is meaningless to look for viability in segments that are obviously scarred (thin, echodense). The dyskinesia that occurs in such segments during stress should not be mistaken for viability. It is a passive phenomenon related to the increase in intraventricular pressure.

Viability Response

- Sustained improvement during stress
- Biphasic response (improvement at low-dose dobutamine, deterioration at peak levels)

STRESS ECHO IN LOW-FLOW LOW-GRADIENT SEVERE AORTIC STENOSIS

NOTES

Low-Flow Low-Gradient Severe AS - Definition

Severe aortic stenosis in the setting of reduced left ventricular function and a valve area ≤1.0 cm2, where the aortic velocity is <4.0 m/s or the mean transvalvular pressure gradient is ≤30-40 mmHg.

Principle of Stress Testing in Low-flow Low-gradient Aortic Stenosis

Augmentation of stroke volume with dobutamine should increase flow across the aortic valve and cause a significant increase in gradients without a change in aortic valve area (<1.0 cm2).

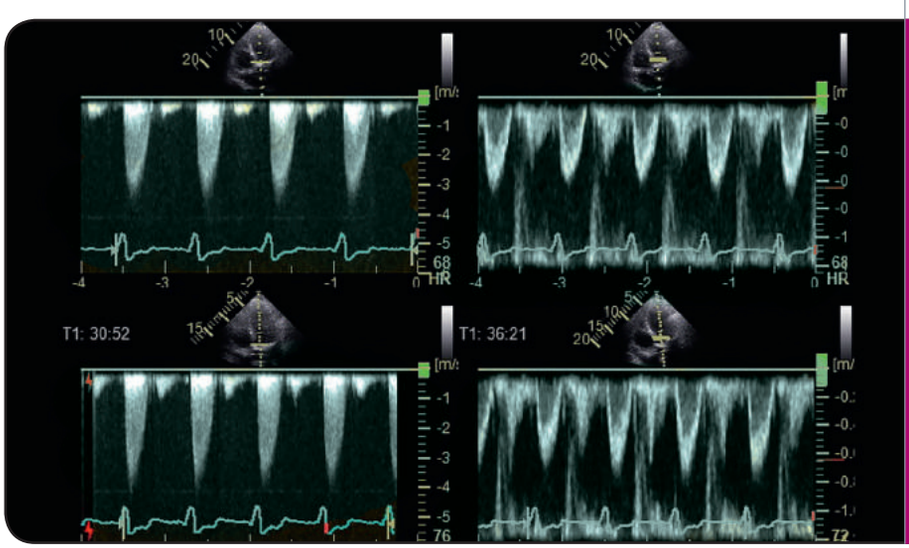
Stress echo can be used to distinguish true (severe) low-flow low-gradient aortic stenosis from "pseudo-severe" low-flow low-gradient aortic stenosis. Patients with left ventricular contractile reserve and true severe low-flow low-gradient aortic stenosis have an acceptable surgical risk. Valve replacement is recommended in the majority of these patients and usually improves their functional status and survival.

Changes in Echo Parameters During Stress

	True severe AS	Pseudo-severe AS
Stroke volume (LVOT velocity)	+	+
Transvalvular gradients	+++	(+)
Aortic valve area	-	(+)

Even moderate aortic stenosis can be relevant when patients have ischemic or dilated cardiomyopathy. The increased afterload in aortic stenosis is an additional burden to the ventricle.

Patients with pseudo low-flow low-gradient AS have a small aortic valve area because the low stroke volume does not push the valve open. In contrast, in patients with true severe aortic stenosis the aortic valve area is fixed and does not increase when stroke volume rises.



LOW-FLOW LOW-GRADIENT AORTIC STENOSIS – apical four-chamber view/CW & PW Doppler

Increase on LVOT velocity and AV velocity in a patient with true low flow low gradient AS

NOTES

STRESS ECHO IN LOW-FLOW LOW-GRADIENT SEVERE AORTIC STENOSIS

Dobutamine Protocol in Low-Flow Aortic Stenosis

- Start with a low dobutamine dose (at 5 µg/kg/min)
- Increase stepwise (+2.5–5 µg/kg/min) to maximum of 20 µg/kg/min
- Duration of each step: 3-5 min
- Monitor blood pressure and ECG (arrhythmias, ischemia)

The differentiation between true severe and pseudo-severe aortic stenosis may be improved by calculating the projected aortic valve area (the aortic valve area that would be present if the flow rate were normal).

More detailed information can be found in Blais et al. Circulation 2007

Echocardiographic Examination

Parameter	Applied for
LVOT diameter (at rest)	Aortic valve area (AVA) (continuity equation)
LVOT velocity signal (PW Doppler)	AVA (continuity equation), stroke volume
Doppler signal aortic valve (CW Doppler)	Maximum and mean gradient, AVA continuity equation
Representative 2D images	Visual assessment or calculation of ejection fraction/contractile reserve
(Color Doppler of mitral regurgitation)	Dynamic mitral regurgitation?

Stroke volume can actually drop when heart rate increases excessively. This will also affect the gradients and can lead to misinterpretation. Sometimes submaximal stress provides the highest gradients.

Endpoints in Low-Flow Aortic Stenosis

- The maximum dobutamine dose has been reached (20 µg/kg/min)
- Obvious inotropic response and positive outcome of the test
- Ventricular arrhythmias (ventricular tachycardia/increasing frequency of polymorphic ectopic beats
- (Increase in heart rate ≥ 10-20 beats/min)

A dobutamine response is present when the forward stroke volume increases by ≥20% (= 20% increase in the velocity time integral).

Things to Consider

- An increase in mitral regurgitation under stress can counterbalance an increase in cardiac output during stress.
- Tachycardia during stress may offset an increase in stroke volume
- Difficulties in obtaining adequate Doppler signals during stress may lead to an underestimation of the increase in gradients
- Determine the average of several beats in the presence of atrial fibrillation

022 // Contrast Echocardiography

CONTENT

NOTES

PRINCIPLES

Injected air or gas bubbles can generate a very strong ultrasound signal when hit by an ultrasound wave. This signal is used to opacify (contrast) the blood pool during echocardiography.

Contrast agents are micro-bubbles, which consist of an outer shell and encapsulated inner gas.

Intravenous injection of contrast results in pronounced contrasting of right heart chambers. Contrast agents with the following characteristics have been developed to achieve adequate opacification of the left heart as well:

- Small bubble size (1-8 µm)
- To allow passage through the pulmonary circulation and myocardial microcirculation
- A durable shell and gas with high density, high molecular weight, and low solubility
- Non-toxic
- No side effects
- High echogenicity
- Strong ultrasound reflectors
- Contrast effects that last for 3-10 minutes; the contrast medium can be applied as a bolus, repeat bolus, or an infusion.
- Can be destroyed with high-power ultrasoundTo study replenishment of contrast in myocardial microcirculation

Reactions of Bubbles to Ultrasound

Linear oscillation	At low acoustic power (MI<0.2)	Compression and rarefaction are equal in amplitude	No special contrast signal is achieved
Non-linear oscillation	At intermediate acoustic power (MI 0.2–0.5)	Ultrasound waves are created at harmonics of the delivered frequency	Micro-bubble-specific signal
Destruction	At high acoustic power (MI >0.5)	Bursting of the bubbles, resulting in transient emission of a high-intensity signal	Intermittent imaging allows visualization of capillary refill

MI = mechanical index – the power of the signal to which the bubbles (or any tissue) are exposed.

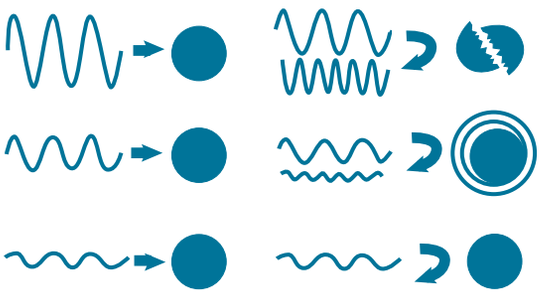
PRINCIPLES

Acoustic Power and Microbubble Responses

High Power (MI >0.5)

Low Power (MI 0.2–0.4)

Very Low Power (MI <0.1)



Very low power does not affect the contrast bubbles (no signal). Intermediate power causes the bubbles to resonate and generate a signal. High-power ultrasound leads to the destruction of bubbles, generating a very strong return signal.

NOTES

Some destruction of micro-bubbles is always present, even at a lower acoustic power.

Imaging of (Left Heart) Contrast Requires Special Settings

Low mechanical index (real-time imaging)	Less bubble destruction, weak tissue signal, simultaneous assessment of function and perfusion is possible
High mechanical index (ECG-triggered intermittent imaging)	Intentional destruction of bubbles to generate high-intensity signals and study replenishment

Low mechanical real-time imaging is used to study left ventricular function.

CONTRAST AGENTS

Right Heart Contrast Agents

(Do not cross the pulmonary circulation)

Agent	Dose
Agitated saline (+blood and air)	8 ml 0.9% saline (+1 ml blood + 1 ml air)
Dextrose 5% water	10 ml
D-galactose microparticle solution (Echovist®)	5–10 ml
Urea-linked gelatin (Haemaccel®)	10 ml
Oxypolygelatine (Gelifusin®)	10 ml
Sonicated albumin (5%)	10 ml

NOTES

CONTRAST AGENTS

Left Heart Contrast Agents

(Cross the pulmonary circulation and enter the myocardial microcirculation)

Agent	Dose Bolus	Dose Infusion
Perflutren protein type A microsphere (Optison®)	0.5 ml, < 1 ml/s, flush of 0.9% sodium chloride injection, or 5% dextrose injection, maximum dose 8.7 ml in any patient	Not more than 5ml in a 10-min period and do not exceed the maximum cumulative dose of 8.7 ml per study
Perflutren lipid microsphere (Definity®)	10 microliters (microL)/kg of the activated product by intravenous bolus injection within 30-60 seconds, followed by a 10-mL saline flush	Activated Definity® via intra-venous infusion of 1.3 mL added to 50 mL of preservative-free saline. The rate of infusion should be initiated at 4 mL/minute, but titrated as necessary to achieve optimal image enhancement; should not exceed 10 mL/minute.

Potential Side Effects

- Back pain
- Headache
- Urticaria
- Rarely: anaphylactic reactions (estimated rate of 1 per 10,000)
- Modern

The ultrasound return signal generated by micro-bubbles is several million times more effective in scattering ultra-sound than red blood cells.

Contraindications for Left Heart Contrast Agents

- Hypersensitivity to Perflutren
- Intra-arterial injection
- Right to left or bidirectional intracar-diac shunts
- Hypersensitivity to blood or albumin (for Optison only)

Thirty minutes of monitoring is required only for patients with pulmonary hyper-tension and unstable cardiopulmonary disease.

APPLICATIONS OF ECHO CONTRAST

Interrogation		Effect
Detection of shunts	ASD	Washout, contrast passage through the ASD
	PFO	Contrast into the left atrium through PFO
	Intrapulmonary shunt	Rapid contrasting of LA via pulmonic veins (≥ 4 cycles)
Doppler signal enhancement	Tricuspid regurgitation	Enhancement of the signal, measurement of maximum TR velocity
Cavity delineation	Ventricular function	Enhanced endocardial delineation
	Heart tumor and masses	Better delineation of the masses; flow within the mass?
Congenital abnormalities	Persistent vena cava sin.	Contrast injection via a left cubital vein results in contrasting of the right atrium via the coronary sinus.

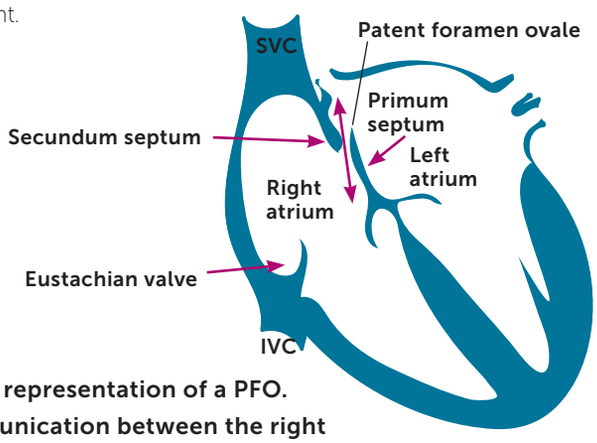
NOTES

Vascularized tumors show opacification when contrast is applied.

RIGHT HEART CONTRAST

Patent Foramen Ovale (PFO)

A patent foramen ovale is a channel/flap between the septum secundum and the septum primum that allows oxygenated blood from the mother to bypass the pulmonary circulation and reach the systemic circulation of the infant during fetal development.



Schematic representation of a PFO. The communication between the right and the left atrium is formed by a channel/flap between the primum and the secundum septum.

NOTES

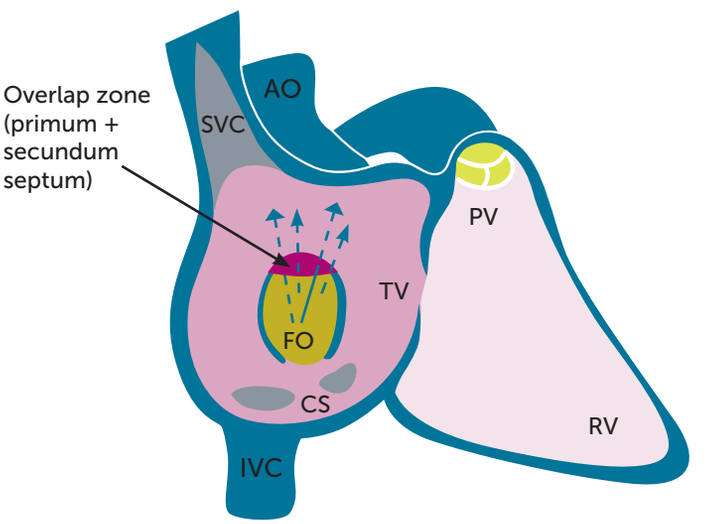
Paradoxical embolism may be associated with acute pulmonary embolism. The presence of a deep vein thrombosis and a sudden rise in right atrial pressure predisposes to right-to-left shunting.

Elevated right atrial pressures (as they occur in pulmonary hypertension) may cause significant right-to-left shunt and hypoxemia.

Patients with high left atrial pressures have pure left-to-right shunts. In this setting the contrast study will be negative (even using a Valsalva maneuver), but the condition can be seen with color Doppler.

RIGHT HEART CONTRAST

- A PFO persists in approximately 30% of adults.
- Its prevalence declines with age.
- The prevalence of PFO is higher in (young) patients with cryptogenic stroke (paradoxical embolism).
- Atrial septal aneurysms are frequently associated with PFOs and/or atrial septal defects (ASDs).
- A prominent Eustachian valve or Chiari network favors the persistence of PFO.
- Migraine and vascular headache are more common in the setting of a patent foramen ovale with right-to-left cardiac shunting.
- Decompression sickness in scuba divers may lead to air embolism through a patent foramen ovale.
- Paradoxical embolism through a PFO may also occur into the coronaries, renal arteries, retinal arteries, or other sites of systemic vascular circulation.

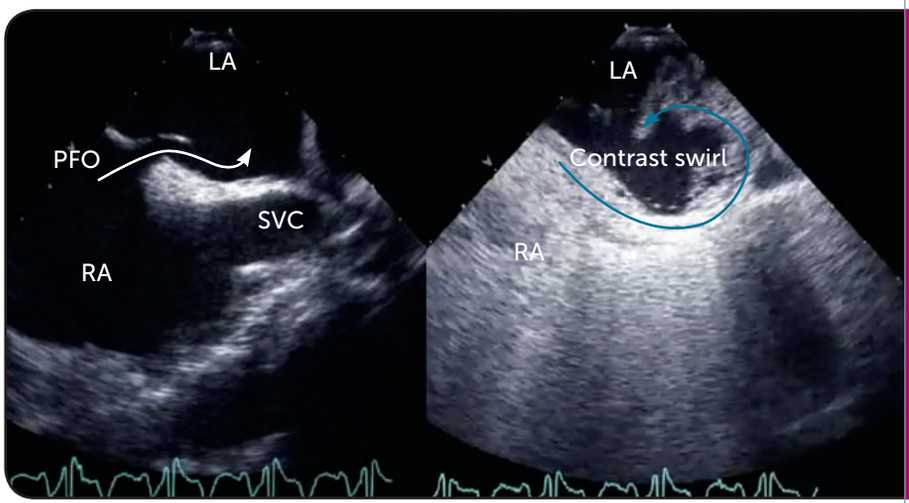


Fossa ovalis (FO) and a patent foramen ovale as seen from the right atrium. The connection between the right and the left atrium occurs through an "overlap" zone between the secundum and the primum septum, which is located cranially in the fossa ovalis.

The degree and direction of the shunt depend on the following factors:

- Size of the PFO
- Pressure gradient between the right and the left atrium
- Respiratory phase (influences pressure gradient from left to right)
- Mechanical factors; distortion of cardiac anatomy (interatrial septum) may increase the degree of shunting (i.e. platypnea-orthodeoxia syndrome)

RIGHT HEART CONTRAST



NOTES

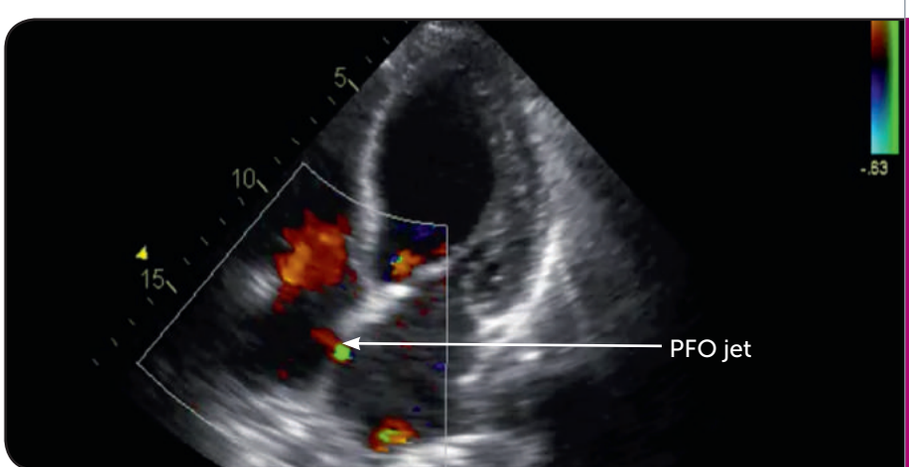
CONTRAST IN PFO – TEE bicaval view/2D & contrast

Large PFO and hypermobile interatrial septum; the separation between the primum and secundum septum is visible in 2D (left side). Pronounced contrast opacification of the left atrium occurs after the injection of oxypolygelatine (right side).

Detection of a Patent Foramen Ovale with Color Doppler

- Best seen on a slanted four-chamber view and a modified parasternal short-axis view
- Located cranial portion of the interatrial septum in proximity to the aortic valve and superior vena cava
- Size of color jet (degree of shunting) may vary with respiration
- Not always possible to differentiate between a PFO and a small atrial septal defect

Right atrial inflow from the inferior vena cava "bouncing" off the interatrial septum may mimic an ASD/PFO jet.



COLOR DOPPLER in PFO – slanted apical four-chamber view/color Doppler

A small jet (PFO) is passing through the interatrial septum

How to Perform an Adequate Transthoracic Contrast Study for Identification of a Patent Foramen Ovale

- Use an apical four-chamber view.
- The interatrial septum and the right upper pulmonic vein should be visible.
- Perform a bolus (intravenous) injection of contrast.
- Look for contrast crossing the interatrial septum.

Contrast that enters the left atrium via the pulmonic circulation (normal) usually comes late and bubbles appear smaller. A negative transthoracic contrast study does not rule out a PFO. The sensitivity and specificity is much lower than that of a transesophageal contrast study.

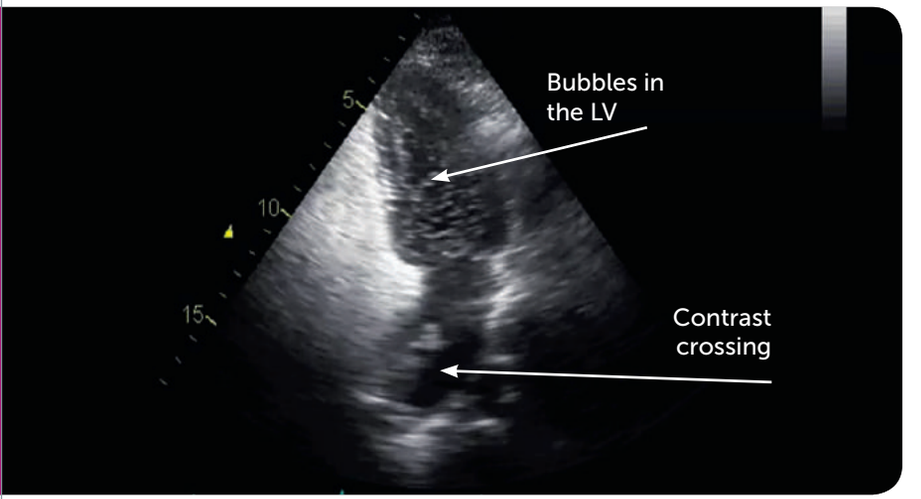
Consider hepatopulmonary syndrome in the setting of severe hepatic disease, volume overload, and low oxygen saturation. Right contrast echo can detect intrapulmonary shunts (pronounced contrasting of the left heart via the pulmonic veins after ≥ 4 cardiac cycles).

NOTES

POSITIVE TRANSTHORACIC CONTRAST STUDY – apical four-chamber view/2D contrast

Positive contrast study with Oxypolygelatine (Gelifusin®) used as contrast agent. A small "cloud" of contrast enters the left atrium via the interatrial septum.

RIGHT HEART CONTRAST



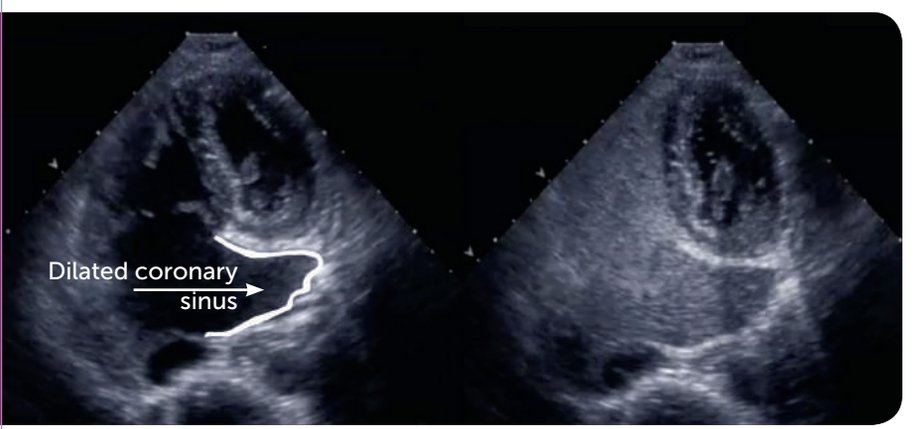
Contrast injection may aid procedures such as pericardiocentesis (position of the needle in the pericardium or the heart).

If Negative, Repeat the Study using a Valsalva Maneuver

- Use a four-chamber view (image from as far medial as possible to avoid lung interference when the patient inhales).
- Let the patient exhale.
- Ask the patient to perform a Valsalva maneuver.
- Inject contrast.
- Let the patient release the "Valsalva pressure" as soon as contrast appears in the right atrium.
- Let the patient inhale (to a normal level) – too vigorous inspiration will result in poor image quality.

PERSISTENT LEFT SUPERIOR VENA CAVA – apical four-chamber view/2D & contrast

Patient with a dilated coronary sinus (right); contrast (Oxypolygelatine - Gelifusin®) injected via a left cubital vein demonstrates contrasting of the right atrium via the coronary sinus, suggestive of a persistent left superior vena cava.



Platypnea-Orthodeoxia Syndrome

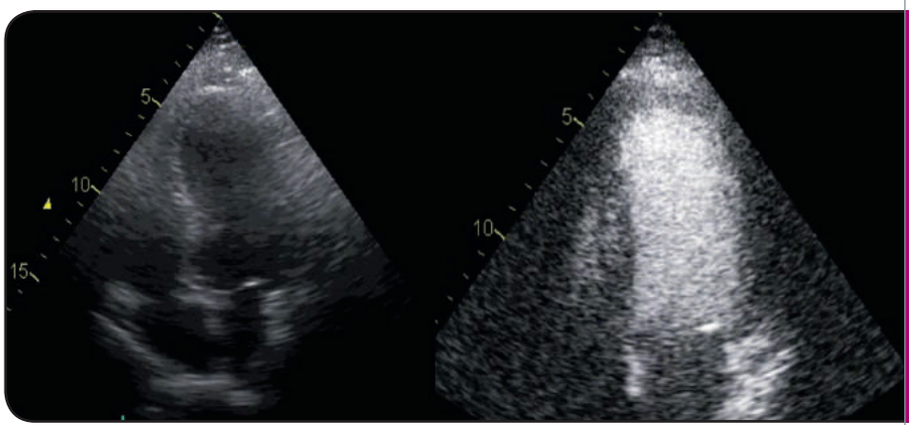
Right-to-left shunt that leads to dyspnea and oxygen desaturation when patients are brought into upright position. The upright position increases the degree of shunting by anatomically stretching the interatrial communication.

Predisposing Factors

- Aortic dilatation/aneurysm
- Chest surgery (pneumectomy)
- Pulmonary emphysema, diseases of the pericardium

QUANTIFICATION OF LEFT VENTRICULAR FUNCTION

Left heart contrast opacification greatly enhances the visibility of the endocardial border and thus improves assessment of global and regional function.

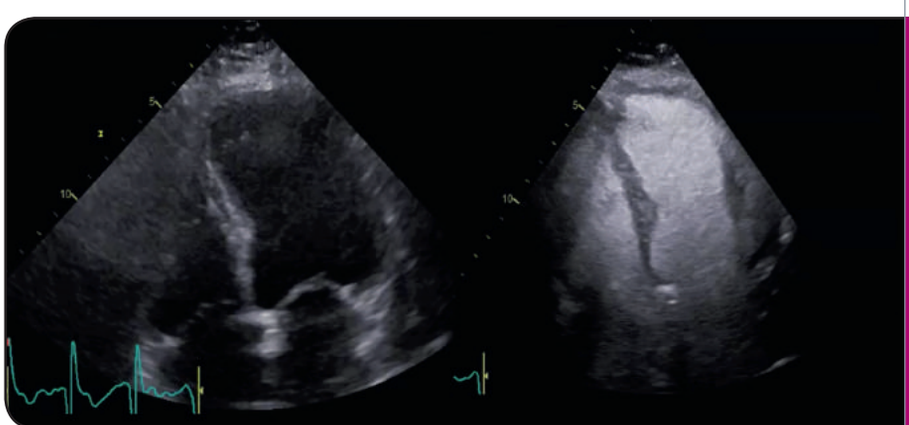


IMPROVEMENT OF IMAGE QUALITY USING CONTRAST – apical four-chamber view/2D & contrast

Difficult assessment of global and regional left ventricular function in a patient with very poor image quality (left). The contrast study greatly improves image quality (right).

Contrast Settings

- Harmonic imaging mode
- Low mechanical index – real-time imaging (MI = 0.5)
- Compression in the medium to high range
- Image focus at the level of the mitral valve or below



CONTRAST AND WALL MOTION – apical four-chamber view/2D & contrast

Contrast study to assess regional wall motion. Akinesia of the anteroseptal region is clearly visible with contrast (see cine loop - www.123sonography.com/echofacts)

Practical Issues

- When injecting intravenously, contrast will first appear in the right heart.
- Consider that too much contrast will cause attenuation of those regions more distal from the transducer (basal parts of the left ventricle when imaging from the apex).
- Contrast in the right ventricle may shadow the left ventricle and lead to deterioration of image quality when parasternal views are used.
- Titrate the contrast dose to achieve optimal filling.
- Bolus injection is adequate for rest studies, whereas continuous infusion of contrast should be given preference during stress studies.
- Freeze the image intermittently to reduce bubble destruction and allow refilling of the ventricle when contrast is low.
- Contrast may be combined with 3D echocardiography.

NOTES

Apical swirling of contrast is a result of excessive destruction of contrast in the near field by ultrasound.

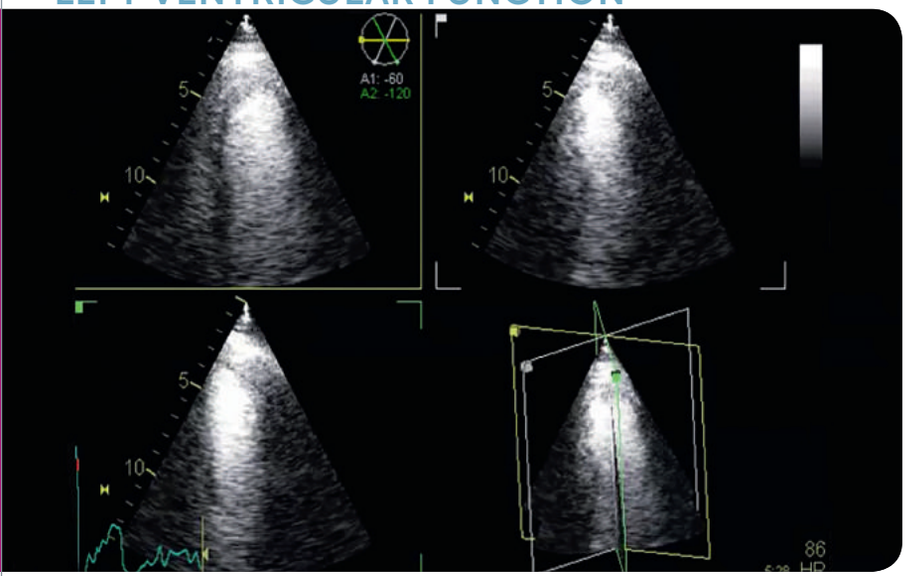
Contrast echocardiography greatly enhances the accuracy of detecting regional wall motion abnormalities, both at rest and during stress.

NOTES

QUANTIFICATION OF LEFT VENTRICULAR FUNCTION

3D CONTRAST STUDY – apical multiplane image acquisition/3D

Contrast study with multiplane 3D, four- (upper left) two- (upper right) and three-chamber views (lower left). The lower right image shows the corresponding cut planes.

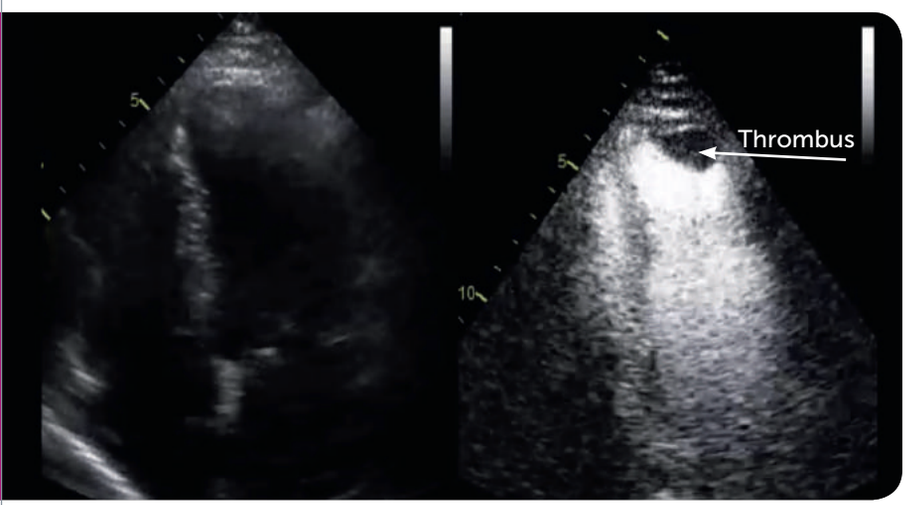


Other Indications for Left Heart Contrast

- Aneurysms and pseudoaneurysms
- Apical hypertrophy
- Ventricular non-compaction
- Apical thrombus (contrast filling defects are visible)
- Masses (increased echo contrast due to vascularization of the mass)
- Pericardial cysts
- Coronary aneurysms and fistulas

CONTRAST AND APICAL THROMBUS – apical four-chamber view/2D & contrast

Patient with suspected apical thrombus (left). Contrast injection demonstrates a filling defect at the apex of the left ventricle, denoting a thrombus (right).



MYOCARDIAL PERFUSION IMAGING

Principle

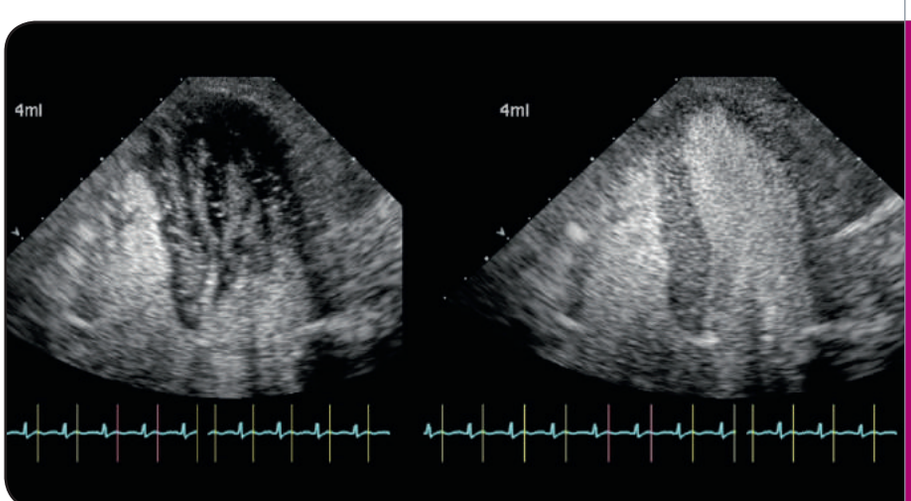
Imaging of contrast within the vascular bed permits assessment of myocardial perfusion at rest and during stress.

Limitations

- Feasible only in patients with very good image quality
- Low concentrations of contrast enter the coronary perfusion bed (5-10% of cardiac output)
- Rapid destruction of bubbles (slow flow in the capillary bed, destruction caused by high intramural pressure)
- Difficult to discern contrast from myocardial tissue

How to Perform Myocardial Perfusion Imaging

- Perform power Doppler imaging (high mechanical index).
- Perform intermittent imaging (one frame imaged every 1-8 cardiac cycles).
- Look at the replenishment of myocardial contrast opacification as an indicator of perfusion.
- Perfusion defects appear as darker areas.



MYOCARDIAL PERFUSION IMAGING – apical four-chamber view/ contrast

Intermittent ECG-triggered imaging. Bubble destruction (left) and replenishment with homogenous contrasting of the myocardium (right) in a healthy patient without perfusion defects.

NOTES

Consider that segments closer to the transducer are destroyed more readily, and that this may mimic a perfusion defect.

Several studies have shown that myocardial contrast echocardiography correlates well with coronary flow reserve. It also predicts recovery of systolic function after reperfusion therapy. However, myocardial contrast echo is technically demanding and involves a learning curve.

NOTES

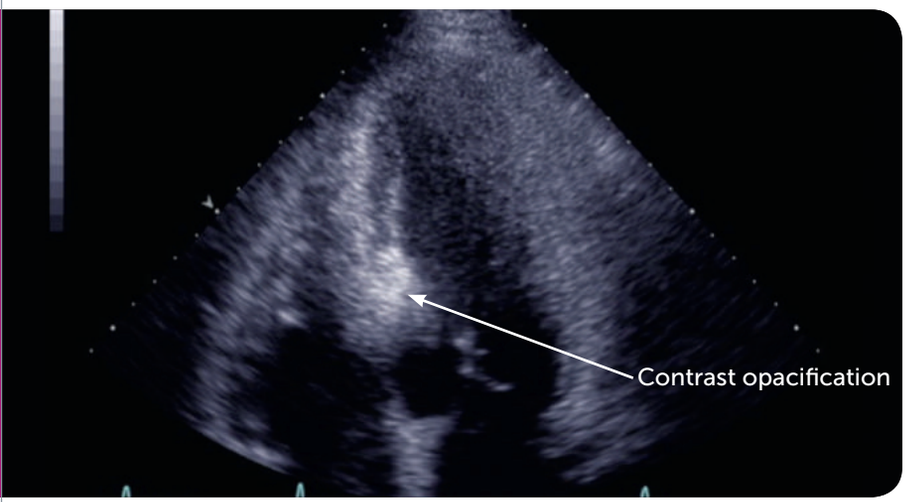
MYOCARDIAL PERFUSION IMAGING

Myocardial Perfusion Imaging in Septal Ablation

Contrast injection into the coronary arteries may be used to study myocardial perfusion. This is applied to define the target perfusion area during alcohol septal ablation therapy in obstructive hypertrophic cardiomyopathy.

CONTRAST AND SEPTAL ABLATION – apical four-chamber view/coronary contrast.

A contrast agent (Optison) is injected into the first septal branch of the LAD during an alcohol septal ablation procedure, resulting in opacification of the basal septum.



023 // 3D Echocardiography

CONTENT

NOTES

BASICS OF THREE-DIMENSIONAL ECHOCARDIOGRAPHY

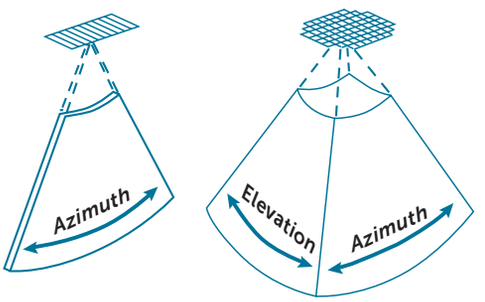
Despite advances in 3D technology, 2D image quality is always better with a 2D transducer than with a 3D transducer. Never base clinical decisions on 3D echo assessment alone.

What is 3D/4D Echocardiography?

3D echo permits 3-dimensional analysis and display of ultrasound data. The term 4D echo is sometimes used to introduce time (moving 3D images) as the "fourth dimension".

How it is done

Data acquisition is achieved by imaging with 3D matrix array transducers.



2D vs. 3D matrix array transducers

3D transducers acquire a pyramidal volume set using more than 3000 independent piezoelectric elements. The beams are formed to a large extent within the transducer. The imaging frequency of transthoracic 3D transducers is between 2 and 4 MHz. In contrast, 2D transducers only scan a two-dimensional sector.

FORMS OF 3D ECHOCARDIOGRAPHY

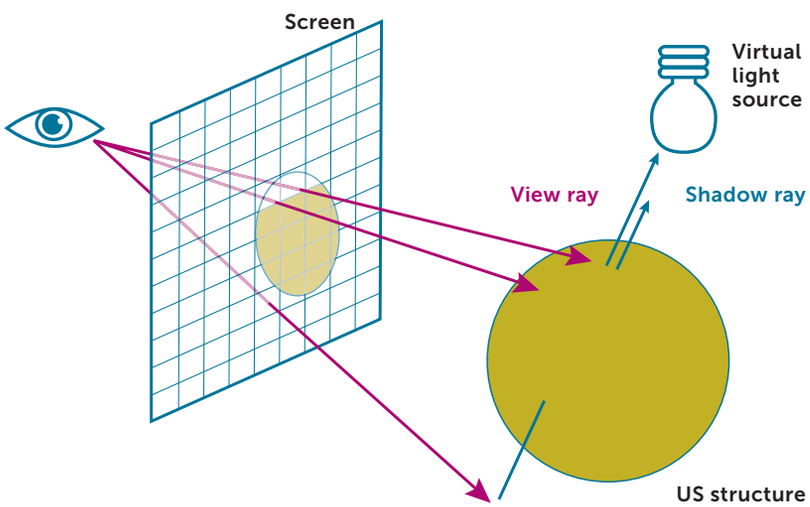
3D-dimensional image "pixels" are known as voxels.

	Description	Advantage	Disadvantage
Real time (live) 3D	Structures are displayed in 3D format while imaging. Acquisition of multiple pyramidal datasets per second.	Immediate results; can be used for monitoring procedures; can be used when RR intervals vary (e.g. atrial fibrillation).	Small sector or zoom mode, low spatial and temporal resolution (frame rate), orientation sometimes difficult.
Triggered multi-beat (full volume) 3D acquisition	A complete dataset is required during several heartbeats.	Higher temporal and spatial resolution, more possibilities of quantification, analysis and display.	Post-processing required, time consuming, stitching artifacts. Only works in sinus rhythm.

FORMS OF 3D ECHOCARDIOGRAPHY

3D Image Representation

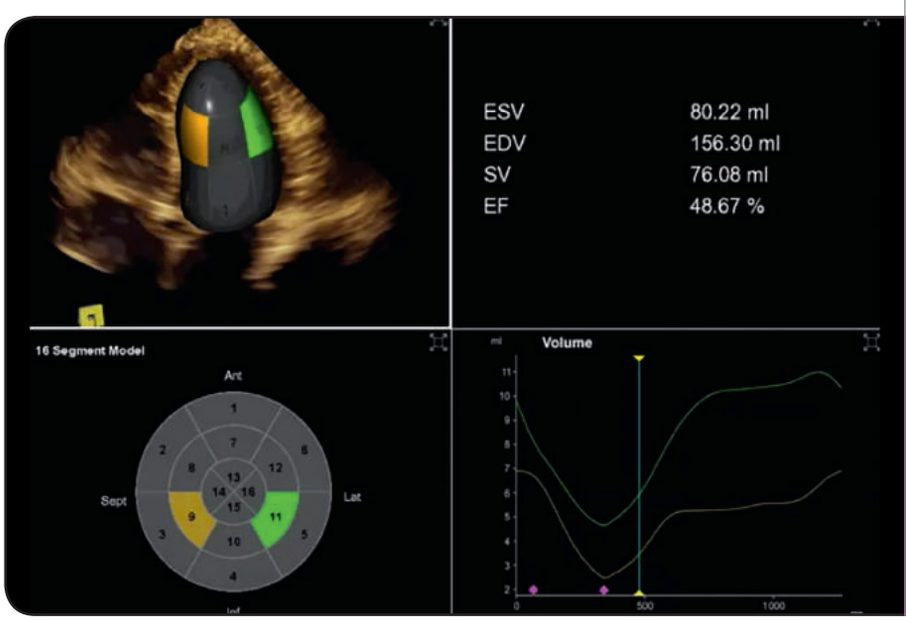
Volume rendering	Rendering algorithms that create the impression of three-dimensionality on a 2D screen (ray casting, shear warp etc.)
Surface rendering	Surfaces are displayed as solid structures or wire frames (i.e. cast of the ventricular cavity).
2D tomographic slices	3D dataset is sliced to reconstruct 2D cut planes (multiplane)



Principle of ray casting (volume rendering technique). Ray tracing is an algorithm that simulates the effects of light as it would be seen by the observer (eye) while it passes through a voxel space.

NOTES

3D rendering algorithms "recode" the original ultrasound pixels/voxels to create a sense of depth (distance shading, gray-level gradient coding etc.). Therefore we lose information concerning the density of tissue and tissue characteristics. In other words, we cannot distinguish fibrosis or calcification from other less echogenic tissue.



SURFACE AND VOLUME RENDERING – apical four-chamber view/3D

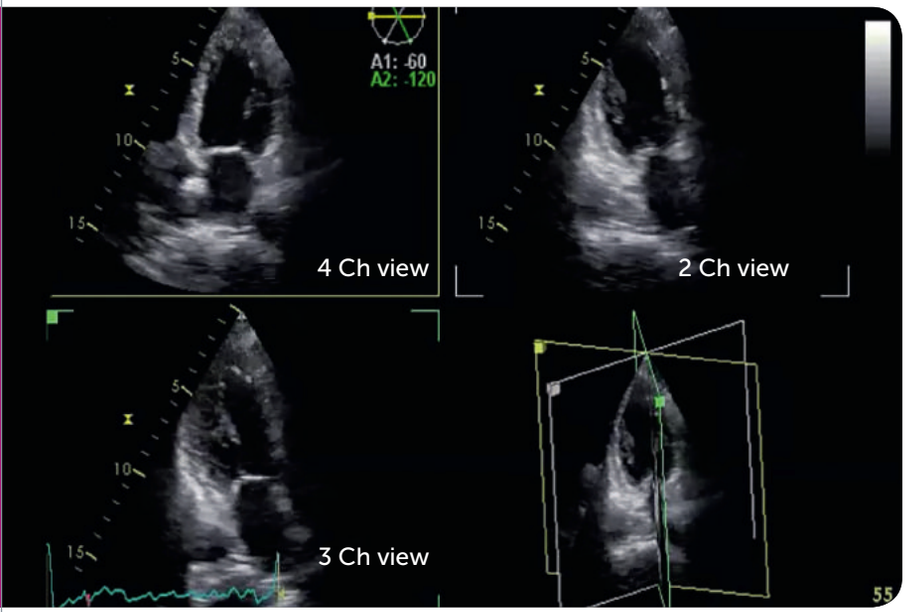
Combination of left ventricular surface and volume rendering with segmental analysis (comparison of two regional volumes). The curves in the right lower quadrant represent the regional volume curves during the cardiac cycle. The bull's eye shows the selected segments (mid lateral segment= green, mid septal segment= orange) in the left lower quadrant.

NOTES

MULTIPLANAR REPRESENTATION – apical views/3D

Simultaneous display of four- (upper right), two- (upper left), and three-chamber views (lower left). The right lower corner shows the corresponding cut planes.

FORMS OF 3D ECHOCARDIOGRAPHY



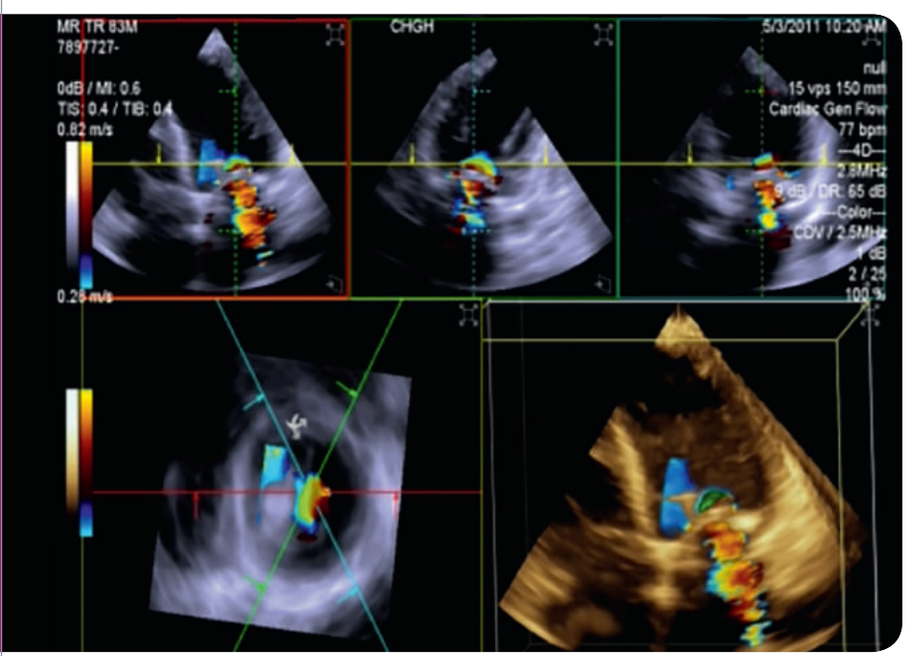
3D color Doppler is still limited by rather low frame rates and small color Doppler volumes.

3D Color Doppler

- Possible with live 3D and multi-beat full volume acquisition
- Still has limited spatial and temporal resolution
- 3D color Doppler with TEE is given preference over TTE
- Permits better appreciation of flow convergence, vena contracta, and jet geometry
- Color jets can also be displayed through reconstructed multi-slice cut planes

3D COLOR DOPPLER – apical full-volume acquisition/3D

The color jet is seen in various cut planes, including a short-axis view (left lower corner), and additionally visualized in 3D (right lower corner).



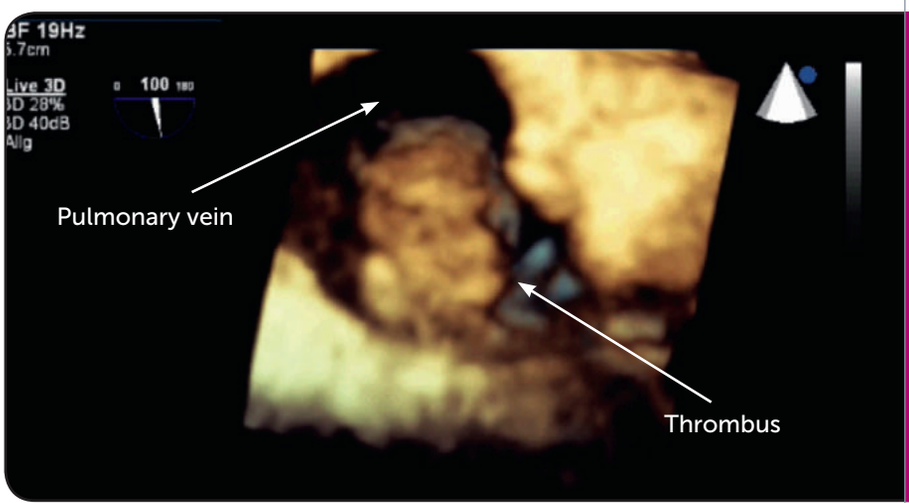
3D IMAGE ACQUISITION

- Make sure you have a good ECG signal (R-wave).
- Aim for best possible 2D image quality (trash in, trash out).
- Try to limit the number of beats in order to reduce stitching artifacts.
- Check for stitching artifacts by viewing a cut plane perpendicular to the sweep plane.
- Acquire images during breath hold to reduce motion and stitching artifacts.
- Select an adequate gain (mid-range) threshold to discern true structures from noise.
- Choose the smallest necessary sector width to achieve the highest possible frame rate.
- Use 2D images as a reference.
- For better orientation on the 3D image, recapitulate cardiac anatomy and topography.

NOTES

Cropping techniques (software algorithms) allow the operator to "cut away" structures that obscure one's view of the structure of interest (i.e. cut away parts of the left ventricle to view the mitral valve or the septum).

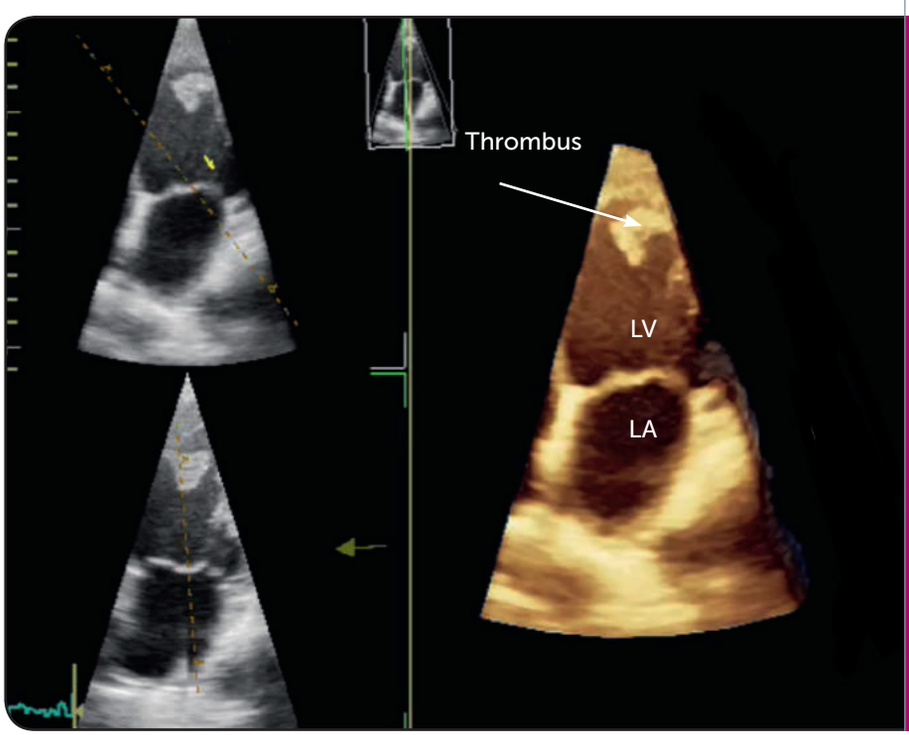
You can reconstruct in 3D only those structures that can also be visualized in 2D.



THROMBUS IN THE RIGHT UPPER PULMONARY VEIN – cropped image/3D TEE

Patient after lung transplantation. Cropped image techniques were used to cut away the left atrium and permit visualization of the right upper pulmonary vein, in which a highly mobile thrombus is seen.

CLINICAL APPLICATIONS OF 3D ECHOCARDIOGRAPHY



APICAL THROMBUS – apical multiplanar image acquisition/3D TTE

3D echocardiography showing a highly mobile apical thrombus.

NOTES

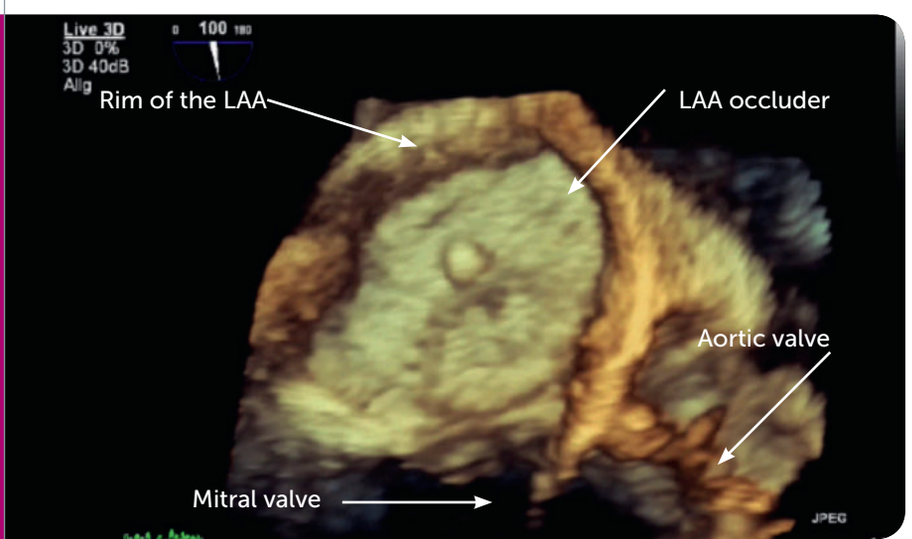
Live 3D – TEE imaging employs higher frequencies (5- 7 MHz) and has better spatial resolution than transthoracic 3D echo. It is the method of choice to monitor interventional procedures (e.g. MitraClip, ASD closure, left atrial appendage occlusion).

CLINICAL APPLICATIONS OF 3D ECHOCARDIOGRAPHY

- Calculation of true volumes (heart chambers)
- Calculation of myocardial mass
- Display several cut planes simultaneously (multiplane)
- Reconstruction of imaging planes that cannot be displayed with conventional 2D echocardiography
- Display structures (e.g. valves) in a more realistic format (volume rendering)
- 3D display of color jets (quantification of regurgitant lesions)
- Monitor interventional procedures on live 3D
- 3D deformation imaging (strain, strain rate)

LEFT ATRIAL APPENDAGE OCCLUDER – 3D TEE

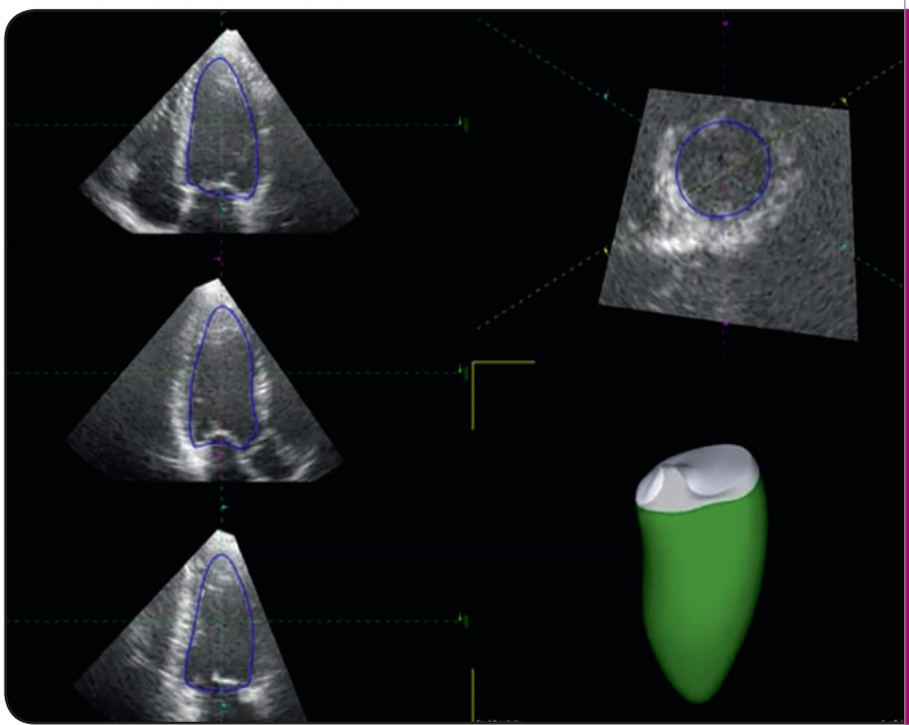
An Amplatzer Cardio Plug System is deployed in the left atrial appendage.



Advanced Quantification Tools

3D speckle tracking	Calculation and visualization of 3-dimensional deformations
Heart chamber segmentation algorithms	Semi-automated methods for endocardial border detection (ventricles, atria)
Regional wall motion analysis	Allows calculation of regional ejection fraction and regional timing of contraction
Parametric display	Color-coded display of various parameters, such as wall motion, contraction timing, strain, etc.

CLINICAL APPLICATIONS OF 3D ECHOCARDIOGRAPHY



ENDOCARDIAL SURFACE RENDERING – apical full volume acquisition/3D

Surface rendering is performed in accordance with semi-automated endocardial tracing on apical and short-axis views. The resulting volume (bag) is seen in the right lower corner.

Calculation of Ejection Fraction and Volumes of the Left Ventricle

- 3D volumes do not require geometric assumptions and are superior to all other echocardiographic methods
- 3D volume assessment can be combined with contrast to enhance endocardial border delineation
- 3D volume computation also allows computation of "regional" ejection fractions
- Semi-automated edge detection algorithms are usually employed to define endocardial borders
- Foreshortening of the left ventricle affects volume computations
- Exclude trabeculations when tracing the LV cavity

NOTES

While the accuracy of semiautomated endocardial border detection algorithms has been greatly improved, it is often still necessary to manually correct the contours.

Assessment of Dyssynchrony

- Regional volume curves are plotted against time. These plots are used to determine the time difference between the individual segments to minimal volumes (end-systole). The degree of dispersion of timing correlates with the degree of dyssynchrony
- The systolic dyssynchrony index is a measure of dyssynchrony. It is calculated as the standard deviation of regional ejection times (time to minimal regional volume).
- Dyssynchrony can also be visualized by dynamic tracking of regional contraction on a bull's eye display.

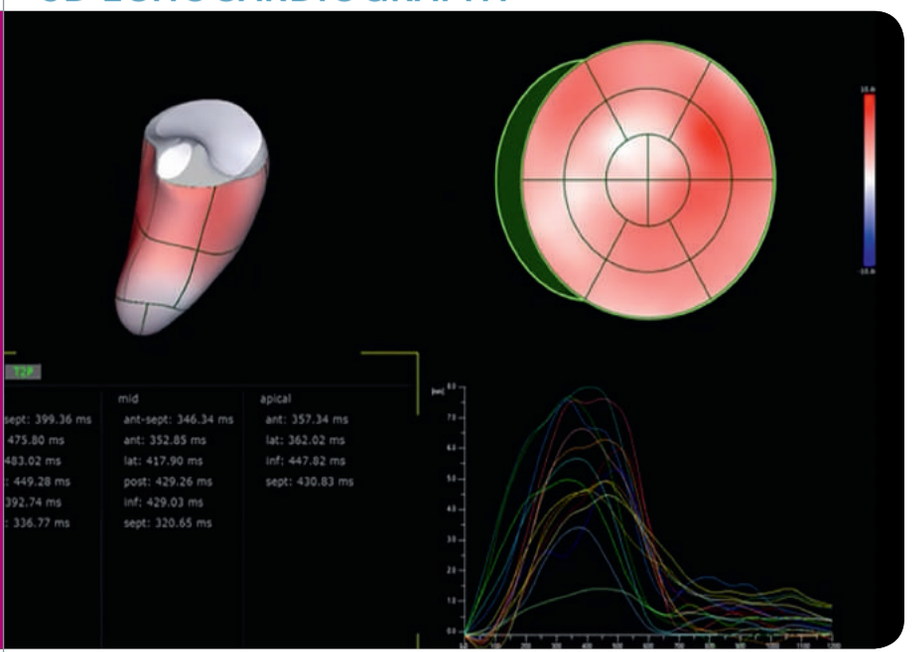
There is currently no recommendation to select patients for cardiac resynchronization therapy based on 3D analysis of dyssynchrony.

CLINICAL APPLICATIONS OF 3D ECHOCARDIOGRAPHY

NOTES

TIMING OF CONTRACTION – apical full-volume acquisition/3D

Timing of contraction in a normal patient. All segments reach their lowest volume (end systole) at (almost) the same time.



Currently the major limitation of 3D in stress echo is its low frame rate.

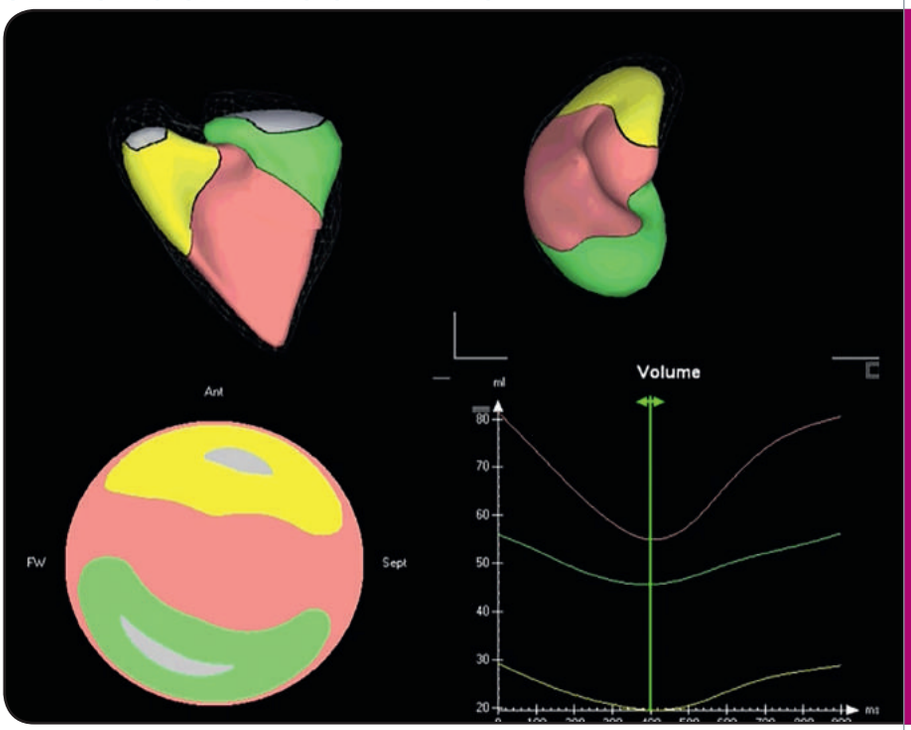
3D Stress Echocardiography

- Facilitates image acquisition by multiplane imaging
- Better visualization of the apex
- Is limited by its low frame rate
- Can be combined with contrast

Assessment of Right Ventricular Function

- 3D volume computation of the right ventricle is superior to 2D methods (complex morphology of the right ventricle).
- Semi-automated edge detection algorithms are applied to detect the endocardial border.
- Right ventricular volume and function computations with 3D have clinical impact (diagnosis and prognostic information) in many diseases (e.g. cardiomyopathy, atrial septal defect, tetralogy of Fallot, pulmonic regurgitation).

CLINICAL APPLICATIONS OF 3D ECHOCARDIOGRAPHYS



NOTES

3D RIGHT VENTRICULAR VOLUMES – apical RV full-volume acquisition/3D

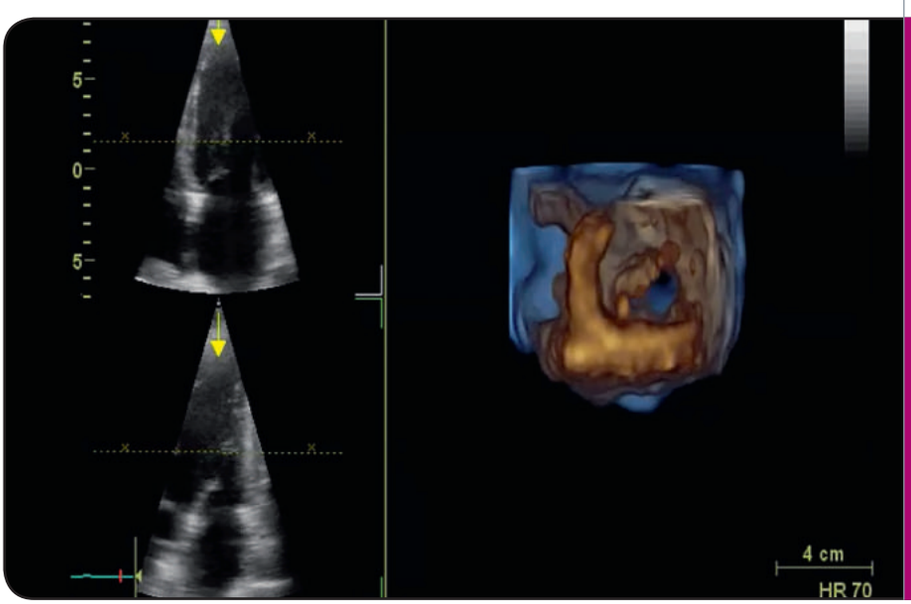
Regional volumes are divided into outlet (yellow), inlet (green) and apical (red) parts, which allows regional volume computations (curves in the right lower corner)

Mitral Valve Morphology in 3D Echocardiography

- 3D valve assessment can be performed with 3D- TTE and 3D-TEE
- 3D TEE is superior to 3D TEE
- Allows detection of structural defects and lesions (prolapse, flail, restriction, vegetations)
- May be combined with 3D color Doppler
- Can define the exact location (leaflet scallop) of the defect
- May be useful in patients who have undergone mitral valve replacement and repair (e.g. detection of paravalvular leaks)
- Is used to select patients for the MitraClip procedure and monitor them during the procedure
- Allows the investigator to study the motion and geometry of the mitral valve apparatus

The mitral valve is best studied from a surgical view (en face view from the left atrium).

While 3D imaging of the aortic, tricuspid, and pulmonic valves is feasible and may sometimes provide relevant information, the 3D image quality of these valves is usually inferior to that of the mitral valve.



3D RECONSTRUCTION OF THE MITRAL VALVE – apical full-volume acquisition/3D

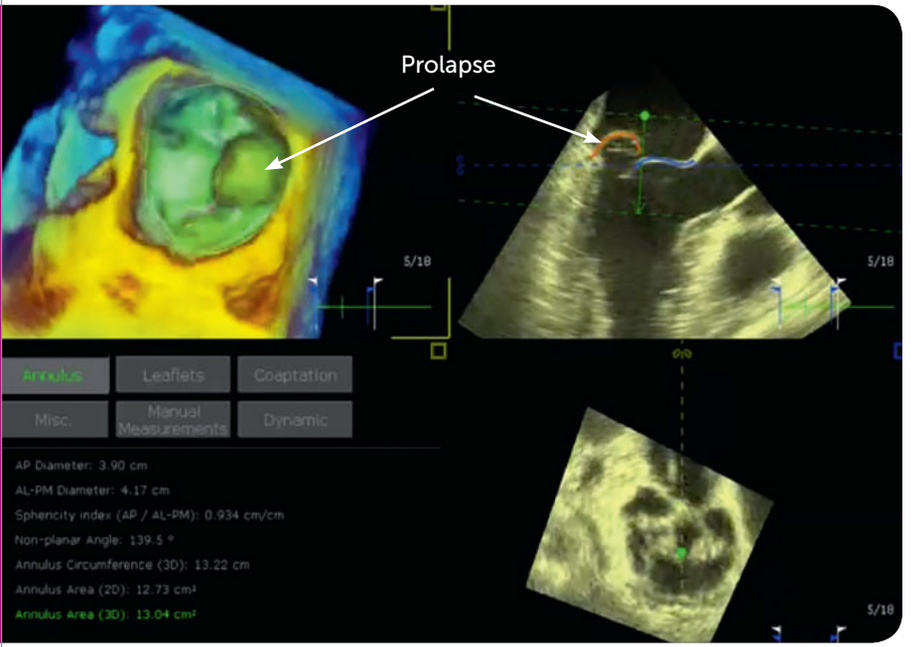
The mitral valve is viewed from the left ventricle.

NOTES

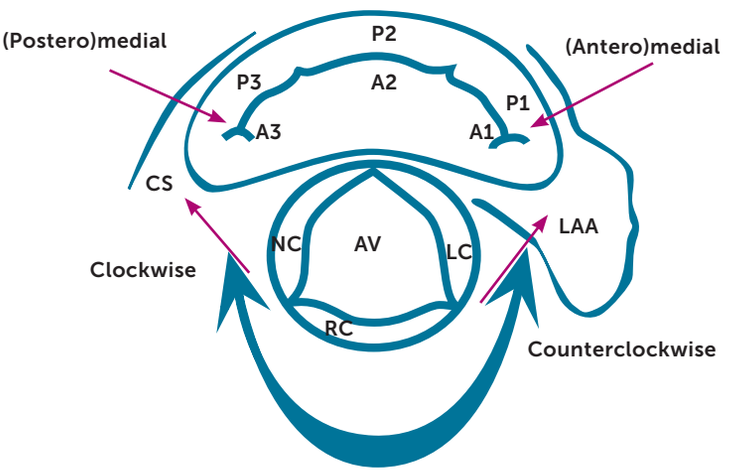
CLINICAL APPLICATIONS OF 3D ECHOCARDIOGRAPHY

3D MITRAL VALVE PROLAPSE – 3D TEE

Visualization of mitral valve prolapse in the medial posterior leaflet (P2) using 3D TEE.



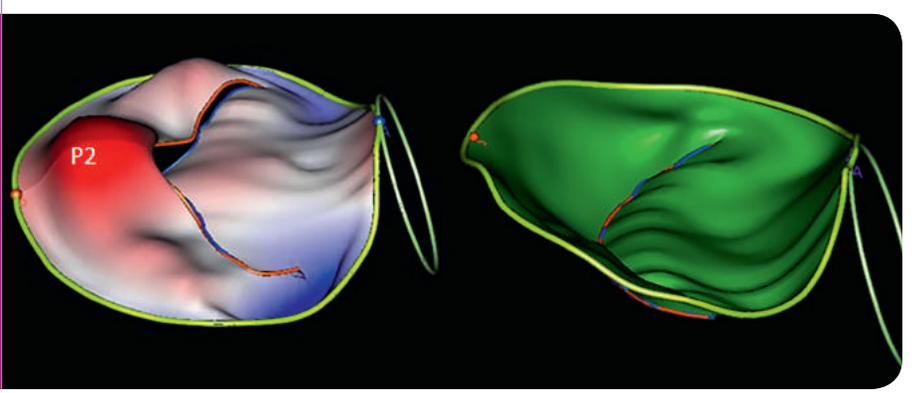
Always try to get the aortic valve on the 3D image; it permits you to determine the orientation (medial or lateral) of the mitral valve.



The anterior mitral leaflet is always adjacent to the aortic valve and is quadrangular in shape. The posterior mitral leaflet is shorter, but arises from a larger circumference than the anterior leaflet. The (postero-)medial portion of the valve is always oriented clockwise to the aorta while the (antero-)lateral portion is positioned counterclockwise from the aorta. The left atrial appendage is always adjacent to the (antero-) lateral commissure.

RECONSTRUCTION OF MITRAL VALVE PROLAPSE – 3D reconstruction of a mitral valve prolapse in the medial posterior leaflet (P2).

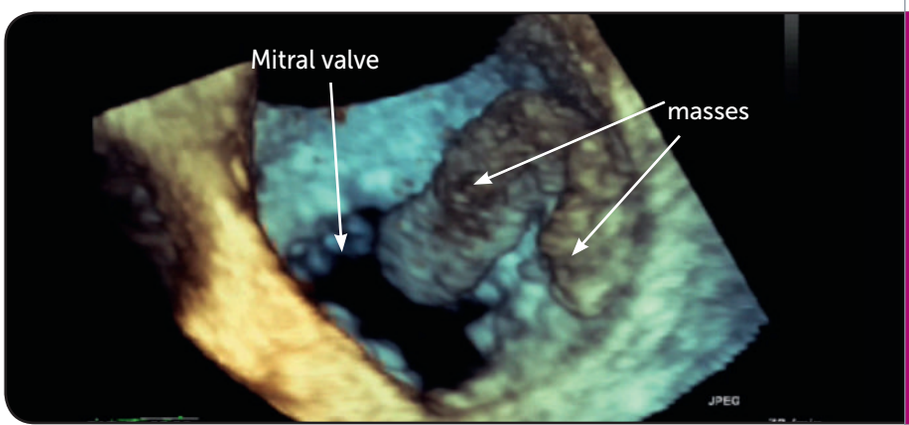
This technique allows calculation of mitral valve distances (commissural diameter), areas (leaflet area), angles, and volumes (tenting volumes).



CLINICAL APPLICATIONS OF 3D ECHOCARDIOGRAPHY

Further Clinical Applications of 3D Volume Rendering of Structures

- Endocarditic vegetations and complications
- Pacemaker lead interference with tricuspid valve closure
- Atrial septal defects – quantification of defect size
- Complex congenital abnormities
- Intra-cardiac masses
- Measurement of the aortic root/ annulus (e.g. TAVR evaluation)

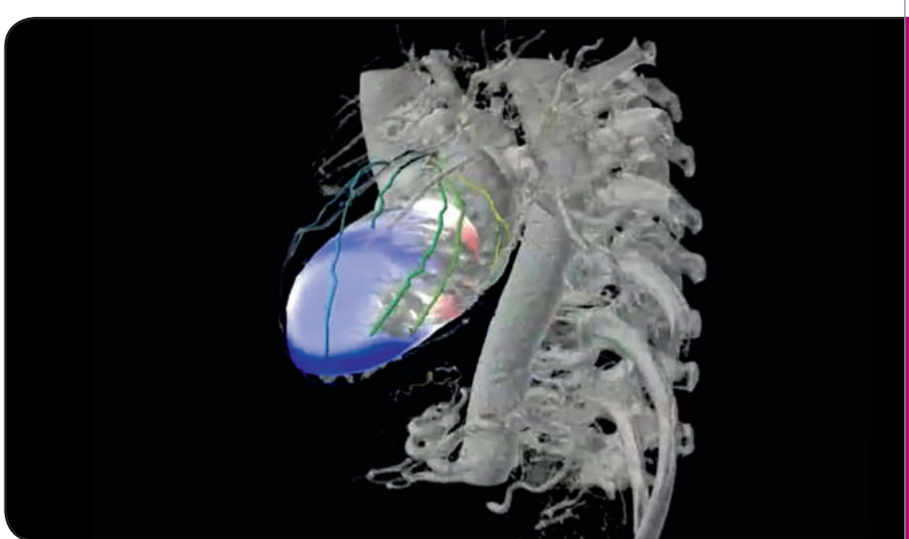


LEFT ATRIAL MASSES – 3D TEE

Two large masses originating from the left atrial appendage, which extend towards the mitral valve.

Future Perspectives

- Improvements in temporal and spatial resolution
- Smaller transducers/footprint
- Refined analysis tools
- Fusion imaging
- 3D strain



FUSION IMAGING – CT and 3D echocardiography

Fusion of cardiac CT data (showing coronary arteries) with a left ventricular Beutel generated by 3D echo. The LV "Beutel" shows the area of latest contraction in a color coded way (red is the area of late contraction).

NOTES

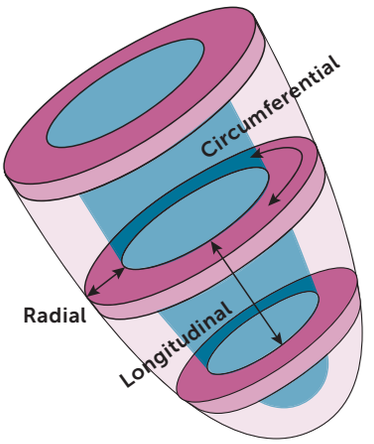
NOTES

024 // Myocardial Deformation Imaging

CONTENT

NOTES

PRINCIPLES OF MYOCARDIAL MECHANICS



- The orientation of myocardial fibers in the left ventricular wall ensures equal distribution of regional stress and strains.
- The left ventricle undergoes a twisting motion, which decreases the radial, circumferential and longitudinal length of the left ventricular cavity.
- During isovolumetric contraction, the apex initially performs clockwise rotation.
- During the ejection phase the apex then rotates counterclockwise while the base rotates clockwise when viewed from the apex.
- In diastole, relaxation of myocardial fibers and subsequent recoiling (clockwise apical rotation) contributes to active suction.

MEASURES OF MYOCARDIAL DEFORMATION

Displacement

- Displacement is the distance the myocardium (or any cardiac structure) travels between two consecutive image frames.
- Displacement is measured as a distance and therefore expressed in centimeters.

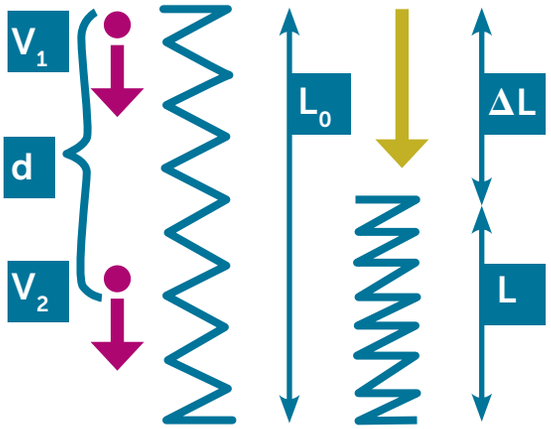
Tissue Velocity

- The speed (displacement per unit of time) of movement of a myocardium (or any cardiac structure)
- Tissue velocity is reported in cm/s

Strain and Strain Rate

- Strain is defined as the fractional change in the length of a myocardial segment.
- Three perpendicular axes (i.e. longitudinal, circumferential, and radial) represent different directions of left ventricular myocardial contraction. Strain is not expressed in units; it is usually expressed as a percentage.
- Strain values can be obtained for each segment (segmental strain), as an average value for all segments (global strain), or for each of the theoretical vascular distribution areas (territorial strain).
- Strain rate is the rate of change in strain and is usually expressed as 1/second.

MEASURES OF MYOCARDIAL DEFORMATION



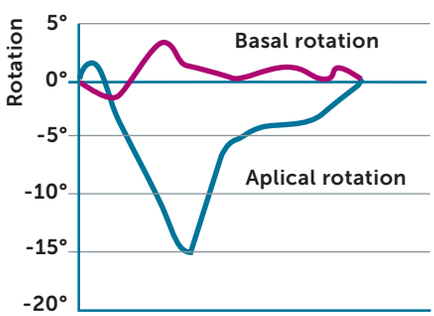
Strain represents the change in fiber length compared to its original length (right side, yellow arrow), whereas strain rate is the difference in tissue velocities at two distinct points in relation to their distance (left side, purple arrows).

$$\text{Strain} = \frac{(L - L_0)}{L_0} = \frac{\Delta L}{L_0} \qquad \text{Strain rate(ST)} = \frac{(V_1 - V_2)}{d}$$

NOTES

Rotation

- Rotation is defined as angular displacement of a myocardial segment on a short-axis view around the LV longitudinal axis, measured in a single plane.
- It reflects rotational displacement; myocardial rotation is expressed in degrees.
- The base and the apex of the ventricle rotate in opposite directions.



Basal and apical myocardial rotation in a healthy patient. The basal segment rotates clockwise, whereas the apical parts rotate more and more counterclockwise.

Basal rotation changes from counterclockwise in infancy to clockwise in adults.

Twist/Torsion

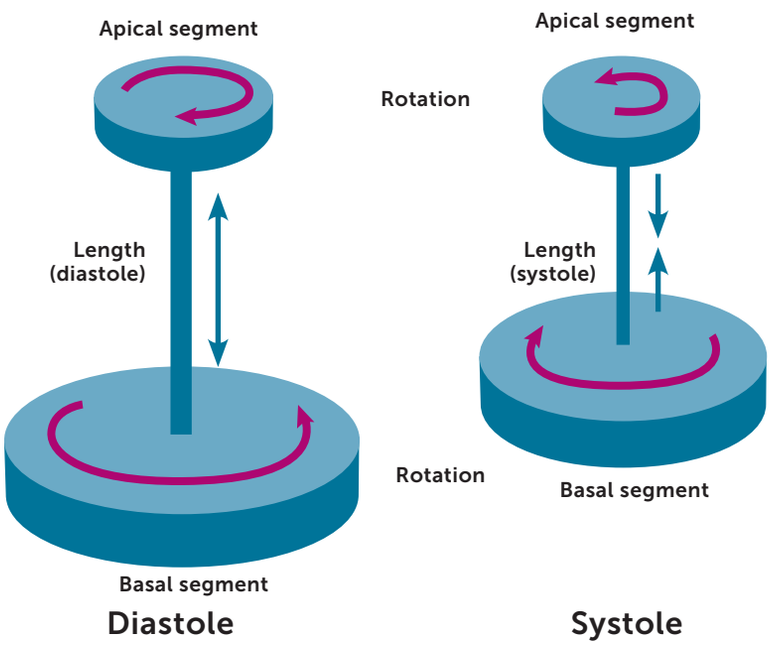
- It is defined as the net difference between apical and basal rotation and is expressed in degrees.
- It is calculated from two short-axis cross-sectional planes of the left ventricle.
- The normal peak LV twist angle is approximately 7.7° (Takeuchi et al. JASE 2006).
- The torsional gradient (degree/cm) is defined as the twist/torsion normalized to ventricular length from base to apex, and accounts for the fact that a longer ventricle has a larger twist angle.

The twist angle increases significantly with age.

NOTES

MEASURES OF MYOCARDIAL DEFORMATION

Rotation of the Left Ventricular Apex and Base During the Heart Cycle



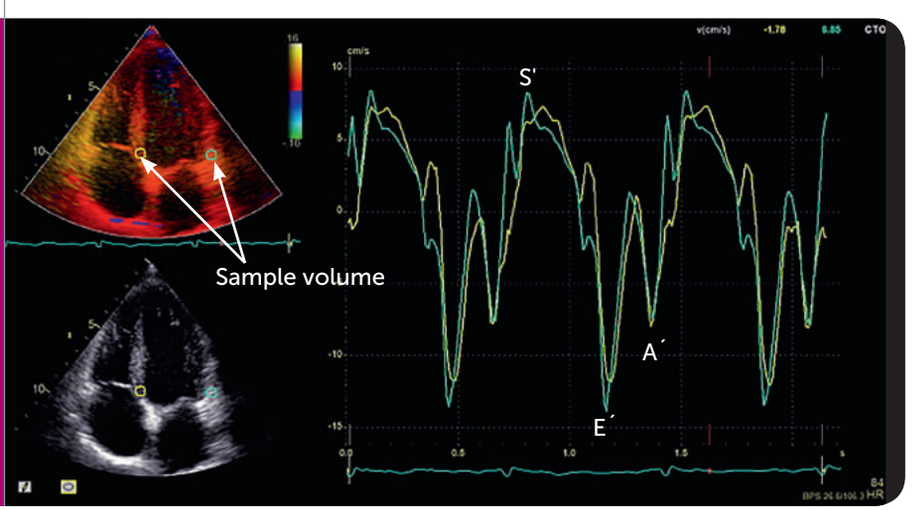
TISSUE DOPPLER IMAGING

- Tissue Doppler velocity estimation of myocardial motion employs the same principle as pulsed-wave and color Doppler echocardiography for blood flow.
- A wall filter is used to distinguish between signals from tissue and blood flow.
- Strain and strain rate can be calculated.

It is contrary to conventional (blood flow encoding) Doppler. TDI focuses on lower velocity frequency shifts.

TISSUE VELOCITY TRACINGS – apical four-chamber view/TDI

Color tissue Doppler imaging of a normal patient with velocity tracings of the basal septal and basal lateral segments



TISSUE DOPPLER IMAGING

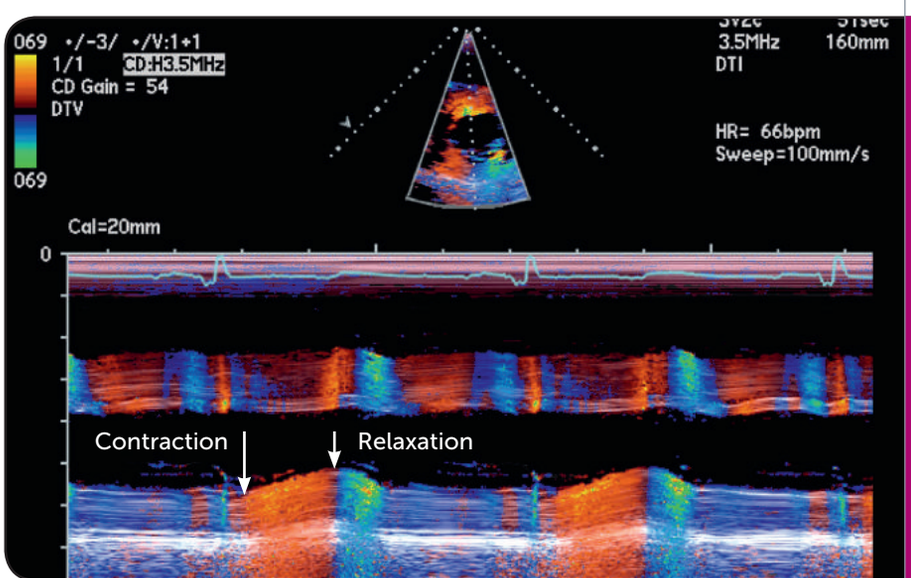
Tissue Doppler Image Acquisition

Spectral Doppler

- Place the sample volume in the region of interest of the myocardium. Make sure that the sample volume is inside the myocardium throughout the cardiac cycle.
- Adapt your sweep speed (slow sweep speed for the assessment of peak values in several beats and high sweep speed for measuring slopes in a few beats).
- Reduce the gain.
- Align the beam to the direction of the interrogated motion.
- On apical views, tissue velocity measurements are performed at the annulus and the basal end of the basal and mid levels of the different walls.

Color Doppler

- High frame rates are needed (> 100 frames/sec).
- Reduce depth and sector width (of both gray scale and Doppler sector) to improve frame rates.
- Avoid reverberation artefacts.
- Record at least 3 beats.



TISSUE DOPPLER MMODE – PSAX/TDI & MMode

MMode for a parasternal short-axis view at the papillary muscle level, combined with tissue Doppler imaging. This form of display can be used to accurately time the start of volumetric contraction and relaxation (arrows).

NOTES

Use the sector tilt function on your scanner; it permits better alignment of the tissue Doppler sample volume with the direction of myocardial motion.

NOTES

With the advance of speckle tracking, TDI has lost much of its appeal. It is only used for few indications.

Global longitudinal contractile function can also be assessed with the conventional MMode, by measuring the excursion of the mitral annular plane during systole (MAPSE).

CURVED MMODE – apical four-chamber view/curved MMode & TDI

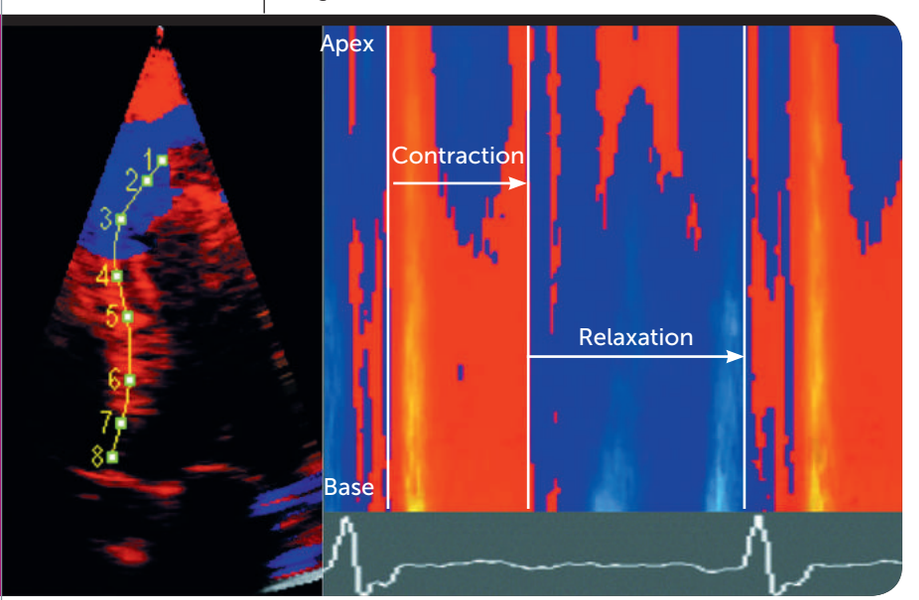
Curved M-mode is a color display format in which functional information (such as velocities, strain, strain rate) concerning different segments of the heart (such as the 4-chamber view) are displayed along an M-mode line, which follows the myocardial walls. The M-mode line „curves" around the myocardium. Starting at the basal inferior segment, it moves to the apex and back to the basal lateral wall. The functional information is color coded.

Modified image views should be used whenever necessary to achieve the optimal imaging angle.

TISSUE DOPPLER IMAGING

Clinical Applications of Tissue Doppler Imaging

Clinical Setting	Measure
Diastolic function	Spectral TDI at the mitral valve annulus (medial + lateral) for the assessment of filling pressures (E/E')
Right ventricular function	Spectral TDI on the lateral side of the tricuspid valve annulus for the assessment of basal right ventricular function (S')
Constriction vs. Restriction	Spectral TDI at the mitral valve annulus shows a large E' in constriction (> 8cm/s) vs. a small E' in restriction (usually below 3cm/s)
Dyssynchrony	Spectral and color Doppler TDI help to quantify and visualize dyssynchronous motion between various segments



Limitations of Tissue Doppler Imaging

- Tissue Doppler velocities may be influenced by global heart motion or by the movement of adjacent structures.
- Imaging artifacts may interfere significantly with TDI accuracy.
- The position of the baseline is automatically defined as the value at the beginning of the QRS complex and might therefore be incorrect under certain conditions (e.g. bundle branch block, suboptimal ECG, atrial fibrillation).
- Tissue Doppler is angle dependent.

SPECKLE TRACKING ECHOCARDIOGRAPHY

- Speckles on the 2D image are stable and have unique myocardial features.
- Speckles result from interference due to the backscatter of the ultrasound wave from structures smaller than the length of the wave.
- Speckles can be tracked from frame to frame and provide information about local displacement, from which parameters of myocardial function (e.g. strain, strain rate) can be derived.
- Speckle tracking is an offline technique applied to recorded 2D images.
- Different components of contraction (longitudinal, circumferential and radial motion) can be studied separately.
- Peak systolic strain is generally used to quantify contractility. It is defined as the maximal shortening (at any region of the myocardium) during systole.

NOTES

Subendocardial function is largely regulated by longitudinal contraction, and may be impaired before the circumferential or radial component deteriorates. Thus, longitudinal function serves as an early marker of left ventricular dysfunction.

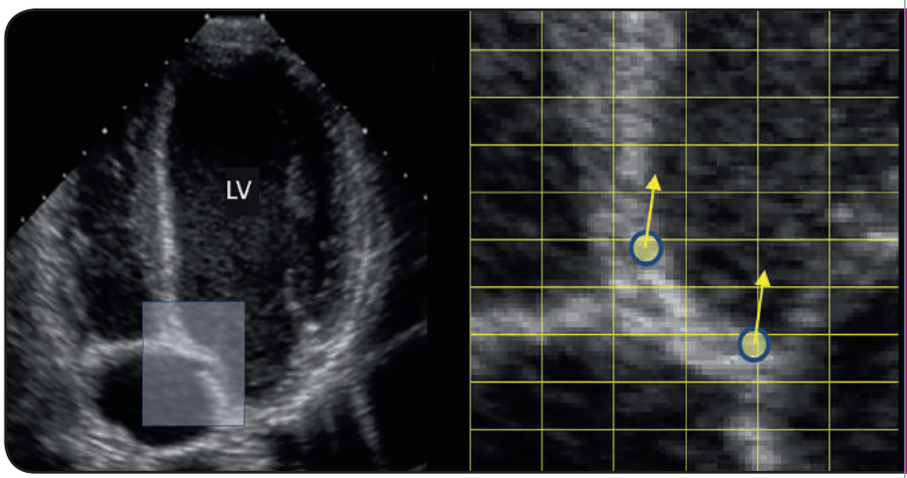


ILLUSTRATION OF MYOCARDIAL SPECKLES – apical four-chamber view/2D

Speckle tracking imaging monitors the local displacement of myocardial speckles and uses the obtained information to derive parameters of myocardial function.

2D Image Acquisition for Speckle Tracking Echocardiography

- Longitudinal strain is calculated from apical views and circumferential strain from short-axis views.
- Frame rates around 80 frames/sec are advised. Low frame rates result in the loss of speckles, whereas high frame rates reduce spatial resolution and image quality.
- Position the focus point at an intermediate depth.
- Adjust sector depth and width to include as little as possible of the areas outside the region of interest.
- Avoid artifacts (any artifact that looks like a speckle pattern will influence the quality).
- Avoid apical foreshortening (apical views) and oval images (short-axis views).

Make sure you have a good ECG signal. Avoid ectopic beats.

NOTES

When aortic valve closure cannot be seen accurately, use a Doppler signal (PW or CW) of the left ventricular outflow to determine aortic valve closure.

For strain representation on a bull's eye display, you need to assess all apical views. All acquired views should have approximately the same cycle length. This may be a problem even in normal (usually young) individuals who have sinus arrhythmia.

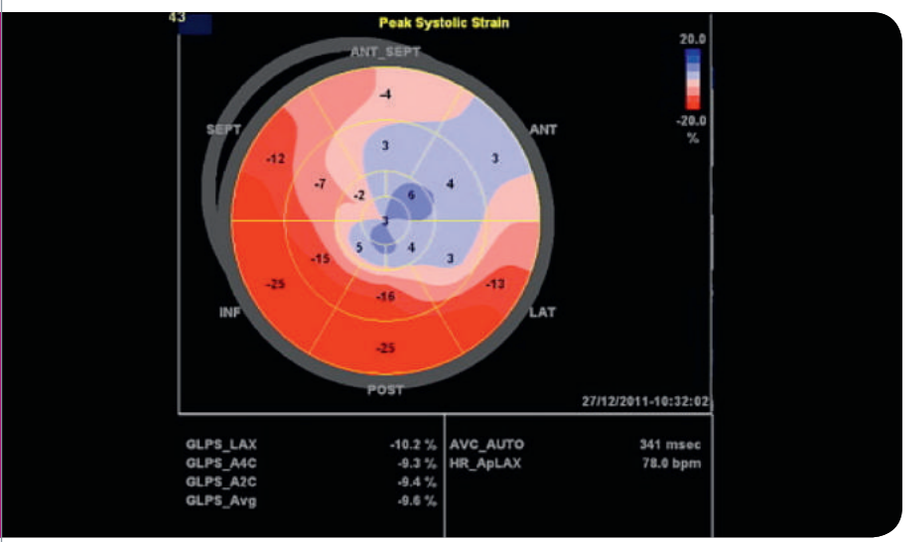
SPECKLE TRACKING ECHOCARDIOGRAPHY

Analysis of Speckle Tracking Images

- Assessment of speckle tracking strain is a semiautomatic method, but requires manual definition of the endocardial border of the myocardium.
- The region of interest should cover most of the myocardial wall thickness. The pericardium should be avoided.
- Adjust the region of interest manually until optimal tracking is accomplished.
- Define end systole (aortic valve closure as seen on the apical long-axis view).
- Speckle tracking strain can be obtained for both ventricles and the atria.
- Strain can be obtained for each individual segment (segmental strain) by averaging all segments (global strain), or for each of the theoretical vascular distribution areas (territorial strain).
- Segmental strain is typically shown in a bull's eye representation.

BULL'S EYE REPRESENTATION – apical views/2D STE

Bull's eye representation of segmental peak systolic longitudinal strain in a patient with anterior myocardial infarction. Longitudinal contraction is significantly impaired in the apical region, the anterior wall and the anterior septum, with preserved longitudinal contraction in the remaining segments. The global average longitudinal strain is reduced (-10%).



Advantages of Speckle Tracking Echocardiography over Tissue Doppler

- STE is angle independent
- Only reflects active contraction (no tethering effects)
- More robust and less influenced by frame rate
- Easy to perform
- All components of myocardial deformation can be assessed

Strain and strain rate are not load independent.

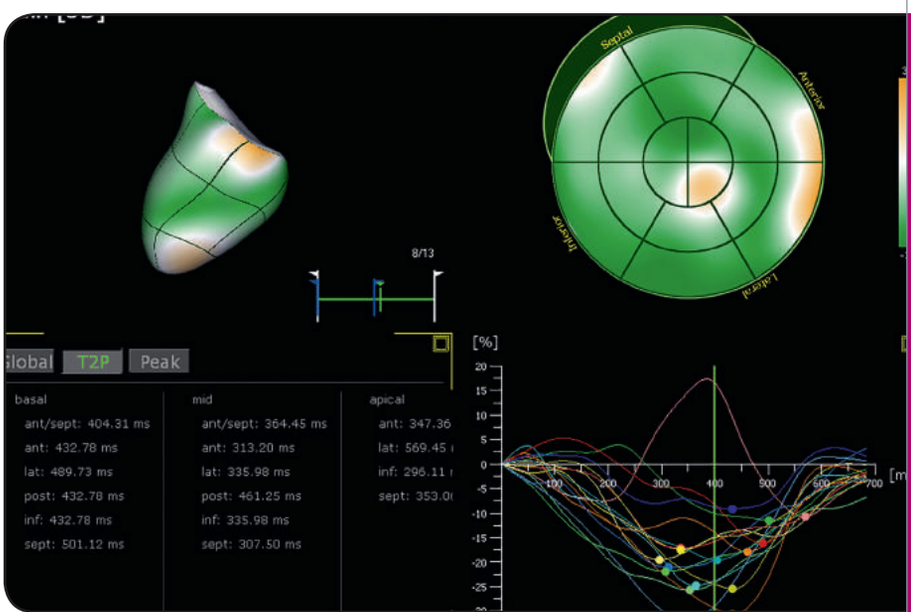
Limitations of Speckle Tracking Echocardiography

- Low image quality, imaging artifacts (e.g. acoustic shadowing, reverberations) and suboptimal tracking of the endocardial border may lead to underestimation of myocardial deformation.

SPECKLE TRACKING ECHOCARDIOGRAPHY

Three-Dimensional Speckle Tracking Echocardiography

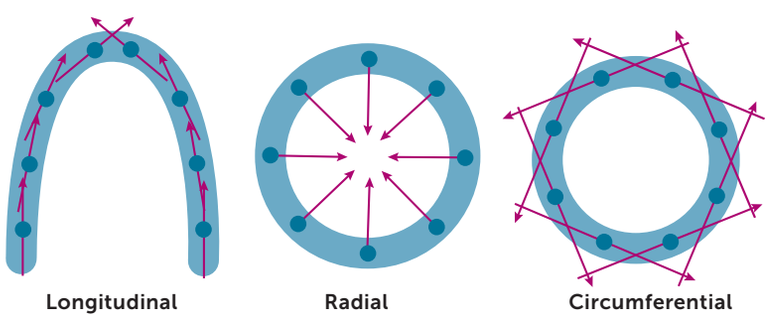
- Speckles can be tracked irrespective of their direction.
- 3D STE results correlate well with strain values derived from MRI
- Relatively low temporal and spatial resolution.
- 3D STE can be assessed in apical 3D full-volume samples acquired over consecutive cardiac cycles during breath hold.
- Information about left ventricular motion (e.g. displacement, rotation) and deformation (e.g. longitudinal/circumferential/radial strain) is calculated automatically.



3D TIME TO PEAK CONTRACTION – Full-volume acquisition/3D

3D full-volume acquisition may be used to assess peak longitudinal function as well as the timing of contraction. The time to peak contraction is shown in this patient.

Directions of Contraction using Speckle Tracking Echocardiography



Three perpendicular axis (i.e. longitudinal, circumferential, and radial) represent the main directions of left ventricular myocardial contraction.

NOTES

Be sure to include the entire LV cavity in the pyramidal 3D full-volume, and always optimize the automatically detected myocardial borders manually.

3D strain is still in its infancy. Its major limitations are low frame rates, stitching artifacts, and vendor dependency.

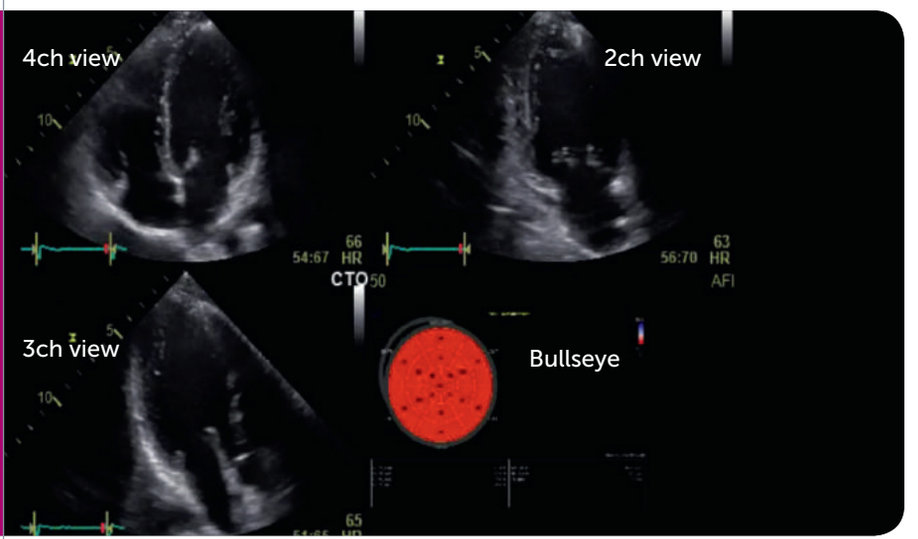
NOTES

SPECKLE TRACKING ECHOCARDIOGRAPHY

Longitudinal Strain

GLOBAL LEFT VENTRICULAR LONGITUDINAL STRAIN – apical views/2D STE

Global left ventricular longitudinal strain is calculated using two-, three-, and four-chamber views. Bull's eye representation (lower right corner) shows normal longitudinal contraction, indicated in red.



As a simplified approach, just remember that normal longitudinal systolic strains are usually ≥ 18%.

The assessment of longitudinal strain is more robust than radial and circumferential strain. Currently it has the greatest impact on clinical echocardiography.

Longitudinal strain is usually higher in the apical region than in the basal region (apical to basal gradient), and higher in subendocardial layers.

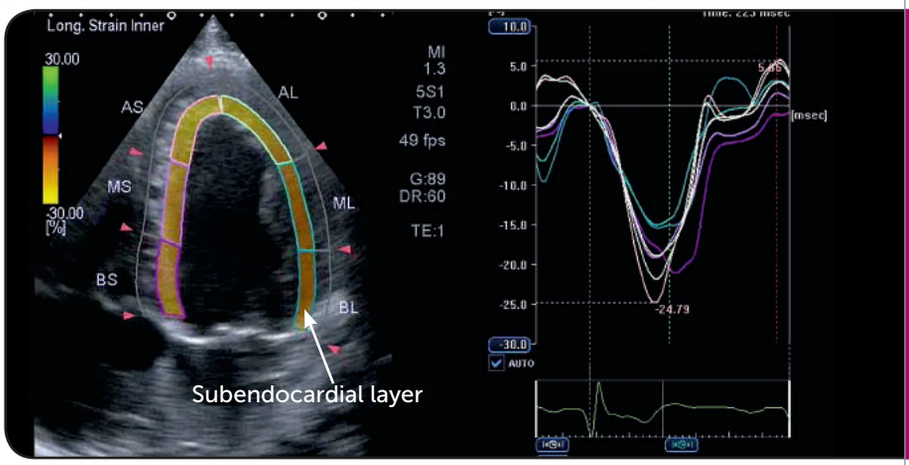
Some propose that quantification of subendocardial longitudinal strain should be the preferred method to study subclinical dysfunction.

Reference Values of Left Ventricular Longitudinal Systolic Strain

	All levels	Apical	Mid	Basal
All walls	−18.6 ± 5.1	−20.2 ± 5.6	−18.7 ± 3.8	−17.0 ± 5.2
Anterior	−19.5 ± 4.2	−19.4 ± 5.4	−18.8 ± 3.4	−20.1 ± 4.0
Anteroseptal	−18.8 ± 4.2	−18.8 ± 5.9	−19.4 ± 3.2	−18.3 ± 3.5
Inferior	−20.0 ± 4.5	−22.5 ± 4.5	−20.4 ± 3.5	−17.1 ± 3.9
Lateral	−18.3 ± 4.7	−19.2 ± 5.4	−18.1 ± 3.5	−17.8 ± 5.0
Posterior	−16.3 ± 6.3	−17.7 ± 6.0	−16.8 ± 5.0	−14.6 ± 7.4
Septal	−18.3 ± 5.3	−22.3 ± 4.8	−18.7 ± 3.0	−13.7 ± 4.0

Marwick et al. JACC Cardiovasc Imaging 2009

SPECKLE TRACKING ECHOCARDIOGRAPHY

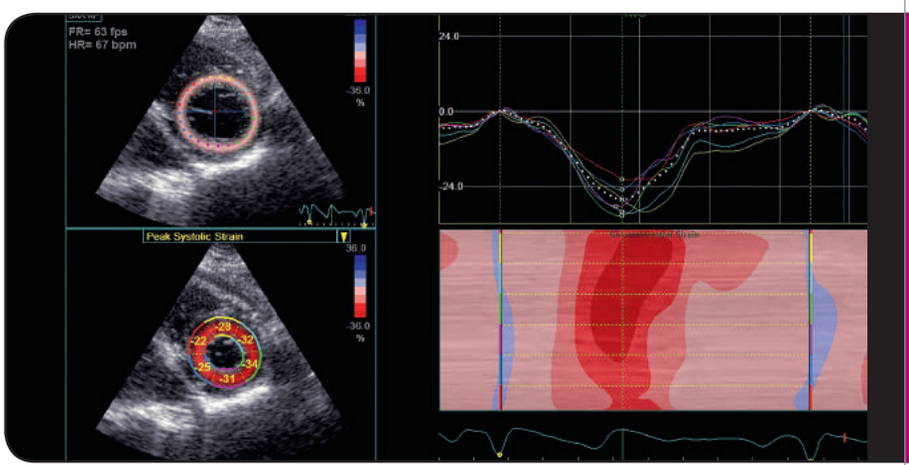


NOTES

SUBENDOCARDIAL TRACKING – apical four-chamber view/STE

Selective quantification of longitudinal strain of the subendocardial (inner) layers of the myocardium.

Circumferential Strain



CIRCUMFERENTIAL STRAIN – PSAX apical/2D STE

Circumferential strain of the apical part of the left ventricle in a normal patient. Peak systolic segmental circumferential strain values are shown in the lower left corner.

Reference Values of Left Ventricular Systolic Circumferential Strain

Segment	Mean peak systolic circumferential strain (%)
Anterior	-24±6
Lateral	-22±7
Posterior	-21±7
Inferior	-22±6
Septal	-24±6
Anteroseptal	-26±11

Reference: Hulburt et al. Echocardiography 2007

Circumferential strain is usually higher in apical and mid-ventricular segments compared to the left ventricular base.

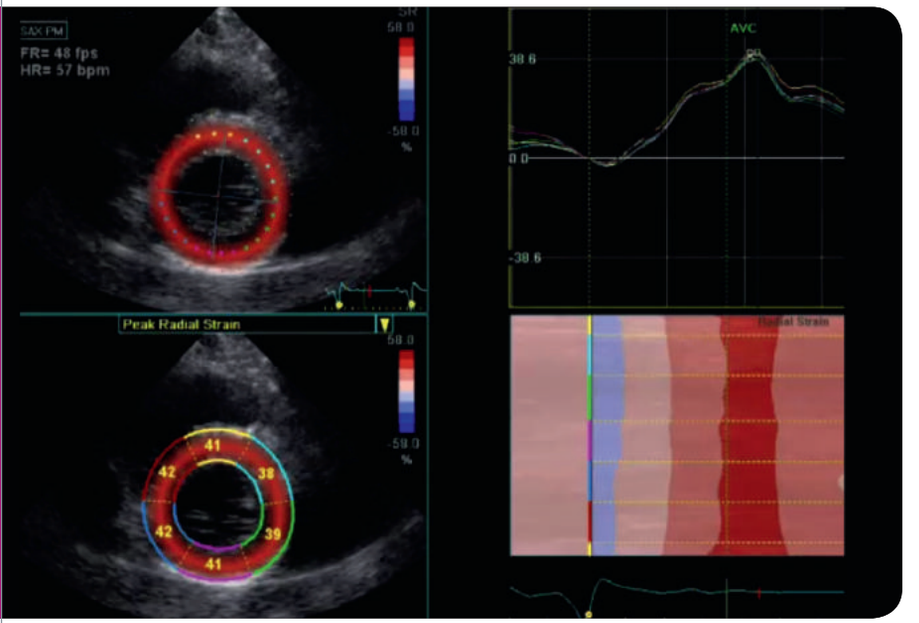
NOTES

SPECKLE TRACKING ECHOCARDIOGRAPHY

Radial Strain

RADIAL STRAIN –
PSAX mid-ventricle/2D STE

Radial strain of the apical part of the left ventricle in a normal patient. Peak segmental radial strain values are shown in the lower left corner.



Radial strain is higher in the subendocardium compared to the subepicardium.

Reference Values of Left Ventricular Systolic Radial Strain

Segment	Mean peak systolic radial strain (%)
Anterior	39±16
Lateral	37±18
Posterior	37±17
Inferior	37±17
Septal	37±19
Anteroseptal	39±15

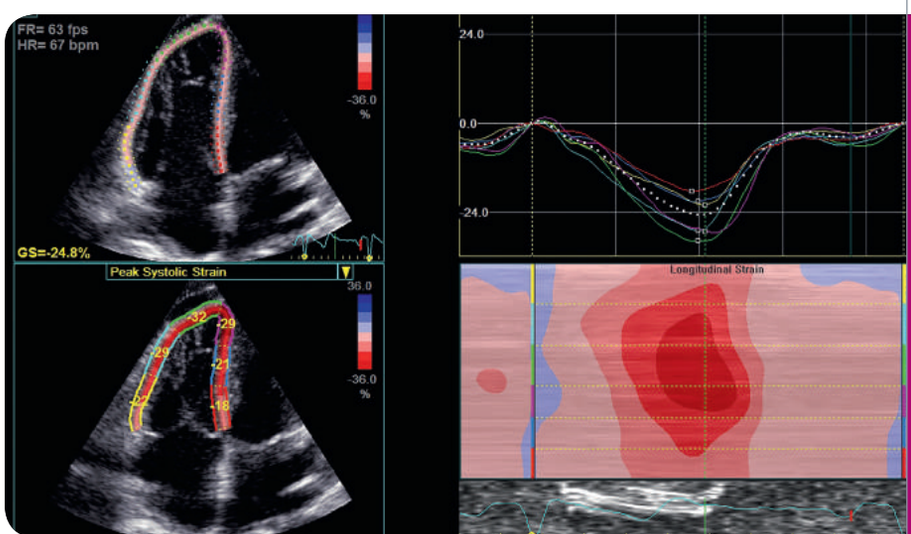
Hulburt et al. Echocardiography 2007

CLINICAL APPLICATIONS OF MYOCARDIAL DEFORMATION IMAGING

Myocardial Deformation in the Assessment of Right Ventricular Function

Principles

- Normal right ventricular contraction is a peristaltic wave directed from the inflow tract to the infundibulum.
- Longitudinal shortening is the key component in overall right ventricular performance, with equal contributions of the free RV wall and the interventricular septum.
- Right ventricular longitudinal strain and strain rate correlate well with radionuclide right ventricular function.
- A right ventricular longitudinal strain ≥ 25% or a right ventricular longitudinal strain rate ≥ -4 sec-1 indicates normal right ventricular function.

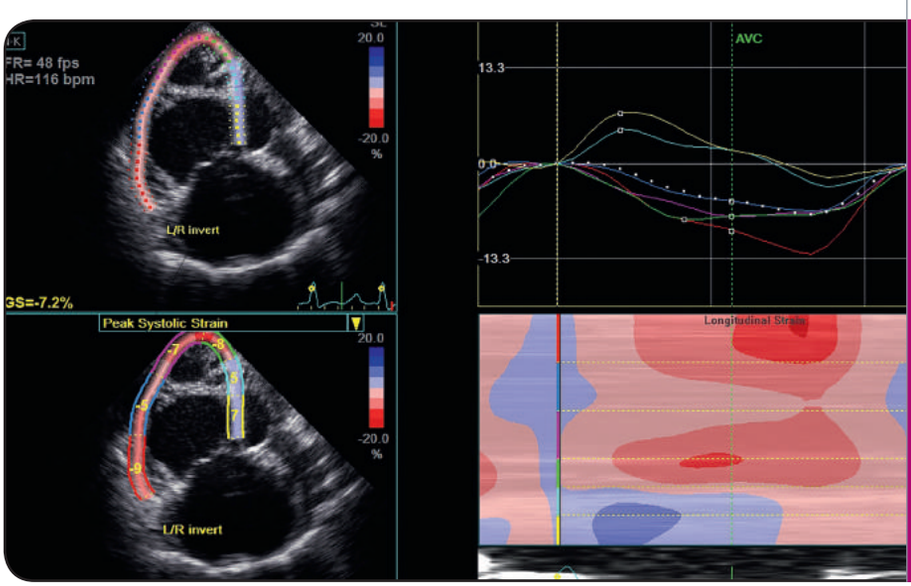


RIGHT VENTRICULAR LONGITUDINAL STRAIN – optimized four-chamber view/2D STE

Longitudinal strain of the right ventricle in a normal patient with a mean longitudinal strain of -24.8%. Peak systolic longitudinal strain values are shown in the lower left corner.

2D Image Acquisition for Speckle-Tracking of the Right Ventricle

- Use an apical four-chamber view optimized for the right ventricle.



RV STRAIN IN PULMONARY HYPERTENSION - optimized four-chamber view/2D STE

Reduced right ventricular longitudinal strain in a patient with severely reduced right ventricular function due to severe pulmonary hypertension. Peak systolic longitudinal strain values are shown in the lower left corner; the mean longitudinal strain is -7.2%.

NOTES

The use of right ventricular strain has not been fully validated for clinical practice.

NOTES

CLINICAL APPLICATIONS OF MYOCARDIAL DEFORMATION IMAGING

Myocardial Deformation Imaging in Various Clinical Settings

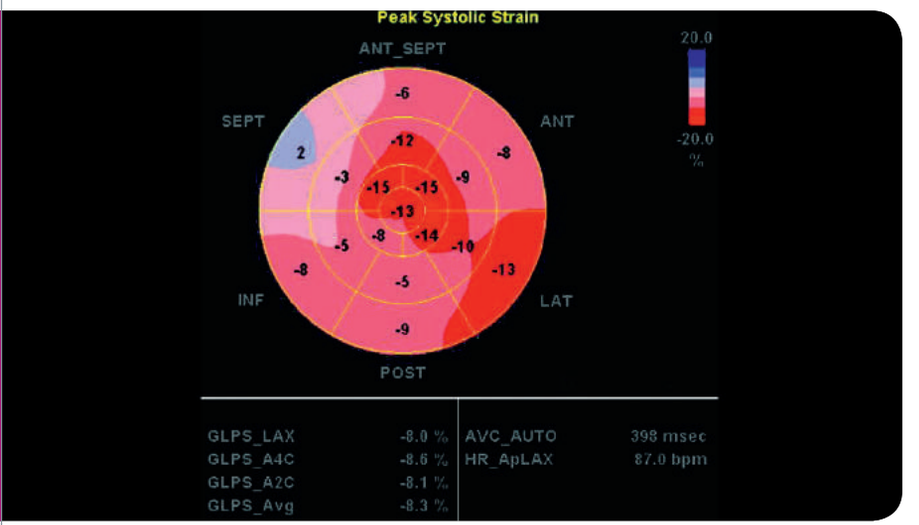
An increasing body of data shows that deterioration of longitudinal strain is an early marker of left ventricular dysfunction, and that it could be an important parameter for the timing of valve surgery (such as surgery for aortic stenosis).

Aortic Stenosis

- Decreased longitudinal strain (especially in the basal regions) and increased circumferential strain
- Reduced left ventricular twist
- Global longitudinal strain correlates with the severity of aortic stenosis and exercise tolerance
- Impairment in longitudinal contraction is partly reversible after aortic valve replacement

AORTIC STENOSIS – apical views/2D STE

Bull's eye presentation of segmental longitudinal strain in a patient with severe asymptomatic aortic stenosis and normal systolic left ventricular function (ejection fraction > 60%). Global longitudinal systolic function is significantly reduced, especially in the basal segments.



Aortic Regurgitation

- Reduction in longitudinal and radial strain and strain rate
- Reduction improves after aortic valve replacement

Mitral Regurgitation

- Early left ventricular dysfunction is characterized by a reduction of global longitudinal strain
- Reduction of longitudinal, circumferential and radial strain rate
- Delayed untwisting motion of the left ventricle

Use speckle tracking if you are uncertain about the presence of wall motion abnormalities. In some cases it might even be superior to the naked eye.

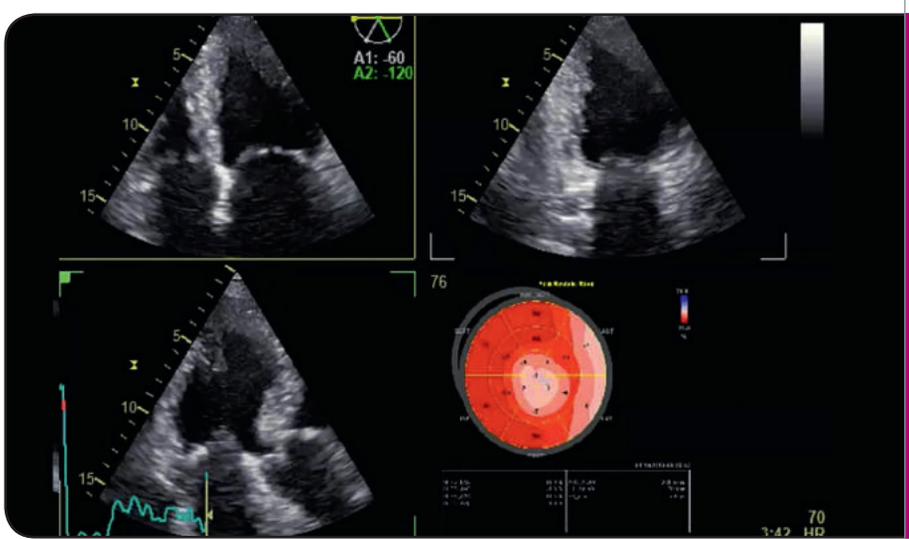
Coronary Artery Disease

- Longitudinal strain is compromised at an early stage in coronary artery disease
- Simplifies the detection of regional wall motion abnormalities
- Pronounced post-systolic shortening after aortic valve closure is a common finding in acute ischemia
- Residual longitudinal strain in akinetic or severely hypokinetic regions indicates sustained viability

CLINICAL APPLICATIONS OF MYOCARDIAL DEFORMATION IMAGING

Hypertrophic Cardiomyopathy

- Longitudinal function is reduced, whereas circumferential and radial function is elevated
- Often paradoxical systolic lengthening detectable
- Regional heterogeneity (typically basal and mid septal longitudinal strains most affected)



APICAL HYPERTROPHIC CARDIOMYOPATHY – apical views/2D STE

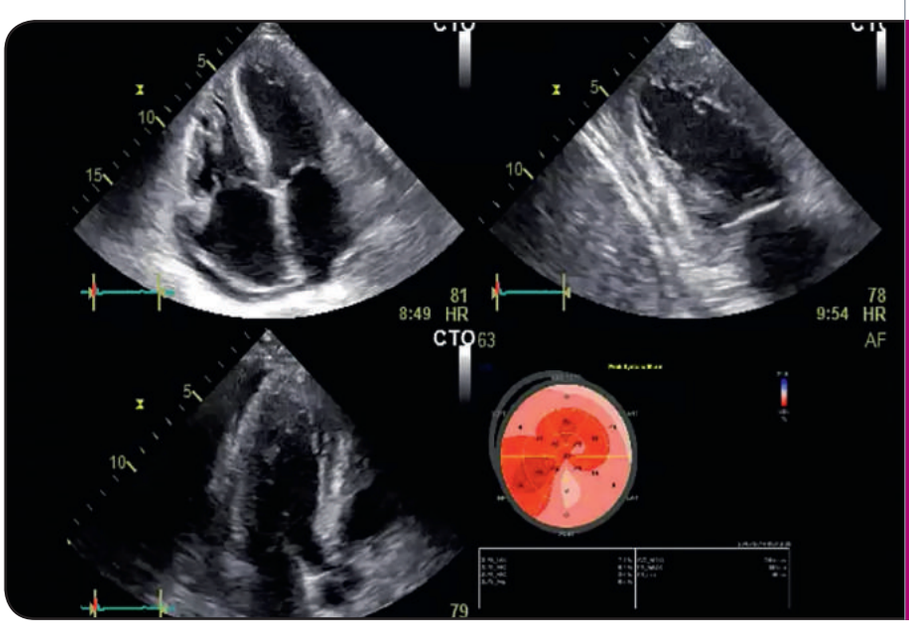
Typical strain pattern in a patient with apical hypertrophic cardiomyopathy. Strain is reduced at the apex in the region of hypertrophy.

Dilated Cardiomyopathy

- Reduced strain in all directions
- Reduced left ventricular twist/torsion

Restrictive Cardiomyopathy

- Reduced longitudinal strain, but preserved circumferential strain
- Preserved left ventricular twist/torsion



AMYLOIDOSIS – apical views/2D STE

Typical longitudinal strain pattern in a patient with amyloidosis. Longitudinal strain is preserved at the apex and severely reduced in (most of) the mid and basal segments.

NOTES

In hypertrophic cardiomyopathy, longitudinal strain is most severely reduced in areas of pronounced wall thickness and fibrosis.

NOTES

CLINICAL APPLICATIONS OF MYOCARDIAL DEFORMATION IMAGING

Constrictive Pericarditis

- Preserved longitudinal function but reduced circumferential strain
- Reduced left ventricular twist/torsion

Dyssynchrony

- Allows quantification of dyssynchrony and has the potential to optimize cardiac resynchronization therapy (CRT)

Left Atrial Deformation

- Correlates with the recurrence of atrial fibrillation after radiofrequency catheter ablation

Hypertensive Heart Disease

- Reduced basal longitudinal strain
- Reduced strain, especially of the basal anterior septum

Patients with hypertensive heart disease and left ventricular function frequently show reduced longitudinal function despite a normal ejection fraction.

NOTES