

# Echo examen TEE

- Inhoud
  - TEE
  - Echocardiografie theorie
  - Boek Sidebotham
  - Guidelines

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## Method

A 9 mm nonfocused Aerotech 3.5 MHz transducer was designed and instrumented to permit easy swallowing by

adults (fig. 1). The transducer is placed in a 1.9 cm  $\times$  1.3 cm  $\times$  0.6 cm casing with rounded edges for easy esophageal passage. Its blunt end is attached to a calibrated 3 mm coaxial cable which permits sufficient control of rotation at 30–40 cm length, the level of cardiac echoes.

Prior to the study, subjects were fasted. After gargling 20 cc of 2% viscous xylocaine, they swallowed the transducer. Subjects usually fed the transducer to themselves. Most found the procedure innocuous, but three of the 38 subjects did complain of discomfort. No complications were encountered. Cardiac echoes were easily obtained, and the entire procedure required approximately 10 minutes. The transducer's position was identified by the aortic root echo. From this position, advancement and a small degree of left lateral rotation were used to scan the anterior mitral valve leaflet. Esophageal echoes were obtained in sitting and supine positions, and recorded on standard echocardiographic equipment. Figure 2 shows a PA and lateral chest X-ray of the esophageal transducer in place.

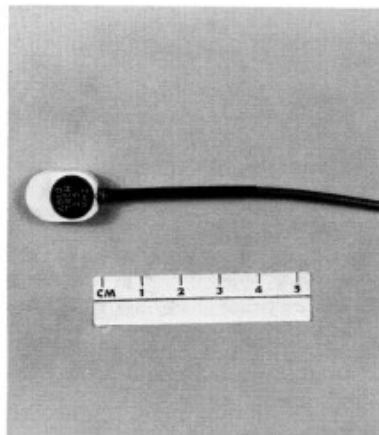


FIGURE 1. Photograph of esophageal 3.5 MHz nonfocused transducer.

# Contra-indicaties

- Absoluut
  - Aortadissectie type A!
  - Oesofagus
    - Obstructie
    - Divertikel, laceratie, fistel
  - Actieve maag- of oesofagusbloeding
  - Niet nuchter ( < 4 uur)
    - < 6 uur bij DM (cave gastropathie)
  - Allergie lidocaine / midazolam

# Contra-indicaties

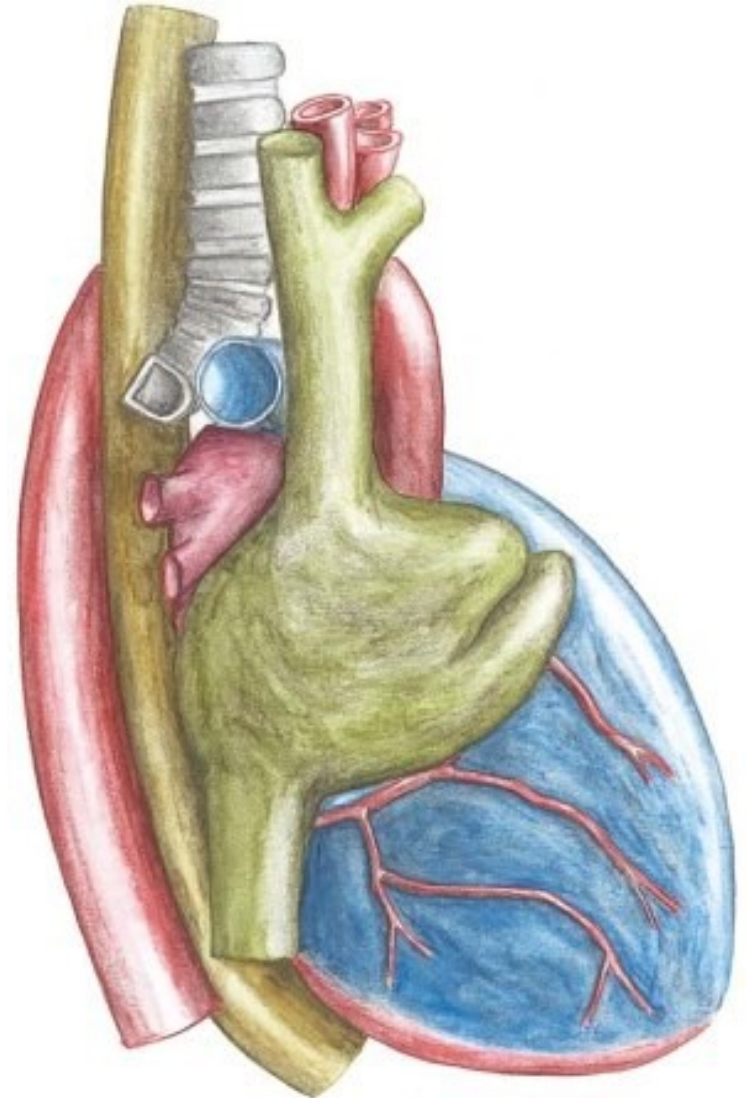
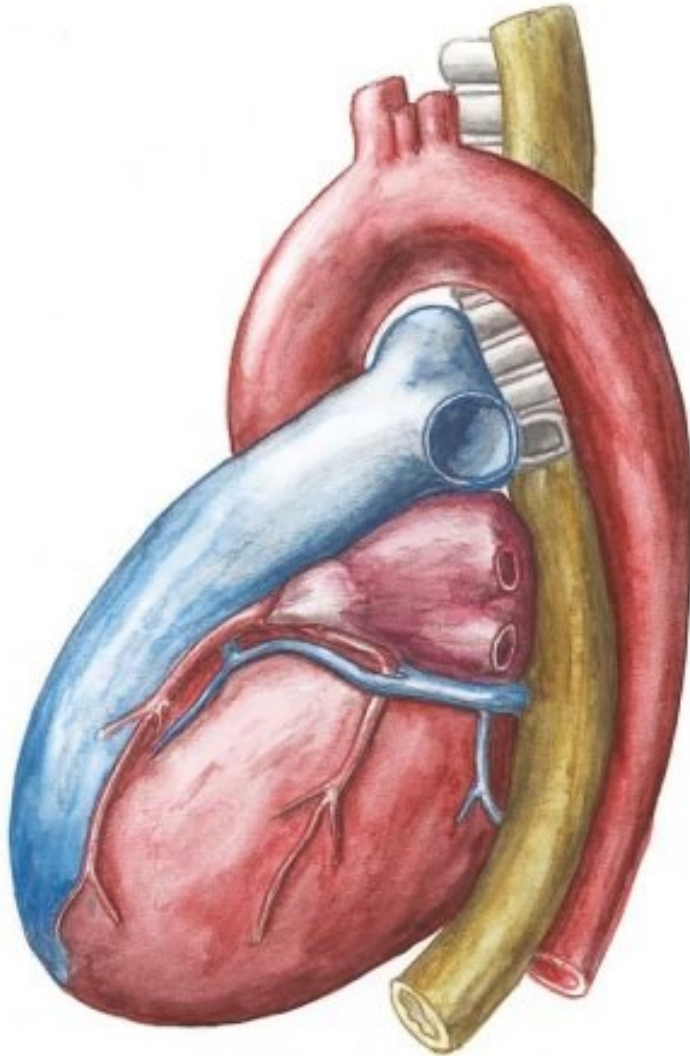
- Relatief
  - Oesofagitis
  - Oesofagusvarices
  - Recente maagchirurgie
  - Maagulcus
  - Hiatus hernia
  - Aandoening cervicale wervelkolom met beperkte flexie
  - Dysmorphie gebit/mond-keelholte met bemoeilijkte passage
  - Verhoogde bloedingsneiging
    - Trombocytopenie  $< 50$ , INR  $> 4$

# Probe

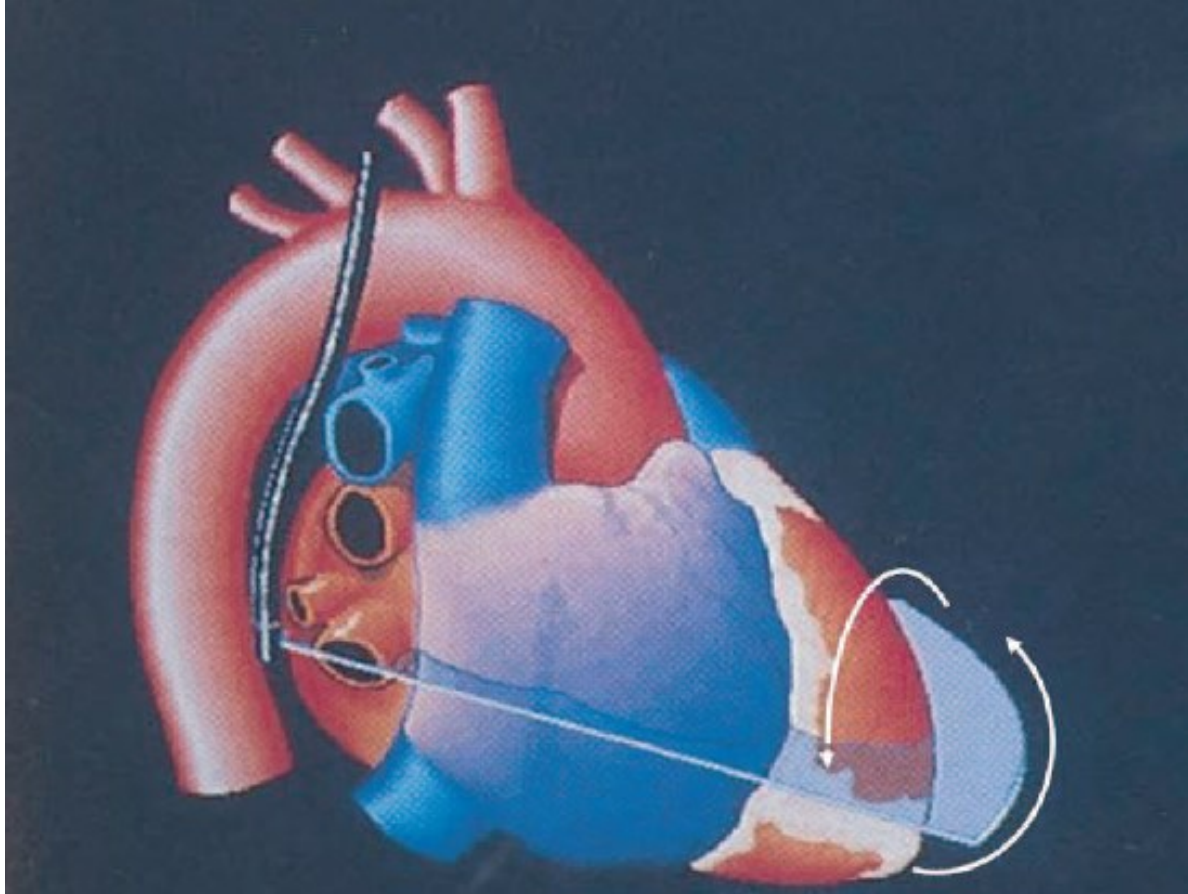
- Draaiing
- Plane rotation (graden)
- Anteflexie/retroflexie
- Sideward flexion



# Anatomie



# Anatomie



Transversaal  $0^\circ$  links = Re pt

Longitudinaal  $90^\circ$  links = onder pt

# Vorbereiding

- Gel in beschermhoes voor probe
  - Impermeabel voor micro-organismen
  - Tip luchtvrij
- Mondbit, kunstgebit verwijderen
- Lidocainegel op probe
- Uitleg patient
  - Na onderzoek 1 uur niet eten/drinken, daarna water
  - Na 1 dag nog klachten: contact afdeling
- Eventueel midazolam
  - O<sub>2</sub>, SpO<sub>2</sub>, RR, short stay verkoever



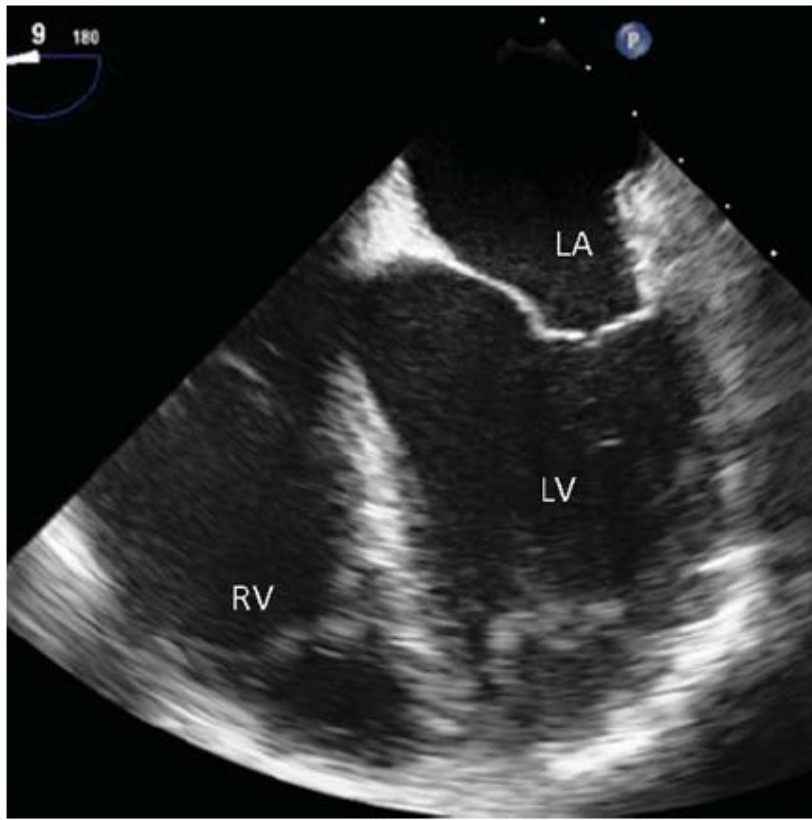
# Verslaglegging

- Introductie probe
- Atriale septum
- LAA
- Kleppen
  - Aortaklep
  - Mitralisklep
  - Tricuspidalisklep
- Aorta thoracalis

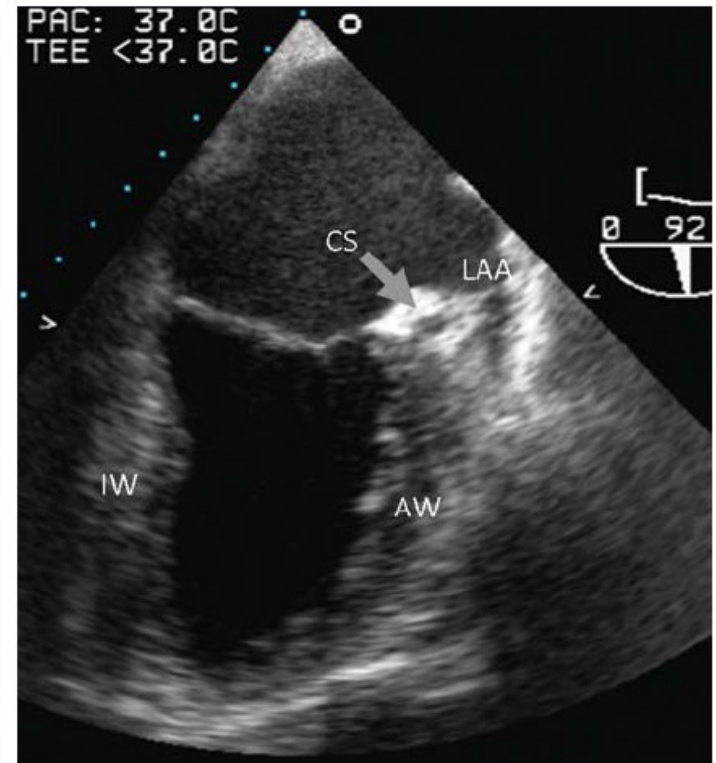
# Opnames

- EAE update 2010
  - Transoesofageaal
  - Transgastrisch
  - Aorta
- Duur afhankelijk van tolerantie patiënt en vraagstelling

# Transoesophageaal

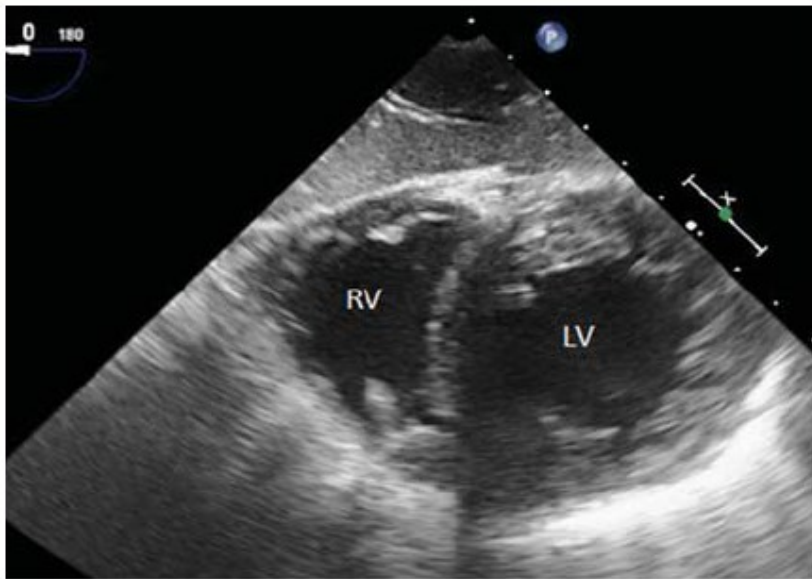


**Figure 2** Transoesophageal four-chamber view. LA, left atrium.

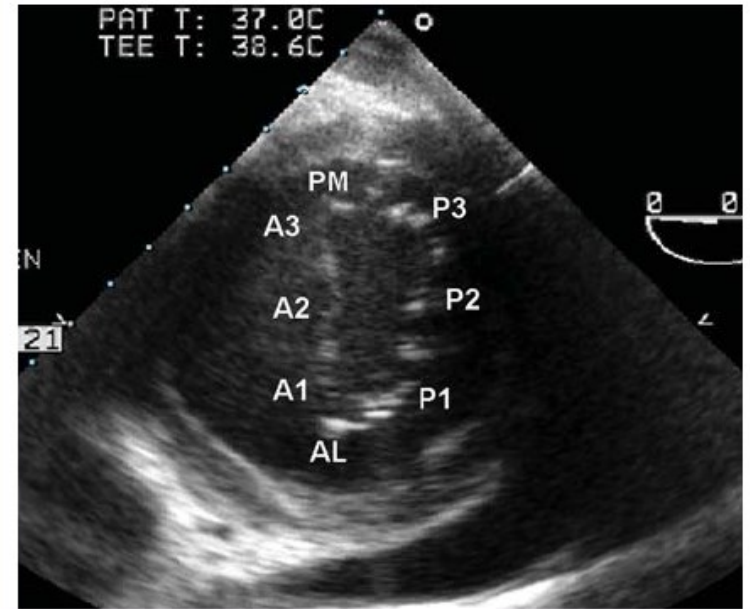


**Figure 3** Transoesophageal two-chamber view. AW, anterior wall; IW, inferior wall; LAA, left atrial appendage; CS, coronary sinus.

# Transgastrisch

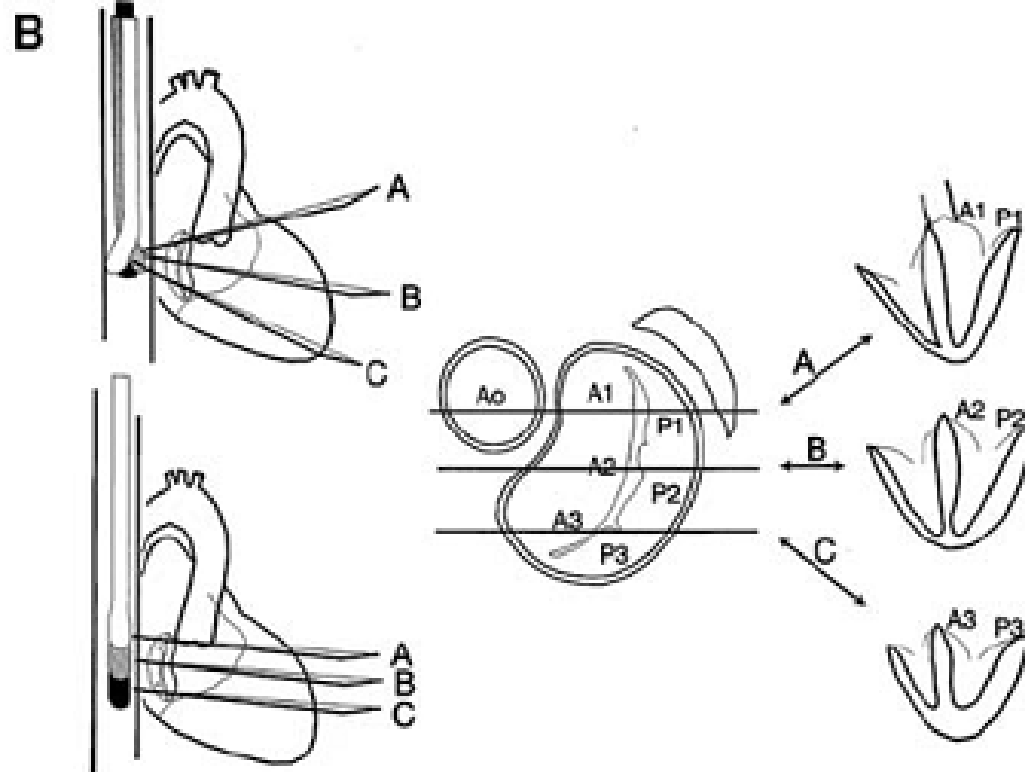


**Figure 13** Transgastric short-axis view of the left (LV) and right ventricle (RV).

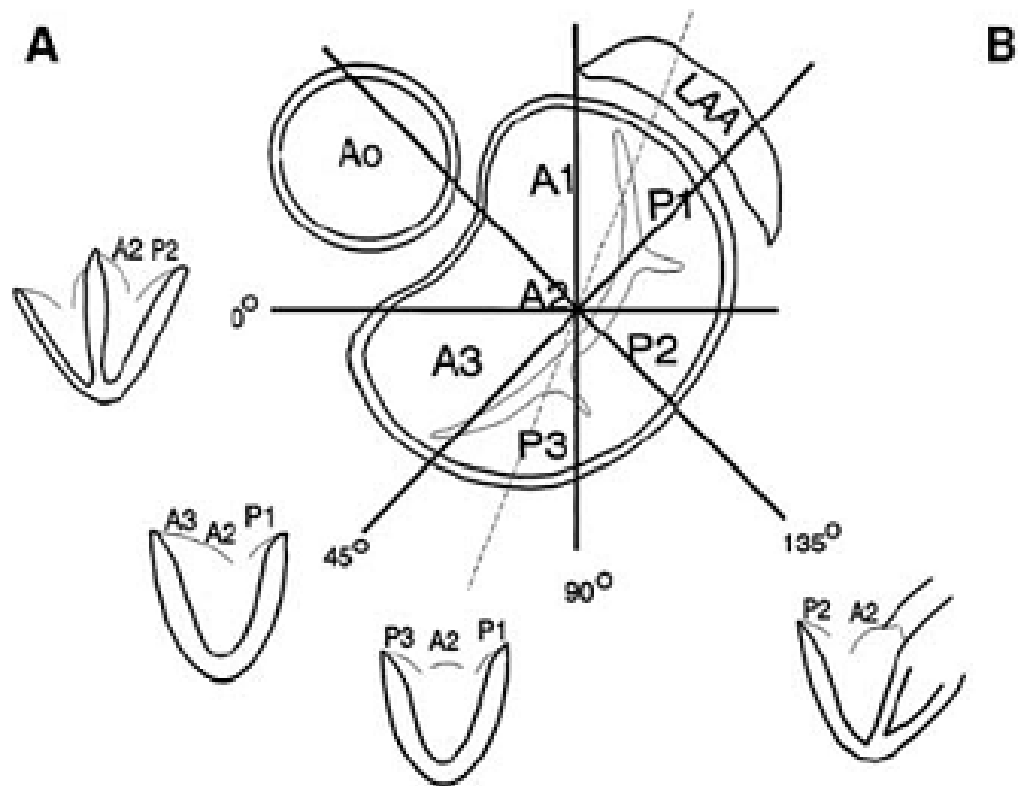


**Figure 17** Short-axis view of the open mitral valve from the transgastric position. AL, anterolateral; PM, posteromedial commissure. A1–A3 and P1–P3 denominate the respective leaflet scallops.

# Mitralisklep

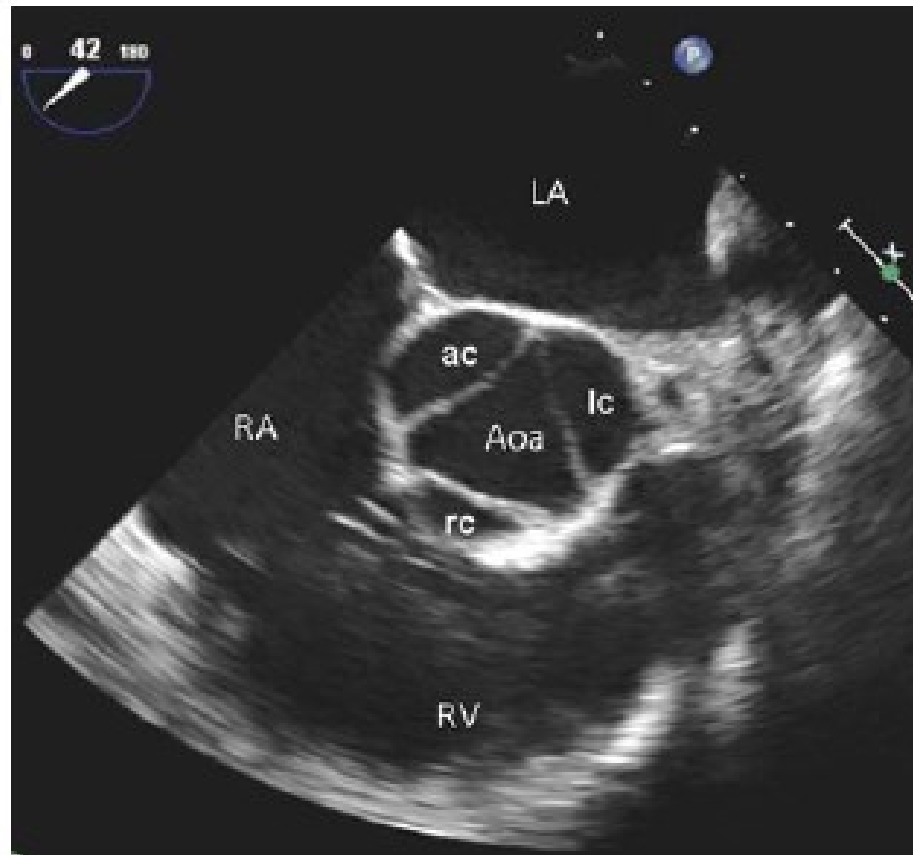


**Figure 20** Examination of the mitral valve. Screen depiction of relative position of mitral leaflets and segments/scallops in typical transoesophageal cross-sections created by three different examination manoeuvres. Note that individual anatomy, especially scallop morphology, is variable, and so is the relation of image plane orientation to individual anatomy; the schematic drawings should therefore be understood as approximations. A1–A3, anterior leaflet segments; P1–P3, posterior leaflet segments; Ao, aortic valve; LAA, left atrial appendage. (A) Examination by rotation of imaging cross-section with fixed transducer position positioned at the level of the mitral valve centre. (B) Examination by flexion/withdrawal and retroflexion/advancement of the transesophageal transducer, while rotation angle is fixed in a transverse orientation ( $0^\circ$ ). (C) Examination by probe shaft rotation (counterclockwise from plane A to C), while rotation angle is fixed in an orientation approximating the mitral closure line ( $45\text{--}90^\circ$ ). Note that the aortic valve is not imaged in these planes. Reproduced, with permission, from Foster et al.<sup>47</sup>



**Figure 20** Examination of the mitral valve. Screen depiction of relative position of mitral leaflets and segments/scallops in typical transoesophageal cross-sections created by three different examination manoeuvres. Note that individual anatomy, especially scallop morphology, is variable, and so is the relation of image plane orientation to individual anatomy; the schematic drawings should therefore be understood as approximations. A1–A3, anterior leaflet segments; P1–P3, posterior leaflet segments; Ao, aortic valve; LAA, left atrial appendage. (A) Examination by rotation of imaging cross-section with fixed transducer position positioned at the level of the mitral valve centre. (B) Examination by flexion/withdrawal and retroflexion/advancement of the transesophageal transducer, while rotation angle is fixed in a transverse orientation (0°). (C) Examination by probe shaft rotation (counterclockwise from plane A to C), while rotation angle is fixed in an orientation approximating the mitral closure line (45–90°). Note that the aortic valve is not imaged in these planes. Reproduced, with permission, from Foster et al.<sup>47</sup>

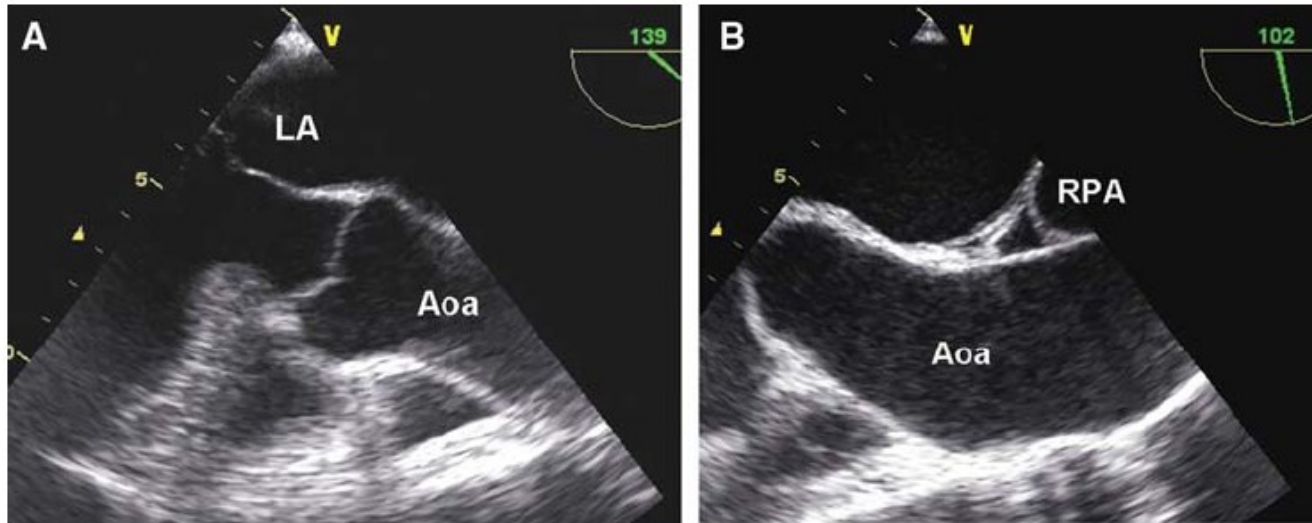
# Aorta(klep)



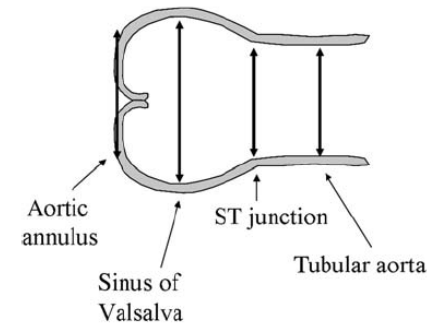
**Figure 5** Aortic valve short-axis view (ac, a coronary; lc, left coronary; rc, right coronary cusp and sinus).



# Aorta(klep)

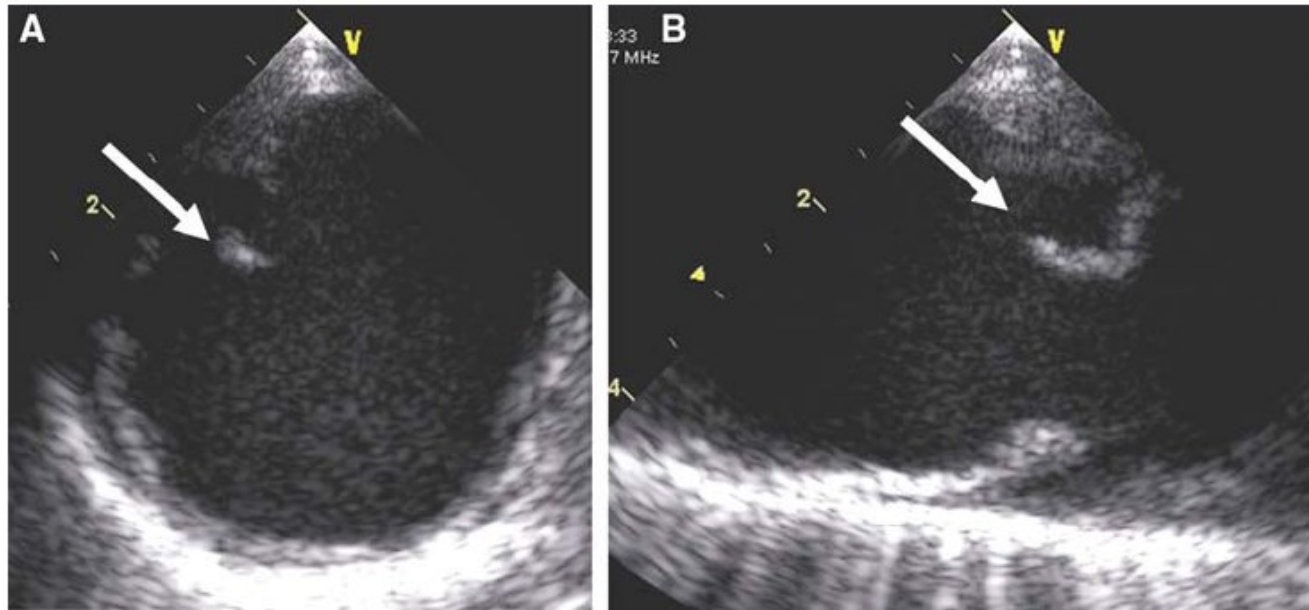


**Figure 7** Long-axis view of the ascending aorta. (A) Proximal ascending aorta. (B) In the same patient, after retraction of the probe and adjustment of the plane orientation, a long portion of the dilated ascending aorta is seen. RPA, right pulmonary artery.



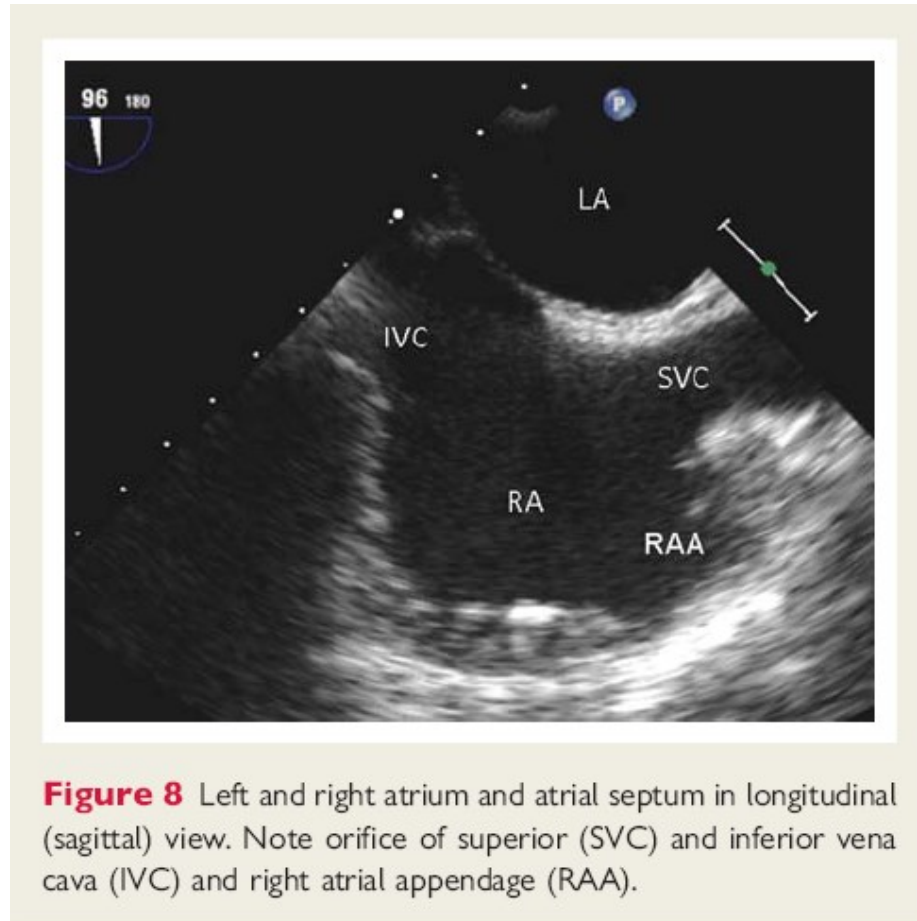
**Figure 24** Typical measurements of the aortic root apparatus. ST, sinotubular.

# Aorta(klep)

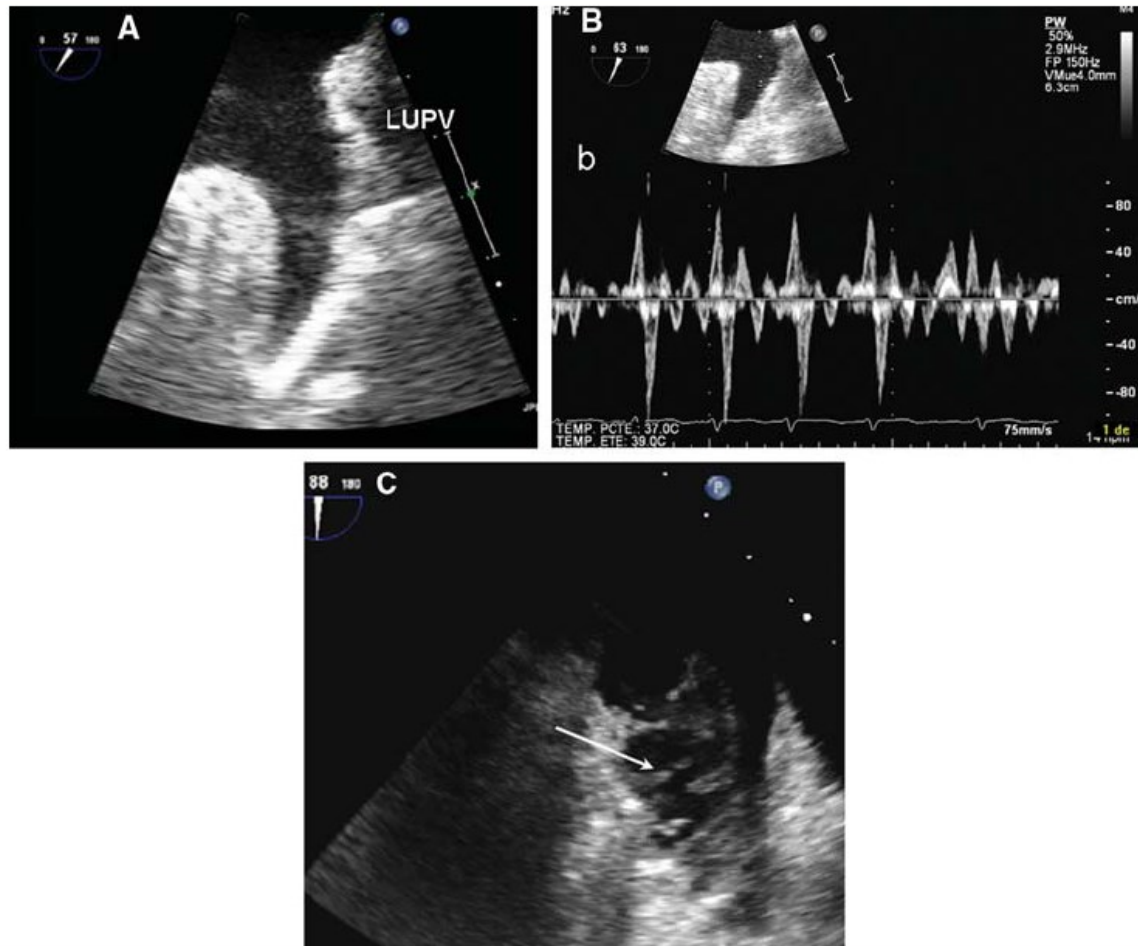


**Figure 18** Descending aorta: (A) short-axis view; (B) long-axis view. The aorta shows atherosclerotic lesions, some of which (arrow) have superimposed mobile thrombus.

# Cardiale emboliebron 2



# Cardiale emboliebron



**Figure 9** (A) Left atrial appendage. (B) Pulsed wave Doppler recording of emptying (upward) and filling (downward) velocities in atrial fibrillation. The velocities are quite high ( $>25$  cm/s), indicating relatively low risk of thrombus generation. LUPV, left upper pulmonary vein. (C) Example of left atrial appendage with marked pectinate muscles (arrow). There is no thrombus.

# Virtueel

- <http://pie.med.utoronto.ca/TEE/index.htm>

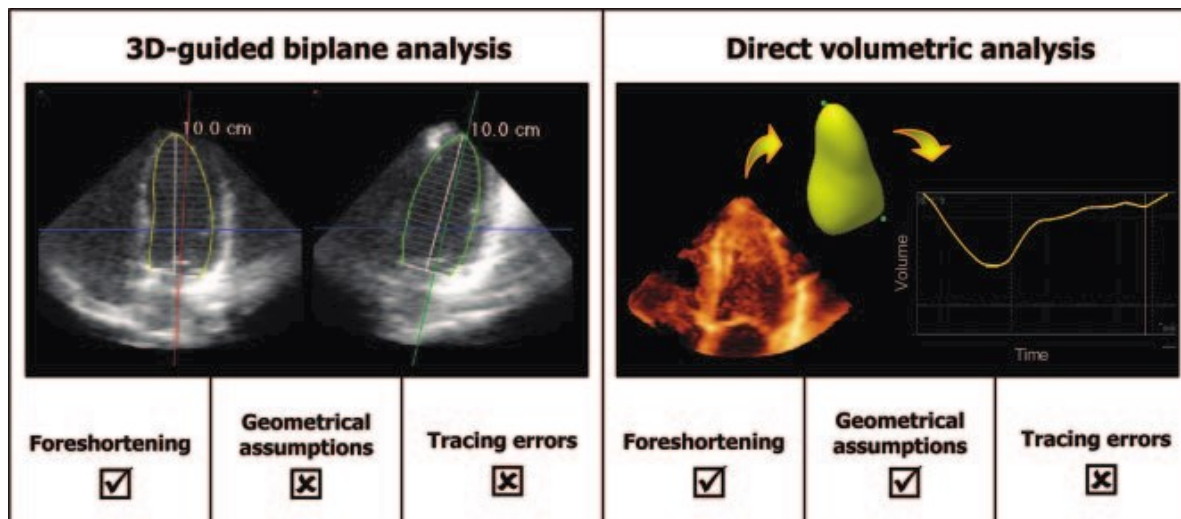


# TEE views

- [http://pie.med.utoronto.ca/TEE/TEE\\_content/TEE\\_guideSheet.html](http://pie.med.utoronto.ca/TEE/TEE_content/TEE_guideSheet.html)

# Klinische toepassing

- Meting LV-volume en ejectionfractie
  - Nadeel 2D: onbetrouwbaar
    - Foreshortening
    - Abnormale vorm of contractie LV
    - Suboptimale scheiding bloed en weefsel

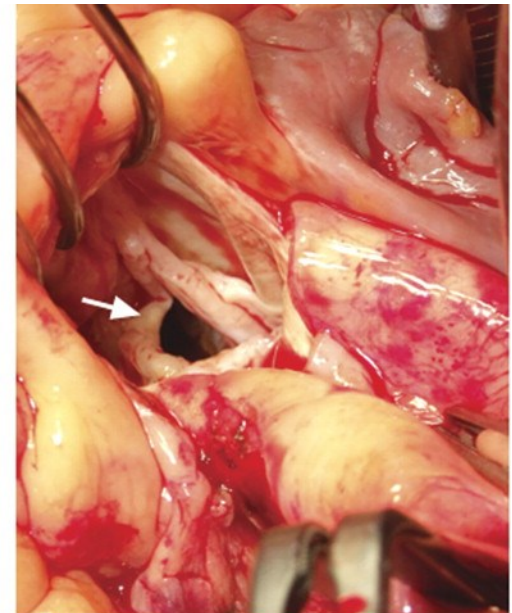
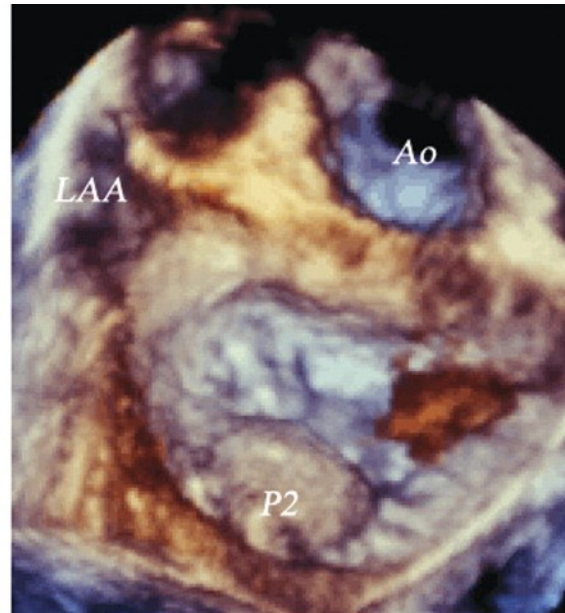
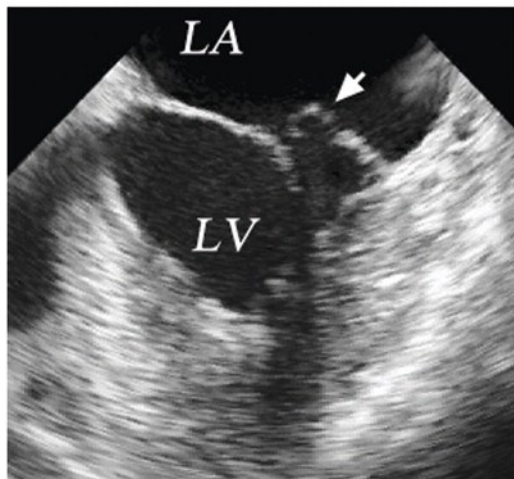


# Klinische toepassing

- LV-massa
- Bepaling RV-volumina en massa
- Atriale volumina
- Resynchronisatie
- Morfologie kleppen
- Aangeboren hartafwijkingen
  
- TEE: peri-operatieve planning van mitralisklepchirurgie

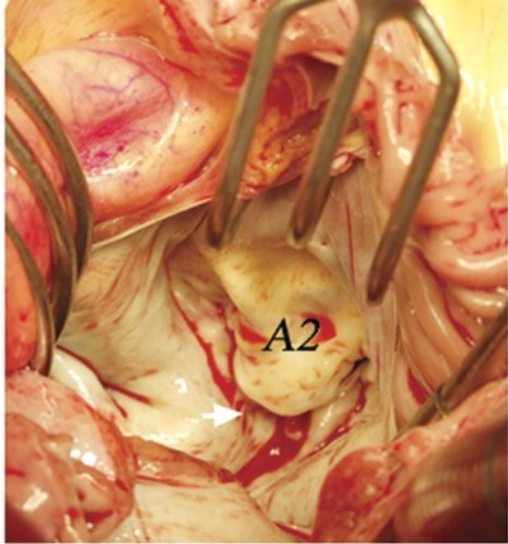
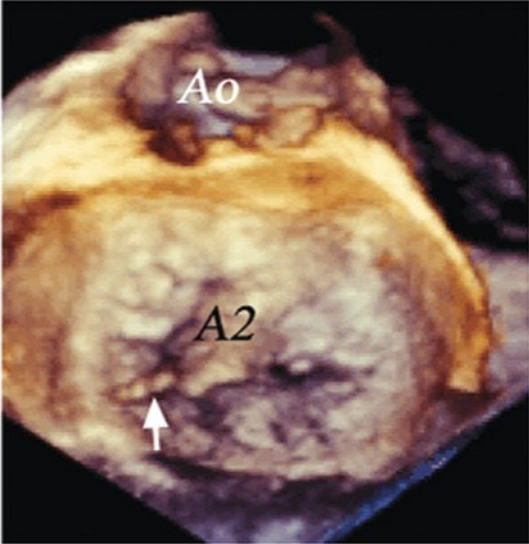
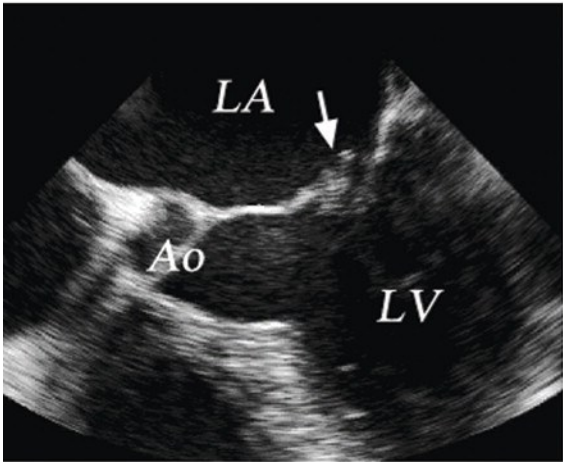


**Left frame: prolapse of the middle segment of the posterior mitral leaflet. 2D TEE shows bulging of PML (arrow).**



**Wei J et al. Eur J Echocardiogr 2010;11:14-18**

**Ruptured chordae tendineae of the middle segment of the anterior mitral valve leaflet (AML).**

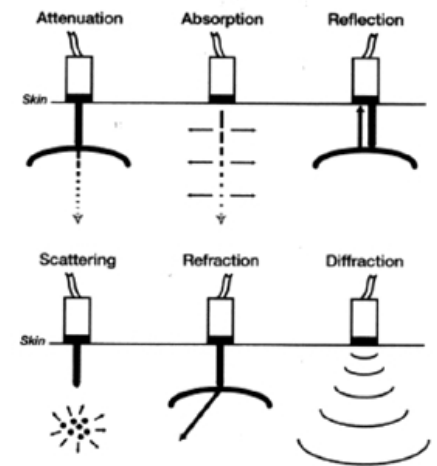


**Wei J et al. Eur J Echocardiogr 2010;11:14-18**

# *Echocardiografie theorie*

# Fysiologie

- Echogolven: backscatter, refractie en transmissie
  - Backscatter produceert het meeste van het ultrasound beeld
  - Hoe hoger de frequentie, hoe kleiner de golflengte, hoe meer backscatter
  - Refractie en transmissie komen n naar de transducer



# Fysiologie formules

- $V = f \times \text{wavelength}$ 
  - Snelheid ultrasound soft tissue 1540 m/s
  - Gemakkelijke formule 1: elke cm duurt 6.5ms
  - Gemakkelijke formule 2:  $1.54 / \text{Mhz} = \text{wavelength}$  in mm
  - Snelheid geluid in bot hoger dan in bloed, soft tissue, lucht
- $L (\text{length of the nearfield}) = r^2 / \text{wavelength}$ 
  - Hoe hoger de frequentie, hoe langer de nearfield
- Doppler shift =  $2 \times f_0 \times v \times \cos \text{hoek} / c$

# Doppler

- Doppler shift ( $f_d$ ) =  $2 \times f_0 \times v \times \cos \text{hoek} / c$ 
  - $V$  = velocity of blood
  - $C$  = speed of propagation in medium = 1540 m/s
  - Elke mHz bij  $v$  van 1 m/s = Doppler shift van 1.3kHz
- Nyquist limiet =
  - Aliasing when  $f_d$  exceeds  $\frac{1}{2} * \text{PRF}$
  - $\text{PRF} / 2$
  - $77.000/\text{depth in cm}$

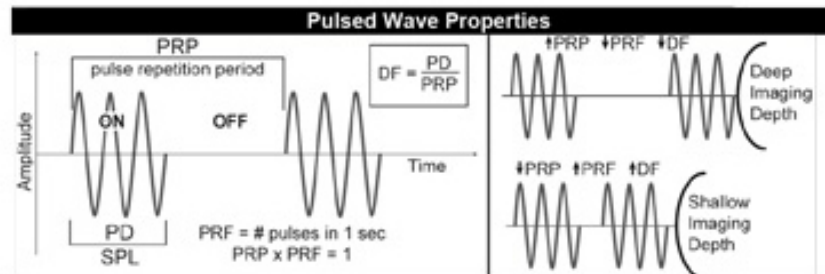
# Fysiologie

- Pulse duration or length (mm)
  - Physical length that pulse occupies
  - Affected by source of ultrasound
  - Frame rate = temporele resolutie = pulse duration / PRF
- PRF
  - Rate at which pulses are emitted from transducer
  - Affected by source of ultrasound
  - PRP = time from one pulse to another (pulse repetition period)
  - Nyquist limit =  $PRF / 2$
  - $77.000/\text{depth in cm}$
- Duty factor (N 0.1%)
  - Fraction of time that transducer is emitting ultrasound
  - Pulse duration / dead time
- Impedance
  - Rays: density in  $\text{kg/m}^3$  x speed of sound in  $\text{m/s}$
  - Differences in impedance determine the ratio of transmitted versus reflected sound
- Aliasing
  - Can be reduced by decreasing depth, increasing PRF, reducing transducer frequency, changing to CW

# Fysiologie 2

- **Attenuatie coefficient voor soft tissue**
  - $\frac{1}{2}$  x frequency (mHz)
  - In dB/cm
- **Impedantie**
  - Density of medium x propagation speed
  - In rayls
  - Soft tissue 1.630.000 rayls
- **Intensity**
  - Amplitude in het kwadraat
  - mW/cm<sup>2</sup>
- **Flow resistance**
  - Formule =  $8 * L * \text{viscositeit} / \pi * \text{radius tot de } 4^{\text{e}} \text{ macht}$
- **Flow rate**
  - Formule =  $\text{pressure difference} * \pi * \text{diameter}^4 / 128 * \text{length} * \text{viscosity}$





	Units	US Value	Determinant	Adjustable
<b>Pulse Duration (period)</b>	time	0.5 - 3.0us	source	no
<b>SPL (λ)</b>	distance	0.1 - 1.0mm	source, medium	no
<b>PRP (time)</b>	time	0.1 - 1.0ms	source	yes
<b>PRF (f)</b>	cycles/sec	1 - 10kHz	source	yes
<b>Duty Factor</b>	none	0.1 - 1%	source	yes

- US Pulse = collection of cycles that travel together
- Pulse Duration (PD): time from start to end of a pulse, pulse "on", PD = #cycles x time
- Spatial Pulse Length (SPL): distance from start to end of a pulse, SPL = #cycles x λ
- Pulse Repetition Period (PRP): time from start of 1 pulse to start of next pulse (on + off)
- Pulse Repetition Frequency (PRF): # of pulses in 1 second (irrespective of # cycles in pulse)
- Duty Factor (DF): ratio time that transducer produces pulse (on)

- **PRP and PRF are inversely related (PRP x PRF = 1) and depend on imaging depth.**
  - **PRP** is the time for transmitting and receiving the pulse (on + off)
    - In clinical US, PRP ranges from **0.1-1.0ms** (longer times for deeper structure)
  - **PRF** is the rate at which bursts of energy are emitted from the transducer:
    - Commercial machines have a PRF 200 - 5000/sec (200-5kHz).
    - **PRF (in Hz) = c/2d = 154,000cm/s / 2d(in cm) = 77,000/ d (cm)**
  - At a greater imaging depth more time is spent listening so PRP is longer, PRF and DF shorten.

**Acoustic Impedance**

- Different tissues have different resistance to the passage of sound, termed **acoustic impedance (Z)**. In biologic media, typical values for Z in Rayls (Kg/m<sup>2</sup>/s) are 1,250,000 to 1,750,000Rayls (1.25-1.75MRayls).
- Acoustic impedance is calculated (not measured) as the product of tissue density and the velocity of sound in that tissue, each of which can be measured and multiplied together.
- Impedance is characteristic of the medium only, and increases when:
  - 1) density increases, 2) propagation speed increases

Tissue	Velocity m/s	Impedance Rayl
Bone	4080	7.8
Blood	1570	1.61
Fat	1450	1.38
Kidney	1560	1.62
Liver	1550	1.65
Soft Tissue	1540	1.63
Air	330	0.0004

<b>Impedance (rayls) = density (kg/m<sup>3</sup>) x propagation speed (m/s)</b>	A medium has a high impedance when: <ul style="list-style-type: none"> <li>• It is very dense and/or</li> <li>• It has a fast propagation speed</li> </ul>
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**Reflection and Transmission**

- At an interface between tissues of differing acoustic impedance, part of the sound wave is reflected, and part continues deeper into tissues (transmitted).
- At the boundary between two media there is "**conservation of intensity**", so that adding the reflected and transmitted intensity obtains the incident intensity.

# Resolutie

- Spatial resolution
  - Axial resolution (Z)
    - Pulse length, transducer frequency
  - Lateral resolution (X, Y)
    - Beam width, depth, gain
- Temporal resolutie
  - Synoniem = frame rate
  - Depth, sweep angle, line density, PRF

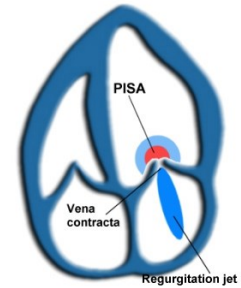
# Belangrijke formules

- PISA
- Continuity equation
- Bernouilli
- Flow en CO
- PHT

# Achtergrond

- Bloed is een vloeistof met een *flow* die de *pressure gradient* volgt, welke geschat wordt met de *velocity*, door de eenvoudige Bernouilli vergelijking

# PISA



- Principe = PISA flow gelijk aan MR flow
  - $2 \pi r^2 \times V_n$  (Q in cc / sec) = ERO x MR Vmax
- ERO = flow rate / MR Vmax
  - MR velocity in cm / s
- ERV = ERO x VTI MR

# Bernouilli

- Snelheid is een schatting van de drukgradient over een klep, naar aanleiding van de eerste wet van behoud van energie (Newton)
- Vereenvoudigde vergelijking
  - Drukgradient =  $4 \times v^2$
  - Niet meegenomen: deceleratie na klep en viscositeit bloed
- Wel voor berekening:
  - Stenotische klep (AS)
  - PH met TI signaal + RAP (5/10/15)
  - LVOT contour en VTI
  - Snelheid VSD:  $LV_{sp} - PG_{jet} = RV_{sp} = sPAP$ 
    - Een lage snelheid over VSD betekent een hoge sPAP
  - Eind diastolische snelheid PI jet: drukberekening is  $edRV_{druk} + RAP$
  - Schatting PVR
  - MR (Dp/Dt): LV pressure increase early systolic:  $EH \text{ mmHg/sec} = 32/\text{tijdsinterval in sec}$
- Niet te gebruiken bij dubbele klepvitiae, bij aorta ascendens van minder dan 30mm (pressure recovery), bij  $V1 > 1 \text{ m/s}$

# Continuity equation

- Tweede wet van Newton: law of conservation of mass
  - Mass cannot be destroyed, flow rates are the same at different locations in a flow stream
- Flow rate op punt 1 is gelijk aan punt 2
- $A_1 \times V_1 = A_2 \times V_2$ 
  - LVOT area x LVOT VTI = AVA x aortic VTI
  - AVA = LVOT area x LVOT VTI/aortic VTI

# Flow en CO

- Flow rate = CSA x flow velocity (Vmax)
- $SV = CSA \times VTI = \pi \times r^2 \times VTI$ 
  - Assuming a circular shape
  - Ook voor berekening RV / R fractie / Qp : Qs (intracardiac shunt)
- $CO = SV \times HF$
- Fick
  - Less than transaortic flow (Fick + RV)



# Doppler

- Aliasing
  - Nyquist limit is highest obtainable velocity
  - When frequency is higher than Nyquist
  - Nyquist limit =  $PRF/2$
- Doppler tissue imaging
  - Filter out low amplitudes and high Doppler shifts
- MPI CW 4/5CH: IVCT (systole)/IVRT (diastole) / ET

# Hemodynamiek

- Flow: laminair/turbulent
- Vroege sluiting MK: Aol
- B-hump: vertraagde sluiting MK, verhoogde einddiastolische LV druk
- Vroegsystolisch naar beneden bewegen IVS: LBTB
- Afplatting IVS: systolisch (drukbelasting RV), diastolisch (volumebelasting RV)
- IVC dilatatie: verhoogde RA druk

# Artefacten

- Side lobes
  - Posterieure mitralisklepannulus
- Reverberaties
  - Posterieure pericard
- Shadowing
  - Kunstkleppen
- Near-field clutter
  - Lijkt op LV thrombus

# Normaalwaardes

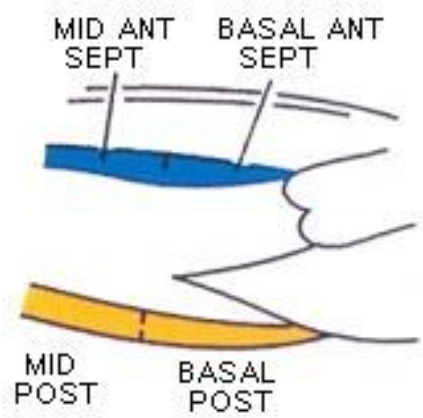
- Bij mannen van volwassen leeftijd

# LV

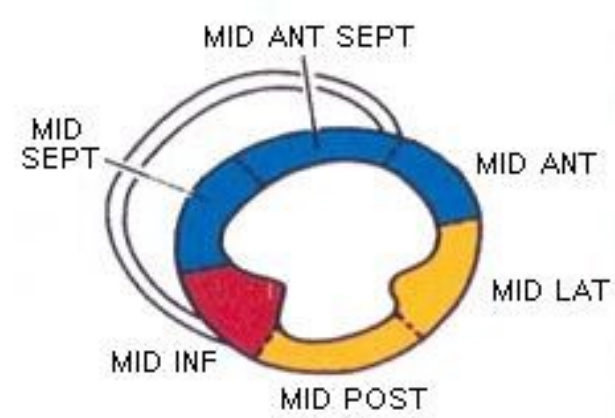
- $FS = (LVEDD - LVESD) / LVEDD \times 100\%$
- $LV \text{ massa} = 0.8 \times 1.04 \times (IVS + LVPW + LVEDD)^3 + 0.6g$
- $LVMI = LV \text{ massa} / BSA$

# LV

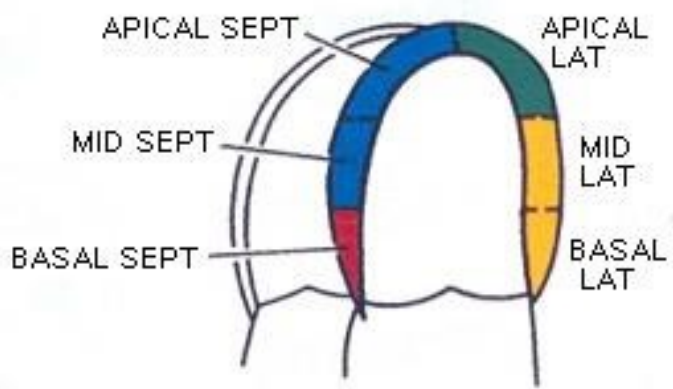
- Septum < 10mm
- Posterior wand <10mm
- LVEDD <59mm
- LVEDV <155ml
- LVESV <58ml
- EF >55%
- FS >25%
- LV massa <224g
- LVMI <115g/m<sup>2</sup>
- WMSI <1.5
- dP/dT >1200mmHg/s



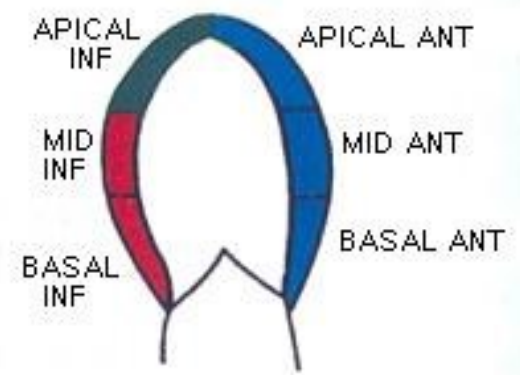
**LAX**



**SAX  
PM**

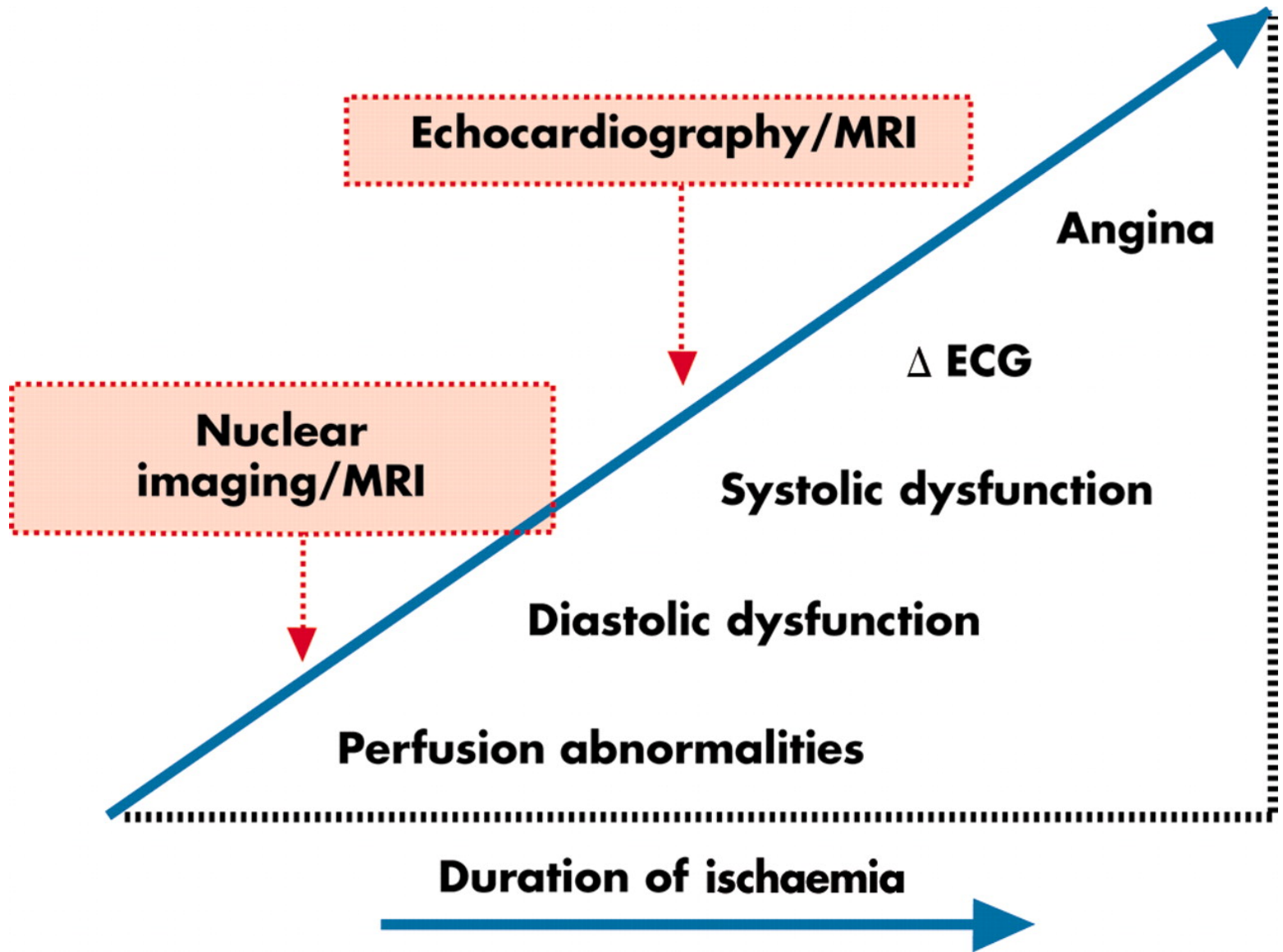


**4C**



**2C**

- Left anterior descending distribution
- Right coronary artery distribution
- Circumflex distribution
- Left anterior descending / circumflex overlap
- Left anterior descending / right coronary artery overlap





# RV

- RV mediolateraal midventriculair d 35mm
- RV lengte d <79mm
- RV lengte s <63mm
- RV oppervlakte <28cm<sup>2</sup>
- RV dikte < 5mm
- TAPSE >16mm
- RVOT <2.3mm

# Septum

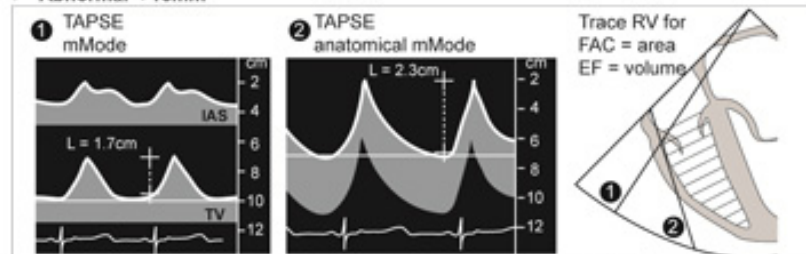
- Normally the interventricular septum is curved towards the RV in both systole and diastole, it moves towards the RV in diastole and towards the LV in systole
- Volume overload
  - RV dilatation, diastolic flattening (D-shaped LV), paradoxical diastolic movement (towards LV in diastole)
- Pressure overload
  - Ventricular hypertrophy and paradoxical septal movement in systole
- Interatrial septum bows toward RA throughout most of the cardiac cycle (not if  $RAP > LAP$ )

## Right Ventricle

RV Systolic Function Indices	
RV Fractional Area Change (FAC)	RV Ejection Fraction (EF)
$RV\ FAC = \frac{EDA - ESA}{EDA}$	$RV\ EF = \frac{EDV - ESV}{EDV}$
<ul style="list-style-type: none"> <li>ME 4C view trace endocardial border include TV, trabeculae, pap muscles</li> <li>Normal 35 - 60% (49%)</li> <li><b>Abnormal &lt; 35%</b> <ul style="list-style-type: none"> <li>Mild 25 - 35%</li> <li>Moderate 18 - 24%</li> <li>Severe <math>\leq</math> 17%</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>ME 4C view trace endocardial border include TV, trabeculae, papillary muscles</li> <li>Use method of disc technique for volume</li> <li>Assesses RV body but not infundibulum</li> <li>Normal EF 44 - 71% (58%)                             <ul style="list-style-type: none"> <li><b>Abnormal EF &lt; 44%</b></li> </ul> </li> <li>3D better estimates RV volumes                             <ul style="list-style-type: none"> <li>RVEDV 89ml/m<sup>2</sup></li> <li>RVESV 45ml/m<sup>2</sup></li> </ul> </li> </ul>

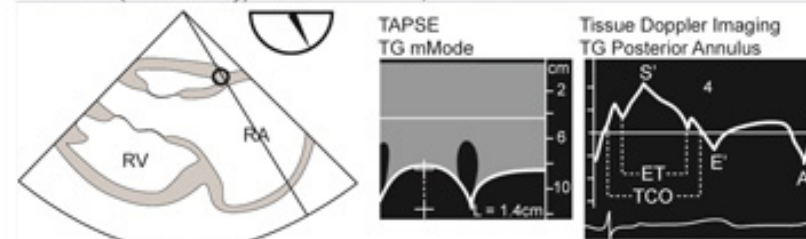
### Tricuspid Annular Plane Systolic Excursion (TAPSE)

- Distance of systolic excursion of lateral TV annulus in longitudinal plane
- Surrogate measure of global RV function, correlates with angio, biplane MOD, FAC
- Measure using M-mode
  - Difficult alignment in ME4C with m-Mode
  - Anatomical m-Mode for ME4C improves assessment
  - TG RV inflow better alignment
- Normal 16 - 30mm (23mm)
- Abnormal < 16mm**



### Tricuspid Valve Peak Annular Velocity (S')

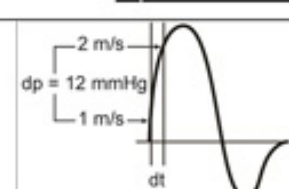
- Systolic excursion velocity of the TV annulus (mid RV wall less reliable)
- Use TG LAX view, posterior TV annulus
- Use tissue Doppler imaging (TDI) either
  - Pulsed spectral (peak velocity): Normal 10-19 cm/s, **Abnormal < 10 cm/s**
  - Color (mean velocity): Normal 6 - 14 cm/s, **Abnormal < 6 cm/s**



### dP/dt

$$dP/dt = \frac{12\text{mmHg}}{\text{time}}$$

- Rate of pressure rise in RV
- TR trace, time for TR velocity to increase 1 to 2m/s
- Abnormal < 400mmHg/s



Diagnostic Criteria Arrhythmogenic RV Dysplasia (ARVD)		
Major Criteria	RV Dysfunction	Minor Criteria
<ul style="list-style-type: none"> <li>Severe RV dilatation + ↓ RV EF with little or no LV impairment</li> <li>Localized RV aneurysm</li> <li>Severe segmental RV dilatation</li> <li>Fibrofatty replacement of myocardium on endomyocardial biopsy</li> </ul>	<ul style="list-style-type: none"> <li>Mild global RV dilatation ± ↓ EF with normal LV</li> <li>Mild segmental RV dilatation</li> <li>Regional RV hypokinesis</li> </ul>	
<b>ECG</b>		
<ul style="list-style-type: none"> <li>Epsilon waves in V1 - V3</li> <li>Localized prolongation of QRS (&gt; 110 ms) in V1 - V3</li> </ul>	<ul style="list-style-type: none"> <li>Inverted T waves in V2 + V3 without RBBB</li> <li>Late potentials on signal averaged EKG</li> <li>Ventricular tachycardia with a LBBB</li> <li>Frequent PVCs (&gt; 1000 PVCs / 24h)</li> </ul>	
<ul style="list-style-type: none"> <li>Familial disease confirmed on autopsy or surgery</li> </ul>	<ul style="list-style-type: none"> <li>Family hx of sudden cardiac death &lt; age 35</li> <li>Family history of ARVD</li> </ul>	

**Left Ventricular Non-Compaction (LVNC)**

- LVNC is characterized by prominent LV trabeculae and deep intertrabecular recesses (spaces). There is no other coexisting cardiac structural abnormality.
- LVNC is often familial. It occurs in isolation and in association with congenital cardiac disorders (Ebstein's anomaly or complex cyanotic heart disease) and some neuromuscular diseases.
- Two echocardiographic criteria (Chin and Jenni) are used for diagnosing LVNC by quantifying the depth of penetration of the intertrabecular recesses. The Jenni criteria describes the LV wall as being made up of 2 layers, an outer compacted layer, contiguous with the epicardium, and an inner non-compacted layer.

**TEE in Left Ventricular Non-Compaction (LVNC)**

- Numerous, excessively prominent trabeculations and deep intertrabecular recesses
  - Segments involved are mid LV (especially inferior + lateral) and apical
  - Perfused intertrabecular recesses are supplied by intraventricular blood on color Doppler
  - LV systolic function is preserved, but may have global or regional LV and RV dysfunction
- |  |  |
|--|--|
| <p><b>Chin criteria:</b> X-to-Y ratio <math>\leq 0.5</math> in LVNC<br/>                 X = epicardial surface to trabecular recess trough<br/>                 Y = epicardial surface to trabecular peak</p> | <p><b>Jenni criteria:</b> End-systolic thickness of non-compacted (NC) and compacted (C) layer is taken at the area of maximal LV wall thickness in the LV SAX view. A ratio of NC/C &gt;2 is LVNC</p> |
|--|--|



# RV

- **Dysfunctie postoperatief:**
  - Stunning door te weinig myocardiale protectie
  - Luchtembolus RCA
  - Pulmonale hypertensie preexistent
  - Acute PH door protamine
  - RV compressie tijdens chest closure

# Diastologie

- Diastole bestaat uit:
  - IVRT
  - Early filling (reservoir)
  - Diastasis
  - Atrial systole (pomp)
- TTE:
  - E/A (LA-LV gradient,  $N > 1$ )
  - Deceleratietijd (compliance, prognosis MI,  $N$  140-240ms)
  - E' (ventricular relaxation, LV filling pressure in diastolic dysfunction, lateraal hoger dan septaal,  $N > 12$ cm/s)
  - E/E' (LV filling pressure=LVEDP=LA,  $N$  5-10, verhoogd boven 16)
  - IVRT (LA pressure, early active LV relaxation,  $N$  80ms)
  - S/D/A pulmonaalvenen ( $N$   $S > D > A$ ), Ar-A (LV filling pressure,  $N$  0)
  - LA volume (chronicity, prognosis,  $N < 29$ ml/m<sup>2</sup>)
  - Vp (early diastolic relaxation, estimation PCWP) ,  $N > 50$ cm/s)
  - LV functie
  - sPAP
  - Valsalva (differentiating stages, in pseudonormal stage E decreases, A increases)

# Diastologie

- Zie volgende tabel +
- E 72 cm/s
- A 40 cm/s
  
- Verhoogde LA druk bij:
  - $S/D < 1$
  - Toegenomen AR-duur (meer dan transmitral A wave duration) en amplitude ( $>0.35\text{m/s}$ )
  - $E/E' > 15$
  
  - *LA dilatatie*
  - *IVRT  $< 70\text{ms}$*
  - *$E/A > 2$*
  - *Deceleratietijd  $< 150\text{ms}$*

# EAE

**Table 1** Normal values for Doppler-derived diastolic measurements

Measurement	Age group (y)			
	16-20	21-40	41-60	>60
IVRT (ms)	50 ± 9(32-68)	67 ± 8(51-83)	74 ± 7(60-88)	87 ± 7(73-101)
E/A ratio	1.88 ± 0.45(0.98-2.78)	1.53 ± 0.40(0.73-2.33)	1.28 ± 0.25(0.78-1.78)	0.96 ± 0.18(0.6-1.32)
DT (ms)	142 ± 19(104-180)	166 ± 14(138-194)	181 ± 19(143-219)	200 ± 29(142-258)
A duration (ms)	113 ± 17(79-147)	127 ± 13(101-153)	133 ± 13(107-159)	138 ± 19(100-176)
PV S/D ratio	0.82 ± 0.18(0.46-1.18)	0.98 ± 0.32(0.34-1.62)	1.21 ± 0.2(0.81-1.61)	1.39 ± 0.47(0.45-2.33)
PV Ar (cm/s)	16 ± 10(1-36)	21 ± 8(5-37)	23 ± 3(17-29)	25 ± 9(11-39)
PV Ar duration (ms)	66 ± 39(1-144)	96 ± 33(30-162)	112 ± 15(82-142)	113 ± 30(53-173)
Septal é (cm/s)	14.9 ± 2.4(10.1-19.7)	15.5 ± 2.7(10.1-20.9)	12.2 ± 2.3(7.6-16.8)	10.4 ± 2.1(6.2-14.6)
Septal é/á ratio	2.4*	1.6 ± 0.5(0.6-2.6)	1.1 ± 0.3(0.5-1.7)	0.85 ± 0.2(0.45-1.25)
Lateral é (cm/s)	20.6 ± 3.8(13-28.2)	19.8 ± 2.9(14-25.6)	16.1 ± 2.3(11.5-20.7)	12.9 ± 3.5(5.9-19.9)
Lateral é/á ratio	3.1*	1.9 ± 0.6(0.7-3.1)	1.5 ± 0.5(0.5-2.5)	0.9 ± 0.4(0.1-1.7)

Data are expressed as mean ± SD (95% confidence interval). Note that for é velocity in subjects aged 16 to 20 years, values overlap with those for subjects aged 21 to 40 years. This is because é increases progressively with age in children and adolescents. Therefore, the é velocity is higher in a normal 20-year-old than in a normal 16-year-old, which results in a somewhat lower average é value when subjects aged 16 to 20 years are considered.

\*Standard deviations are not included because these data were computed, not directly provided in the original articles from which they were derived.



# Grading

- 1
  - Impaired relaxation
    - Delayed LV early diastolic active relaxation, normal LA pressure
    - IVRT lengthens, MV opening is delayed, LV pressure is maintained longer than normal in diastole, which diminished the gradient resulting in a low E
- 2
  - Pseudonormalisation
    - *High filling pressure combined with impaired relaxation*
    - Mildly elevated LA pressure, low LA-LV gradient, reduced LV suction force
    - LA pressure rises, MV opens earlier, IVRT is shortened, gradient for early filling greater, increasing Emax, rise in mid diastolic ventricular pressure, Edec shortened
- 3
  - Restrictive filling
    - *Reduced chamber compliance in association with an elevated LA pressure*
    - Noncompliant LV chamber, increased stiffness, elevated LA pressure, high opening LA-LV pressure gradient, failing LA contractility, rapid increase in ventricular pressure as the ventricle fills, shortening Edec, responds positively to preload reduction (Valsalva, diuresis)
    - Ar-A > 30ms
- 4
  - Restrictive filling, irreversible
    - Inflow by pushing blood

# Longveneflow

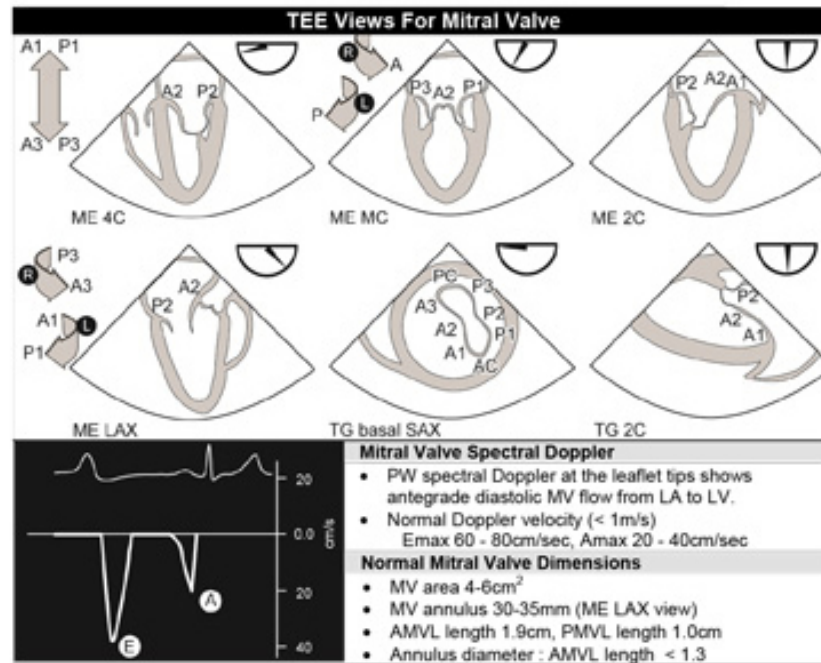
- S
  - Flow in LA during ventricular systole due to atrial relaxation and suction effect of base to apex movement of the heart
- D
  - Fall in atrial pressure secondary to ventricular filling, which promotes forward flow
- A
  - Atrial systole
- D
  - Graad 1: afname D
  - Graad 2/3: toename D door higher LA pressure

# Constrictief vs restrictief

- Restrictief (diastolische dysfunctie graad 3, amyloidose)
  - LA >
  - LVEF N
  - E/A > 1.5 (geen verandering op preloadvermindering)
  - DT < 160ms
  - Es' < 8cm/sec
  - PH, TI > MI, inspiratoire diastolische leverveneflowomkering
- Constrictief
  - Valsalva: >25% verandering E
  - IVRT variatie met ademhaling

# Atria

- LA <30mm
- RA <45mm
- LA <20mm<sup>2</sup>
- LA volume minder dan 58ml
- LA-volume/BSA < 28ml/m<sup>2</sup>
- Flowsnelheid bij TTE N > 64cm/sec
  - Verhoogde kans op thrombusvorming bij >30cm/sec
- Schatting RA-druk:
  - 5 RA N
  - 10 Vmax TI 2.6-4m/s VCI gedilateerd
  - 15 Severe TI Geen respiratoire  
variatie VCI



**Mitral Regurgitation (MR)**

**Etiology**

- Failure of any single component of the MV apparatus can result in MR.
- Carpentier classified the mechanisms of MR according to range of leaflet motion (next page).
- **Functional MR** occurs with structurally normal MV leaflets causing central or eccentric MR.
  - Annular dilatation from LA or LV enlargement may cause incomplete leaflet closure with central MR beginning in the annular plane.
  - RWMA or an infarcted papillary muscle alters the LV geometry. Fixed leaflet and chordal length tethers one leaflet causing eccentric MR in the same direction as the tethered leaflet. Though no leaflet tissue crosses the annular plane in systole, there is "relative" prolapse in relation to the other.
- **Diastolic MR** occurs when LV pressure exceeds LAP during diastole.
  - Pressure gradient is often small as is the regurgitant volume.
  - Diastolic TR often accompanies diastolic MR.
- **Mitral valve prolapse (MVP)** is defined by systolic displacement of one or more MV segments beyond the annular plane into the LA. An appreciation of the saddle-shaped structure of the mitral annulus is crucial to avoid over-diagnosing MVP. In most cases, subtle MVP is diagnosed correctly only when mitral leaflet tissue moves beyond the annular high points, in the ME AV LAX (120°) view, so that both mitral leaflets, LA, LVOT, and the aortic valve are seen.

**Etiology MR**

**1° Structural**

- MV prolapse (MVP)
- Ruptured chordae
- Rheumatic
- Mitral annular calcification (MAC)
- Mass (myxoma, vegetation)

**2° Functional**

- LV/LA dilatation
- Papillary muscle dysfunction
- Regional wall motion
- Aortic stenosis

**Diastolic MR**

- A flutter, PVC, 1°, 2°, 3° heart block
- ↑ LVEDP (AI, cardiomyopathy)

# Longvenen

- S1: atriale relaxatie
- S2: inflow door RV contractie
  
- S-flowsnelheid 40-60 cm/sec
- D-flowsnelheid 35-45 cm/sec

# Aorta

- Boog <30mm
- Descendens <30mm
- Abdominalis <30mm

# Schatting sPAP

- $sPAP = 4 \times V_{max} TI + RA \text{ druk}$
- Pulmonale acceleratietijd  $> 120ms$



# Sidebotham

**Table 5.1** Common causes of incorrect diagnosis with TOE, classified by location

<b>Right atrium</b>	
Crista terminalis	Right atrial appendage
Eustachian valve	Enlarged coronary sinus
Thebesian valve	Pectinate muscles
Chiari network	Catheters or wires
<b>Left atrium</b>	
Pectinate muscles	Inverted LA appendage
Warfarin (Coumadin) ridge	Accessory lobe of LA appendage
LA membrane	Native LA following heart transplantation
Stapled-off LA appendage	
<b>Interatrial septum</b>	
Double membrane fossa ovalis	Atrial septal aneurysm
Lipomatous hypertrophy	
<b>Right ventricle</b>	
Trabeculae	Moderator band
<b>Left ventricle</b>	
Trabeculae	Lobulated or bifid papillary muscles
False tendons	Spurious segmental wall motion abnormalities
Calcified papillary muscles and chordae	Subvalvular apparatus
<b>Valves</b>	
Valvular strands	Caseous calcification of MV annulus
Lipomatous hypertrophy of TV annulus	
<b>Pericardial space</b>	
Transverse sinus	Oblique sinus
<b>Extracardiac</b>	
Hiatus hernia	Aortic aneurysm
Pleural effusion	
<b>Great vessels</b>	
Persistent left-sided SVC	Aortic-Innominate apposition

# Klepprothesen

- Typen
- Bileaflet
  - Ring zichtbaar op annulusniveau
  - Mitralisklep leaflets goed zichtbaar
    - Open 80 graden, dicht 25 graden
  - Meerdere jets
- Regurgitatie: paravalvulair door endocarditis (rocking valve?), loslating hechting
- Gradient
  - M Vmax < 2.5 m/s, A V max < 3.5 m/s

# Klepprothesen en gradient

- Aorta prothese  $V_{max} > 3$  m/s
  - Mean gradient  $> 15$ mmHg abnormaal
  - DVI  $< 0.25$  abnormaal
  - Jetcontour driehoekig
  - Bereken de EOA per m<sup>2</sup>
- Mitralis prothese
  - Mean gradient  $> 5$ mmHg abnormaal
- DD hoge gradient:
  - High flow, obstructie (thrombus, endocarditis, pannus, degeneratie), regurgitation

# Tamponade

- RV en LV diastolische collaps
- RA en LA systolische collaps
- TV E verhoogt met inspiratie > 40%
- MV E verlaagt met inspiratie > 25%

# Optimale klepmorfologie voor PMVR

- Pathologie in segment 2
- Geen calcificatie
- Klepoppervlakte  $> 4\text{cm}^2$
- Lengte posterieure klepblad  $> 10\text{mm}$
- Coaptatie diepte  $> 11\text{mm}$
- Normale dikte en mobiliteit van de klepbladen
- MI met prolaps, flail size  $< 15\text{mm}$ , flail gap  $< 10\text{mm}$

# Guidelines

# MI

**Table 3** Grading the severity of organic mitral regurgitation

Parameters	Mild	Moderate	Severe
Qualitative			
MV morphology	Normal/Abnormal	Normal/Abnormal	Flail leaflet/Ruptured PMs
Colour flow MR jet	Small, central	Intermediate	Very large central jet or eccentric jet adhering, swirling and reaching the posterior wall of the LA
Flow convergence zone <sup>a</sup>	No or small	Intermediate	Large
CW signal of MR jet	Faint/Parabolic	Dense/Parabolic	Dense/Triangular
Semi-quantitative			
VC width (mm)	<3	Intermediate	≥7 (>8 for biplane) <sup>b</sup>
Pulmonary vein flow	Systolic dominance	Systolic blunting	Systolic flow reversal <sup>c</sup>
Mitral inflow	A wave dominant <sup>d</sup>	Variable	E wave dominant (>1.5 cm/s) <sup>e</sup>
TVI mit /TVI Ao	<1	Intermediate	>1.4
Quantitative			
EROA (mm <sup>2</sup> )	<20	20–29; 30–39 <sup>f</sup>	≥40
R Vol (mL)	<30	30–44; 45–59 <sup>f</sup>	≥60
+ LV and LA size and the systolic pulmonary pressure <sup>g</sup>			

CW, continuous-wave; LA, left atrium; EROA, effective regurgitant orifice area; LV, left ventricle; MR, mitral regurgitation; R Vol, regurgitant volume; VC, vena contracta.

<sup>a</sup>At a Nyquist limit of 50–60 cm/s

<sup>b</sup>For average between apical four- and two-chamber views.

<sup>c</sup>Unless other reasons of systolic blunting (atrial fibrillation, elevated LA pressure).

<sup>d</sup>Usually after 50 years of age;

<sup>e</sup>in the absence of other causes of elevated LA pressure and of mitral stenosis.

<sup>f</sup>Grading of severity of organic MR classifies regurgitation as mild, moderate or severe, and sub-classifies the moderate regurgitation group into 'mild-to-moderate' (EROA of 20–29 mm<sup>2</sup> or a R Vol of 30–44 mL) and 'moderate-to-severe' (EROA of 30–39 mm<sup>2</sup> or a R Vol of 45–59 mL).

<sup>g</sup>Unless for other reasons, the LA and LV size and the pulmonary pressure are usually normal in patients with mild MR. In acute severe MR, the pulmonary pressures are usually elevated while the LV size is still often normal. In chronic severe MR, the LV is classically dilated. Accepted cut-off values for non significant left-sided chambers enlargement: LA volume <36 mL/m<sup>2</sup>, LV end-diastolic diameter <56 mm, LV end-diastolic volume <82 mL/m<sup>2</sup>, LV end-systolic diameter <40 mm, LV end-systolic volume <30 mL/m<sup>2</sup>, LA diameter <39 mm, LA volume <29 mL/m<sup>2</sup>.

# AoS

**Table 3** Recommendations for classification of AS severity

	Aortic sclerosis	Mild	Moderate	Severe
Aortic jet velocity (m/s)	≤2.5 m/s	2.6–2.9	3.0–4.0	>4.0
Mean gradient (mmHg)	—	<20 (<30 <sup>a</sup> )	20–40 <sup>b</sup> (30–50 <sup>a</sup> )	>40 <sup>b</sup> (>50 <sup>a</sup> )
AVA (cm <sup>2</sup> )	—	>1.5	1.0–1.5	<1.0
Indexed AVA (cm <sup>2</sup> /m <sup>2</sup> )	—	>0.85	0.60–0.85	<0.6
Velocity ratio	—	>0.50	0.25–0.50	<0.25

<sup>a</sup>ESC Guidelines.

<sup>b</sup>AHA/ACC Guidelines.



# AoI

**Table 2** Grading the severity of AR

Parameters	Mild	Moderate	Severe
Qualitative			
Aortic valve morphology	Normal/Abnormal	Normal/Abnormal	Abnormal/flail/large coaptation defect
Colour flow AR jet width <sup>a</sup>	Small in central jets	Intermediate	Large in central jet, variable in eccentric jets
CW signal of AR jet	Incomplete/faint	Dense	Dense
Diastolic flow reversal in descending aorta	Brief, protodiastolic flow reversal	Intermediate	Holodiastolic flow reversal (end-diastolic velocity >20 cm/s)
Semi-quantitative			
VC width (mm)	<3	Intermediate	>6
Pressure half-time (ms) <sup>b</sup>	>500	Intermediate	<200
Quantitative			
EROA (mm <sup>2</sup> )	<10	10–19; 20–29 <sup>c</sup>	≥30
R Vol (mL)	<30	30–44; 45–59 <sup>c</sup>	≥60
+LV size <sup>d</sup>			

AR, aortic regurgitation; CW, continuous-wave; LA, left atrium; EROA, effective regurgitant orifice area; LV, left ventricle; R Vol, regurgitant volume; VC, vena contracta.

<sup>a</sup>At a Nyquist limit of 50–60 cm/s.

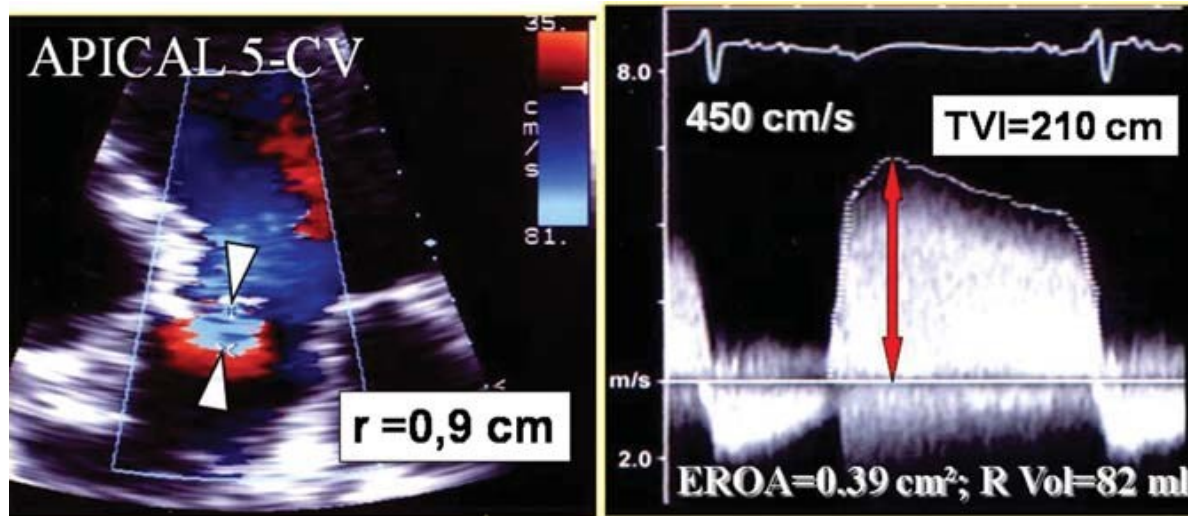
<sup>b</sup>PHT is shortened with increasing LV diastolic pressure, vasodilator therapy, and in patients with a dilated compliant aorta or lengthened in chronic AR.

<sup>c</sup>Grading of the severity of AR classifies regurgitation as mild, moderate or severe and subclassifies the moderate regurgitation group into 'mild-to-moderate' (EROA of 10–19 mm<sup>2</sup> or an R Vol of 30–44 mL) and 'moderate-to-severe' (EROA of 20–29 mm<sup>2</sup> or an R Vol of 45–59 mL).

<sup>d</sup>Unless for other reasons, the LV size is usually normal in patients with mild AR. In acute severe AR, the LV size is often normal. In chronic severe AR, the LV is classically dilated. Accepted cut-off values for non-significant LV enlargement: LV end-diastolic diameter <56 mm, LV end-diastolic volume <82 mL/m<sup>2</sup>, LV end-systolic diameter <40 mm, LV end-systolic volume <30 mL/m<sup>2</sup>.

# AoI

- Meting mogelijk met Doppler volumetrische metingen
  - LVOT SV – mitral inflow volume



$$\text{ERO} = \text{Flow/Peak velocity} = 178/450 = 0.39 \text{ cm}^2$$

$$\text{R Vol} = \text{EROA} \times \text{TVI} = 0.39 \text{ cm}^2 \times 210 \text{ cm} = 82 \text{ mL}$$

**Figure 11** Quantitative assessment of aortic regurgitation severity using the proximal isovelocity surface area method from the apical five-chamber view (CV). Arrows: white: PISA radius, Red: peak regurgitant velocity.

# TI

**Table 5 Grading the severity of TR**

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

CW, continuous-wave; EROA, effective regurgitant orifice area; RA, right atrium; RV, right ventricle; R Vol, regurgitant volume; TR, tricuspid regurgitation; VC, vena contracta.

<sup>a</sup>At a Nyquist limit of 50–60 cm/s.

<sup>b</sup>Baseline Nyquist limit shift of 28 cm/s.

<sup>c</sup>Unless other reasons of systolic blunting (atrial fibrillation, elevated RA pressure).

<sup>d</sup>In the absence of other causes of elevated RA pressure.

<sup>e</sup>Unless for other reasons, the RA and RV size and IVC are usually normal in patients with mild TR. An end-systolic RV eccentricity index >2 is in favour of severe TR. In acute severe TR, the RV size is often normal. In chronic severe TR, the RV is classically dilated. Accepted cut-off values for non significant right-sided chambers enlargement (measurements obtained from the apical four-chamber view): Mid RV dimension ≤33 mm, RV end-diastolic area ≤28 cm<sup>2</sup>, RV end-systolic area ≤16 cm<sup>2</sup>, RV fractional area change >32%, maximal RA volume ≤33 mL/m<sup>2</sup>.

An IVC diameter <1.5 cm is considered normal.

# TS

- Etiology: RA, carcinoid, congenital, functional by mass RA
- 2D TEE: immobility leaflets, thickening, diastolic doming, commissural fusion
- CW Doppler: peak and mean transvalvular velocity and pressure gradients
  - E wave velocity: 0.3-0.7 m/s
  - Mean pressure gradient: >5mmHg

# PI

**Table 4 Grading the severity of PR**

Parameters	Mild	Moderate	Severe
Qualitative			
Pulmonic valve morphology	Normal	Normal/ abnormal	Abnormal
Colour flow PR jet width <sup>a</sup>	Small, usually <10 mm in length with a narrow origin	Intermediate	Large, with a wide origin; may be brief in duration
CW signal of PR jet <sup>b</sup>	Faint/slow deceleration	Dense/variable	Dense/steep deceleration, early termination of diastolic flow
Pulmonic vs. Aortic flow by PW	Normal or slightly increased	Intermediate	Greatly increased
Semi-quantitative			
VC width (mm)	Not defined	Not defined	Not defined
Quantitative			
EROA (mm <sup>2</sup> )	Not defined	Not defined	Not defined
R Vol (mL)	Not defined	Not defined	Not defined
+RV size <sup>c</sup>			

PR, pulmonic regurgitation; CW, continuous wave; EROA, effective regurgitant orifice area; PW, pulse wave; RV, right ventricle; R Vol, regurgitant volume; VC, vena contracta.

<sup>a</sup>At a Nyquist limit of 50–60 cm/s.

<sup>b</sup>Steep deceleration is not specific for severe PR.

<sup>c</sup>Unless for other reasons, the RV size is usually normal in patients with mild PR. In acute severe PR, the RV size is often normal. Accepted cut-off values for non-significant RV enlargement (measurements obtained from the apical four-chamber view): Mid RV dimension  $\leq 33$  mm, RV end-diastolic area  $\leq 28$  cm<sup>2</sup>, RV end-systolic area  $\leq 16$  cm<sup>2</sup>, RV fractional area change  $> 32\%$ , maximal.

# PI

- Colour Doppler: jet area, width VC
- CW Doppler: intensity regurgitant signal
- PW Doppler: holodiastolic flow reversal PA
- Leading to RV volume overload and TR

# PS

- Secondary to congenital obstruction
- Subvalvular, valvular, supravvalvular
- Peak or mean transvalvular pressure gradient with CW Doppler
- Ernstige PS > 80mmHg



# MS

- Ernstige MVA  $< 1.0\text{cm}^2$  en gradient  $>12\text{mmHg}$
- $MVA = 220/PHT$
- Bij prothese obstructie of mismatch bij mean gradient  $>10\text{mmHg}$  en PHT  $>160\text{ms}$

**Table 9** Recommendations for classification of mitral stenosis severity

	Mild	Moderate	Severe
Specific findings			
Valve area (cm <sup>2</sup> )	>1.5	1.0–1.5	<1.0
Supportive findings			
Mean gradient (mmHg) <sup>a</sup>	<5	5–10	>10
Pulmonary artery pressure (mmHg)	<30	30–50	>50

<sup>a</sup>At heart rates between 60 and 80 bpm and in sinus rhythm.

**Table 5** Assessment of mitral valve anatomy according to the Wilkins score<sup>64</sup>

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	Midleaflets 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 in diastole	Considerable thickening of all leaflet tissue (>8–10 mm)	Extensive brightness throughout much of the leaflet tissue	Extensive thickening and shortening of all chordal structures extending down to the papillary muscles

The total score is the sum of the four items and ranges between 4 and 16.