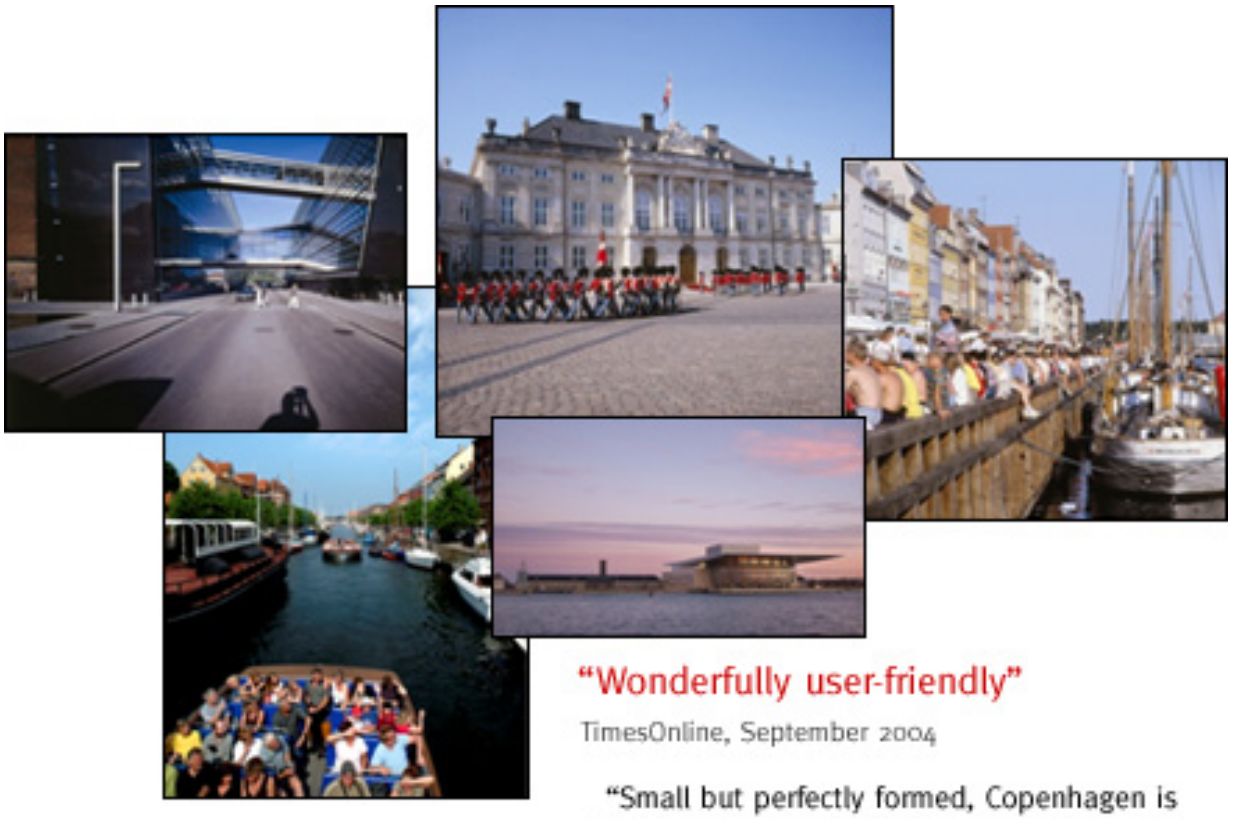
EACTA ECHO'07
Focus on accreditation

Book of Abstracts

EACTA echo 2007
Copenhagen
Denmark

Edited by
Carl-Johan Jakobsen, Aarhus, Denmark



"Wonderfully user-friendly"

TimesOnline, September 2004

"Small but perfectly formed, Copenhagen is Europe's most user-friendly capital; an enciting smorgasbord of 18thcentury housing, sea, design shops and polite, bicycle-minded residents speaking flawless English"

DEAR FRIENDS AND COLLEAGUES

It is a great pleasure for us to see you all at the 6th Annual Course on perioperative echocardiography, held in Copenhagen, Denmark September 22nd -25th, 2007. Since the first EACTA course on echocardiography in 2002, there has been a tremendous increase in technical developments, necessitating new skills and opening up a huge range of new possibilities.

Our intention was a tailor-made course to these developments, but we have also focused on the needs for both those less experienced in transthoracic echocardiography (TTE) and, for those more experienced in TEE, a didactic course tailor made for revision for the European echo accreditation and examination.

In order to realize the benefit of TTE for cardiopulmonary optimization, we aim to provide extensive opportunities for hands-on TTE. To achieve our goals, we shall be using a variety of different teaching modalities, including DICE-sessions, teamwork, and working and training in small groups.

With this course EACTA is pleased to be able to highlight its commitment to providing educational opportunities for those wishing to develop special competence in perioperative echocardiography by way of achieving a European accreditation in TEE - a joint program of European Association of Cardiothoracic Anaesthetists (EACTA) and the European Association of Echocardiography (EAE).

We do hope that the course is fulfilling your expectations and that you will enjoy the social programme. Further we hope you will meet old friends and colleges, create new liaisons and lastly take the opportunity to visit the "Wonderful Copenhagen".

Erik Sloth
Chairman EACTA-ECHO' 07

Carl-Johan Jakobsen
EACTA Past-president

Rob Feneck
Chairman EACTAECHO Committee

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EACTA/EAE ACCREDITATION COURSE

Saturday September 22nd

08.30 - 09.00 Registration
Chairmen: Jan Hultman & Joachim Erb
09.00 - 09.45 Physics and machine settings/controls, *Joachim Erb, Germany*
09.45 - 10.15 Comprehensive perioperative TEE examination, *Jan Hultman, Sweden*
10.15 - 10.45 Coffee
Chairmen: Fabio Guarracino & Rob Feneck
10.45 - 11.15 Safety and complications of TEE, *Rob Feneck, UK*
11.15 - 11.45 LV Systolic function, *Fabio Guarracino, Italy*
11.45 - 12.15 LV Diastolic function, *Patrick Wouters, Belgium*
12.15 - 13.15 Lunch
Chairmen: Justiaan Swanevelder & Isabelle Michaux
13.15 - 13.45 Pulmonary and Tricuspid valve, *Isabelle Michaux, Belgium*
13.45 - 14.15 Rt Ventricular function & the pulmonary circulation, *Isabelle Michaux, Belgium*
14.15 - 14.45 ICE and TEE for guidance of transcatheter ASD and PFO closure. *Jens Erik Nielsen-Kudsk, Denmark*
14.45 - 15.15 Aortic valve disease, *Justian Swanevelder, UK*
15.15 - 15.45 Coffee / Tea
Chairmen: John Kneeshaw & Marco Ranucci
15.45 - 16.30 Haemodynamic Calculations I, *Marco Ranucci, Italy*
16.30 - 17.30 Question and answer, clips and theory MCQs I. *(digital interactive technology). John Kneeshaw, UK*

Sunday September 23rd

Chairmen: Dominique Bettex & John Kneeshaw
08.30 - 09.00 Mitral stenotic disease, *Andreas Ziegler; Germany*
09.00 - 09.30 Mitral regurgitation, *Dominique Bettex, Switzerland*
09.30 - 10.00 Guided Mitral repair, *John Kneeshaw, UK*
10.00 - 10.30 Coffee
Chairmen: Manfred S Seeberger & Erik Sloth
10.30 - 11.00 Assessment of prosthetic valves, C*hristian Hassager, Denmark*
11.00 – 11.30 Ischaemic Heart Diseases, *Manfred Seeberger-Stucky, Switzerland*
11.30 - 12.00 Adult congenital heart diseases, *Keld Sørensen, Denmark*
12.00 - 12.30 TEE and Endocarditis, *Christian Hassager, Denmark*
12.30 - 13.15 Lunch
Chairmen: Rob Feneck & Marco Ranucci
13.15 - 13.45 Artefacts and illusions, *Joachim Erb, Germany*
13.45 - 14.00 The EACTA / EAE Accreditation process, *Rob Feneck, UK*
14.00 - 15.30 Haemodynamic Calculations II and interactive question and answer, clips and theory MCQs II *(digital interactive technology), John Kneeshaw, UK & Marco Ranucci, Italy*

EACTA SPECIAL LECTURES

Sunday September 23rd

Chairmen: Jan Hultman & Heinz Tschernich
16.00 - 16.30 Deformation imaging - new modalities with focus on global and regional function, *Heinz Tschernich, Austria*
16.30 - 17.00 Real-time L3D TOE, *Jan Hultman, Sweden*
17.00 - 17.30 4D TEE and the Mitral Valve assessment, *Andreas Ziegler, Germany*

EACTA SPECIAL COURSE

Monday September 24th

Chairmen: Erik Sloth

08.30 - 09.00 Physics and machine settings/controls (Knobology) in TTE. *Frank Steensgaard-Hansen, Denmark*
09.00 - 10.00 How to make a TTE. *Frank Steensgaard-Hansen, Denmark*
10.00 - 10.30 Coffee
10.30 - 11.00 Echocardiography as a tool in haemodynamic optimization. *Erik Sloth, Denmark*
11:00 - 11:30 The FATE protocol, *Erik Sloth, Denmark*
11.30 - 12.30 Hands-on TTE
12.30 - 13.15 Lunch
13.15 - 14.15 Hands-on TTE

Chairmen: Heinz Tschernich & Patrick Wouters

14.15 - 14.45 Important clinical "pictures", *Erik Sloth, Denmark*
14.45 - 15.15 TTE Doppler Echo, *Frank Steensgaard-Hansen, Denmark*
15.15 - 15.45 Coffee/Tea
15:45 - 17:00 Principle of myocardial velocity and deformation imaging. *Heinz Tschernich, Austria, Fabio Guarracino, Italy & Patrick Wouters, Belgium*

Tuesday September 25th

Chairmen: John Kneeshaw & Justiaan Swanevelder

08.30 - 09.45 Dissections, Asc, Desc and Stents, *Justian Swanevelder, UK*
09:45 - 10.15 Coffee
10.15 - 11.00 Congenital diseases, *Bent Østergaard Kristensen, Denmark*
11.00 - 12.00 Hands-on TTE
12.00 - 13.00 Lunch
13.00 - 14.00 Hands-on TTE

Chairmen: Erik Sloth & Heinz Tschernich

14.00 - 14.30 The Echo Report, *Heinz Tschernich, Austria*
14.30 - 15.00 Echo clips/MCQ's and discussion, *Faculty*
15.00 Closure of EACTAecho 2007

Tivoli Gardens – social event

Tivoli is many different things: Denmark's most popular tourist attraction is simultaneously an institution, a bearer of tradition, a national symbol and a limited company. Not only is it one of the world's oldest amusement parks – the one that gave Walt Disney the idea for his Disneyland – it is also the third most visited in Europe.

Tivoli has a tradition of renewal. As Tivoli's founder, Georg Carstensen, once said in 1843: "Tivoli will never be finished". Every year, there is some new addition – a new ride, a new restaurant, a new kind of entertainment – and the old must make room for the new. But Tivoli is more than just amusements, good food and entertainment. Tivoli is also fairy lights, flowers and above all, romance. Tivoli is at its most romantic when darkness falls, but no matter when you choose to visit the Gardens, it's like stepping into a magic universe.

The fairy tale starts the moment you leave the outside world.

The EACTAecho 2007 dinner will take place in the famous

Vise-vers huset in Tivoli

After the dinner you can visit Tivoli Gardens the most varied attractions. Sunday September the 23rd is Tivoli's last opening day in 2007. From 22.00 one of the most famous Danish rock bands will perfume live on the big garden stage and the season is finally closed with with the world famous Tivoli fireworks 23.30.

f you have not booked a ticket for this great event – please ask the staff. Tickets may be available for 60 Euro

Physics of Ultrasound, Machine Settings and Controls

Joachim M. Erb, Berlin, Germany

Characteristics of (ultra)sound waves

*Sound waves are mechanical vibrations that induce alternate reductions and compressions on their passage through **any** physical medium. Sound waves are described by the following terms:*

- *Frequency (f): number of cycles per second (Hz)*
- *Wavelength (λ): distance between cycles (mm)*
- *Amplitude: extension of cycles, „loudness" (decibels)*
- *Propagation velocity (c): depending on the medium in which the sound waves travels (m/sec)*

(Ultra)sound propagation velocity and carrying medium

Medium	Air	Lung	Fat	Water	Blood	Muscle	Bone
V ($m \cdot s^{-1}$)	330	600	1450	1450	1560	1580	4080

*Average propagation velocity of (ultra)sound in soft tissue: **1540 $m \cdot s^{-1}$***

Ultrasound is defined as sound with frequencies above the for humans audible range between 20 Hz and 20.000 Hz. Diagnostic medical ultrasound uses frequencies between 1.000.000 and 20.000.000 Hz = 1 to 20 megahertz (MHz). By selecting transducers with different frequencies and by adjusting the frequency on the machine display one can select the emitted wave length. According to the wave equation: $c = \lambda \bullet f$, a change in frequency results in a reciprocal change in wave length. As the propagation velocity in the heart is 1540 $m \cdot s^{-1}$, the emitted wavelength can be calculated as $\lambda = c / f$ or λ (mm) = 1,54 / f (f in MHz).

Importance of wavelength for the ultrasound diagnostic:

- *Image resolution: maximal 1 -2 wavelengths (approx. 1 mm). The shorter the wavelength, the higher the image resolution will be.*
- *Depth of penetration: proportional to the wavelength, inversely related to the frequency: short waves travel short distances, long waves travel long distances.*

Knobology: ***Select transducer and adjust frequency to match the required penetration depth while allowing for optimal image resolution.***

Interaction of ultrasound waves with tissue

- *Reflection: a part of the ultrasound wave is thrown back towards the transducer by an object.*
- *Scattering: a part of the ultrasound wave is diffused into all directions by an object.*
- *Refraction: the direction of the ultrasound wave is deflected from the straight path by an object.*
- *Attenuation: the energy of the ultrasound wave is absorbed by conversion into heat.*

Reflection of ultrasound

Reflection is the basis of ultrasound imaging. Ultrasound is reflected at tissue boundaries and tissue interfaces. The amount of reflected ultrasound energy depends on the difference in acoustic impedance Z between tissues ($Z = \sigma \bullet c$). Smooth tissue boundaries with a lateral dimension greater than the wavelength act as specular reflectors („mirror-like"). The angle of incidence equals the angle of reflection. Optimal reflection is achieved if the direction of the ultrasound beam is perpendicular to the object boundary. This assures optimal image quality.

Scattering of ultrasound

Small structures (lateral dimension < 1 wavelength) and rough surfaces diffuse ultrasound into all directions. Only a small amount of the ultrasound wave energy is reflected towards the transducer. The amplitude (energy) of the scattered signal is 100

to 1000 times (40 - 60 dB) less compared to a reflected signal. Scattering of ultrasound from moving blood cells is the basis of Doppler echocardiography.

Refraction of ultrasound

Ultrasound waves are refracted on their passage between tissues of different acoustic impedance e.g. they are deflected from their initial straight path (comparable to light refraction through optical lenses). Refraction allows enhanced image quality if used for acoustic focusing in a transmitter. Unplanned and unrecognized refraction in the tissue is the source of ultrasound imaging artefacts (for example double image artefact).

Attenuation of ultrasound

During penetration of tissue the ultrasound signal strength is attenuated by conversion of ultrasound energy into heat, additionally by reflection and scattering. The overall attenuation is depending on the acoustic impedance and is frequency dependent. The penetration depth for adequate imaging is limited to approximately 200 wavelengths and requires adaptation of the ultrasound frequency to the examination conditions.

Transducer frequency 1 MHz:	*approximate penetration depth 30 cm*
Transducer frequency 5 MHz:	*approximate penetration depth 6 cm*
Transducer frequency 20 MHz:	*approximate penetration depth 1,5 cm*

Ultrasound technology

The piezoelectric crystal (quartz or titanate ceramic) is build of polarized particles with the property of spatial orientation if an electric current is applied. The crystal rapidly expands and compresses if an alternating electric current is applied, generating an ultrasound wave. If an ultrasound wave impacts on a piezoelectric crystal, the rapid sequence of compressions and decompressions creates an electric current by changes in the spatial orientation of the polarized particles. Therefore the piezoelectric crystal serves as transmitter and receiver.

Transducers are build by putting piezoelectric crystals in a case with damping material and a specially designed acoustic lens. As linear transducers with multiple crystals (sequenced array) have a large aperture, small aperture phased array sector scanners are used in echocardiography. As the ultrasound beam is not an ideal, linear beam, but has a cylindrical near zone ($F_n = D^2 / 4\lambda$) and a cone shaped far field ($\sin \theta = 1{,}22\ \lambda / D$), focused transducer are build using either a concave piezoelectric crystal, an acoustic lens or, most lately, the electronic beam formation with multiple crystals which allows for an adjustable focus. The focal area can be narrow or spread apart using multiple foci. This focus position has to be frequently corrected during image acquisition in order to achieve optimal image quality.

Knobology:	***Chose between a single focus and multiple foci. Adjust focus to area of interest on the screen and readjust if you have changed the penetration depth.***

Ultrasound beam and side lobes

Fractions of ultrasound energy are dispersed laterally at an angle from the main beam, with $\sin \theta = n\ \lambda / D$. The reflection of side lobe ultrasound waves off specular reflectors creates image artefacts.

Practical application of ultrasound in 2D echocardiography

The transmitter sends a directed short sound pulse and listens. Sound is reflected at the object and travels back to the receiver, where it is picked up. Knowledge of the direction in which sound was send and the time delay from sending to receiving (travel time) allows locating the object. The reflected sound energy is proportional to the size of the object. The reflected sound energy is displayed on the image screen as a point, whereby the brightness of the dot reflects the received energy using a gray scale. This is called B (brightness) – mode. The position of the point on the screen correlates to the point of origin of the reflection (reflector).

2 D echocardiography

The sector scanner in B-mode electronically sweeps across a plane, creating a tomographic image. Time for image acquisition is proportional to the number of scan lines used, for example with r 20 cm, 128 scan lines ⇒ 33 ms. The image repetition frequency (frame rate) is proportional to the scan line density. A high frame rate (timely resolution) is necessary to assess rapidly moving structures. Modern technology allows to send several scan lines at one time, thus increasing image repetition frequency and thereby timely resolution.

Knobology: ***Select sector width and penetration (image) depth to allow for optimal frame rate. Reduce scan line density if higher timely resolution is needed.***

2 D echocardiography motion mode (M - mode)

Only one scan line is utilized (biopsy cylinder information), and the signal is recorded in brightness mode against time. This allows for a minimal sampling time interval, for example with r 20 cm, c = 1540 $m \cdot s^{-1}$ ⇒ 0,3 ms, enabling a high pulse repetition frequency, with a timely resolution up to 2 KHz. M - mode is ideal for imaging of rapid cardiac motion.

Signal processing up to the 2 D echocardiographic image
Several processing steps of the reflected sound signal take place before the image is displayed in real time on a sector screen. The most important ones are signal amplification (gain), compensation for penetration depth (time gain compensation), filtering, compression and rectification. Coordinates are coding direction and penetration depth (time delay), while the signal amplitude is coded with a gray scale.

Spatial image resolution

The details of an image are determined by the spatial resolution, which has three components:
Axial resolution (along the length of the ultrasound beam): this is the most precise resolution. The smallest measurable distance equals 1-2 wavelengths. All quantitative measurements should be made in axial (perpendicular) alignment.
Lateral resolution (side to side across the ultrasound beam): is mostly dependent on the beam width (2-3 λ) and therefore decreases with the distance of the reflector from the transducer.
Elevational (sagittal) resolution: the „slice thickness" of the ultrasound image is 3-10 mm depending on selected penetration depth.

Knobology : Important controls in 2 D mode

- ***Imaging depth: adjusts the size of the image on the screen and selects the maximal penetration. An increased imaging depth reduces image repetition frequency and thus timely resolution.***
- ***Sector width: reduced sector width allows for higher frame rates***
- ***Gain: regulates the overall brightness of the display***
- ***Time gain controls: regulate the gain in a given distance from the transducer***
- ***Lateral gain controls: regulate the gain along a set of neighbouring scan lines***
- ***Ultrasound frequency: influences penetration and resolution***
- ***Contrast: post processing application adjusting the gray scale use***
- ***Focus: improves image quality and measurement in the focal area***
- ***Freeze button, trackball: necessary for measurements***

Doppler echocardiography

If a source of sound moves towards the listener, the sound frequency increases. If a source of sound moves away from the listener, the sound frequency decreases (Chr. Doppler, 1842). This principle also applies to the reflection of sound. If sound is reflected by moving objects, the frequency of the reflected sound is changed. With the object moving towards the transducer, the frequency is increasing (positive Doppler shift),

while with the object moving away from the transducer, the frequency is decreasing (negative Doppler shift). In echocardiography, Doppler shift is caused by the velocity of blood cells as well as myocardium and heart valves. As Doppler echocardiography normally focuses on the reflection of the ultrasound signal at moving blood cells, filters are used to remove signals from myocardium and valves. The resulting frequency change is proportional to the flow direction, velocity and flow characteristics.

- *Doppler equation: $F_d = 2 \times F_0 \times V \times \cos \alpha / c$*
- *F_d = Doppler shift (Hz)*
- *F_0= transmitted sound frequency (Hz)*
- *V = blood flow velocity ($m \cdot s^{-1}$)*
- *cos α = angle between blood flow direction and ultrasound beam*
- *c = velocity of sound (1540 $m \cdot s^{-1}$)*
- *Doppler shift ≈ transmitted sound frequency*
- *Doppler shift ≈ velocity of objects*
- *Doppler shift ≈ intercept angle*
- *$V = F_d \times c / 2 \times F_0 \times \cos \alpha$*

Knobology: ***Minimize emitted US-frequency to allow for maximal Doppler shift measurable!***

Intercept angle and Doppler shift

The intercept angle between blood flow and ultrasound beam should be as parallel as possible. Keep in mind that you see a 2D image, but you are measuring a 3D flow. An intercept angle up to 20 ° may be tolerated, as the resulting error is not more than 6 % of the actual velocity.

Knobology: ***Angle correction: this feature is used for vascular Doppler, but should not be used to correct cardiac flow velocities for which the 3D characteristics are uncertain.***

Analysis and display of Doppler signals

The primary signal is a complex mixture of the transmitted frequency and multiple overlying Doppler shifts. It is broken down into individual frequencies by fast Fourier transformation. Frequencies of Doppler shifts in the heart are below 20 KHz and therefore audible. The Doppler signal comprises a spectrum of frequencies with varying intensities. The graphic display of the spectral analysis shows velocities recorded against time. The signal intensity is coded through the brightness of the spectral display. In the acoustic display, the sound frequency codes velocity, and the volume codes signal intensity.

Continuous wave Doppler (CW)

Two separate crystals in one transducer are transmitting and receiving ultrasound continuously and independently. All velocities along one scan line are measured, with the result being a filled-in spectrum, as many different velocities are measured along the scan line at one time. The origin of the signal can not be precisely located

- *Advantage: very high velocities (Doppler shifts) can be measured accurately*
- *Disadvantage: the origin of the velocity can not be located (no time delay)*

Knobology: ***The icon on the CW Doppler line marks the focus and should be set to the point where you want to measure the velocities of interest.***

Pulsed wave Doppler (PW)

One crystal sends a short ultrasound bursts and acts intermittently as transmitter and receiver. Only velocities within a defined sample volume are measured. The result is a framed spectrum if laminar flow is measured (identical velocities at one time), allowing precise location of velocity with a low V_{max}.

- *Advantage: velocity can be measured at a precise location (depth of interest)*

- *Disadvantage: the maximal measurable velocity is limited by the pulse repetition frequency (Nyquist limit, at 10 cm depth correct measurable V_{max} is app. 1,5 $m \cdot s^{-1}$)*

Knobology: ***If high pulse repetition frequency is chosen in PW Doppler mode, the machine displays multiple sample volumes. This can lead to erroneous measurements!***

Colour flow Doppler (CF)

Pulse wave Doppler with many sample volumes in a freely adjustable sector, where the V_{mean} in the sample volume is displayed. The flow direction is coded by colour.

- *Red: flow towards the transducer*
- *Blue: flow away from the transducer*

A homogeneous colour spectrum is seen with laminar flow, while the addition of yellow/green colours appears with turbulent flow profiles. Flow data are superimposed on the two dimensional B-mode image.

- *Advantage: velocity and flow direction is displayed in relation to the anatomy*
- *Disadvantage: low image update frequency (frame rate)*

Knobology: ***Adjust colour gain to optimal level just below limit of spontaneous colouring. Minimize colour sector and penetration depth to allow for sufficient frame rate.***

Aliasing phenomenon

At least two observations per cycle are necessary in order to correctly describe a periodic movement. Less observations in time lead to misinterpretations of direction and velocity of the respective movement. Aliasing occurs if F_D >1/2 PRF

➔ *Nyquist Limit = PRF / 2*

Aliasing with PW Doppler and colour flow Doppler

Velocities above Nyquist limit are displayed incorrectly: with PW-Doppler with inverted +/- signs, with colour flow Doppler with inverted colours.
Aliasing can be prevented by use of
Low ultrasound imaging frequency
Short distance between sampling volume and transducer (depth of interest)
Maximum PRF (small colour flow sector)
Increased velocity scale with baseline shift

Knobology: ***Use baseline shift to minimize aliasing!***

Contradictions of 2D / M – Mode and Doppler echocardiography

	2D-Mode, M-Mode	Doppler-Mode
Ultrasound frequency	*as high as possible ⇒ best resolution*	*as low as possible ⇒ high velocities measurable*
Intercept angle	*as perpendicular as possible ⇒ optimal reflection*	*as parallel as possible ⇒ smallest error*

Comprehensive perioperative TEE examination

Jan Hultman, Stockhom, Sweden

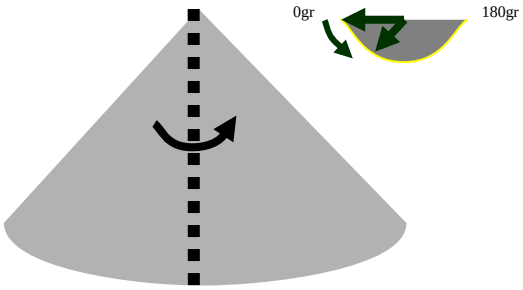
The great importance of echocardiography in open heart surgery is undisputable. Moreover, development of new clinically applicable techniques has further shown its value. However, in order to use all the advantages for diagnosis and monitoring the user has to be familiar with the basic views and what a comprehensive examination should include. The OR and ICU setting differ considerable from the echolab which emphasises the importance of what the focus is for the examination. Despite all modern techniques they are all based on the standard basic views and therefore this lecture is focused on how they are obtained. It is well known that TOE examination and report in the OR and ICU often is undertaken under time limited and stressed circumstances. Nevertheless the whole heart including the pericardium should be examined in each case. With training it does not take more than approximately 5 minutes for an overview. Therefore it is important to acquire and use a routine for the procedure and report.

Advantages

- *Less risk of missing important information*
- *You learn more with documentation and report*
- *Documentation and report makes second opinion possible. Remember that no one can blame you for making assessment errors. However you are to blame if you cannot show what your report is based upon.*
- *Echocardiography is a picture medium and no lab answer i.e. several observers and a discussion could be necessary for a consensus*

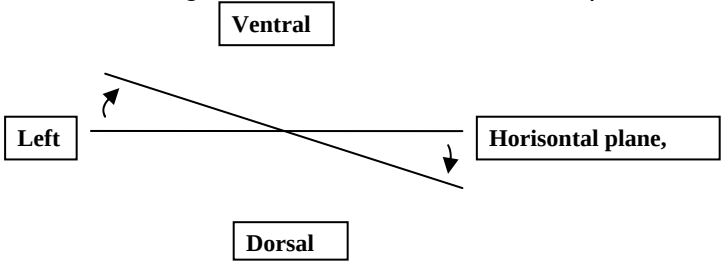
My suggestion for the routine examination/report
Follow the blood through the heart!

A pathologic finding should always be examined in several views otherwise there is a considerable risk for misinterpretation. With the regular 2D image it should be kept in mind that structures are seen in one plane only. The new technical development in 3 dimensional echocardiography facilitates correct assessments but its relevance in clinical practise needs more evaluation. Still in 2D imaging the TOE investigator has to build a three-dimensional picture in the mind. With the multiplane probes it is easy to obtain numerous images/views from the same position of the probe by just rotating the crystal (fig 1). Try to place the focus of interest in the middle of the image (the dotted line) which enables multiple views of the object you want to study in detail.



Figur 1

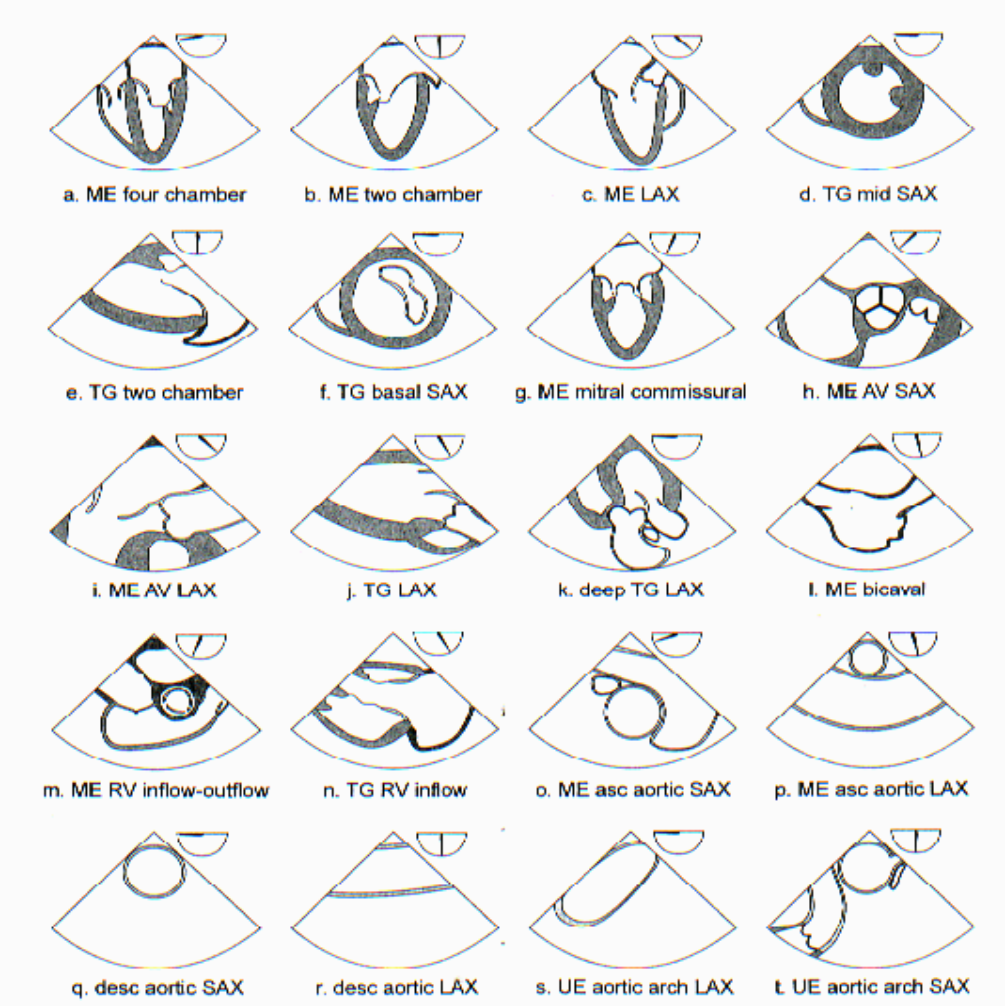
The rotation of the crystal is clockwise like in figure 2 with reference to the handle/proximal part of the probe/and from the anaesthesiologists view from the head of the patient.



Figur 2

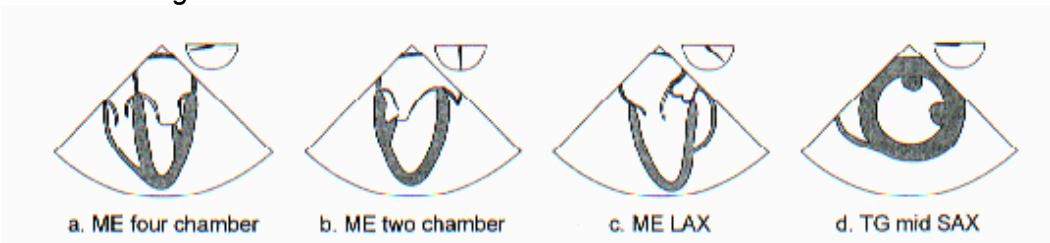
For the beginner this is one of the main obstacles in the beginning of the learning process. The best way to learn how the anatomy of the heart is pictured in the different standard TOE views is during a wet lab situation. To overcome the confusions in TOE terminology for the views the

ASE and SCA have agreed to the standards as in figure 3.



Figur 3 From Shanewise et al, Anesthesia and Analgesia 1999. ME = midesophageal, TG=transgastric, SAX= short-axis, LAX=long-axis, UE=upper esophageal, RV=right ventricle

With each view also the rotation of the crystal is shown. To obtain these views with this rotation/angle of the crystal the starting-point is a horizontal plane of the crystal window which has to be lined with the horizontal plane of the patient. Of course, for example to find the optimal ME 4 chamber view the rotation of the crystal has to be slightly modified from patient to patient. Therefore in most textbooks on TOE images a considerable range in the rotation of the crystal is mentioned. Most used views/standard views/"starting views"



ME=mid esophageal, LAX=long-axis, TG=transgastric, SAX=short-axis

These views are standards because the LV is often the focus of interest and they are usually easily obtained. Moreover for example from the ME four chamber view it is easy to make an assumption about what is wrong. The depth in the oesophagus is not mentioned in the summaries below because it varies considerable between patients. However the ME views are mostly found in the range of 25-30cm from teeth.

To obtain

ME four chamber
Retroflexion of the probe and a rotation of the crystal to 10-20degrees. Both the mitral and tricuspid valve should be visible.
ME two chamber
Retroflexion of the probe and crystal rotation to 90 degrees.

ME LAX
Retroflexion of the probe and crystal rotation to 120-140 degrees

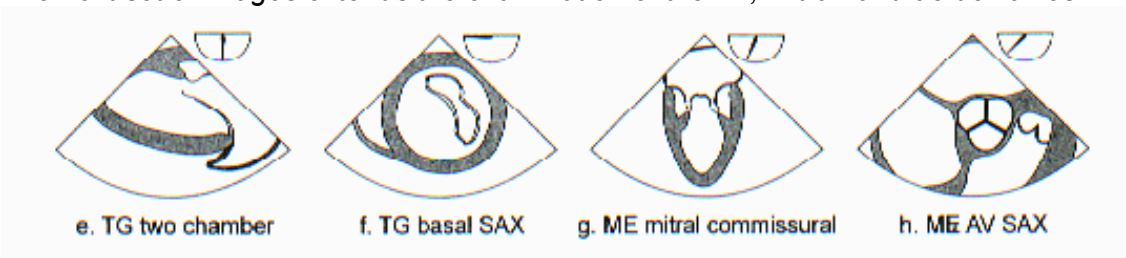
TG mid SAX
The transgastric probe position with the probe antiflexed. The papillary muscles should be visualized to avoid an oblique view.

The use

2D/Mmode/+ colour
Morphology, dimensions (preload), wall thickness and function of the LV (RV), tricuspid-, mitral- and aortic valves. Also the dimensions of left atrium and left atrial appendix and atrial septum morphology.

Doppler
Transvalvular flow in the tricuspid- and mitral valves. Aortic subvalvular Doppler.

The next set of images extends the examination of the LV, mitral- and aortic valves



AV= atrioventricular and refers to the atrioventricular plane

To obtain

TG two chamber
Rotate the crystal to 90 degrees from the TG SAX view.

TG basal SAX
Keep the probe anteflexed and pull the probe slowly from the TG mid SAX view. Observe that it sometimes is difficult to obtain a plane where both mitral leaflets are seen.

ME mitral commissural
This view is a modification from the ME two chamber by rotating the crystal from 90 to approximately 60-70degrees.

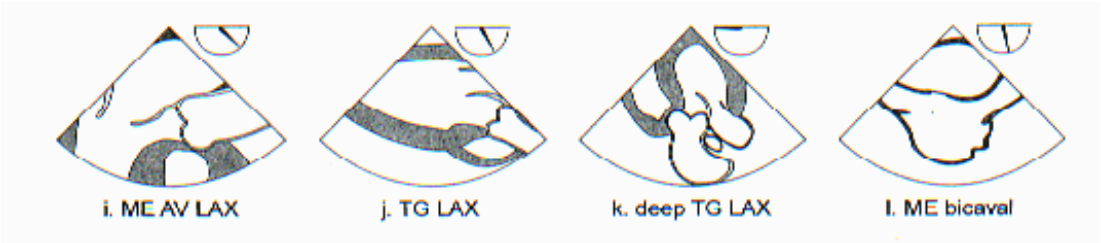
ME AV SAX
In this view the focus of interest is usually the aortic valve. Start with the ME 4 chamber view and focus on the aortic valve. After that rotate the crystal to approximately 40 degrees.

The use

2D/Mmode + colour
LV morphology, dimension, wall thickness and function. Mitral- and aortic valve morphology and function. Morphology of the left atrial appendix, RV outflow tract and atrial septum.

Doppler
RV outflow tract

The next set of images are focused on a more detailed examination of the subaortic area of the LV, aortic valve, sinus Valsalva, sinotubular junction and the first part of the ascending aorta. Also on the LA/RA, inferior- and superior vena cava



To obtain

ME AV LAX
Rotate the crystal to 120-140 degrees from the ME AV SAX but keep the aortic valve in the midline of the image (See fig 2).

TG LAX
Extend the rotation of the crystal to 120-130 degrees from the TG two chamber view

Deep TG LAX
Go deep into the ventricle and anteflex the probe in O degrees

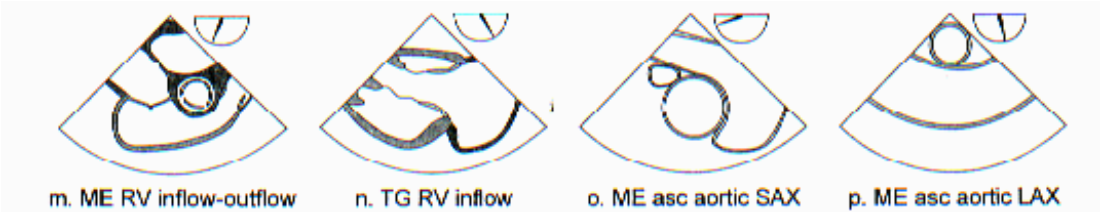
ME bicaval
Start with the ME 4 chamber view and withdraw the probe to focus on the atria. Thereafter rotate the crystal to approximately 100 degrees.

The use

2D/Mmode + colour
LV outflow tract, subaortic, aortic valve, sinus Valvsalva and ascending aorta morphology dimensions and function. Inferior- and superior vena cava and atrial septum morphology. LA/RA morphology dimension and function.

Doppler
Subaortic and aortic valve flow. It could be difficult to line the Doppler signal adequately in both the TG LAX and deep TG LAX views. Moreover it is not taken for granted that a perfect 2D image is a guarantee for an optimal lining.

More images are needed to examine the RV in more detail and also the aorta and the pulmonary artery.



To obtain

ME RV inflow-outflow
Use the ME AV SAX and extend the rotation of the crystal to 60-70degrees

TG RV inflow
With the probe in the TG LAX just rotate the whole probe to the right side of the patient

ME asc aortic SAX
This is a basal view and a withdrawal of the probe is needed from the ME 4 chamber image. The probe should be antefexed.

ME asc aortic LAX
Extend the rotation of the crystal from the ME asc aortic SAX to approximately 120 degrees.

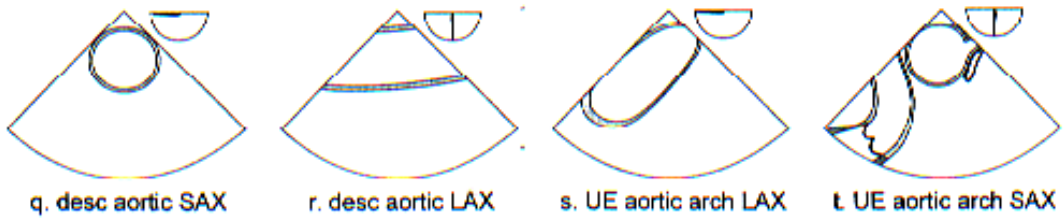
The use

2D/Mmode + colour

RV, tricuspid and pulmonary valve morphology, dimension and function. Ascending aorta and pulmonary artery (including the bifurcation and right pulmonary artery) morphology and dimension. The left pulmonary artery is difficult to see because of airway interference.

Doppler
Tricuspid valve and pulmonary artery flow.

The following images are focused on the aorta



UE= upper oesophagus

To obtain
Desc aortic SAX
Rotate the probe with the crystal window facing dorsal/posterior

Desc aortic LAX
Rotate the crystal to 90 degrees from the probe positioned as in the desc aortic SAX

UE aortic arch
Follow the desc aorta proximal until the aortic arch is reached. Thereafter rotate the probe slightly to the right. Often the aortic arch is depicted more horizontal in the image compared to the image in this figure.

UE aortic arch SAX
This is the distal part of the aortic arch with the left subclavian artery. Because of the near field location of the left subclavian artery it is sometimes difficult to find and the image can be poor.

The use
2D/Mmode + colour
Aortic and left subclavian entry, morphology and dimension.

Doppler
Usually of no use

All presented views should be regarded as standard views and all cardiothoracic anaesthesiologists should be familiar with how to find and interpret them. Once this skill and knowledge are acquired every view could be modified to optimize the examination. With the multiplane probes any structure could be investigated from 0 to 360 degrees by rotating both the probe and the crystal. However the image could be lost especially with a rotation of the probe because of lost contact with the surrounding structures. Therefore some investigators prefer larger probes for improved image quality. However, introducing a large size probe into the oesophagus means increased risk for hurting the patient. Modern TOE probes have become smaller but still with improved image quality. The beginner should as soon as possible focus on the image and not how far into the oesophagus the probe is positioned and how much the crystal is rotated. The standard views are used as reference. Also if you are lost- always rotate the crystal to 0 degrees and face the window of the probe ventral/anterior.

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Safety and complications of TEE

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Introduction.

TEE is rightly regarded as essentially a safe procedure. It is sometimes described as "semi-invasive" to distinguish it from invasive intravascular techniques. However, TEE involves the same degree of invasiveness as a gastroscopy, and therefore usually requires topical local anaesthesia, most commonly with intravenous sedation, or is undertaken during general anaesthesia.

The morbidity and mortality of TEE is comparable with that of upper GI endoscopy, i.e complication rate of 0.08-0.13%, and a mortality rate of 0.004%. (1,2) Some of the data on the safety of TEE comes from patients undergoing diagnostic TEE in the echo lab, rather than patients in the OR. Many of the complications of sedative and local anaesthetic drug overdosage are confined to the setting of the awake patient. Nonetheless there are a number of other safety issues, and there are now sufficient data to evaluate safety both in the operative and non-operative setting.

Safety Issues

1. The use of drugs that may produce inadvertent general anaesthesia in an inappropriate setting has been the subject of concern, and many national and institutional guidelines exist to encourage safe practice. These generally involve dosage guidelines, minimum monitoring requirements for patients undergoing procedures under intravenous sedation, and facilities for resuscitation and recovery.

2. Upper GI trauma may be related to "blind" insertion, and the size of the probe and probe tip relative to the size of the oesophagus. Persistent attempts to place the probe in the oesophagus despite resistance may be associated with pharyngeal trauma which may be serious. In the worst scenario, oesophageal perforation may occur which carries a high morbidity and mortality.

3. TOE must be undertaken with extreme caution in patients with pre-existing gastrointestinal pathology. Many would regard oesophageal stricture, diverticula, varices, achalasia, Mallory-Weiss tear, tumour and recent oesophageal surgery as absolute contraindications to TOE. Although evidence of such disease may often be available in the cardiac surgical patient, it is not invariable.

4. In cardiac surgery patients, prolonged intubation tracheal and IPPV may mask signs of trauma.

5. TEE may be responsible for some minor oesophageal and gastric trauma, but the degree of bleeding may be worsened by heparinization and perioperative coagulopathy. Other conditions (e.g. acute gastric erosion) may also be responsible.

6. The incidence of adverse oesophageal symptoms following TOE is still unclear. Short term symptoms of dysphagia (difficulty in swallowing) and odynophagia (painful swallowing) have been shown to be similar in cardiac surgery patients with and without TOE (3). In contrast, others have shown an increased incidence of dysphagia (4) and a further study identified age, duration of endotracheal intubation, and use of TEE as significant predictors of postoperative dysphagia.(5)

7. Airway obstruction may occur, particularly in paediatric patients. An incidence of 1-2% has been reported (6), suggesting that this is a frequent problem that demands vigilance. In adults, airway obstruction has been reported, particularly in small elderly patients who had undergone a prolonged or traumatic procedure.

8. Successful passage of the TOE probe is not invariable. Failure has been reported in 0.7-1.9% of awake sedated patients (1), 0.8% of anaesthetised paediatric patients (7), and 0.18% of anaesthetised and ventilated adult patients.(8)

9. There appears to be no reliable data yet to question the biological safety of ultrasound in the context of TOE. Patients are protected against thermal injury by automated systems to control the temperature at the probe tip.

10. The development of bacteraemia, particularly in patients at risk of endocarditis, is an issue, although perioperative practice invariably involves prophylactic antibiotics.

11. Cleaning the TOE probe and prevention of cross infection are important considerations. Most institutions in the UK require active bactericidal cleaning of the probe between cases whether a protective sheath is used or not.

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LV Systolic function

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Introduction

The evaluation of the size and function of the left ventricle (LV) in patients with known or suspected heart disease is a central diagnostic issue in several clinical situations. In contrast with other methods that have been developed over the last decade to assess both qualitatively and quantitatively the left ventricular function (LVF), such as right heart catheterization, angiocardiography, radionuclide ventriculography and more recently nuclear magnetic resonance, cardiac ultrasonography is a widespread and readily available procedure. It is a non-invasive technique that can provide morphologic and functional information of the heart, with no stress for the patient and no exposure to contrast medium or to radiation. In perioperative clinical scenarios the posterior approach by transoesophageal echocardiography (TOE) provides high-quality real-time images of the beating heart by M-mode and two-dimensional echo, and qualitative and quantitative assessment of blood flow in the heart and vascular structures by Doppler technology.

In the management of critical patients, as a monitor of cardiac patients in cardiac and non cardiac surgery, and of ICU patients with acute haemodynamic decompensation, and also in some special settings, either elective either emergent (i.e. mitral valve repair, aortic dissection), TOE is an important tool for the evaluation of LVF. It has an important role to disclose causes of acute and chronic haemodynamic disturbances, by elucidating systolic performance, preload conditions and diastolic function, and by detecting changes in regional contractility which may lead to diagnosis of acute myocardial ischemia in patients at risk for it or suffering from obstructive coronary artery disease. The TOE evaluation of the LV is based on a systematic study through M-mode, 2-D and Doppler flow investigation. Such a study can lead to a whole anatomical and functional understanding, so allowing the assessment of global and regional systolic LVF and of LV diastole.

***The study of systolic function**.*

The term "contractility" is often used to describe the systolic LVF. However we should consider that contractility refers to the inotropic state of the myocardium, that is the intrinsic strength of the muscle fibers. This intrinsic property of the myocite depends on the neurohormonal and metabolic milieu surrounding it. In particular, sympathetic nerve activity, pH and Ca^{++} release from the sarcoplasmic reticulum mainly influence cardiac fibers contractility. A change in contractility is defined as an alteration of the inotropic state independent of preload, afterload and heart rate. So it is important to remember that most of the indices used in clinical practice to study LV performance fail to assess intrinsic myocardial contractility, being influenced by heart rate and loading conditions.

Systolic evaluation in clinical practice.

From a clinical point of view, where the practice of evaluating the LVF is aimed to support clinical management, TOE systolic evaluation takes into account the complex LV ellipsoidal geometry. This leads to estimate LVF through methods based on the assessment of changes in diameters, areas and volumes of the LV chamber both on systole and diastole.

Such evaluation is routinely performed by registration of LV images through standard views that can be easily obtained with common omniplane transducers, as reccomended by the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists.

Systolic function: changes of LV diameters

Based on diameter change, TOE allows measurement of Shortening Fraction (SF). This is obtained in the mid-papillary view of the LV, by M-mode measurement of the

difference between end-diastolic and end-systolic diameters normalized for end-diastolic diameter (fig. 1). The normal value is ≥ 30%.

With the measure of SF, simple linear dimensions can provide an estimate of overall performance of LVF. This is certainly an advantage of this method in routine evaluation,

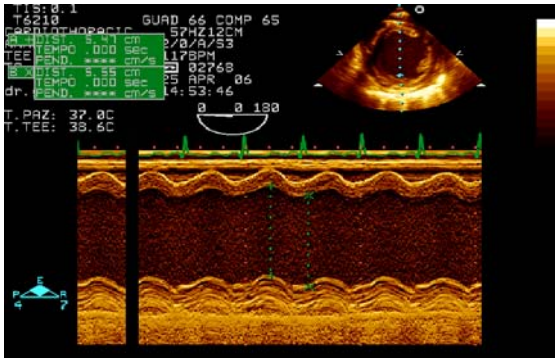


Fig. 1 M-Mode: SF measurement

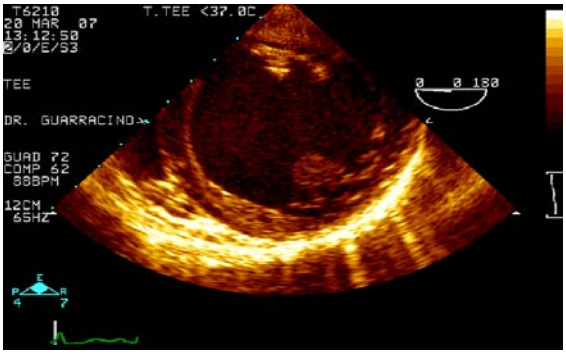


Fig. 2 a Midpapillary TG view of the LV

where time sparing and easy-to-do methods are searched for, but some important limiting aspects have to be taken into consideration. This measurement assesses only one portion of the LV, that is the mid-papillary one. This implies that SF can reflect global LV function only if LV has a uniform contraction in all other parts. For instance, the presence of a basal hypokinesia or apical aneurism would make it impossible for SF to reflect global LV systolic function. Other conditions that need to be considered are bundle branch block and right ventricle dilation, which too can affect uniform and sinergic LV pattern of contraction.

Systolic function: changes of LV area

The TOE assessment of LV systolic phase on area change is based on calculation of the Fractional Area Change (FAC). It is obtained in midpapillary short-axis view (fig. 2 a) by 2-D measurement of end-diastolic and end-systolic areas normalized for end-diastolic area (fig. 2 b). The normal value is ≥ 45%.
The 70% of the LV stroke volume depends on mid-papillary portion contraction. This is why FAC can provide a reasonable global estimate of LVF, which correlates with ejection fraction.

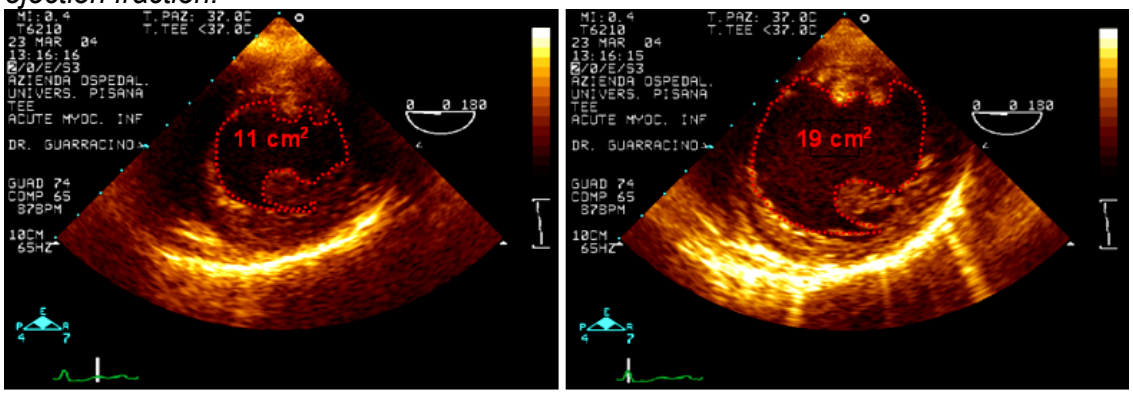


Fig. 2 b Measurement of LV end-diastolic and end-systolic areas.

But, as underlined for SF, this measurement has some limitations that have to be considered. FAC at midpapillary level may not correlate with global function in patients with myocardial infarction in other areas of the ventricle that are not investigated through this view. Moreover, changes in loading conditions can alter FAC measurement. The measurement of ventricular areas can be also obtained by automatic detection of myocardial border, as with acoustic quantification (see Automatic Border Detection).

Systolic function: changes of LV volume

TOE evaluation of LVF based on changes of LV chamber volumes, consists of measurement of ***Ejection Fraction*** *(EF) and* ***Stroke Volume*** *(SV).*

Ejection Fraction
EF is a well accepted and useful index of quantitative LVF, but it is influenced by changes in load conditions, both pre- and after-load, and contractility. So it is not an index of contractility! EF can be considered as a measure of the interaction among preload, afterload and contractility in determining ventricular performance. In this sense it can be a good estimate of LVF.
EF calculation is obtained in long-axis views of the LV, by measurement of end-diastolic and end-systolic volumes normalized for end-diastolic volume (fig. 3). The normal value

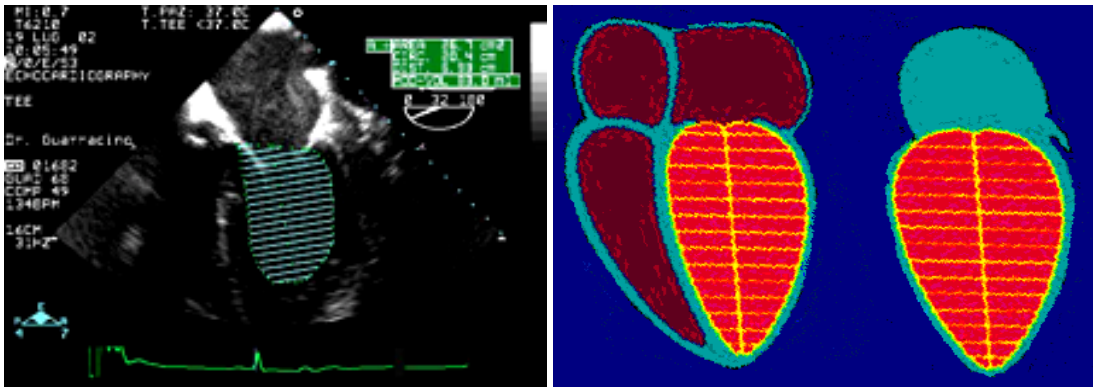


Fig. 3 Long-axis four chamber view of LV for EF measurement.

is ≥ 60%. This measurement can be performed with two methods: the area-length method and the Simpson's rule.
With the area-length method the LV is assumed to be an ellipsoid, and volumes are obtained by measuring LV areas and lengths at both end-diastole and end-systole. Those measurements require a long axis view of the chamber. A problem that often arises with TOE is the difficulty to obtain a good visualisation of the true apex in the four-chamber view at 0-30° from the oesophagous.
The Simpson's rule assumes that the LV volume can be obtained by summing the volumes of multiple slices of known thickness that compose the ventricle itself. Each slice is considered as a ellipsoid cylinder. The measurement requires two different long axis views of the ventricular chamber. This method, known also as "disk summation method", is integrated in the software of echo machines (fig. 3).
Whatever the method used, who uses the echo tool has to remember some important points that can influence EF measurement and help in obtaining a correct quantification:

- ✓ Obtain the best apical view you can by searching for the true long-axis view;
- ✓ Adjust settings in order to have the best detection of endocardial border;
- ✓ Always select appropriate sinus beats. End-diastole occurs at the peak of R-wave, and end-systole at half of the T-wave;
- ✓ Take three heart cycles in case of sinus rhythm; average nine-ten cycles in case of atrial fibrillation.

Stroke volume
The SV is a useful index for the evaluation of the haemodynamic status of patients and in assessing the response to cardiovascular therapy. It can be obtained with 2-D TOE by measuring LV volumes, as decribed above for EF, or by using Doppler technology, which allows measurement of forward flow through any transverse section area within the heart, combined with 2-D echo measurement of a cross sectional area (CSA).
Like EF, SV is influenced by load conditions, contractility and heart rate too. Once determined the end-diastolic and end-systolic volumes as described for EF calculation, their difference will give the SV. Normal value is 50-80 ml.
All limitations described for EF are to be considered in SV measurement also.
Based on combination of Doppler sampling of flow and 2-D measurement, the calculation of the SV is obtained from the integration of the instantaneous blood flow velocity over the CSA of the outflow tract (LVOT) or the aortic valve (AV) during one

cardiac cycle. So the formula for SV calculation is: SV = CSA • TVI, where TVI is the time velocity integral detected with the Doppler.
What we need to measure are the CSA with 2-D and the velocity of the blood flow by Doppler sampling the flow through the choosen cross section (LVOT, AV). The Doppler sampling of the LVOT or the AV is performed in the deep transgastric view or the longitudinal transgastric view at about 90-100°, where a good alignment of ultrasound beam and blood flow is possible (fig. 4 b). From a midoesophageal long-axis view at 120° the LVOT or AV diameter is measured (photo 11), and the CSA can be calculated by the formula:

CSA = π • (diameter / 2)^2 (fig. 4 a).

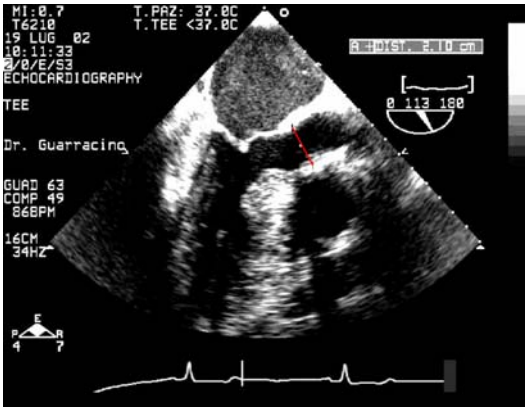


Fig. 4 a LAX at 120°: measurement of CSA

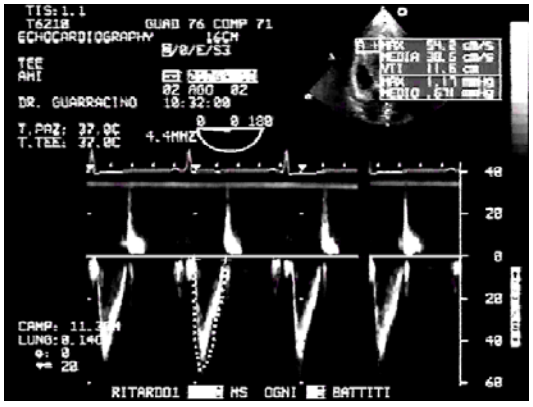


Fig. 4 b VTI measurement by Doppler

Based on the continuity equation, SV can be measured by sampling any other valve. Normal value of SV = 60-90 ml.
SV times heart rate provides the Cardiac Output. This is of importance in many clinical situation, when Cardiac Output can be assessed by serial sampling of flow. As the area of anatomic structures as LVOT or AV does not change over time, it is enough to determine the TVI of forward flow to monitor cardiac output. A change of TVI as a consequence of any treatment allows the phisician to understand the response to therapy, so influencing the clinical management of hemodynamics. Of course this method cannot be considered alternative to pulmonary artery catheterization, as in many perioperative scenarios the two methods are to be considered complementary. The usefulness of TOE calculation of SV and cardiac output relies on the quick and non invasive measurement, which makes it relevant in the prompt assessment of hemodynamics.
Other indices of LVF, such as Isovolumic Contraction Time and dP/dt, both indices of isovolumic phase, the maximal power and the peak aortic flow acceleration at early systole can be determined by TOE, but their use in everyday practice is limited by complexity of measurements and time-consume, but also by dependence on load conditions and inotropic state changes. For dP/dt calculation mitral regurgitation has to be present too.

End-systolic indices in LVF evaluation.

A different approach to myocardial contractility is based on the investigation of the relationship of end-systolic indices. Such indices reflect the intrinsic inotropic state of the myocardium and are not influenced by loading conditions. The end-systolic volume to peak systolic pressure and the end-systolic area to peak systolic pressure are two of the relationship more widely studied in this field. The left ventricular end-systolic pressure is red as end-systolic arterial pressure at dicrotic notch, while end-systolic volume, measured with methods described for EF calculation, and end-systolic area are obtained by TOE. By plotting end-systolic pressure and end-systolic area or volume, and then obtaining a new pressure-area or volume curve with manipulation of preload or afterload, it is possible to assess the inotropic state of the myocardium through the end-

systolic pressure-volume relationship. In the clinical use of TOE these indices of myocardial performance are not currently used, but future technological progress in the automation of such pressure-volume curves will surely lead to their routine use.

Regional contractility: the study of left ventricular wall motion.
During myocardial ischemia or after myocardial infarction, the myocardial territory involved will contract abnormally or even stop contracting. Therefore a change in regional wall motion (RWM), as detected by echocardiography, is a reliable sign of myocardial ischemia. Ultrasound is very sensitive in detecting myocardial ischemia, and TOE is reported to be more sensitive even than changes detected by ECG and pulmonary artery catheter, but certain limitations can make some assessments difficult. Assessment of wall motion abnormalities (WMA) is mostly subjective, and it takes a certain degree of experience to accurately assess WMA. Based on a semiquantitative "eyeballing" evaluation, severity is graded from no WMA (normal) to akinesis or even dyskinesis. The appearance of a normally contracting wall on echo is that of motion and thickening. Description of location and extent of RWMA requires a segmental model of the LV.

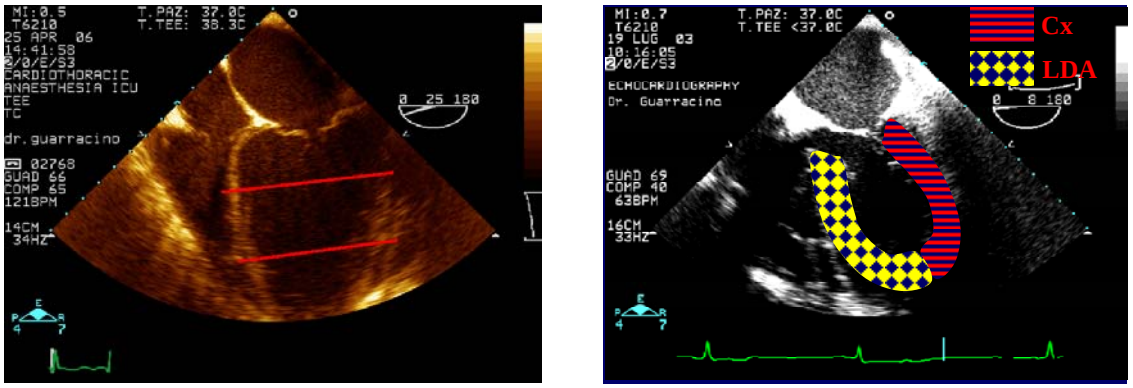


Fig. 5 LV segmentation for regional wall motion analysis

Based on the recommendations of the American Society of Echocardiography Standard Committee, the ASE/SCA guidelines for performing a TOE exam suggest a division of the LV into three levels from base to apex (fig. 5): basal level, extending from mitral valve annulus to the tips of the papillary muscles, mid level, extending from the tips to the basis of the papillary muscles, and apical level, representing the remainder part of the LV. Basal and mid levels are then divided into six segments, the apical level into four. So the LV is divided into 16 segments. This segmentation allows the evaluation of each single segment for motion and thickening during the systolic phase of the cardiac cycle. The 16 segments can be investigated through the four-chamber, the two-chamber and the long axis view, and the transgastric basal and midpapillary. The analysis of WM can be quantitative by using a grading scale for motion and thickening, which leads to a scoring system. The grading of motion is based on the evaluation of segmental radial shortening of the distance from the endocardium to the centre of the LV cavity during systole. Thickening is graded on the increase in the distance between endocardial and epicardial border during systole. This increase is estimated by eyeball on a scale from + to +++ (tab.1). The evaluation of WM is highly dependent on the observer experience. The LV wall movement can be due to other causes than contraction alone. The heart shifts during respiration, and also has rotational and translational movements. In intraoperative scenario other possible movements related to surgery may be present and affect the evaluation. Although based on an arbitrary estimation, systolic thickening is of great importance in the evaluation of WM because it is poorly influenced by rotational and translational movements, and has a high correlation with the extent of normally perfused myocardium.

For routine purposes, when monitoring of myocardial ischemia is needed, the transgastric midpapillary short-axis view is the most useful. It is easy to obtain and to keep thanks to the papillary muscle morphology; it allows a good visualization of myocardial regions supplied by all three coronary arteries, so that any change in WM can be readily matched with coronary anatomy or information from coronary angiogram; sensitivity and specifity for WM are very high at this level of LV.

Tab.1	% radial shortening	Thickening	Score
Normal	**> 30**	**+++**	**1**
Mild hypokinesis	**10 - 30**	**++**	**2**
Severe hypokinesis	**<10, >0**	**+**	**3**
Akynesis	**0**	**0**	**4**
Dyskinesis	**paradoxical**	**thinning**	**5**

Based on WMA, criteria for diagnosis of myocardial ischemia are the following:

- the WMA is a new finding (i.e., hypokinesis of a previously normally contracting myocardial segment, or a akinesis of a previously hypokinetic area),
- it worsens by at least two scores,
- it has a duration > 1 minute at least.

Non ischemic WMA *can be due to normal regional heterogeneity, altered loading, tethering or systolic dysfunction, bundle branch block, and ventricular pacing. Also CAD related causes, such as infarction, stunning and hibernating, can create WMA.*

New Advances in global and regional LV evaluation.

In the last recent years the progress of industry in ultrasound technology has led to new advances in the field of ventricular evaluation. Main progress involved automatic detection of tissue-blood interface and measurement of myocardial tissue velocities.

Automatic Border Detection: acoustic quantification

In some modern echo machines a new software for automatic detection of endocardial border is included. With this technology, called Acoustic Quantification, blood and tissue are clearly discriminated by integrated analysis of backscattered signal from endocardium and blood. A coloured border indicator allows delineation of the endocardial borders. By drawing a region of interest (ROI) including the LV cavity, it is possible to get continuous, real time determination of areas and volumes (fig. 6). This method can reduce variability in border detection among operators, and is able to provide a real-time measurement of the LV areas and volumes, and so of FAC and EF. An accurate assessment, however, is strongly influenced by the quality of images and by the settings of the echo machines.

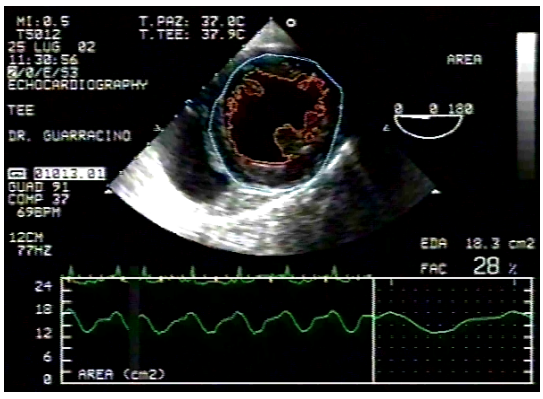


Fig. 6 Acoustic quantification of LV

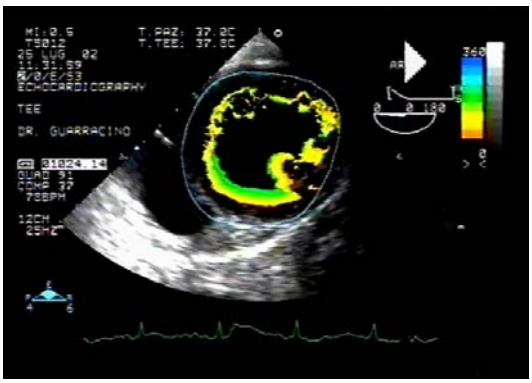


Fig. 7 Colour Kinesis of LV

Color Kinesis

This tool is based on acoustic quantification technology, of which it represents an extension. This ultrasound technology tracks the motion of the endocardium in systole

and provides color-ancoded images reflecting the magnitude and timing of endocardial motion. The duration of systolic phase of the cardiac cycle is divided in several moments. Each of this sub-phases is represented with a different colour starting from orange at isovolumetric contraction, going through yellow during early ejection, then green, and finally blue at late systole (fig. 7). At each color corresponds a different velocity of the endocardial wall towards the cavity. Normal segments show a full color pattern from orange to blue, reflecting an increase in velocity from 0 to 360 cm sec^{-1} at end systole. The color-encoded image is very helpful in evaluating systolic movement of LV walls, so allowing a global and regional qualitative assessment of systolic LVF.

Tissue Doppler Imaging

The Tissue Doppler is a recent application of Doppler sampling to detect myocardial tissue velocity through cardiac cycle. The Doppler technology is modified to detect low velocities from tissue movement: consecutive phase shifts of ultrasounds reflected from myocardium are detected, and intramural myocardial velocities are determined.
Myocardial movement is represented with color Doppler by changes from blue to red according to standard pattern. No color is detected from akinetic segments.
The analysis can be performed with M-mode to assess myocardial velocities over time. Pulsed Doppler application allows registration of myocardial velocities throughout cardiac cycle both in systole and diastole (fig. 8).
The pulsed-TDI provides a velocity map of the myocardial wall during both systole and diastole. During systole a velocity (V_s) wave is recorded, whose reduction is observed during myocardial ischemia. The registration of V_s from ventricular segments leads to regional assessment of WM. Preliminary studies report high sensitivity and reproducibility, and indicate pulsed-DTI as a promising method to assess regional LV contractility.
The usefulness of DTI examination relies on 1) the possibility to perform a complete evaluation of any segment of LV wall, 2) the possibility to have appropriate information on LV systolic function also in those situation in which standard 2-D exam can be misleading, such as bundle branch block, pacemaker implanted heart, pericardial abnormalities, that is all those settings causing apparent or true abnormal motion of LV segments.
For further clinical application of tissue Doppler in evaluation of myocardial deformation see chapter on Strain and Strain Rate echo.

Conclusion.

Perioperative TOE evaluation of LV systolic function, both global and regional, provides insight into hemodynamic impairment. This is useful in cardiac and non cardiac surgical settings, and also in intensive care unit any time a reliable and repeatable evaluation of cardiac function is needed.
TOE evaluation of LV systolic function requires a systematic and complete study of anatomic and functional features. This is possible by performing a standard exam and by applying all ultrasound methods, 2-D, M-mode, Color Doppler and spectral Doppler in order to obtain a whole qualitative and quantitative evaluation of systolic function. Application of modern technologies allows detection of new indeces of ventricular function, that are promising to play a relevant role in the assessment of LVF.

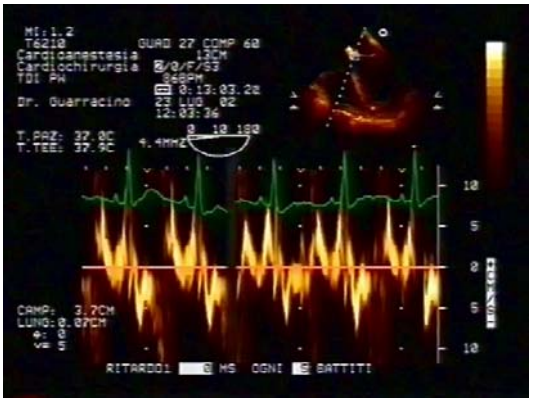


Fig. 8 Tissue Doppler evaluation of LV

TOE assessment of LV Diastolic Function

Patrick F Wouters, Gent, Belgium

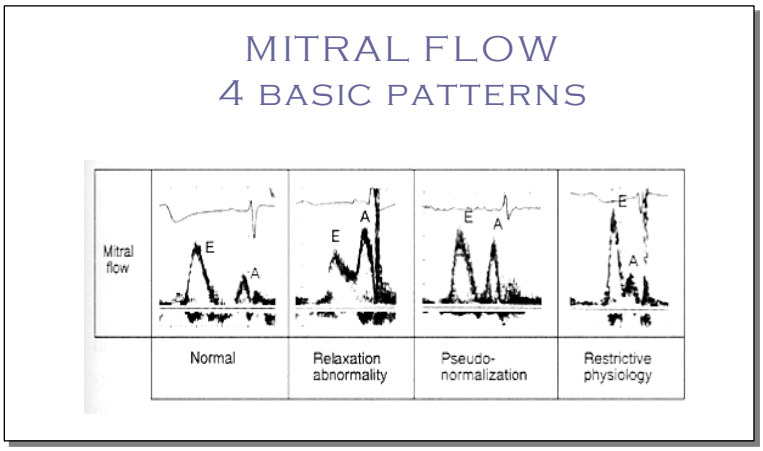
In cardiology the diagnosis of diastolic heart failure requires three conditions: (1) the presence of signs or symptoms of heart failure; (2) a normal or slightly reduced LV ejection fraction and (3) increased diastolic filling pressures. Large-scale epidemiological studies show that the incidence of primary diastolic heart failure is rather high, particularly in the elderly, and accounts for one third of all patients presenting with congestive heart failure. The pathophysiology of diastolic dysfunction includes delayed relaxation and/or increased stiffness. These conditions produce an upward displacement of the diastolic pressure–volume relationship with increased end-diastolic, left atrial and capillary wedge pressure leading to symptoms of pulmonary congestion. The prognosis of diastolic heart failure is thought to be better than for systolic dysfunction (lower annual mortality rate of approximately 8% vs 19% - although this has recently been challenged) but morbidity rates can be substantial. There is no specific therapy to improve LV diastolic function directly. Medical therapy of diastolic dysfunction is often empirical and lacks clear-cut pathophysiological concepts. Treatment of the underlying disease (myocardial ischemia, arterial hypertension, aortic valve stenosis) is currently the most important therapeutic approach although data suggest now that calcium channel blockers, beta-blockers, ACE-inhibitors and AT2-blockers as well as nitric oxide donors may be beneficial. Invasive assessment of diastolic function allows determination of the time constant of relaxation from the exponential pressure decay during isovolumetric relaxation, and the evaluation of the passive elastic properties from the slope of the diastolic pressure–volume (constant of chamber stiffness) and stress–strain relationship (constant of myocardial stiffness). Doppler Echocardiography has become the best available non-invasive tool to evaluate diastolic performance. There is no evidence yet of its utility in the surgical patient, however, the knowledge and technology now being available in the operating theatre should help us identify patients at risk for - or with diastolic dysfunction and adapt our hemodynamic management accordingly. The difficulty in evaluating diastolic function in surgical patients is the high variability in loading conditions in this setting and most diagnostic echocardiographic measurements/variables are influenced by preload.

Basically there are four techniques to evaluate diastolic performance i.e. analysis of
- *Transmitral flow patterns using pulsed wave Doppler*
- *Pulmonary venous flow patterns with pulsed wave Doppler*
- *Mitral annular motion using tissue Doppler imaging (either with pulsed or color wave Doppler)*
- *LV inflow propagation velocity using color Doppler M-mode*

Also atrial dimensions provide important information. An enlarged left atrium often is the signature of longstanding diastolic dysfunction.

The transmitral flow pattern remains the starting point: it is easy to acquire and rapidly categorizes patients into normal, delayed relaxation, and restrictive filling patterns. It is important to always position the sample volume correctly at the level of the mitral valvular (MV) tip using the 4-chamber midesophageal view, with the Doppler beam slightly angulated to the lateral wall. Signal gain needs adjustment to obtain a clear spectral envelope and the ECG tracing must be displayed on the screen to properly relate the signals to phases in the cardiac cycle. Mitral inflow velocities reflect the instantaneous pressure gradient between left atrium and left ventricle. Velocities of the early diastolic inflow (E-wave) relate to the pressure drop occurring during isovolumetric relaxation (and the amount of blood present in the atrium before MV opening). In healthy adults peak E velocities are typically higher than the second component of the mitral inflow signal that relates to atrial contraction (A-wave). With impaired isovolumetric relaxation, the magnitude of peak E-velocities decreases and a compensatory rise in atrial velocities occurs. Hence, early stage diastolic dysfunction

typically is associated with reduction of E velocities, a lower ratio between E and A max velocities and a prolonged deceleration time of the E wave component. The natural response to the body to adapt to this condition is to raise atrial pressures and restore the transmitral pressure gradient. As a result, transmitral flow variables (I.e. E and A velocities, E/A ratio and E deceleration time) will return to normal values despite the further progression of disease. This stage is referred to as 'pseudonormalization' since the mitral inflow profile cannot be distinguished from the normal pattern except for the fact that it occurs at elevated atrial pressures. At this stage, additional measurements are required – either invasive assessment of filling pressures or by using one of the other echo-techniques described below that do offer incremental information on diastolic function. With further progression of diastolic dysfunction mitral flow analysis regains its diagnostic potential: further increases of filling pressures produce very high E-velocities, supranormal E/A ratios and a shortened E-deceleration time which indicate restrictive filling.(Figure 1)



Firgure 1

<u>Pulmonary venous (PV) flow</u> velocities reflect the instantaneous pressure differences between pulmonary veins and atrium. They consist of a systolic (often biphasic), a diastolic and an atrial component. The diagnostic utility of the diastolic D-wave is comparable with the mitral E-wave.(Figure 2) The atrial component however will increase both in magnitude and duration when atrial pressures rise. Hatle et al. showed that the difference in duration between the PV atrial wave and the MV atrial wave was directly related to atrial pressures (Rossvoll and Hatle JACC 1993). Hence, this variable adds useful information to discriminate a normal from pseudonormal MV flow signal. However, A-wave characteristics are also affected by atrial function and are absent in atrial fibrillation.

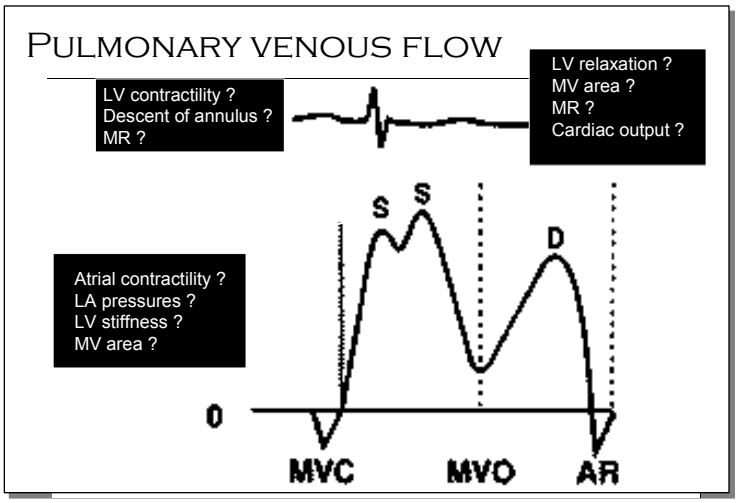
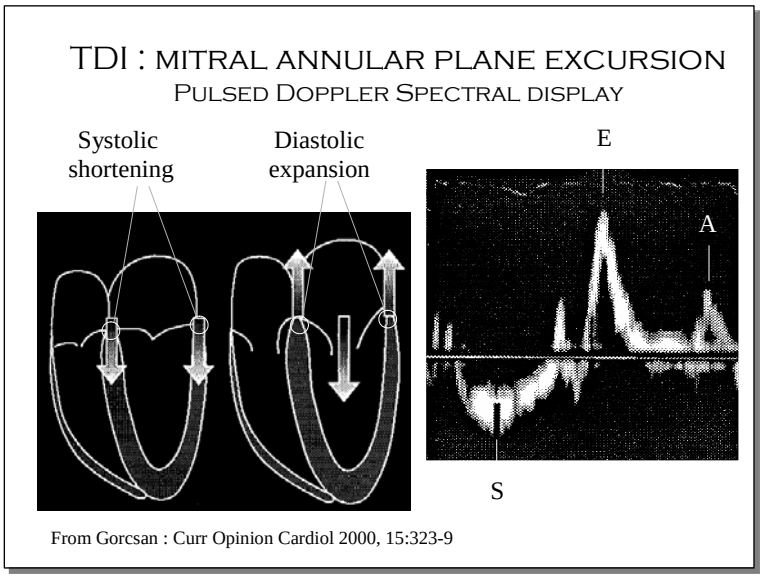


Figure 2

Newer techniques have recently become available and are claimed to have more diagnostic potential for the analysis of diastolic function because they are less affected by altered loading conditions and have a more direct relationship with the severity of diastolic disease.

<u>Tissue Doppler imaging is used to analyse the motion of the MV annulus</u> as it reflects global long axis LV function. It shows a systolic component (S' or Sm) during motion of the MV annulus towards the apex (away from the transducer in TOE imaging– towards the TTE transducer) and two diastolic components (E' or Em and A' or Am) that relate



to early and atrial diastolic filling respectively. (Figure 3). The maximum E' velocity is inversely related to the time constant of isovolumetric relaxation tau (Am J Cardiol 1997;79:928) and is less dependent on loading conditions than the corresponding transmitral E flow component (this has been challenged for healthy adults – but appears to hold true in diseased patients - see J Appl Physiol 2001;90:299). The relationship between E and E' velocities even appears to correlate with LV filling pressures but most importantly, E' max velocities show a linear decrease with the progress of diastolic disease and can be used to quantify diastolic function in patients with atrial fibrillation. An important limitation of Doppler-based measurements is the error made with improper alignment of the beam with respect to the motion studied. In fact, the position of the TOE probe is often suboptimal to allow such an alignment with respect to MV annular motion. Tissue velocity imaging can be performed with pulsed Doppler as well as with the Color Doppler modality. The former can be performed with nearly every echo machine – it requires removal of the high pass filter and a reduction of the gain settings of the pulsed wave Doppler application. Scales should be adjusted since tissue velocities are about 1/10th of flow velocities. Color Doppler tissue imaging requires specific software modules.

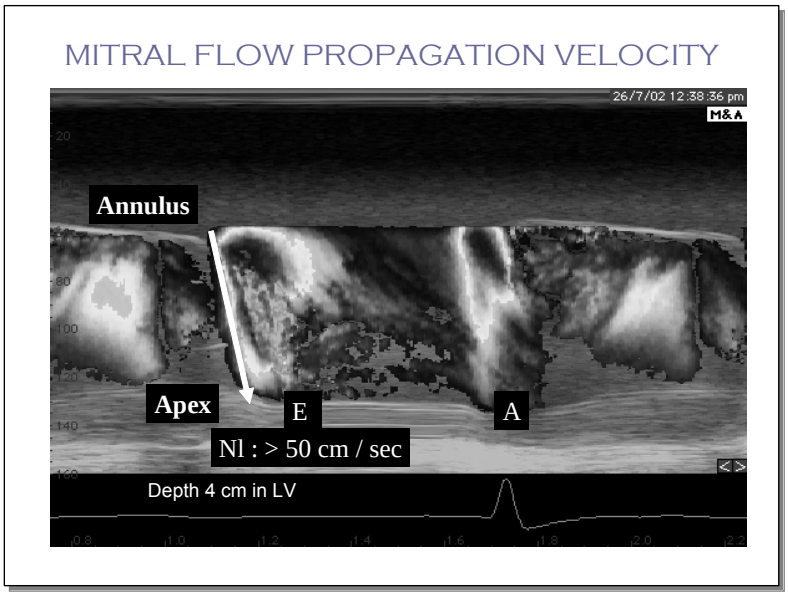


Figure 4

Finally, <u>the propagation velocity of early mitral inflow</u> (VpE) has been shown to correlate with diastolic function in a load-independent way (for healthy and diseased subjects – see JACC 2000 36: 1664-9). This variable can be assessed and quantified using the color Doppler M-mode application.(Figure 4) Several techniques have been published which differ with regard to the identification and choice of the isovelocity line – see JASE 2002; 15: 339-48. Although experimental data appear promising, a consensus is clearly needed to stratify the current published data and to objectively assess the clinical value of this method.

A combination of the above-mentioned methods is needed to accurately assess diastolic performance in the clinical setting – JASE 1999; 12:609-17. This can be performed in a few minutes by any trained echocardiographer but a fair understanding of the pathophysiology of diastole and attention to detail for using each of the techniques properly is required.

Pulmonary and Tricuspid valve

Isabelle Michaux, Yvoir, Belgium

Structure and function of the tricuspid valve.

The tricuspid valve (TV) has 3 membranous leaflets separated by indentations rather than true commissures. These are anterior, septal and posterior leaflets. Each leaflet is attached to the papillary muscles by chordae tendinae. Two papillary muscles can be described: anterior and posterior muscles. The area of the TV, 7-9 cm^2, is larger than that of the other valves and the tricuspid annulus is in a more apical position than the mitral annulus.

Echocardiographic evaluation of the tricuspid valve.

2-D views

TV can be visualised by TOE in 5 main views and by TTE in 3 main views. In TOE, the MOE 4-chamber view or the modified 4-chamber view (obtained by slightly advancing and turning the probe to the right until the TV appears in the centre of the display) show the septal leaflet on the right of the display, and either the anterior or posterior leaflet on the left, depending on the degree of probe retroflexion. The MOE RV inflow-outflow view at 60-75° shows the anterior leaflet on the right of the display and the posterior leaflet on the left. From the MOE bicaval view at 100-110°, we observe the anterior leaflet on the right of the display and the septal leaflet on the left. From the stomach, we obtain the TG SAX view of the TV at 30-45° that allows for evaluation of the 3 leaflets: septal leaflet on the right, posterior leaflet on the upper part of the display and anterior leaflet on the left. Finally, the TG RV inflow view at 120° provides a view of the posterior leaflet on the upper part of the display and anterior leaflet on the lower part of the display. In this view, we can also correctly observe the chordae tendinae and papillary muscles. In TTE, TV can be observed in the apical 4-chamber view, the parasternal short-axis view of the aortic valve and in the sub-costal view.

Doppler evaluation

Because of the cyclic changes of the RV function associated with respiration, all the right-sided Doppler signals should be recorded at end-expiration.

The modified 4-chamber view and the TG RV inflow view allow for a close alignment between the blood flow through the TV and the ultrasound beam. In these views, by placing the PW Doppler sample volume through the leaflets of the TV, the inflow of the right ventricle can be recorded. The velocities across the TV are lower than those across the mitral valve. As for the mitral inflow, the tricuspid inflow shows E and A waves. Similar to the measurements of transmitral velocities, peak E and A velocities, corresponding velocity-time integrals, deceleration time of the E wave (DT_E), and ratio of peak E/A velocities, can be determined. The tricuspid inflow changes with increasing age as does the mitral inflow: E wave decreases, A wave increases, leading to a gradual decrease in the E/A ratio and an increase of the E wave deceleration time (Table 1).[1] The E/A ratio increases in the case of elevation of the central venous pressure, in severe tricuspid regurgitation (TR) and for RV dysfunction (RV infarction or advanced restrictive myocardial disease).[2]

CW Doppler allows for estimation of the severity of TV stenosis (read below) or estimation of pulmonary systolic pressures. A colour Doppler superimposed on TV is used to detect TR, which is a common finding in mechanically ventilated patients. When a TR is present, it is possible to measure the maximal velocity of the regurgitant jet by CW Doppler. By application of the simplified Bernoulli's equation ($\Delta P = 4V^2$), the peak pressure gradient between right ventricle and right atrium can be calculated. RV systolic pressure then equals this gradient plus central venous pressure. In the absence of pulmonary valve stenosis, the pulmonary systolic pressure is equivalent to the RV systolic pressure. The accuracy of this pressure's estimation depends on the recording of a complete envelope of the regurgitant velocity by CW Doppler.

Tricuspid inflow

TTE[1]	< 50 years	> 50 years
E (cm/s)	51 ± 7	41 ± 8
A (cm/s)	27 ± 8	33 ± 8
E/A	2 ± 0.5	1.3 ± 0.4
TOE[3]	Male	Female
E	34 ± 10	35 ± 9
A	23 ± 7	22 ± 5
E/A	1.5 ± 0.6	1.7 ± 0.5

Table 1. Normal values for tricuspid inflow measured by PW-Doppler in TTE and TOE.

Acquired disease of the tricuspid valve.

Tricuspid regurgitation: TR is a turbulent systolic jet with a high velocity (2-3m/s) visualised by colour Doppler through the TV. A lower jet velocity (< 2 m/s) is a sign of severe TR or RV dysfunction, because of the near equalisation of right ventricle and right atrial pressures. The most common causes of significant TR are: 1) annular dilatation due to RV dilatation in case of volume or pressure overload, or RV dysfunction; 2) valvular destruction by rheumatic or carcinoid disease, or endocarditis; 3) traumatic rupture of the papillary muscles by chest trauma or endovascular leads. Assessment of the gravity of TR relies on 2-D echocardiography, colour and PW-Doppler (Table 2). Visualisation of the vena contracta is easy, a jet width > 0.7 cm identifies severe TR with a sensitivity of 89% and a specificity of 93%.[4] Visualisation of the flow convergence zone (PISA) is more challenging for TR than for mitral

	minimal	light	moderate	severe
Jet length (cm)	< 1.5	1.5-3.0	3.0-4.5	>4.5
Jet area (cm^2)[$]	< 2.0	2.0-4.0	4.0-10.0	>10.0
Vena contracta (cm)[4]	Not defined	Not defined	< 0.7	> 0.7
Pisa radius (cm)[5]		≤ 0.5	0.6 – 0.9	> 0.9
Hepatic vein flow	Systolic dominance	Systolic dominance	Systolic blunting	Systolic reversal

[$] *Not valid in eccentric jet*

regurgitation and was validated for TR only in small studies.[5] On the other hand, both the PISA and vena contracta methods are more accurate for determination of the severity of central regurgitation compared to eccentric regurgitation. In moderate and severe TR, right ventricle and right atrium are dilated and the systolic wave of the hepatic vein flow is blunted or reversed.

Tricuspid stenosis: The common cause of tricuspid stenosis is rheumatic disease, that also involves the mitral valve. Characteristic findings on 2-D examination are doming opening of the leaflets during diastole, thickening of the leaflets, restricted leaflet motion and commissural fusion. CW-Doppler evaluation of tricuspid inflow shows an increased peak velocity of the E wave (> 1.5 m/s). Few studies validated the calculation of the TV area based on the pressure half-time (PHT) method of Hatle et al[6]: TV area = 220/ PHT (or = 190/PHT)[7] but they showed errors of a magnitude that limits its clinical application.[8] Practically, a mean pressure gradient > 7 mmHg and a PHT > 190 msec are signs of severe tricuspid stenosis.

Carcinoid heart disease: Fibrosis of the right-side valves occurs in patients with hepatic metastasis of their carcinoid tumors. 2-D examination of the TV shows thickened, retracted leaflets that create a tricuspid stenosis without the typical doming aspect of the TV stenosis. The leaflets are often immobile in a open position creating a severe TR, that can lead to cardiac failure and death.

Tricuspid endocarditis: Tricuspid endocarditis is less frequent than mitral or aortic endocarditis. Tricuspid endocarditis is observed on the atrial side of the valve in intravenous drug abusers or in association with venous foreign bodies (venous catheter or wires). Inflammatory process (systemic lupus erythematosus) can also destroy the TV: Libman-Sacks vegetations are sterile masses located on the ventricular surface of the TV in association with thickened leaflets. TOE is preferable to TTE in the evaluation of infective endocarditis because of the low negative predictive value of the latter.[9] TOE allows close evaluation not only of the leaflets but also of the chordae tendinae and the papillary muscles.[9]

Assessment of the pulmonary valve

Structure and function of the pulmonary valve.

The pulmonary valve (PV) has 3 leaflets (or cusps): anterior, left and right. The left and right cusps are situated in posterior. Similar to the structure of the aortic valve and because of their common embryologic origin, the valvular apparatus of the PV consists of 3 cusps, sinuses of Valsalva and a sino-tubular junction. The annulus of the PV is more distensible that that of the aortic valve; the surface of the PV is approximately $2cm^2/m^2$.

Echocardiographic evaluation of the pulmonary valve.

2-D views

PV can be visualised by TOE in 3 main views and by TTE in 2 main views. The MOE RV inflow-outflow view at 60-75° shows the right and anterior cusps of the PV and the main trunk of the pulmonary artery (PA) to the right of the aortic valve. The UOE SAX view of the aortic arch (at 90°) shows the PV and the main trunk of the PA on the left side of the display. This view offers a close alignment between the blood flow through the PV and the ultrasound beam; however in our clinical practice, this UOE SAX view of the aortic arch is difficult to obtain. The deep TG view of the right ventricle (anteflexing the probe at 0°) shows the RV outflow tract, the PV and the beginning of the PA. In TTE, we can observe the PV in the parasternal short-axis view of the aortic valve and in the sub-costal view.

Doppler evaluation

The PA flow can be recorded with PW Doppler in the UOE or TG views placing the sample volume in the RV outflow tract or just above the PV. A normal flow pattern exhibits a gradual acceleration and deceleration, with a peak velocity occurring close to the mid-ejection (symmetric shape). By measuring the velocity-time integral of the PA flow signal and the diameter of the PA, it is possible to calculate RV stroke volume and shunt fractions. In the same views, C-W Doppler can be placed through the PV to assess pulmonary stenosis or end-diastolic gradient of pressure between right ventricle and PA in the presence of pulmonary regurgitation (PR). Normal peak flow through the PV ranges between 0.5 and 1.0 m/s. A peak velocity > 3.0m/s is a sign of pulmonary stenosis (read below).

Colour Doppler superimposed on the PV is helpful to detect pulmonary stenosis or PR. In the presence of PR, the diastolic pulmonary pressure can be calculated as the end-diastolic pressure gradient ($\Delta P = 4V^2$) across the PV plus central venous pressure.

Acquired disease of the pulmonary valve.

Pulmonary regurgitation: In adult, acquired PR is most often observed in patients with pulmonary hypertension, due to the dilatation of the PA and the right ventricle. Severe PR is usually observed in patients with anatomic abnormalities of the PV or after valvotomy. Because of the low prevalence of severe PR, few validation studies are available. 2-D visualisation of the entire PV could be difficult even in TOE. Colour Doppler mapping of the PV is the most widely used method. Trivial PR are very small jets (jet length < 1cm) with a central origin (leaflets coaptation) and a slow deceleration rate of the end-diastolic velocity of the PR measured by CW Doppler. Severe PR are very large regurgitant jets with a rapid deceleration rate of the end-diastolic velocity of the PR. Standards for the pulmonary vena contracta have not been established.

Pulmonary stenosis: The common cause of pulmonary stenosis is congenital disease or homograft dysfunction. 2-D examination shows a doming opening of thickened leaflets with restricted motion of these leaflets. Indirect signs of pulmonary stenosis are post-stenotic dilatation of the main trunk of the PA, RV hypertrophy and signs of RV pressure overload. Severity of the stenosis is evaluated by the transvalvular peak pressure gradient measured by CW Doppler: a peak gradient < 25-30 mmHg is a sign of mild stenosis, peak gradient between 30 and 65 mmHg reflects moderate stenosis and peak gradient > 65 mmHg is considered as a severe stenosis.[10]

Pulmonary endocarditis: Isolated pulmonary endocarditis is rare and usually occurs in patients with multivalvular endocarditis and predisposing factors: abnormal PV, PV homograft, venous foreign bodies (venous catheter or wires), immunosuppression and intravenous drug abusers. Complications of tricuspid or pulmonary endocarditis are septic pulmonary emboli or infarcts and RV failure.

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Right ventricular function & the pulmonary circulation

Isabelle Michaux, Yvoir, Belgium

Information about the structure and function of the right heart, including right ventricle, right atrium (RA), pulmonary artery (PA) and the great veins (superior [SVC] and inferior caval [IVC] veins) can be vital in the perioperative period. During this time, mechanical ventilation with an increase in intrathoracic pressure and various adverse factors (hypoxaemia, acidosis, embolism, and volume overload) may produce important alterations of right ventricular (RV) preload and afterload and affect RV function. Perioperative RV failure has an important effect on outcome and a rapid diagnosis followed by specific therapy is a prerequisite for favourable outcome. The search for the cause of haemodynamic instability and refractory hypotension represents a category I indication for perioperative TOE (Task Force 1996).[1] TOE also provides invaluable information on the presence of intracardiac shunts, thrombi, vegetations, foreign bodies or valvular dysfunction, and, allows for a non-invasive estimation of systolic and diastolic pulmonary pressures as well as RV stroke volume by Doppler techniques.

Anatomy of the right heart

The systemic venous return enters the RA via SVC and IVC and the coronary venous blood via the coronary sinus. The hepatic veins insert into the IVC 2-3 cm distally from the insertion of the latter into the RA. The right atrial appendage is located medially on the top of the RA adjacent to the ascending aorta. Anterior to the insertion of the IVC, a rudimentary but variable valve (Eustachian valve) can be seen. Occasionally, this valve is large and perforated, giving rise to a lace-like structure known as network of Chiari. The right ventricle can be depicted as a tetrahedron where tricuspid ostium, interventricular septum, and anterior and inferior parts of the free wall represent the four walls. A strong muscle band divides the RV cavity into two parts: the inflow tract and the outflow tract (RVOT). This U-shaped band is called crista supraventriculars and extends from the upper interventricular septum along the anterior tricuspid annulus into the RV free wall. In contrast to the trabeculated inner wall of the inflow tract, the surface of the RVOT is smooth. The RVOT is separated from the trunk of the PA by the pulmonary valve (PV). The bifurcation of the PA is located to the left of the ascending aorta with the right PA traversing toward the right hilus behind the aorta.

Physiology of the right heart

Most of the venous return collected in the RA during RV systole enters the right ventricle in early diastole immediately after opening of the TV. After the onset of RV contraction, the RV pressure quickly exceeds the diastolic pulmonary artery pressure, and, consequently, the time between the closure of the tricuspid valve (TV) and the opening of the PV (isovolumic contraction time) is very short. The right ventricle operates as a low pressure, thin-walled volume pump, moving the blood across the low resistance pulmonary bed into the left heart. Consequently, the right ventricle is very sensitive to increases in its afterload, caused by increases in pulmonary vascular resistance and pressure. The right ventricle responds to the increase in afterload by mobilising its contractile and preload (dilation) reserve and fails when the reserve becomes exhausted. High right-sided filling pressures and RV dilatation impair the filling of the left ventricle (LV) by the mechanism of ventricular interdependence (competing for space within the pericardium and by leftward shift of the ventricular septum). The RV coronary blood flow is physiologically continuous but becomes limited to diastole when the systolic RV pressure increases. This is associated with a reduction of the coronary flow reserve in the presence of increased myocardial O_2 demand (because of high RV wall stress) and renders the right ventricle vulnerable to myocardial ischaemia. The thin and compliant wall of the normal right ventricle allows a large increase in end-diastolic volume (preload) without a corresponding increase in RV end-diastolic and right atrial mean pressures. During acute increase in RV volume it is not only the RV wall but also the pericardium that increasingly opposes further filling and causes a steep increase in right filling pressures.

The contraction of the right ventricle follows a specific, "peristaltic" pattern with the outflow tract contracting 25-50 ms after the inflow, giving rise to an intraventricular systolic pressure gradient between the proximal and distal parts of the right ventricle.

Two-dimensional examination of the right heart

Right ventricle

The right ventricle can be visualised by TOE in four main views and by TTE in 3 main views. They include the modified 4-chamber view at 0°, obtained from the MOE 4-chamber view by slightly advancing and turning the probe to the right until the TV appears in the centre of the display. This view shows the anterior part of the free wall, with its apical segment on the right, and the basal segment on the left of the display. The MOE RV inflow-outflow view at 60-75º shows the inferior and anterior parts of the free wall on the left of the display (basal) and the RVOT on the right. From the stomach we obtain the TG SAX view of the right ventricle (TG RV SAX) that allows for evaluation of the basal segments of the anterior (lower part of the display) and inferior (upper part of the display) free wall. Finally, the TG RV inflow view at 120° provides a view of the inferior free wall (basal and apical segments at the upper part of the display), the anterior free wall (at the lower part of the display). In TTE, the right ventricle can be observed in the apical 4-chamber view, the parasternal long-axis view and short-axis view of the aortic valve.

Right atrium and Vena Cava

The RA and the atrial septum can be studied in the 4-chamber view as well as in the MOE bicaval view (plane at 110°). The utility of these two views is in searching for patent foramen ovale (using colour Doppler and echo contrast) and thrombi. On the MOE bicaval view, the SVC is displayed on the right and the IVC on the left. The SVC can also be seen in the UOE SAX view of the ascending aorta (at 0°).

Hepatic veins

From the modified 4-chamber view, by rotating the probe to the right, we visualise the junction between the IVC and the three hepatic veins in the liver: the left vein on the right, the right vein on the left, and the middle vein between them. The use of the colour Doppler, with a reduced Nyquist limit, can help to localise the hepatic veins.

Right ventricular outflow tract and the pulmonary artery

The RVOT can be observed on the MOE RV inflow-outflow view as well as on the deep TG view of the right ventricle. The PA is interrogated in the UOE views with the plane at 0° or 90°. The UOE view at 0° shows the main trunk of the PA as well as its bifurcation and the right branch. The UOE SAX view of the aortic arch (plane at 90°) shows the main trunk of the PA.

Evaluation of right ventricular function

Because of the cyclic changes of the RV size and function associated with respiration, all the right-sided Doppler signals should be recorded at end-expiration. The normal values of RV size and function were mostly derived by TTE in awake and spontaneously breathing subjects. A normal RV function implies transfer of venous return across pulmonary circulation into the left heart with physiological right heart volumes and pressures. RV dysfunction means that the right ventricle is still able to fulfil its physiological pumping function by activating its reserve or compensatory mechanisms. When the right ventricle is unable to function properly anymore despite the fully activated compensatory mechanism, RV failure develops, manifesting as systemic venous congestion, underfilling of the LV, low cardiac output syndrome or cardiogenic shock.

a) Systolic function

The RV fractional shortening expresses the change of the RV diameter (free wall to septum) between diastole (EDD) and systole (ESD) in percent (RV FS% = [RV EDD – RV ESD/RV EDD] x 100). The diameters are measured in the MOE 4-chamber view just

below the TV and perpendicular to the major axis of the right ventricle. The normal value of RV FS% is >30%.

The area of the right ventricle can be measured by planimetry in the modified 4-chamber view or TG RV SAX view. The RV/LV end-diastolic area (EDA) ratio, which is measured on the MOE 4-chamber view, is more important for the diagnosis of RV dilation than the absolute RV dimensions.[2] In acute pulmonary embolism, a RV/LV EDA ratio between 0.6 and 1 denotes a mild RV dilatation while a ratio > 1 is associated with a severe dilation.[3]

The RV fractional area change (FAC) is calculated as EDA-ESA/EDA (where ESA = end-systolic area). A RV FAC lower than 35% before coronary artery bypass graft surgery is associated with an increased postoperative mortality.[4] The wide range of normal values for the RV FAC (40-74%)[5] and its poor relationship with the severity of haemodynamic instability limit its use in our daily practice.

The tricuspid annular plane systolic excursion (TAPSE) is the maximal systolic excursion of the tricuspid lateral annulus measured with a M-mode placed on the lateral annulus. To reduce the angle between the ultrasound beam and the longitudinal displacement of the annulus, we recommend that it be recorded in the TG RV inflow view. In TTE, there is a good correlation between the TAPSE and the RV ejection fraction (RV EF) measured by radionuclide ventriculography or right heart catheterisation; a TAPSE >14 mm separates normal from reduced RV function.[6]

Tissue Doppler imaging (TDI) of the tricuspid lateral annulus measures the systolic and diastolic velocities of the lateral annulus of the TV. In our experience, the TG RV inflow view allows for recording of the tricuspid TDI with the narrowest angle between the probe and the longitudinal movement of the annulus. The typical RV velocity pattern is characterised by two systolic velocity and two diastolic velocity peaks. The peak systolic velocity of the tricuspid annulus (Sa) appears to be a useful measure of global RV function. Based on TTE data, a systolic velocity > 15 cm/s separates normal from reduced RV function[7] and a systolic velocity < 11.5 cm/s predicts a RV EF < 45% with a sensitivity of 90% and a specificity of 85% in patients with dilated cardiomyopathy.[8]

The PA flow: The pulmonary artery VTI is used for calculation of stroke volume and cardiac output. By rule of thumb, pulmonary artery VTI > 15 cm suggests a normal stroke volume.[9]

Measurement of pulmonary artery pressure

Echocardiography offers non-invasive access to the pressures in the right heart and eliminates the use of a pulmonary artery catheter. When a tricuspid regurgitation is present, which is quite common in mechanically ventilated patients and in patients with abnormal RV function, it is possible to measure the maximal velocity of the regurgitant jet by CW Doppler. By application of the simplified Bernoulli's equation ($\Delta P = 4V^2$), the peak pressure gradient between right ventricle and RA can be calculated. RV systolic pressure then equals this gradient plus central venous pressure. In the absence of PV stenosis, the pulmonary systolic pressure is equivalent to the RV systolic pressure. The accuracy of this pressure's estimation depends on the recording of a complete envelope of the regurgitant velocity by CW Doppler. The Doppler signal can be enhanced by intravenous injection of an echocardiographic contrast agent. Similarly, in the presence of pulmonary regurgitation, visualised by colour Doppler on the deep TG view of the right ventricle, the diastolic pulmonary pressure can be calculated as the end-diastolic pressure gradient across the PV plus central venous pressure.

For pulmonary hypertension, the flow pattern resembles that of the aortic ejection, with a short acceleration time and early peak (asymmetric shape). An abrupt decrease in velocity in midsystole (notch) can be observed in some patients with severe pulmonary hypertension. Total RV ejection time (ET) and acceleration time (RV AcT) (time to peak) are shortened while RV isovolumic relaxation time (time between the closure of PV and opening of the TV) and pre-ejection time (time between the onset of QRS and the onset of the pulmonary ejection flow) are prolonged. Finally, the index of RV AcT divided by

RV ET also correlates with the pulmonary artery pressure. Patients with pulmonary hypertension often present an abnormal tricuspid flow pattern with prolonged deceleration time of the E velocity and low E/A ratio. RA and right ventricle are dilated as a result of the increased RV afterload. The presence of RV hypertrophy allows for differentiating between acute and chronic pulmonary hypertension. The diastolic RV wall thickness is measured by M-mode echocardiography, using the MOE inflow-outflow view or the TG RV inflow view. RV hypertrophy can be considered if RV wall thickness is > 6 mm.[10] In acute pulmonary hypertension, the RV thickness is typically < 6 mm, with clear visualisation of the RV trabeculations. In chronic pulmonary hypertension, the RV hypertrophy is more pronounced with a RV wall thickness of about 10 mm.

Preload and afterload of the right ventricle

The right ventricle typically dilates in response to an increase in preload or afterload or in both. Because the two ventricles share myocardial fibres of the common septum and are enclosed in the same stiff pericardium, any change in the RV filling can affect the diastolic function of the LV (diastolic interdependence). TOE provides valuable information on the shape and motility of the interventricular septum that is a mediator in this ventricular interaction.

Volume overload: With increasing intensity of RV volume overload, the RV end-diastolic pressure equals and eventually exceeds the left ventricular end-diastolic pressure. The septum follows the abnormal diastolic transseptal pressure gradient and becomes flat and even bows toward the LV in end-diastole. In diastole, in the TG SAX view, the right ventricle assumes a circular shape, whereas the LV becomes crescentic. These changes are more pronounced if the pericardium is closed. During systole, when the pressure generated by the LV exceeds the RV pressure, the septum moves swiftly towards the right ventricle and the LV resumes its normal circular shape. This abnormal early systolic outward movement is called paradoxical septum motion.

Pressure overload: For pressure overload, the high RV end-diastolic pressure and volume affect LV filling and shape in the same way as during diastole. However, in severe pulmonary hypertension, the RV may exceed the LV pressure even during systole. In this case, the abnormal shift of the septum towards the LV also persists throughout systole.

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Illustrations - *Two-dimensional TOE examination of the right cavities.*

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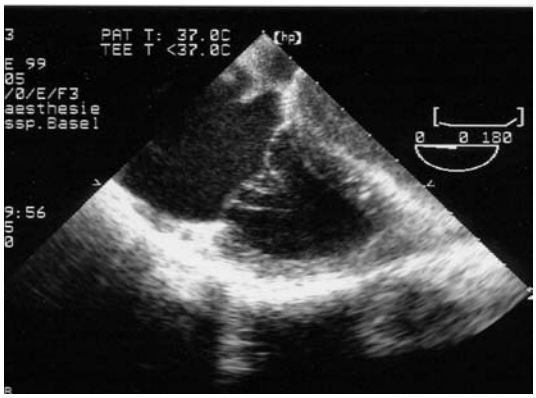
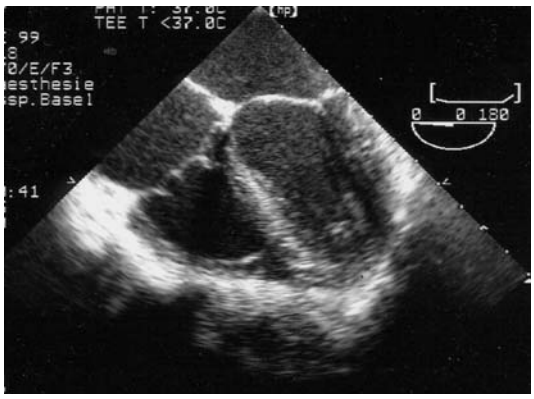
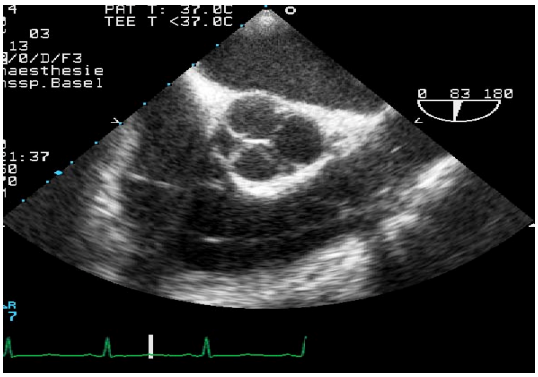


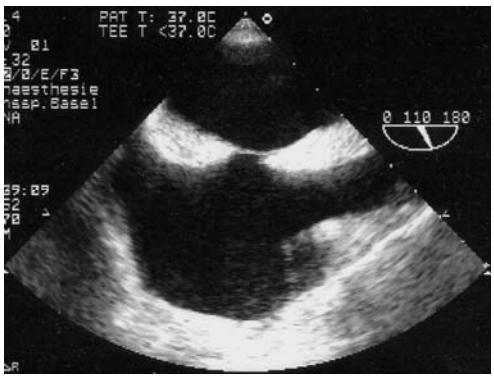
Modified MOE 4-chamber view



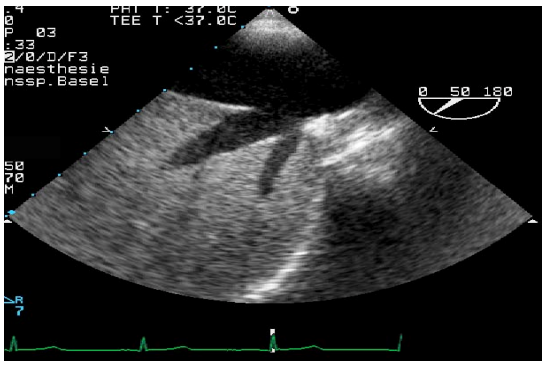
MOE 4-chamber view



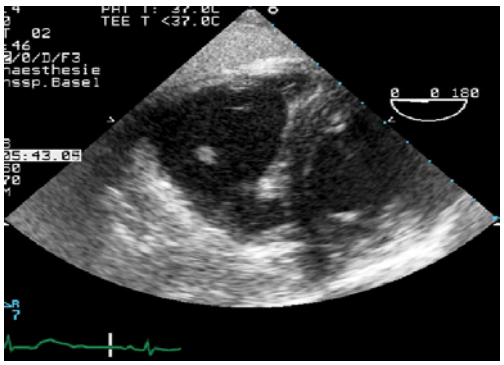
MOE RV inflow-outflow view



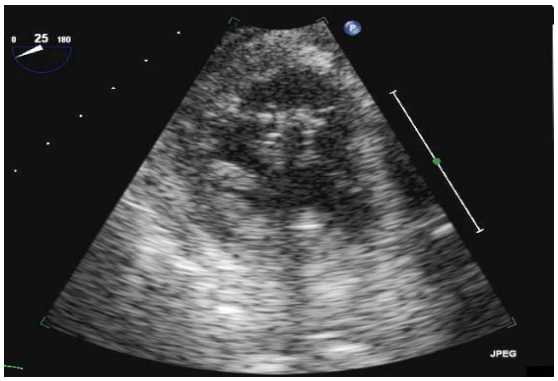
MOE Bicaval view



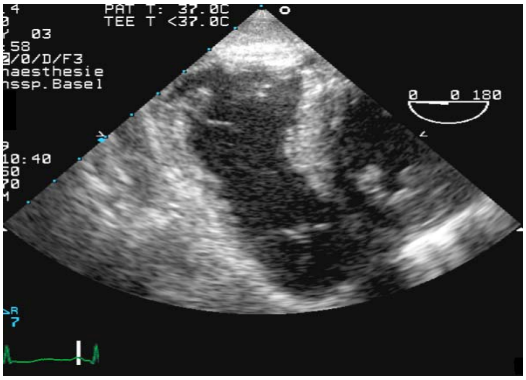
Inferior vena cava and hepatic veins



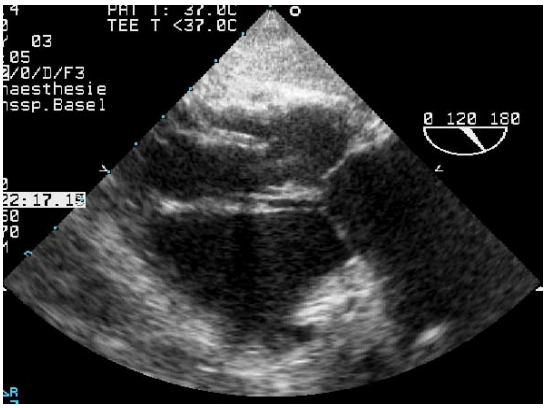
TG RV SAX view



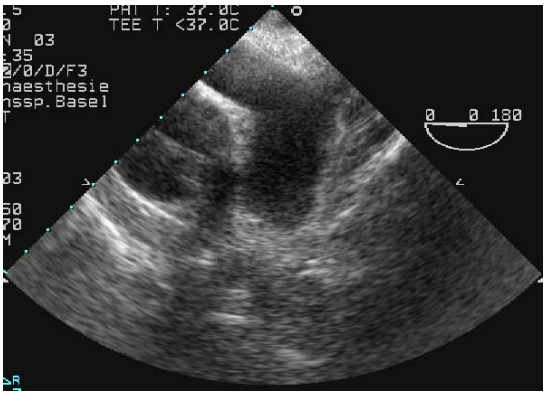
TG TV SAX view



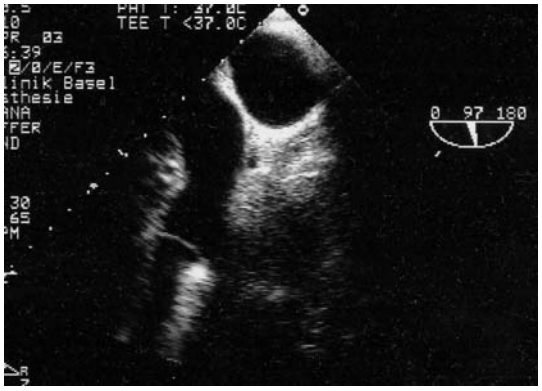
Deep TG RV outflow view



TG RV inflow view



MOE ascending aortic SAX view



UOE aortic arch SAX view

Abbreviations list

A	*late peak diastolic velocity of the tricuspid inflow*
Ac T	*acceleration time of the pulmonary artery flow*
CW	*continuous wave*
E	*early peak diastolic velocity of the tricuspid inflow*
EDA	*end-diastolic area*
EDD	*end-diastolic diameter*
EF	*ejection fraction*
ESA	*end-systolic area*
ESD	*end-systolic diameter*
ET	*ejection time*
FAC	*fractional area change*
FS	*fractional shortening*
IVC	*inferior vena cava*
LV	*left ventricle or left ventricular*
MOE	*mid-oesophageal*
PA	*pulmonary artery*
PR	*pulmonary regurgitation*
PV	*pulmonary valve*
PW	*pulsed wave*
RA	*right atrium*
RV	*right ventricular*
RVOT	*right ventricular outflow tract*
SAX	*short axis*
SVC	*superior vena cava*
TDI	*tissue Doppler imaging*
TG	*transgastric*
TOE	*transoesophageal echocardiography*
TTE	*transthoracic echocardiography*
TR	*tricuspid regurgitation*
TV	*tricuspid valve*
UOE	*upper-oesophageal*
VTI	*velocity time integral*

ICE & TEE for guidance of transcatheter ASD & PFO closure

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Background: *TEE has been extensively used to guide transcatheter device closure of atrial septal defects. However, continuous imaging by TEE during such interventions in supine patients requires general anesthesia and endotracheal intubation. Intracardiac echocardiography (ICE) with an ultrasound catheter introduced percutaneously into the heart is a new technique that allows transcatheter closure of atrial septal defects to be performed in local anesthesia in adults and larger pediatric patients.*

Aim: *To describe our initial experience with ICE for guidance of transcatheter ASD and PFO closure.*

Method: *The 10F AcuNav ICE catheter (Acuson; Siemens) coupled to an Acuson Cypress ultrasound machine was used. The tip of this catheter is equipped with a 64-element phased array 5.5-10 MHz transducer that scans in a single plane longitudinal to the catheter with a 90-degree sector image and a depth penetration up to 12 cm. It has 2-dimensional and color Doppler imaging modes. The tip is steerable in four directions. It was introduced in an 11-F sheath usually through the right femoral vein which was also used for the device delivery sheath. An 8-F AcuNav ICE catheter became available very recently and was used during the latest 5 ASD/PFO closures.*

Results: *We previously performed 175 transcatheter ASD closures and 39 PFO closures (sept. 1997 to sept. 2007). The majority of these interventions were done in general anesthesia with endotracheal intubation and guided by TEE. From march 2004 to september 2007 we did 31 ASD closures and 19 PFO closures guided by ICE (n=50). In the first 3 patients we used both ICE and TEE. All the following septal defect closures in adults and larger pediatric patients were guided by ICE and fluoroscopy and performed in local anesthesia. The devices used were Amplatzer ASD (n=29), Amplatzer PFO (n=17) and Helex (n=4) occluders. All defects were single and sized with and without a stationary balloon-catheter by fluoroscopy and ICE. No patients received more than one occluder device. The procedural time and fluoroscopy time (mean±SD) were 29.5±13.1 and 9.0±5.7 min for the PFO closures and 51.8±22.0 and 13.9±11.8 min for the ASD closures. All defects except for 2 were successfully closed without any complications relating to the ICE procedure or device closure. In one case a Helex occluder got entangled with the tricuspid valve during deployment (diagnosed by ICE) and had to be removed by surgery. In another case with a large ASD in an aneurismal septum an Amplatzer occluder embolized to the pulmonary artery due to unintended release of the device from the delivery cable during manipulations with the delivery sheath. No residual shunts were detected at 3 months post-procedural follow-up (TTE). ICE gave high-quality near-field imaging of the atrial septum, the position and size of the defects, rims and distance to surrounding structures. It was superior to TEE in imaging the inferior rim and the captured septal rims inside the device before deployment. Fluoroscopy was necessary for precise measurement of the balloon-stretched diameter which was often difficult with ICE.*

Conclusion: *50 transcatheter ASD/PFO closures were guided by intracardiac echocardiography (ICE). It produced high-quality near-field imaging superior to TEE and rendered general anesthesia and endotracheal intubation unnecessary. There were no complications associated with the ICE procedure itself but 1 complication related to a closure device and 1 operator-related complication. ICE has become our preferred imaging technique to guide transcatheter closure of atrial septal defects in adults and larger children.*

Aortic valve disease

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Every complete transoesophageal echocardiographic (TOE) examination should include a careful assessment of the aortic valve. TOE can define the severity and mechanisms of aortic stenosis (AS) and aortic regurgitation (AR). Although the highly experienced surgeon may feel comfortable to make decisions by direct surgical inspection alone, most appreciate the ability of TOE to define the abnormality pre-operatively, confirm their intra-operative impression and assess the postoperative results, even in routine valve replacement procedures.

Aortic valve anatomy

The aortic valve (AV) forms part of the aortic root together with the three sinuses of Valsalva, two coronary ostia and sinotubular junction. It consists of a crown-shaped aortic annulus (annulus fibrosa) and three similar semilunar cusps called the right coronary cusp (RCC), left coronary cusp (LCC) and the noncoronary cusp (NCC). The leaflets are composed of a dense collagen layer, covered by a thin avascular collagen layer, and endothelium. The RCC and right sinus of Valsalva is positioned anterior and give rise to the right coronary artery (RCA). The left coronary artery (LCA) originates from LCC and left sinus of Valsalva. The posterior of the three cusps is called the NCC and lies adjacent to the interatrial septum. The fibrous thickening seen at the central portion of the normal leaflets are called the nodules of Arantius. After years of function the leaflets may develop this thickening of the edges, as well as filamentous strands on them. These small filamentous strands connected to the aortic valve (up to 5mm in length) may appear in the left ventricular outflow tract during diastole, or on the aortic side during systole. They are referred to as Lambl's excrescences and may be misinterpreted as vegetations. This is usually an incidental finding in elderly patients who are otherwise well (Bollen).

The sinotubular junction (STJ) connects the root to the proximal ascending aorta. It is circular and thicker than the adjacent sinuses, defining the start of the ascending aorta. The STJ plays an important role in suspending the aortic valve leaflets. The upper limit of the aortic valve annulus diameter is 2.6cm and the sinotubular junction is 3.4cm. Together with the sinuses of Valsalva and ascending aorta diameters these are important measurements for surgical decisionmaking. The plane of the AV is oblique with the right posterior side more inferior to the left anterior side. Therefore the origin of the LCA is superior to the RCA.

The normal AV area is 2.5-3.5 cm^2 with a normal echo pressure gradient across the valve of 2-4 mmHg (flow velocity of 60-100 cm/sec). The opening and closing of the leaflets inside a normal aortic root is smooth and symmetrical. During systole in a compliant aorta, root dilatation precedes, and aids in the opening of the leaflets. This root dilatation pulls the closed leaflets apart and reduces the frictional forces at the commissures (Robicsek). A minimal pressure gradient of around 2 mmHg is therefore enough to open the aortic valve. At maximum displacement of the leaflets during early systole, the aortic valve opening is circular, which is followed by a triangular shape in later systole. The echocardiography appearance of the normal valve orifice may therefore be circular or triangular, depending on whether it was observed earlier or later in systole. There is a gap between the body of the leaflets and the aortic wall (sinuses of Valsalva). If the pressure gradient is increased in a compliant root from 2-8 mmHg, the valve area increases strikingly by about 25%. This effect is absent in a stiff, noncompliant root. When the cardiac output is increased under certain physiological conditions (e.g. exercise), the normal aortic valve therefore copes by increased dilatation of the root, and increased pushing and bending of the leaflets towards the aortic wall.

In a stiff, noncompliant aortic root the valve opening tends to be asymmetric and delayed, with considerable wrinkling of the leaflets (Sripathi). The systolic aortic root dilatation with the active "pull-release" opening mechanism of the leaflets is absent. The leaflets show a lot of inertia and therefore open much later after the development of a gradient between LV and the aorta. The valve opening remains circular and does not become triangular, as seen in a compliant root. There has been speculation that the leaflet wrinkling inside a noncompliant root may increase leaflet stresses and may be responsible for earlier calcification. A stiff root seems to function at maximum level of efficiency and is not able to increase the valve area during a period of increased cardiac output.

The AV is continuous with the anterior leaflet of the mitral valve (MV). It connects the aortic root to the left ventricular outflow tract (LVOT). The left atrium is immediately posterior to the AV while the pulmonary valve (PV) is anterior. Abnormalities of any of the components of the AV or the adjacent structures can affect the valve function. When evaluating the aortic valve for AS or AR, it is essential to always assess the geometry and contractility of the left ventricle (LV) in all the views.

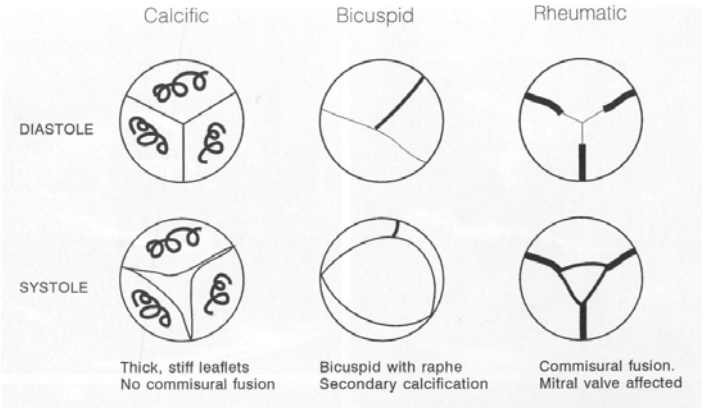
Aortic Stenosis

Aortic valve disease is very common in Western populations. About 25% of people over 65 years of age have aortic sclerosis and 3% over 75 years have severe stenosis (Lindroos). Aortic sclerosis is diagnosed when there is an ejection systolic murmur present in the aortic valve region due to calcification in the ascending aorta, with associated turbulent flow. In this condition there is minor disruption of the aortic valve and minimal obstruction to flow. There may be thickening or calcification of one or more leaflets of a tricuspid AV, but in contrast to aortic stenosis, leaflet opening is not restricted very much and the velocity through the valve is less than 2.5 m/sec. Aortic sclerosis is not innocent because it is an antecedent to clinically significant aortic valve stenosis and it acts as a marker of increased risk of cardiovascular events (Nightingale). Aortic stenosis is differentiated from sclerosis when significant restriction of cusp movement and a raised transaortic peak velocity is seen on echocardiography (Chambers). Calcific "degeneration" is the most common cause of aortic stenosis (AS) in the Western adult population (70-90 years old). It begins with annular and leaflet thickening, which gradually becomes calcified. Although it seems as if most patients with aortic stenosis starts off with aortic sclerosis, the rate of progression is not very clear (Cosmi). The RCC is affected most commonly and often fuses with the NCC. These calcified leaflets have decreased mobility and are very echogenic. AS was once viewed as a "degenerative" disease but is now seen as an active inflammatory process which resembles atherosclerosis, and many of the risk factors are the same for both processes. Inflammation, lipid infiltration, dystrophic calcification, ossification, platelet deposition and endothelial dysfunction have been observed in both diseases and hypercholesterolaemia, lipoprotein Lp(a), smoking, hypertension and diabetes have been reported to be common risk factors for both of them (Mohler). Aortic valve disease is also a marker for coronary artery disease. Although still controversial it seems as if higher doses of lipid-lowering statins may slow down the process of this disease similar to its effect on coronary artery pathology (Newby, Novaro). There is some evidence that this effect occurs without a consistent relationship to cholesterol levels, suggesting that the effects of the statin agents may be caused by their anti-inflammatory effects rather than simple cholesterol lowering (Rosenhek). Other interesting information is that angiotensin converting enzyme (ACE) is involved not only in an unfavourable LV remodelling response in AS patients, but also in the progression of valve degeneration. A potential benefit has therefore been suggested for ACE inhibitors (Caulfield).

The congenital bicuspid AV has a 1-2% incidence making it the most common cardiovascular malformation in humans (Roberts). These patients may present with stenosis as a child or may undergo accelerated calcification of the abnormal AV and present as in adulthood (50-60 years old). The two leaflets are typically arranged in

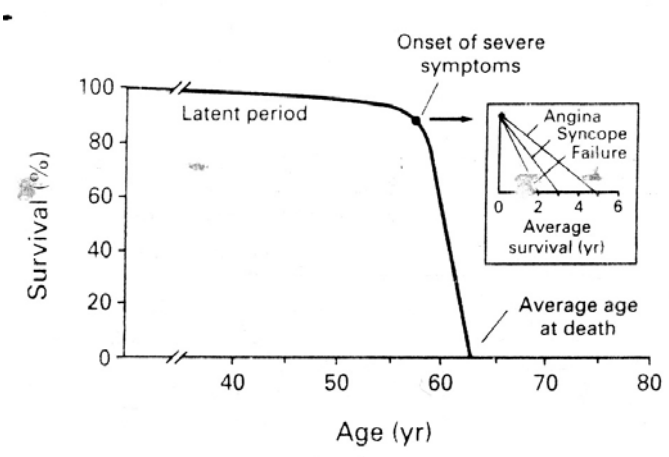
either a right-left or anterior-posterior orientation, with a variation in coronary arrangement. A pseudo-bicuspid AV (three-leaflet valve with fusion of two of the leaflets) will have similar pathophysiology to a truly bicuspid valve. Patients with the most severely malformed bicuspid valves may require intervention during childhood. The bicuspid aortic valve is also associated with other congenital cardiovascular abnormalities including coarctation of the aorta (50-80%), interruption of the aortic arch (36%), and isolated ventricular septal defect (20%) (Duran). The high heritability of the bicuspid aortic valve suggests that its determination is almost entirely genetic (Cripe). Although a nonstenotic bicuspid aortic valve is often considered a benign lesion earlier in life, it has potential complications including aortic regurgitation, infective endocarditis, aortic dilatation and dissection, resulting in considerable morbidity and mortality later in life. Unicuspid valves are rare but may also be a cause of stenosis.

Although rheumatic heart disease may affect the AV with leaflet thickening and decreased movement, it preferentially involves the mitral valve. Rheumatic degeneration of the AV therefore rarely occurs in isolation. In the rheumatic aortic valve commissural fusion occurs together with thickening and fibrosis of the leaflet edges, while the leaflet bodies are less affected in the earlier stage.



Otto Textbook of Clinical Echocardiography

It is very clear that symptoms alone are not an adequate guide for management of valvular heart disease. The lack of symptoms does not predict an uncomplicated course. For example patients with severe aortic stenosis may remain completely asymptomatic but are at risk of sudden death. Once angina, syncope, or heart failure develops, survival is greatly reduced (Ross).



Ross J, Braunwald E. Circulation 1968;38:Suppl 5:61-67

About 75% of patients with symptomatic AS will be dead three years after the onset of symptoms, unless the AV is replaced (O'Keefe). When the peak flow velocity is more than 4.0 m/sec, the likelihood of an asymptomatic AS patient being alive without a valve replacement in two years time is only 21%(+/- 18%) (Otto). In an era of modern echocardiography it is no longer appropriate to wait for a change in symptoms to guide management (Wilkins).

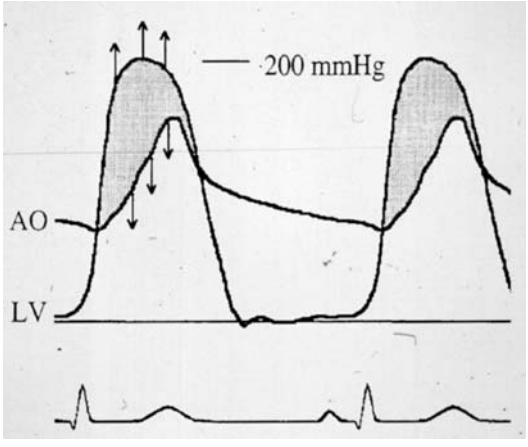
Even in the elderly, the prognosis of a patient with aortic stenosis who receives surgery is excellent. Age should therefore not be a contraindication in these patients in the absence of comorbid conditions (Carabello). The same principle applies to patients with poor LV function due to the AS. Although patients with more severe stenosis, a worse LV function and more severe symptoms experience the greatest benefit from surgical relief of the obstruction (Pereira), they also have a higher operative mortality. Common current practice to establish whether or not truly severe AS is present in patients with an impaired LV and a

low gradient, is to increase the cardiac output pharmacologically and assess ventricular reserve during dobutamine stress echocardiography. Any patient with left ventricular contractile reserve should benefit from surgery (Monin). It is important to distinguish the patient with truly severe AS from the patient with aortic "pseudostenosis", where a LV damaged by another process such as coronary artery disease or cardiomyopathy, is unable to open a mildly stenotic aortic valve (Carabello).

A TOE evaluation of a stenotic AV starts with a 2D examination in the ME SAX and LAX views of the valve. The leaflets may be thickened and calcified with a bright echogenic appearance and reduced mobility. It may be difficult to recognise the "Mercedes Benz" sign and planimetry of the open calcified AV is often very inaccurate but should be performed whenever feasible. The calcification often creates acoustic shadows obscuring certain areas of the valve. Calcification of the annulus is an important finding, which must be communicated to the surgeon because it will affect surgical options. Another important sign of AS is doming of the leaflets during systole. Post-stenotic dilatation of the aortic root and ascending aorta is common. The use of colour flow Doppler usually demonstrates turbulence across the valve but does not quantify its severity. It is useful to look for LV hypertrophy or dilatation, and LA dilatation as result of the pressure overload.

With the 2-D image in the ME LAX AV view the M-mode cursor can be aligned through the aortic valve and used to assess leaflet separation directly and as a percentage of aortic root diameter.

Continuous wave Doppler (CWD) is used to measure flow velocity across the valve and then calculate the pressure gradient with the simplified Bernoulli equation (Pressure Gradient = 4 X V^2). To make an accurate measurement it is very important that the ultrasound wave is parallel to the direction of blood flow. Although it is more difficult to align the ultrasound beam correctly with TOE than with transthoracic echocardiography (TTE), this angle can usually be obtained in the TG LAX and deep TG views. The colour Doppler sector is useful to guide the echocardiographer towards the outflow tract and AV. A meticulous search for the maximal aortic flow velocity signal is essential. The high flow velocity jet of severe AS will have a typical audible sound. The flow pattern is helpful, because in severe AS the peak will occur during mid-systole, while in more moderate AS it will be during early systole. Severe AS is defined as a flow velocity more than 4.5 m/sec, a mean pressure gradient more than 50 mmHg and a peak pressure gradient more than 80 mmHg. The mean pressure gradient is obtained by accurately tracing the outline of the flow velocity signal. Because CWD measures the highest velocity along the line of interrogation, subvalvular obstruction needs to be excluded by placing the PWD cursor proximal to the AV in the LVOT. The LVOT to AV velocity time integral ratio (VTI_{LVOT}/VTI_{AV}) is also a good index of AV stenosis severity. This ratio is independent of any change in stroke volume because both LVOT and AV velocities change proportionally (Oh). If the VTI_{LVOT}/VTI_{AV} ratio is less than 0.25 the stenosis is severe. In any rhythm other than sinus the velocities will vary with each cardiac cycle depending on the preceding RR interval. In this case the average velocities from five to ten cycles should be used to obtain the velocity ratio.



There is excellent anatomical and physiological correlation between echocardiography and cardiac catheterisation findings (Roger). The "peak-to-peak", "peak" and "mean" gradients can be reported from catheterisation data. It is important to distinguish between the maximum instantaneous peak gradient obtained with echocardiography and the peak-to-peak

gradient obtained in the catheter laboratory. The peak-to-peak gradient is obtained by measuring the difference between peak LV pressure and peak aortic pressure with a pressure transducer at different times in the cardiac cycle. The maximum instantaneous echo pressure gradient is higher than the peak-to-peak gradient. It has been shown that the best correlation is between the mean Doppler gradient and the mean cardiac catheter gradient measured simultaneously (Currie). The technical quality of the echocardiography examination however has an important influence on the reliability of this information and subsequent surgical decisionmaking.

The maximum Doppler velocity through the aortic valve will be underestimated if the ultrasound beam is not parallel to aortic blood flow (Hatle). On the other hand the pressure recovery phenomenon may lead to overestimation of Doppler pressure gradient measurements (Baumgartner). In an aortic stenosis jet the pressure will be lowest where the velocity is highest. This is at the vena contracta, which corresponds to the minimal cross-sectional valve area. Distal to the stenosis as velocity decreases, pressure will increase. This total amount of pressure recovery is related to the viscous and turbulence energy losses across the stenotic valve. The Doppler gradients that are measured at the vena contracta will be significantly higher than the catheter measurements taken downstream in the ascending aorta after the pressure has completely recovered (Popescu). The pressure gradient will therefore depend on where the pressure is sampled. In mild to moderate aortic stenosis the three cusps of the valve form a funnel rather than a diaphragm, as would be found in severe stenosis (Chambers). This leads to greater pressure recovery. The pressure recovery phenomenon is more significant in prosthetic aortic valves than in native valves.

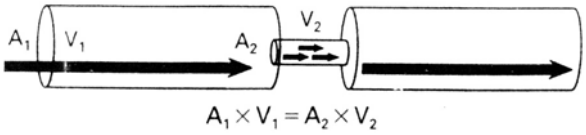
Due to pressure overload, the LV responds to AS with myocardial fibrosis, concentric hypertrophy and impaired diastolic relaxation. This will be followed by systolic LV dilatation, and failure later in the disease. The transvalvular pressure gradient (PG) is only a reliable indicator of severity in the presence of good ventricular function. The PG will decrease if the LV function deteriorates in end stage disease. The effective valve area is far less flow dependent than the pressure gradient, and should therefore be the index of choice for quantifying AS in the presence of abnormal flow rate or elevated blood pressure (Bonow). AS is severe when the AVA is less than 0.8cm^2. Using planimetry is relatively reliable in the valve with healthy leaflet bodies, but more inaccurate in the calcified valve. A perfect short-axis view should be obtained and the three-dimensional anatomy of the crown-shaped AV should be kept in mind when using this technique. The AVA remains fairly constant disregarding the flow across the valve.

The continuity equation is most commonly used to calculate AVA. It is based on the conservation of mass and states that blood flow (area X velocity time integral) through sequential areas of a continuous, intact system must be equal. Therefore blood flow through the left ventricular outflow tract ($Area_{LVOT}$ X VTI_{LVOT}) must equal blood flow across the AV ($Area_{AV}$ X VTI_{AV}).

Therefore AVA = $\dfrac{\mathbf{Area_{LVOT}\ X\ VTI_{LVOT}}}{\mathbf{VTI_{AV}}}$

Pulse wave Doppler is used to measure flow velocity across the LVOT, while continuous wave Doppler will be necessary to measure the much higher flow velocities across the stenotic AV. The $Area_{LVOT}$ is obtained by measuring its diameter in the ME LAX view of the AV during systole, and applying the formula for the area of a circle:

$$Area_{CIRCLE} = \pi r^2$$
$$= \pi \text{ X } (\text{diameter}/2)^2$$
$$= (\pi/4) \text{ X diameter}^2$$
$$= 0.785 \text{ X diameter}^2$$



Carabello, BA, Crawford, FA. New Engl J of Med 1997; 337:32-40

It may be difficult to measure the LVOT diameter accurately because of heavy calcification. This measurement can then easily be underestimated. Another pitfall of the continuity equation is when the ultrasound beam is not parallel to blood flow and the Doppler measurements will therefore be inaccurate. One of the most common causes of misinterpretation of echocardiography findings in this condition is when a mitral regurgitation jet is present. When attempting examination of the aortic valve in the transgastric views, the continuous wave Doppler beam may accidentally cross the mitral valve flows. The orientation and high velocity of a possible mitral regurgitant jet will be similar to that of a possible aortic stenosis jet. A wrong diagnosis will have serious consequences. If a patient is not in sinus rhythm the AV and LVOT flow velocities will vary with each cardiac cycle. Multiple measurements should then be made to get an average. Small errors in measurement will result in large errors in calculated values. The double-envelope continuity equation technique has been described to use in prosthetic valves (Maslow). Using this technique, the high velocity VTI_{AV} is obtained from the outside edge of the continuous wave Doppler signal. Instead of using pulsed wave Doppler to obtain the LVOT velocity, the denser low velocity signal in the same envelope is used to represent the VTI_{LVOT}. This technique seems to be useful also in the stenotic AV. Many patients with aortic stenosis also have aortic regurgitation. This leads to increased transaortic blood flow during systole with a higher gradient for a given aortic valve orifice. The AR however does not affect the continuity equation calculations because the increase in systolic flow is measured in both the LVOT and across the AV. On the other hand will coexisting mitral stenosis cause a low aortic valve gradient because the fixed cardiac output will lead to a decrease in transvalvular blood flow (Troianos).

Systemic hypertension can modify the physical examination findings in the aortic stenosis patient (Chambers). It may also affect the echo findings by inducing a significant reduction in transvalvar flow rate, an increase in effective valve area, and consequently a reduction in pressure gradient (Bermejo). This can be misleading when quantifying stenosis severity.

The differential diagnosis of AS will include LVOT obstruction (sub-aortic membrane or muscular sub-aortic stenosis), hypertrophic obstructive cardiomyopathy (HOCM), and any form of supravalvular stenosis in the root or aorta.

Aortic Regurgitation

Aortic regurgitation (AR) results from a primary valve lesion, an abnormal aortic root and/or ascending aorta, or a combination of both. Primary valve lesions include calcific or rheumatic AV disease, or infective endocarditis. Stenotic AV leaflets often do not completely coapt during diastole, leading to regurgitation. The most common cause of aortic regurgitation in developing countries is rheumatic disease, with clinical presentation in second or third decade of life (Enriquez-Sarano), while in Western countries it is most frequently due to degenerative (aortic root dilatation) or congenital (bicuspid valve) causes with patients presenting in the fourth to sixth decades. The prevalence of aortic regurgitation increases with age.

Infective endocarditis of the AV typically presents with a mobile vegetation connected to a damaged cusp. This is best visible on the ventricular side of the valve when prolapsing into the LVOT during diastole. The diagnosis of aortic valve prolapse is made when any part of a leaflet appears in the LVOT below the level of the aortic annulus. As the endocarditis progresses the cusps are damaged resulting in an

increasing severity of AR. A mycotic aneurysm of the aortic root or a perivalvular abscess may occur and is relatively easy to identify with TOE. In patients with pure aortic regurgitation secondary to rheumatic disease, the essential lesion is retraction and thickening of the edges of the cusps with preservation of the hinge mechanism (Yacoub). The hemodynamic lesion may result in progressive dilatation of the aortic annulus with worsening of the regurgitation over time.

A dilated or abnormal aorta and aortic root may be because of hypertension, Marfan syndrome, trauma or aortic dissection. Aneurysmal dilatation of the ascending aorta can cause AR without annular dilatation, through a tethering effect on the cusps. Movsowitz et al. describes five potential mechanisms of AR in a patient with acute type A aortic dissection (Movsowitz).

a) *Incomplete closure of intrinsically normal leaflets, due to leaflet tethering by a dilated sinotubular junction.*
b) *A bicuspid aortic valve with associated leaflet prolapse unrelated to the dissection process.*
c) *Degenerative leaflet thickening resulting in abnormal coaptation.*
d) *Leaflet prolapse due to disruption of leaflet attachments by a dissection flap that extends below the sinotubular junction and into the aortic root.*
e) *Prolapse of the dissection flap through intrinsically normal leaflets that disrupts leaflet coaptation.*

The first three of these mechanisms (a,b,c) can also occur in patients without aortic dissection. Some patients can have more than one mechanism of AR. In aortic dissection the intimal flap prolapsing through the valve (5th mechanism) may keep the leaflets in the open position causing severe AR. However sometimes the flap acts as a valve during diastole with remarkably little AR although the leaflets are open. The risk of aortic dissection or rupture is increased in patients with a dilated aorta with diameter more than 5.5 cm (Davies). The diameter of the ascending aorta should therefore be routinely assessed and any dilatation should be carefully followed up.

Although the congenitally bicuspid valve usually undergoes premature heavy calcification, a proportion of these patients present with severe regurgitation and pliable cusps. Some of these can be treated by repairing the valve. The bicuspid aortic valve also predisposes to infective endocarditis, which can lead to its destruction with subsequent AR. Another form of congenital AR is where the AV is in close relationship to a perimembranous subarterial VSD. In this situation there is usually a degree of prolapse of more commonly the right coronary cusp due to inadequate fibrous support and in some cases the Venturi effect of the VSD. On current evidence it is very important not only to repair the VSD, but also to resuspend the prolapsing cusp, to prevent rapid progression of aortic insufficiency (Cheung). Quadricuspid aortic valves are rare, but significant aortic insufficiency is common with this lesion due to uneven distribution of mechanical stress leading to incomplete cusp coaptation (Tutarel).

On clinical examination the patient with aortic regurgitation will have a characteristic decrescendo diastolic murmur (Austin Flint) and a third heart sound. This is due to an orifice in the valve, which allows regurgitant flow through out diastole with subsequent rapid filling of the LV. Because of the increased left ventricular end diastolic volume, there is also an increase in the systolic flow across the valve. This leads to a wide pulse pressure with a collapsing pulse and often an ejection systolic murmur. In aortic regurgitation there is both left ventricular pressure and volume overload (Enriquez-Sarano).

The mainstay of aortic regurgitation assessment is based on the integration of the information obtained by 2D echocardiography, colour-flow Doppler, pulsed-wave and continuous-wave Doppler information. Qualitative diagnosis of AR with TOE is relatively easy, but quantitative evaluation is much more difficult, time-consuming, and is used more selectively. There is no one specific technique to accurately quantify AR severity

and it is therefore important to examine multiple imaging planes and use several parameters. The problem to grade severity will probably be overcome in future by three-dimensional (3D) echo.

Assessment of AR starts with an accurate 2D examination in the ME SAX and LAX views of the AV. This includes an evaluation of the cusps, LVOT, aortic root and ascending aorta. Poor coaptation, vegetations, cusp prolapse or perforations, and annular dilatation should be noted. The regurgitant jet causes a rapid rise in LVEDP and may impair opening of the anterior mitral valve leaflet ("reverse doming") by direct restriction leading to a "functional" mitral stenosis (Riedel). Early closure of the MV is sometimes seen before the onset of systole, which can in severe cases even lead to diastolic mitral regurgitation (demonstrated with CFD) (Emi). On M-mode a high frequency fluttering of the anterior MV leaflet may be seen during diastole as result of this jet. Left atrial dilatation would indicate severe AR, except in acute AR in which the chambers have not had time to dilate. The LV should be carefully assessed for function and any dilatation as result of the chronic volume overload, which provides valuable clues as to how acute or chronic the AR is. In chronic aortic regurgitation it will be more dilated and spherical in shape but not necessarily in the acute case. If the LV end-systolic diameter is less than 5.5cm, the patient has a better long-term prognosis. Marked left ventricular enlargement with an end-diastolic diameter of 8 cm or more is associated with increased risk of sudden death (Bonow) and is generally accompanied by overt dysfunction (Klodas). Even in asymptomatic patients surgery is recommended when the end-systolic diameter reaches 5.5 cm or more, or when the ejection fraction decreases below 55% (Bonow). Despite the high risk in patients with severe AR and reduced LV ejection fraction for aortic valve replacement, many will enjoy several years of event-free survival and should not be denied its benefits (Chaliki).

The 2D examination is now repeated with CFD. The colour flow jet is imaged in multiple planes to assess its direction, length, height and width. A central jet usually implies aortic root dilatation while an eccentric jet is caused by leaflet pathology. The severity of an eccentric jet is easily underestimated (Zoghbi). There are three components of a regurgitant jet: the flow convergence in the aorta, the vena contracta through the regurgitant orifice, and the jet direction and size in the LV.

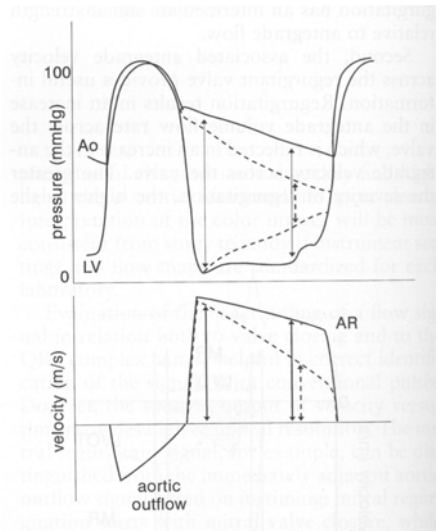
The vena contracta (VC) is the narrowest portion of the jet located at or just distal to the valve orifice, immediately below the flow convergence area (Baumgarten). It is independent of flow rate and driving pressure for a fixed orifice, and should therefore be less influenced by loading conditions than traditional indices of AR severity, such as regurgitant volume and fraction. Its cross-sectional area is equivalent to the effective regurgitant orifice area (Enriquez-Sarano). If planimetry of the jet performed in the AV SAX view is more than 0.3cm^2, AR is severe. To assess the vena contracta appropriately all three components of the jet, the flow convergence, the vena contracta and the jet body, need to be visualized. A vena contracta diameter of more than 0.5cm is highly sensitive, while a diameter of more than 0.7cm has a high specificity as a measure of severe AR (Tribouilloy). It may not be accurate in the presence of multiple regurgitant jets, eccentric jets, or jets with irregular shapes (Zoghbi). The vena contracta has also been shown to correlate well with direct measurements of the regurgitant volume and fraction by aortic flow probe (Willett). In 1987, Perry et al. described the height (width) of the AR jet as a ratio of the LVOT diameter, to have a good correlation with severity (Perry). It is different from the vena contracta, which is measured at the narrowest part of the jet. The jet width in the LVOT is bigger than the vena contracta because the jet expands after passing through the regurgitant orifice. Although this is a semi quantitative index, it became the clinical standard for echocardiographic grading of AR. When the jet width takes up more than 65% of the LVOT diameter it is severe. The jet width is superior to jet length or jet area and is usually accurate in the setting of eccentric jets (Ishii). Limitations of the jet width-LVOT ratio method are that the regurgitant jet orifice may not be in the same imaging plane as

the true LVOT diameter, or the regurgitant orifice may be asymmetric in shape. The jet may therefore be wider in one plane than another, leading to an inaccurate estimate. Both the vena contracta and jet width is evaluated with Nyquist limit set at 50-60cm/s. Colour M-mode can also be used to determine the jet width-LVOT ratio but is more useful to determine the duration of the AR jet during diastole.

The proximal flow convergence area or PISA (proximal isovelocity surface area) can be used to calculate effective regurgitant orifice area and regurgitant volume (Zoghbi). This method is derived from the principle that, as blood approaches an orifice, its velocity increases forming concentric, roughly hemispheric shells of increasing velocity and decreasing surface area (Bargiggia). The one hemisphere corresponding to the Nyquist limit can be used to make the calculations. A zoomed colour flow Doppler image is obtained of the aortic valve and the supravalvular region. The colour flow sector should be as narrow as possible with the least depth, to optimise lateral and temporal resolution. The colour scale is adjusted to achieve the largest possible rounded flow convergence area. The image is frozen and the radius of the proximal isovelocity surface area is measured from the wraparound junction between the blue and red colour, to the regurgitant orifice. A continuous wave Doppler measurement is made of the peak regurgitant blood velocity and velocity time integral across the valve, and used together with the "aliasing" velocity obtained from the colour scale to calculate the effective regurgitant volume and orifice area. Flow rate through the regurgitant orifice is calculated as the product of the surface area of the hemisphere ($2\pi r^2$) and the aliasing velocity (Va). Dividing the regurgitant flow by the peak regurgitant velocity then derives the effective regurgitant orifice (EROA):

$$\text{Regurgitant flow} = 2\pi r^2 \times Va$$
$$\text{EROA} = \text{Regurgitant flow}/Vpeak_{REG}$$

The PISA technique provides a fairly accurate quantitation of the AR (Tribouilloy). The threshold of severe AR is an EROA more than 0.3 cm^2 and a regurgitant volume more than 60 ml. Because of the small values measure, the slightest measurement error may lead to a large percentage calculation error and therefore misclassification of the AR severity. It is less accurate for eccentric than for central jets and a circular orifice is assumed. There is much less experience and evidence with PISA for the assessment of AR compared to MR (Zoghbi).



CWD is used to assess the AR jet from the TG LAX or deep TG views because the beam can be aligned relatively parallel to the blood flow. CFD can be useful to demonstrate the location and direction of the AR jet. The deceleration time (slope) of the diastolic regurgitant jet should now be measured. The rate of decrease of the velocity profile is influenced by the severity of AR, decreasing more rapidly with more severe AR because the larger regurgitant orifice allows a more rapid equilibration of pressures. A slope of greater than 3m/sec demonstrate severe AR, while a pressure halftime (PHT) of less than 200 msec will confirm that fact. The LV compliance and systemic vascular resistance affect these measurements.

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The continuity equation can be used to calculate the regurgitant volume (RV) and regurgitant fraction (RF). The RV is the difference between the systolic stroke volume across the aortic and mitral valves. The stroke volume through each valve is obtained

by multiplying the valve area by the velocity time integral across each valve. In the absence of mitral regurgitation and intracardiac shunts, flow across the mitral valve equals the cardiac output. AR is severe if the RV is more than 60 ml/beat across the AV.

RV = AV stroke volume – MV stroke volume

The RF is the ratio of RV to the stroke volume across the AV. If RF is more than 50%, AR is considered severe.

RF = RV/AV stroke volume

Holodiastolic flow reversal demonstrated with PWD in the descending aorta, is a sensitive confirmation of severe AR (Sutton). The long-axis view of the descending aorta is used and the PWD cursor is placed as much parallel as possible so that flow can be demonstrated.

Patients with severe left-sided regurgitant lesions can remain relatively asymptomatic while the left ventricle dilates and develops irreversible functional impairment. There is both volume and pressure overload and therefore not only an increase in chamber size but also in wall thickness. Left ventricular dysfunction continues to predict a very poor outcome in spite of technically successful valve surgery.

Intraoperative transoesophageal echocardiography in aortic valve surgery

Value of Intraoperative TOE

The routine use of intraoperative transoesophageal echocardiography in patients undergoing valve replacement for aortic stenosis has been validated. A prospective study of 383 patients with aortic stenosis for AV replacement showed the influence of intraoperative TOE (Nowrangi). In six patients a mitral valve replacement or repair was performed on the evidence of the intraoperative examination, although it was not originally planned. In another 25 patients the mitral procedure was cancelled because of the intraoperative findings, while the surgical plan was changed in another 18 patients (13%). Ionescu et al has confirmed the clinical impact and cost-saving implications of routine intraoperative echocardiography during valve replacement operations in a prospective study (Ionescu). Other authors also highly recommend postpump intraoperative TOE as an integral diagnostic modality contributing valuable data in valve replacement surgery (Shapira).

Pre-bypass

Intraoperative assessment of the patient with aortic valve disease provides up-to-date real-time information to the surgical and anaesthetic team. It confirms the preoperative diagnosis, provides detail on the mechanism of pathology, determines the feasibility to attempt a repair procedure and if not possible, provide measurements to plan the size and type of valve to be placed. Many patients will have both aortic stenosis and regurgitation, which complicates their assessment. Severe calcification of the annulus may affect the valve choice and the presence of postoperative paravalvular leaks. In the ME AV LAX view it is easy to measure the diameter of the annulus, sinuses of Valsalva, sinotubular junction and ascending aorta. Although the annular size does not really change during the cardiac cycle, it is good practice to make the measurements just before the aortic valve opens. The pressure in the LVOT and annulus diameter will then be at its maximum. The presence of even mild aortic regurgitation will influence the choice of cardioplegia administration technique for myocardial protection during valve surgery. In the case of AR, cardioplegia cannot be administered through the cardioplegia cannula in the aortic root as routine, because it will not reach the coronary arteries but cause dilatation of the LV through the regurgitant valve. In this case the surgeon will administer the cardioplegia with a special cannula through each individual coronary artery or via a retrograde cardioplegia cannula through the coronary sinus.

Quantifying the severity of disease is usually not necessary in the patient whose diagnosis is well established preoperatively, but is included in the comprehensive examination. This provides an up-to-date baseline for future reference. Remember that the systemic vascular resistance has an influence on flow across the stenotic valve and intraoperative hypotension will therefore increase the transvalvular pressure gradient in the anaesthetised patient. On the other hand will systemic hypertension in the patient with AS lead to a decrease in LV output and thus a reduction in the transvalvular pressure gradient (Kadem). To complicate matters, in the anaesthetised offloaded patient there is a deceased venous return, with a subsequent decrease in LV preload and stroke volume. This will also affect the flow velocity and pressure gradient measured across a diseased valve.

A complete examination also informs the team of any unexpected coexisting pathology. Evaluation of global and regional ventricular function together with inspection of the mitral and tricuspid valves is very important. Preferably the patient would be fully diagnosed and prepared before embarking on surgery, but it is fairly common to find additional mitral regurgitation more severe than expected, an incompetent tricuspid valve or the presence of an undiagnosed patent foramen ovale. Any extra procedures or on-the-table change of surgical plan would have serious implications for the patient. In the ideal situation the surgical and anaesthetic team should be able to interpret and integrate any new echocardiography information in close cooperation with the cardiologist, and make a team decision in the patient's best interest.

The perioperative clinician is often faced with the dilemma of a patient for coronary artery bypass grafting (CABG) surgery in which moderate aortic stenosis is discovered on intraoperative TOE. Quantification of aortic valve disease and the decision to perform a combined CABG and aortic valve replacement procedure can be difficult, especially when the LV function is compromised. Evidence shows that a subsequent AVR after a previous CABG has a higher mortality than a combined procedure (Odell). The degree of stenosis and calcification is important because even in the asymptomatic patient progression of the disease process may be rapid. Functional mitral regurgitation secondary to raised LV pressures from aortic stenosis is also common and will most likely improve after aortic valve replacement. If there is however intrinsic mitral valve disease with abnormal anatomy of the leaflets, an additional procedure to the MV should be considered. In our institution there is a low threshold to proceed with a combined procedure. Evaluation of the ascending aorta may demonstrate atheromatous disease and influence surgical cannulation site and cross-clamp techniques. Moderate dilatation of the aortic root and ascending aorta is also a common finding in patients with aortic stenosis or regurgitation. This may be post-stenotic dilatation due to the long-term turbulent flow pattern distal to the lesion, or secondary to an intrinsic weakness of the aortic wall. Weakness of aortic tissue may require surgical intervention. All this information is very valuable for intraoperative decisionmaking (Troianos).

Post-bypass

During the period immediately after termination of cardiopulmonary bypass TOE provides valuable information on ventricular filling and function, and is very helpful in guiding haemodynamic manipulation. The patient with chronic aortic valve disease often has LV hypertrophy and therefore an inherently poor compliance. This increased wall thickness reduces wall tension as explained by La Place's Law:

$$\text{Wall tension} = \frac{\text{Pressure} \times \text{Volume}}{\text{Wall thickness}}$$

The implication in these patients is that the filling pressure is not a reliable index of preload. They often require intravenous fluid administration in spite of high filling pressures in the immediate postbypass period (Kumar). With severe hypertrophy the LV cavity can almost become obliterated. After valve replacement these patients may sometimes experience systolic anterior motion of the anterior leaflet of the mitral valve. This is due to the acute reduction in afterload in the presence of an underfilled LV. This

patient does not appreciate any positive inotropic agents but usually does well with appropriate volume filling, a vasoconstrictor (phenylephrine) and even a beta blocker (negative inotropy, negative chronotropy).

Intraoperative transesophageal echocardiography is very useful for immediate postoperative evaluation of newly implanted heart valves. A more accurate assessment of valve function can be made if the type of valve prosthesis is known. Any paravalvular leak should be critically examined and its long-term impact should be considered against the risks of a second bypass period. Abnormalities identified in the operating theatre may require immediate surgical correction.

An unusually high peak velocity through a new valve must raise suspicion of prosthesis malfunction. In case of a bileaflet or tilting disc mechanical prosthesis it is of the utmost importance to visualise the full excursion of the leaflets. This is often difficult because of the echogenic shadowing and dropout due to the metal in the valve. Prosthesis malfunction with a leaflet or disc stuck in either the open or closed position can lead to serious perioperative morbidity. If it is not possible to see leaflet motion clearly, fluoroscopy in the catheter suite is indicated to confirm normal function of the valve.

The measurement of a high Doppler flow velocity across a newly implanted mechanical or bioprosthetic aortic valve immediately after the cessation of cardiopulmonary bypass can also be misleading. Several factors may contribute to an increased velocity (Schroeder). A Doppler measurement made through the smaller central orifice of a bileaflet prosthesis will demonstrate a higher peak velocity than through the larger side orifices. Making several measurements can decrease this error. Changes in stroke volume and cardiac output, which occur in the anaesthetised, underfilled, patient can significantly affect "pressure gradients". Immediately after bypass patients are often on inotropic support with a reduced afterload and is therefore in a hyperdynamic state. When a stentless bioprosthesis or homograph is implanted it is quite common to find an increased flow velocity across the valve immediately post-bypass. An aortic stenosis patient with concentric LV hypertrophy may demonstrate a subvalvular/LVOT gradient post-replacement without evidence of anatomic subvalvular obstruction (Morocutti). This is because the hypertrophic LV experiences a relatively "low" filling pressure. This functional "afterload mismatch" tends to normalize after a period of time. The post-cardiopulmonary bypass patient will also have a low hematocrit due to hemodilution from the bypass prime fluid. Blood viscosity is considered in the Bernoulli equation and this situation will therefore lead to calculation of a high pressure gradient across the valve. Technical errors such as patient-prosthesis mismatch may also lead to a high flow velocity (Rahimtoola). The pressure recovery phenomenon (as described earlier) should always be considered in evaluating prosthetic valve function especially when smaller size prosthesis (19, 21 mm) has been placed and when the proximal ascending aorta is small. All of the above factors therefore make it important to calculate valve area using the continuity equation. Moderate gradients therefore do not necessarily indicate imperfect surgical placement and intraoperative TOE is able to discriminate patients with functional phenomena from those with malposition of the prosthesis.

TOE can also confirm successful de-airing after any open-heart procedure (Tingleff). Even the smallest air bubble can cause severe postoperative instability if it enters one of the coronary arteries. With the patient in the supine position air will preferentially go down the anteriorly positioned right coronary artery, which will lead to acute right ventricular failure and arrhythmias, progressing to biventricular failure and cardiac arrest if not managed properly.

Specific procedures

Especially in more complex surgery like homograft or stentless valve replacements with or without root involvement, aortic valve repair, and whenever the ascending aorta is involved, the use of intraoperative TOE is essential. The more complex the attempted procedure, the more important it is that the echocardiographer has a good

understanding of the surgical procedure. The vast majority of aortic valves suitable for repair have regurgitant rather than stenotic lesions. Although the severity of AR is certainly a consideration, identifying the mechanism of AR by TOE may help the surgeon to distinguish those valves suitable for AV repair from the chronic fixed abnormalities requiring aortic valve replacement. A functional classification for aortic regurgitation has been described (El Khoury) to determine the mechanism of disease and to assist in AV repair procedures. The AV is viewed as a functional unit comprising of the annulus, three cusps, sinuses of Valsalva, commissures and the sinotubular junction. Competence of the valve unit depends on the integrity of all its components.

Type I: Normal appearing cusps with functional aortic annulus dilation
Ia: Distal ascending aorta dilation (sino-tubular junction)
Ib: Proximal (Valsalva sinuses) and sino-tubular junction dilation
Ic: Isolated functional aortic annulus dilation
Id: Cusp perforation and functional aortic annulus dilation
Type II: Cusp prolapse: excess of cuspal tissue or commissural disruption
Type III: Cusp retraction and thickening

A full discussion on the surgical repair techniques of the aortic valve is outside the scope of this chapter but can be found in surgical textbooks (Cosgrove) and has been elegantly reviewed recently (Hopkins, Yacoub). Although the severity of AR is certainly a consideration, it is the mechanism responsible for the AR that weighs more heavily in the decision to repair versus replace the AV. Aortic valve repair in the patient with aortic dissection without additional leaflet pathology is relatively easy to perform, and involves resuspension of the cusps (Movsowitz). The quality of an aortic valve repair procedure can be assessed early, even during cardiopulmonary bypass soon after the aortic cross clamp has been removed. The high aortic pressure due to non-pulsatile flow in the arterial cannula of the bypass circuit will indicate an unsuccessful procedure in the form of a continuous regurgitant jet. The presence of any residual aortic regurgitation after a repair procedure is a poor prognostic indicator of longterm outcome and should not be accepted. In such a case a second bypass period and replacement of the valve is indicated.

The Ross procedure involves the replacement of a diseased aortic valve, usually in the younger age group, with the patient's own pulmonary autograft (Kouchoukos). The pulmonary valve is then replaced with a cryopreserved cadaver pulmonary valve. The potential for valve "growth" and annular enlargement together with avoiding permanent anticoagulation, makes this an attractive option in the growing patient (Elkins). It is also associated with lower rates of endocarditis, thromboembolism and degeneration. Although technically demanding, in good surgical hands this procedure has been shown to provide excellent hemodynamic results, and low morbidity and mortality rates in children and young adults (Rubay). Accurate assessment of the aortic and pulmonary anatomy is important for these procedures (Faber). The diameters of the aortic valve annulus, sinotubular junction and ascending aorta should be measured. Severe dilatation of the aortic valve annulus (> 30mm) or the ascending aorta indicates a connective tissue disorder with a risk of future regurgitation of the autograft and is therefore a contraindication to this procedure. Due to its anterior location, the pulmonary valve is often better visualized by epicardial echocardiography rather than the transoesophageal option. The pulmonary valve should be tricuspid and without any degree of regurgitation or stenosis. The diameters of the pulmonary valve annulus and sinotubular junction should also be measured. The pulmonary artery sinotubular junction should match the aortic valve annulus. These measurements should be within 2 mm of each other to avoid distortion and subsequent regurgitation (David). Narrowing of the anastomoses sites would leave a high risk for future complications. Left ventricular contractility and regional function should be carefully evaluated for any sign of ischaemia or dysfunction in the immediate postoperative period. The first septal perforator branch of the left anterior descending coronary artery supplies the important

basal anteroseptal segment of the LV. It is in close proximity and at risk during harvesting of the pulmonary valve. At the end of the procedure the function of the aortic autograft and pulmonary allograft should be assessed. Any regurgitation of either graft indicates possible distortion during implantation and needs an immediate revision of the procedure.

Conclusion

For echocardiography to be fundamental in the success of aortic valve surgery it must be performed at the highest diagnostic level. Those performing intraoperative TOE must have appropriate equipment, skills and knowledge. Systematic examination with accurate recording and reporting, and appropriate professional supervision is the essence to a high-level intraoperative service and improved patient outcome.

According to the ASA/SCA Task Force Guidelines of 1996 valve replacement is a Category II indication for the use of perioperative TOE (ASA/SCA report). That means expert opinion and research studies show that there is weaker evidence and expert opinion that TOE may possibly influence the outcome of these procedures. In 2003 the American College of Cardiology (ACC), American Heart Association (AHA) and American Society of Echocardiograhy (ASE) published updated guidelines for the clinical applications of echocardiography (Cheitlin). In stentless bioprosthesis, homograft, the Ross, and aortic valve repair surgery, intra-operative TOE is recognised to play a very valuable role and these procedures are in the new Class I. Endocarditis and acute, persistent and life threatening hemodynamic disturbances as often seen after cardiopulmonary bypass, is also in Class I. Standard valve replacements, de-airing after cardiotomy and patients at risk of ischemia or haemodynamic disturbances however, fall in Class IIa. This means the weight of evidence/opinion is in favour of the usefulness of TOE in these procedures, although there is a divergence of opinion about its efficacy. With more experience and the availability of TOE on a wider basis, its value as a routine monitor and diagnostic tool will probably become beyond any doubt in the foreseeable future.

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Intra-operative TEE for mitral valve stenosis

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The intra-operative assessment of the mitral valve needs to consider the following:

Leaflet Pathology : Presence on anterior, posterior or both Leaflets, Diffuse or Localised thickening of more than 5 mm due to either Fibrosis oder calcification, Abcess, Vegetations, Perforation, Redundant Tissue, Cleft formation etc.

Leaflet movement: Normal, Restricted, Fixed, Excessive (Prolapse).

Papillary muscles: Normal, Contraction abnormality, Ruptured, Hyper-echoic.

Chordae tendineae: Normal, Shortened, Elongated, Fused, Thickened, Vegetations, Partial rupture.

Mitral ring: Normal, Minimal, moderate or severe calcification.

Mitral ring diameter: Maximum anterior- posterior diameter in the long axis of the left ventricle: Normal value < 35 mm (With average body size).

Quantification of Mitral Stenosis or Regurgitation: Rheumatic heart disease is the most common cause of Mitral Stenosis. In the years following the initial illness a progressive fibrosis and calcification of the valve leaflets with adhesions at the coaptation, shortening of the Chordae tendineae and reduction of the mitral orifice area. Although mitral stenosis following Mitral reconstruction is not a common complication, an intra-operative diagnosis by the cardiac anaesthesiologist is critical!

4 – 5 Square centimetre is the normal value for the mitral orifice area.

A mitral orifice area of between 2,0 und 2,5 square centimetre is mild, between 1,0 und 2,0 Square moderate and under 1,0 square centimetre severe.

The mitral orifice area can be measured using planimetry in a TEE transgastric short axis view at the level of the mitral valve leaflet tips (Fish mouth view).

Using TEE, the typical signs of advanced mitral valve stenosis are insufficient diastolic movement of the valve leaflets during the valve opening. When fusion of the commissures is present, a deformation of the leaflets known as "Doming" can be seen in Diastole. Focal or diffuse fibrosis and calcification of the leaflets is common with calcifications reaching up to 1 cm in thickness and are associated with progressive reduction in leaflet movement.

The left Atrium is often enlarged with Spontaneous contrast (Blood aggregate that appears as "Smoke" due to the stagnant flow in the Atrium). This indicates an increased risk of thrombus in the left Atrium, especially in the Auricle.

Using the transgastric TEE approach, the sub valvular Mitral apparatus should be assessed.
From the four chamber view, the probe should be advanced into the stomach, approx 45cm from the dental line. (Release any flexion fixation) Using ante-flexion and a slight retraction of the probe, the Chordae Tendineae and the Papillary muscles can be visualised at 80 – 100° in the long axis and in the short axis at 0°. In the presence of mitral stenosis, calcification, fibrosis, shortening, thickening and fusion of the Chordae are common, as well as fibrosis and increased echogenicity of the Papillary muscle tips.

CW-Doppler measurements of the diastolic Mitral Valve flow can be used to determine the mean diastolic trans-mitral gradient using the formula $P = 4 \times V_{mean}^2$, although both

the mean velocity and the pressure gradients are automatically calculated be the ultrasound system when a trace of the Doppler signal is performed.

The mean pressure gradient over a Mitral Valve stenosis decreases with decreasing heart rate (Due to a longer diastolic filling phase). Independent of the presence of sinus rhythm or atrial fibrillation, the patient will benefit from a controlled, low to normal heart rate. Tachycardia can result in decompensation and pulmonary oedema.

The mitral orifice area can be determined via the Pressure Half Time (PHT) method from Hatle et al. The PHT is the time required for the initial maximum pressure to fall by half and is inversely proportional to the size of the mitral valve opening area. Using a CW Doppler tracing of the MV inflow the maximum velocity is measured. As the relationship between the pressure difference is related to the square of the velocity, the velocity representing half the pressure difference can be calculated using the formula $V_1 = V_{max}^2/2$ *(or* $V_{max}/\sqrt{2}$*). The pressure half time is then the time difference between* V_{max} *and* V_1 *. The mitral orifice area is then calculated by dividing the pressure halftime into the constant 220 which has been found to the PHT of a mitral orifice area of 1* cm^2*. Modern echocardiography systems have these formula and measurement tools implemented to allow semi-automatic measurement the PHT and mitral orifice area.*

It is important to note that a accompanying aortic regurgitation can decrease the PHT and result in over estimation of the mitral orifice area (i.e. an underestimation of the mitral stenosis). Left ventricular diastolic dysfunction due to left ventricular hypertrophy can increase the PHT and result in under estimation of the mitral orifice area of even normal valves (Over estimation of mitral stenosis or Pseudo-stenosis)

In the absence of significant Aortic or Mitral regurgitation, the continuity equation can be used in addition to the PHT based on the continuity of blood flow through the aortic valve. A2 = A1 x V1 / V2, where A2 = mitral orifice area, A1 = the aortic orifice area, V1 = the aortic velocity and V2 = the mitral velocity.

Conclusion: *According to the Author, using intra-operative TEE and the methods described above, it is possible for the cardiac anaesthesiologist to provide immediate information in the mitral valve, including possible complications of mitral valve reconstruction that have an immediate impact on the surgical decision making.*

Assessment of mitral valve insufficiency with TEE

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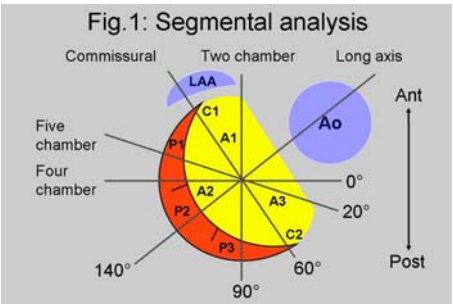
Mitral valve anatomy is a complex entity constituted of 2 leaflets, chordae tendinae (20 on the edges), 2 papillary muscles, a flexible ring, fibrous skeleton of the heart and left ventricular walls. The normal mitral valve function depends upon the complex interaction of all the components of the valve apparatus. They must be scanned along all possible different planes: retrocardiac 0°, 60°, 90°, 140°, and transgastric 0° (short-axis) (Figure 1) (8).



During systole, the MV has to oppose resistance against the high left ventricular pressures (up to 200 mmHg). The intraventricular pressure applies the leaflets again each other on the coaptation surface called zona rugosa. The coaptation surface results from an active balance between closing and tethering forces. A dilatation of the annulus will lead to a diminution of this surface and to an imbalance of these forces; a mitral regurgitation (MR) will ensue.

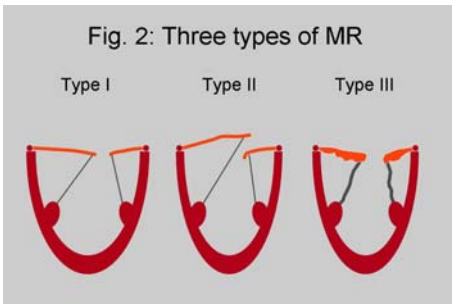
Mitral regurgitation (MR)

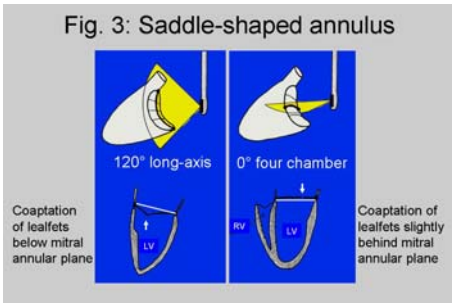
The incidence of the different mitral regurgitation aetiologies vary among the studies but the trend is definitely an increase of the degenerescent mitral valve prolapse (45%) with a progressive decrease of rheumatic diseases (12%). The incidence of ischemic MR (27%) remains quite stable over the years (11).

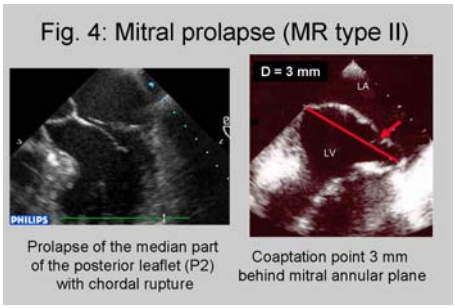
MR is classified in 3 types (Figure 2) (3):

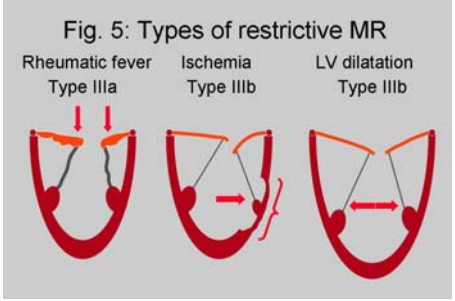
- *Functional MR (type I): normal leaflets, but dilated or calcified ring; the regurgitant jet is mostly central;*
- *prolapse (type II): one or both leaflets are prolapsing into the left atrium (LA), with elongated or ruptured chordae. The coaptation point is > 2 mm behind the mitral annnular level measured on 120° plane (because of the saddle-shape of the mitral annulus) (figure 1 and 3). The jet is excentric, directed away from the prolapsing leaflet. If the coaptation point of the mitral valve is situated at or below the annular plane but the middle part of the leaflet is bulging into the LA, it is called billowing (Figure 4);*
- *Restrictive MR (type III): the leaflets are hold back below the coaptation plane in systole by remodeling due to rheumatic fever (IIIa), systolic wall motion abnormality or LV dilatation (IIIb) (Figure 5) (6).*

The severity of MR and echocardiographic appearance vary depending upon loading









conditions and use of anaesthetics or vasoactive agents. To demonstrate the severity of a mitral regurgitation, we frequently need to manipulate the haemodynamics. By doing that, we should be aware of the different behaviour of different mechanisms of MR in front of haemodynamic variations. Although hypovolemia, decreased systemic afterload or increased inotropy will reduce the severity of most MR, it will increase it in case of a MV prolapse due to elongated chordae tendinae or myxoid degenerescence ; the chordae tendinae or excessive leaflet tissue are further relaxed towards the LA, which induces more prolapse (Figure 6) (1). Under anaesthesia, it is usual for a MR to decrease of 1 or 2 grades (on a 4-grade scale), because of the decrease in sympathetic tone and in venous return, and secondary to IPPV (5). In the restrictive type secondary to ventricular dilatation or ischaemia, the degree of MR is a good marker of the left ventricular functional state.

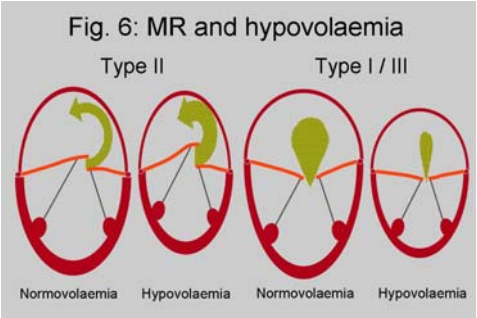


Fig. 6: MR and hypovolaemia

On 2D echo, the evaluation of the severity of MR is best achieved by using an appropriate combination of the following techniques: the anatomy of the heart, the colour and continuous-wave Doppler, the pulmonary venous Doppler flow, the proximal isovelocity area (PISA) and the vena contracta.
The anatomy of the heart in case of a chronic MR is characteristic: dilated LV and LA, systolic bulging of the interatrial septum into the RA, leaflets' non-coaptation or flail leaflet. The definitive diagnosis of MR relies mainly on colour Doppler. It is important to remember that the colour Doppler is a mapping of blood velocities but does not represent an actual blood volume (14). The dimensions of the systolic colour flow jet in the left atrium is the basic mean of eyeballing the severity of a MR; it is a poor way to quantify it, because it depends on different phenomena (7):

- *Colour gain, Nyquist limit, flow filter: machine setting is essential for colour Doppler use. A high Nyquist limit will decrease the jet area because low velocity flows are filtered out, a low Nyquist limit will increase the jet area because lower velocity flows are included. We should standardize the setting by keeping a Nyquist limit between 40 and 50 cm/sec and optimizing the colour gain.*
- *Instantaneous pressure gradient between LV and LA, which is proportional to LV afterload, LV functional status, and LA mean pressure;*
- *Direction of the jet: a central jet will recruit blood already in LA (Venturi effect) and will accordingly overestimate the real regurgitation severity (Figure 7). On the other hand, an excentric jet will underestimate up to 40% the severity of MR; the echo image is a tomography of the flow spread over the atrial wall surface and the jet looses energy by hugging the atrial wall (Coanda effect); it has therefore less velocity and less recruitment (Figure 8) (2).*
- *Regurgitation jet duration. If the jet is not pansystolic, one grade severity should be subtracted.*

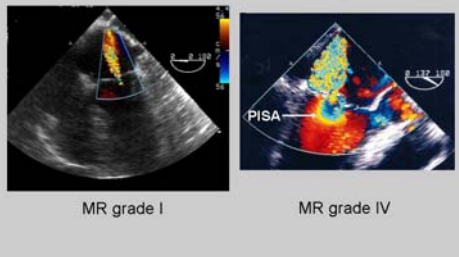


Fig. 7: Central MR flow jets

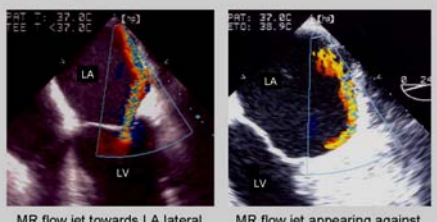
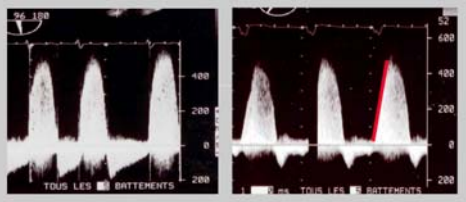


Fig. 8: Excentric MR flow jets

The MR flow should also be evaluated by continuous-wave Doppler; it appears as a large, full, rounded-shaped, high velocity (> 5 m/s) trace. If LV function is depressed, the peak velocity is decreased (< 4 m/s), and the ascending slope is decreased (depressed dP/dt) (Figure 9).

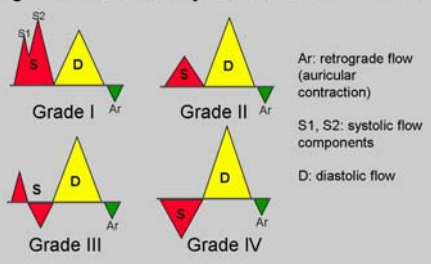
Fig. 9: CW Doppler MR flow



Normal LV function | Depressed LV function

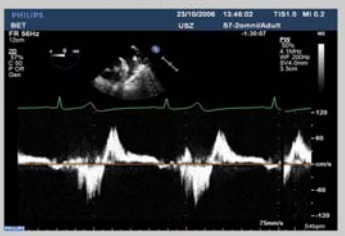
With pulsed-wave Doppler, it is possible to investigate the pulmonary vein flow; with increasing degree of MR, the systolic component of pulmonary venous flow is progressively decreased and reversed, whereas the diastolic component is increased (Figure 10,11) (12). A complete systolic flow reversal is pathognomic of severe MR; however, a severe MR may occur without systolic flow reversal if LA is very large and highly compliant. Pulmonary venous flow may also be asymmetrical in case of excentric MR jets; therefore, pulmonary venous flow must be investigated on both sides, usually in upper pulmonary veins.

Fig. 10: Pulmonary venous flow in MR



We have seen qualitative ways to assess MR. We may also want to quantify MR using the proximal isovelocity surface area (PISA) or the vena contracta (VC) diameter.

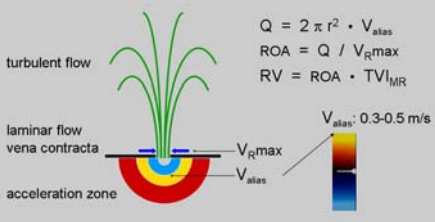
Fig. 11: Systolic pulmonary venous flow reversal



- *PISA (Proximal Isovelocity Surface Area) (Figures 7B and 12): as blood converges into a regurgitant orifice, it forms more or less hemispherical shells of increasing blood velocity and decreasing surface area (proximal flow convergence) (13). The principle of mass conservation assumes that the volume of blood in anyone isovelocity hemisphere is equal to the volume passing through the regurgitant orifice (RO). The continuity equation may be applied: the product of the first aliasing velocity (Valias) by the surface of the corresponding hemisphere is equal to the product of the RO area (ROA) by the maximal MR velocity:*
- $2 \pi r^2 \cdot V_{alias} = ROA \cdot V_R max$
  (r: radius of the hemisphere of first aliasing velocity)

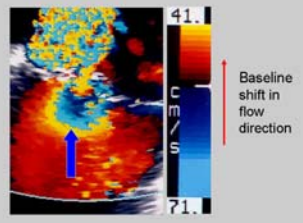
  $ROA = (2 \pi r^2 \cdot V_{alias}) / V_R max$

Fig. 12: PISA



The colour Doppler velocity baseline should be moved upwards in the direction of the flow to get the largest PISA and the scale must be set at values where V_{alias} is 0.3-0.5 m/s (9) (Figure 13); in severe MR (grade IV), the radius (r) is > 1 cm and the ROA > 0.4 cm^2 (7,15).

Fig. 13: PISA



The simplified method measures the aliasing radius by changing colour Doppler baseline to obtain an aliasing velocity of 40 cm/sec and assumes a peak MR velocity of 500 cm/sec (7):

$ROA = r^2/2$

- *In 4-chamber view, the mitral annulus is measured at maximal opening and the velocity time integral (TVI) of the transmitral flow is obtained by placing the Doppler sample volume at the level of the base of the MV leaflets. Mitral stroke volume (MSV) is calculated as the product of MV surface area and TVI.*

  $MSV = (0.785 \times D^2) \times TVI_{mv}$

Similarly, the aortic stroke volume (ASV) may be obtained from the inner LVOT diameter measured at aortic annulus in mid-oesophageal 120° view and the TVI_{LVOT} measured in 120° transgastric view.

$ASV = (0.785 \times D2) \times TVI_{ao}$

The regurgitation volume (RV) is then the difference between these two values.

$RV = MSV - ASV$

The RV may also be obtained from the ROA and the TVI of the MR.

$RV = ROA \times TVI_{MR}$

A simple method to estimate RV may be useful. The relation between TVI and $V_R$max has been shown to remain relatively constant : 1/3.25 (14). Therefore,

$RV = ROA \times TVI_{MR} = 1.9r^2 \times V_{alias}$

Finally, the regurgitation fraction (RF) is the ratio of the RV and the MSV.

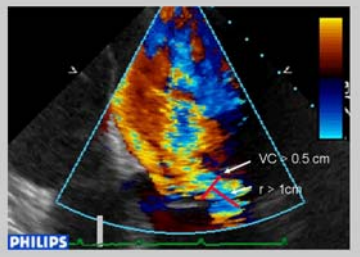
$RF = (RV/MSV) \times 100$

ROA < 0.2 cm^2, RV < 30 ml, RF < 30% = mild MR
ROA 0.2 – 0.4 cm^2, RV 30-60ml, RF 30-50% = moderate MR
ROA > 0.4 cm^2, RV > 60 ml, RF > 50% = severe MR (7)

vena contracta (VC) (Figure 12): the cross-sectional area of the regurgitant jet immediately below MV level or within MV leaflets represents the effective ROA and is called the vena contracta. The flow is laminar and its width is directly proportional to the dimension of the ROA on this plane. The measurement of the VC width may have an advantage over the PISA method for eccentric jets because the VC is affected less by the eccentricity of the jet (10).

VC < 0.3 cm = mild MR
VC 0.3-0.5 cm = moderate MR
VC > 0.5 cm = severe MR (7)

Fig. 14: Severe MR



In summary, a severe (grade IV) mitral regurgitation is characterized by the following echocardiographic signs (Figure 14) (10,15):

- *non-coaptation of leaflets (> 0.5 cm), or flail leaflet*
- *large MR colour flow jet reaching the posterior wall of the LA*
- *systolic flow reversal in pulmonary veins*
- *PISA: first aliasing radius > 1 cm*
- *vena contracta: jet width > 0.5 cm*
- *regurgitant orifice area > 0.5 cm^2*
- *regurgitation fraction > 50%*

The single most efficient sign is the width of the vena contracta (4).

The examination of the mitral valve must always be complete, including 2D scanning of different planes, colour Doppler flow, spectral CW Doppler flow, pulmonary vein flow pattern; semi-quantification must rely on different assessments, including 4-chamber global view, and not on an isolated value. The particular haemodynamic conditions of

anaesthesia and surgery must be taken into account. Best quantification is obtained with measures relatively independent from haemodynamics, like 2D measurements, PISA and vena contracta.

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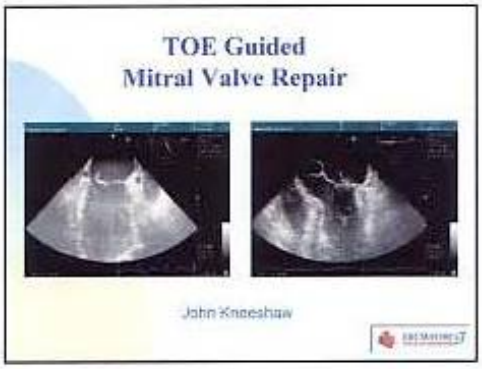
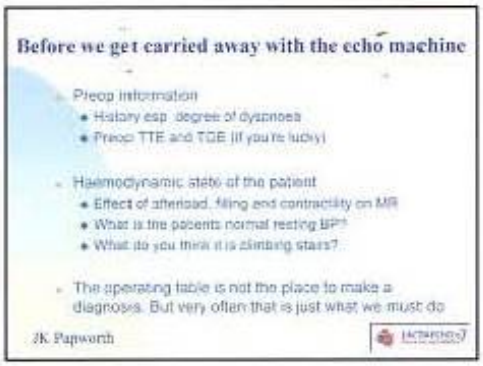
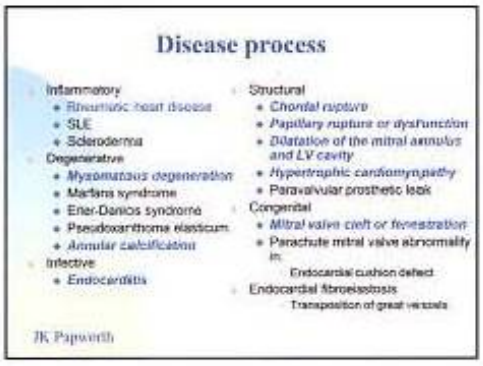
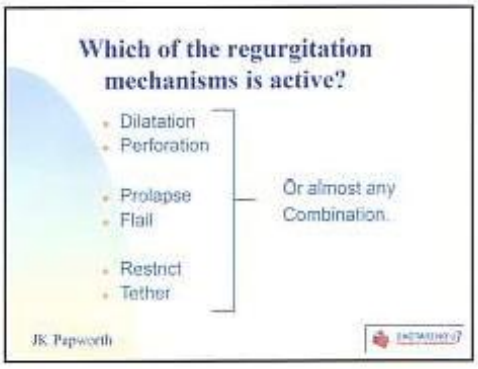
Guided Mitral repair

John Kneeshaw, Papworth, UK









Improved evaluation of the Location and Mechanism of Mitral Valve Regurgitation with a systematic Transoesophageal Echocardiography Examination

5 Ch
4 Ch
2 Ch
Comm
Posterior Comm
TG SAX

96% identification of involved segments
13 patients prospectively
12 patients retrospectively

Lambert et al.; Anaesth Analg 1999;88:1203-12

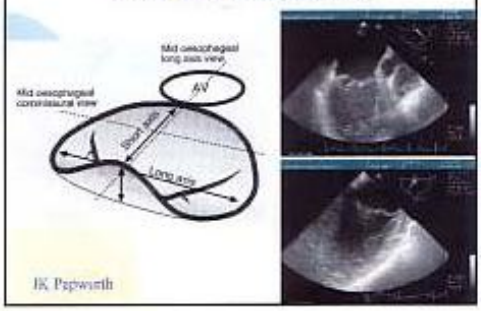
JK Papworth

Localising start simple then refine

- Anterior leaflet
- Posterior leaflet
- Both
- P1
- P2
- P3
- Ant
- AL Comm
- PM Comm

JK Papworth

Then add dimensions



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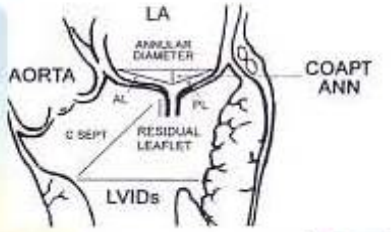
Upper limits of Mitral Annular Dimensions

- Antero posterior 36mm
- Intercommissural 46mm

- Measured at end systole

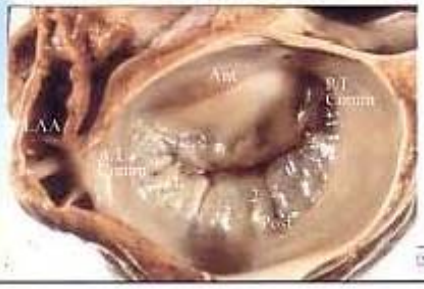
JK Papworth

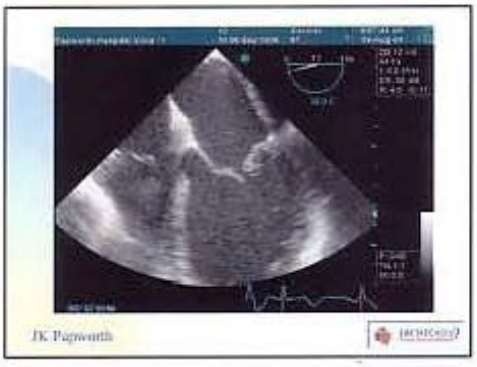
Mitral diameters and coaptation



JK Papworth

How much of the valve can you see in any one view?





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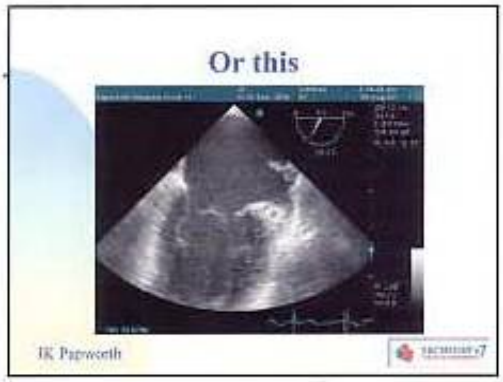
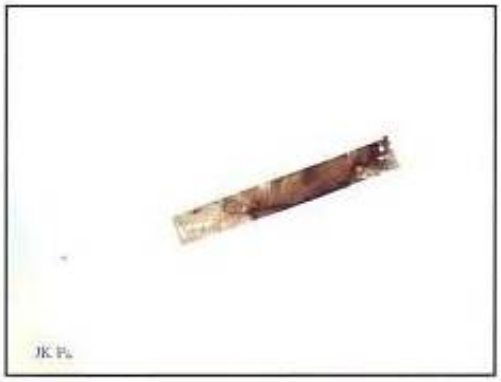
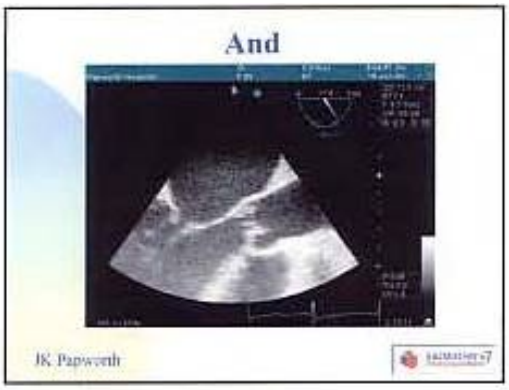
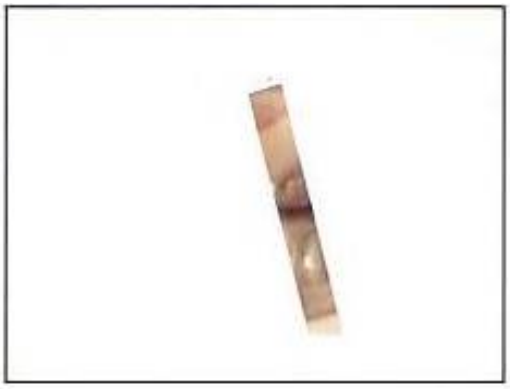
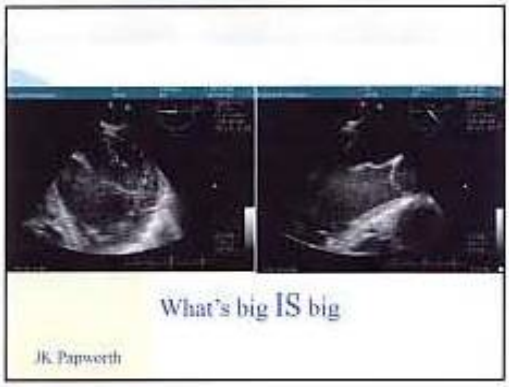
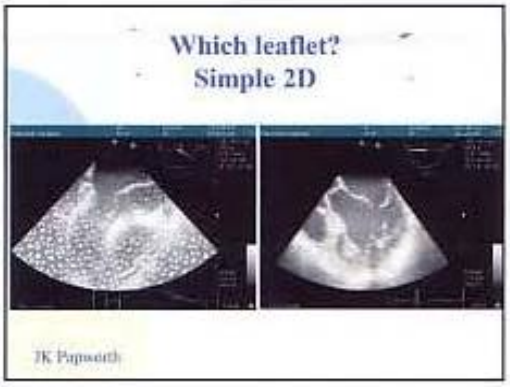

Or this
JK Papworth

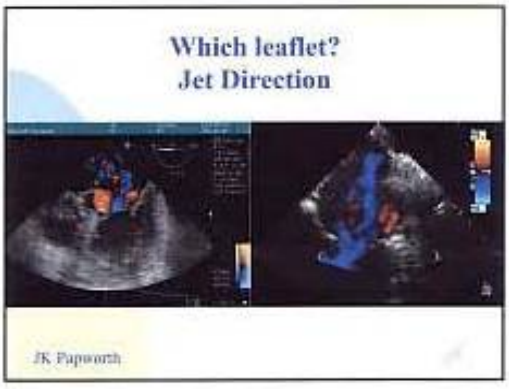

JK Pa

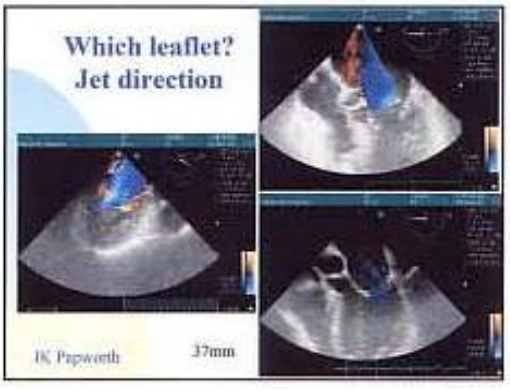

And
JK Papworth




What's big IS big
JK Papworth

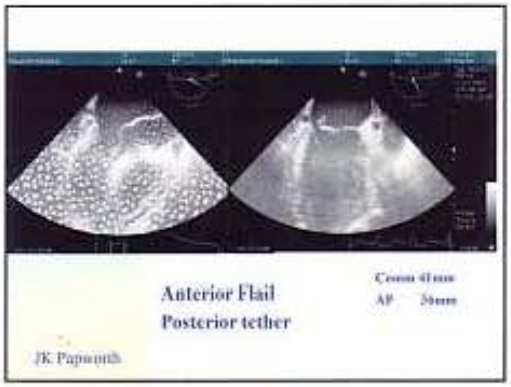

Which leaflet?
Simple 2D
JK Papworth


Which leaflet?
Jet Direction
JK Papworth


Which leaflet?
Jet direction
JK Papworth
37mm

Now can we pin down scallop or scallops

JK Papworth

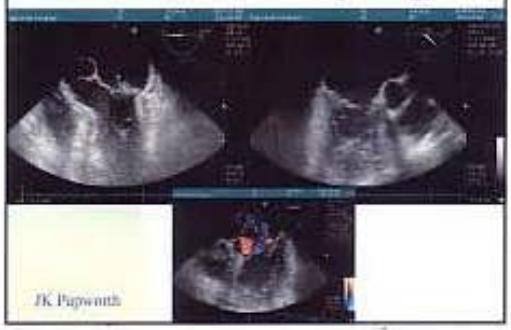


Anterior Flail
Posterior tether

Coom 41mm
AP 36mm

JK Papworth

Describe leaflet morphology



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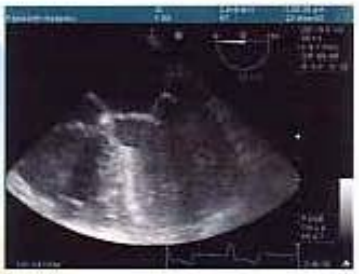
Calcification matters



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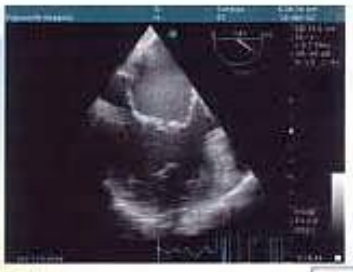
Note atrial size and filling



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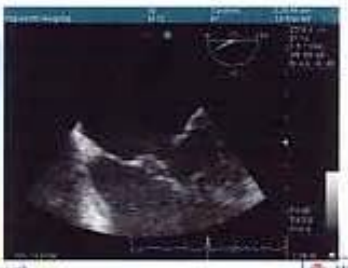
The seagull



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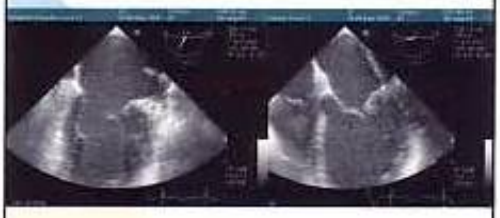
Where do those cords come from?



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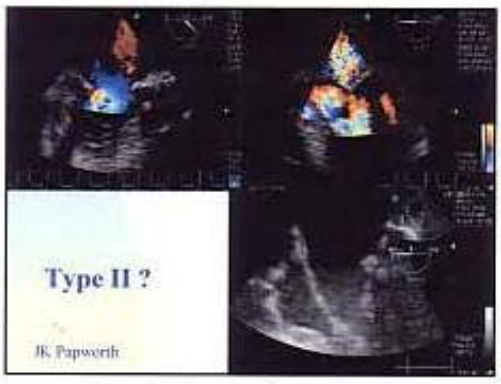

Type II ?

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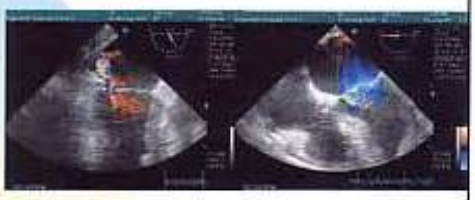
Same patient



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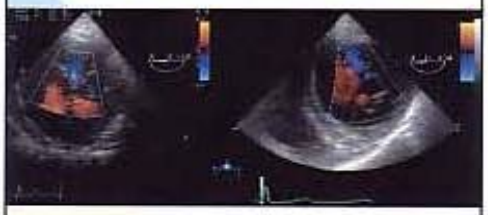
TG SAX all the segments in one view



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Mixed Rheumatic disease

Pliability of ant leaflet
Sub valvar mobility

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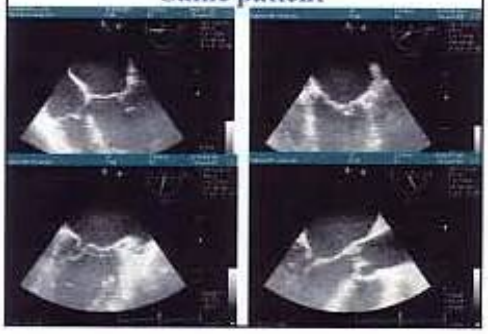
Same patient



Tethering

Ventricular remodelling with MR

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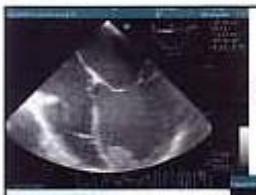
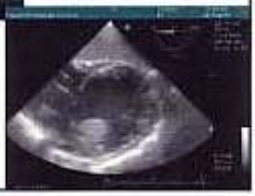
LV failure and dilatation and annular dilation plus apical ventricular thrombus





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Mitral lesions and failing LV

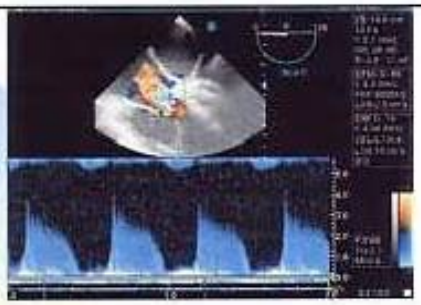
- In patients with severe LV dysfunction undergoing combined CABG and Mitral valve repair, restrictive LV filling pattern is an important marker of high perioperative mortality rate, further negative remodelling of LV and progression of mitral regurgitation late after MV repair

JK Papworth

Don't forget

Pulmonary hypertension

JK Papworth



$4V^2 + RA$

JK Papworth

What does the Surgeon want from us

- Terminology
- Communicating findings
  - Confirm diagnosis
  - Location, and mechanism
  - Feasibility ???
- Loops
- Report

Terminology

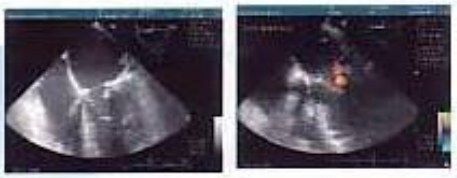
- Make sure you are speaking the same language. Carpentier or Duran.
- Use a clock face if you must. OK prostheses, but native valve anatomy is preferable

JK Papworth

Now I'll nip out for coffee while he fixes it

- Get the surgeon to show you his findings
- If they don't agree. Why not?
  - missed pathology
  - wrongly identified structures
  - Nobody is perfect – we do not always get it 100% right. Neither do the cardiologists!
- Now watch the repair
- Makes post bypass assessment easier

JK Papworth



Assessing the Repair

John Kneeshaw

Post repair evaluation

- Haemodynamic state
- LV function ?deterioration
- New LVWMA
  - Air (RCA)
  - Circumflex artery injury
- Stress if needed to get adequate load
  - Metaraminol (caution in poor ventricles)

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Haemodynamic state

143 patients mitral repair (post bypass findings)

TOE at end surgery vs TTE at discharge

>1 grade discrepancy in mitral regurgitation 17(13%)

Overestimated by TOE : 1 SBP 133mmHg

Underestimated byTOE 16 SBP 90mmHg

Freeman WK et al. J Am Coll Cardiol 1992;20:599-602

Rather old data

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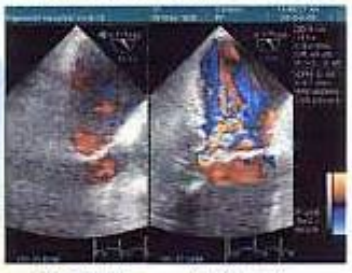
Same view same settings



MAP 65mmHg MAP 90mmHg

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Evaluation

- Residual MR
  - received wisdom accept up to grade 2+ MR (Sets?)
  - graded how?
    - Vena contracta >3.5. Doppler jet area >2cm², etc
- Mitral Stenosis
  - Valve area by planimetry
  - Mitral inflow PHT
  - Mean gradient
    - Evidence that gradient reduces 20-30% over 1st year
- Para annuloplasty leaks
- Not enough Alfieri experience
  - Size of both holes
  - Attention to commissures

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Other issues

- SAM
- Haemolysis
  - Related to flow disturbance ®
- Durability
- Accept or return to bypass

® = Garcia MJ. JACC 1996;27:399-406

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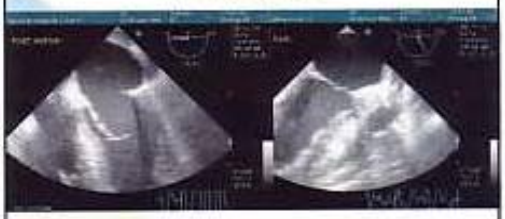
Post repair



Leaflet motion /coaptation

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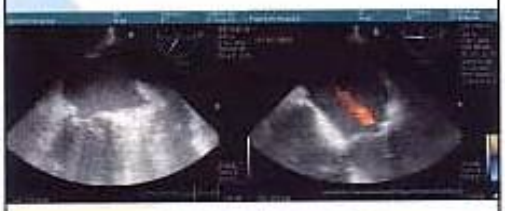
Moving along the posterior leaflet

P2 flail

Cordal rupture "Seagull"

And Annular dilation

Repair posterior resection



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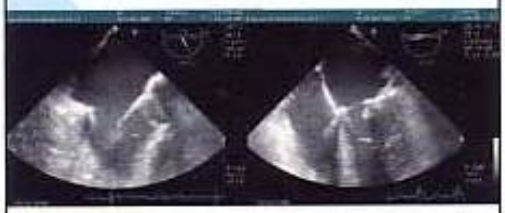
Leaflet motion, coaptation, annuloplasty, grade I MR

Plication Post Leaflet



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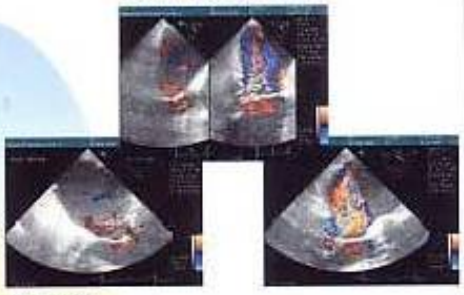
How much MR?



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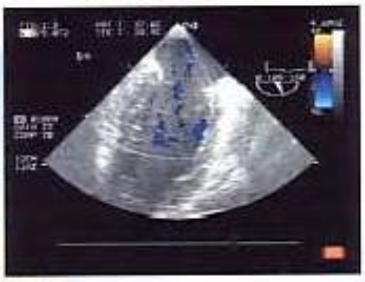
How much MR?



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Pericardial patch ant leaflet

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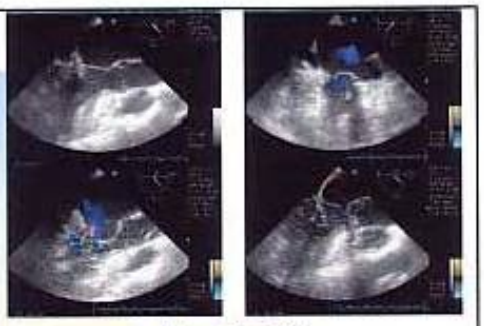

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How tight?

Mitral Stenosis Assessment in Mitral Repair

- Mean gradient
- PHT
- Area by planimetry
- Pisa

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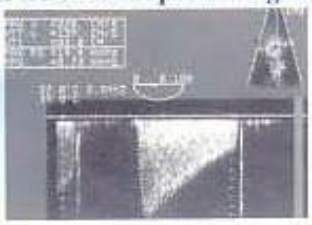
Mean transmitral pressure gradient



$$\text{Mean gradient} = \frac{4(V_1^2 + V_2^2 + V_3^2 + \ldots + V_n^2)}{n}$$

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Problems

- Depends on volume flow rate
- Low stroke volume will produce low gradient
- Angle of Doppler beam
- AF beat to beat variation

Generally

Normal	Mod MS	Mod MS	Severe MS
<3 mmHg	<6 mmHg	6-12 mmHg	>12 mmHg

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MV Area by Planimetry

- Mid diastolic measurement in a short axis view
- Usually an elliptical orifice less complex than the aortic valve
- Beware funnel shaped orifice

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MV Area by pressure half time ($T_{1/2}$ or PHT)

- Rate of pressure decline across orifice is related to orifice area
- Defined as interval between maximal early transmitral pressure gradient and the time when pressure gradient is half that value
- Applied initially to LA & LV catheter measurements and found to be reliable measure of valve area

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Limitations

- Not validated after balloon commissurotomy or through a mitral repair
- Effect of atrial and ventricular compliance
- Concommitant AR

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Broad Para annuloplasty leak

Best described by the clock face

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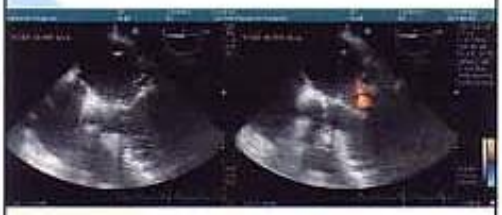
Para annuloplasty leak



Big enough to need fixing?

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SAM

- 2 – 6% Mitral repair surgery
- Venturi theory
- Mitral morphology with direct force on ant leaflet
- Mid oesophageal 5 ch and long axis views
- Treatment strategies
  - Rate, -ve inotropy, +volume, +vascular resistance

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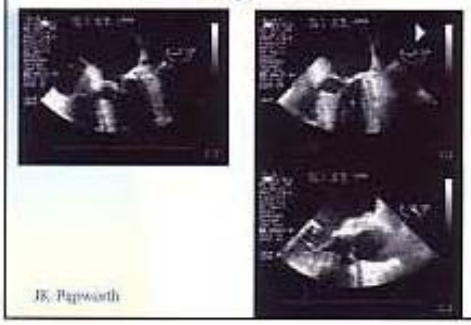
Post repair SAM



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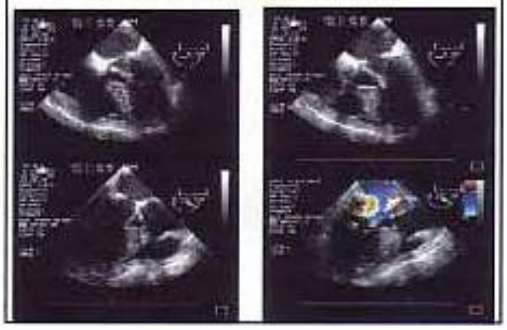
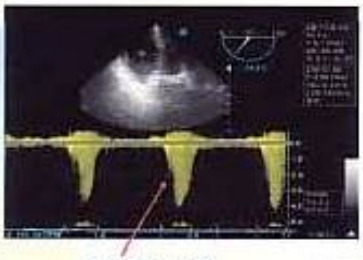
Severe post mitral SAM



Late peaking LVOT velocity



Dagger shape

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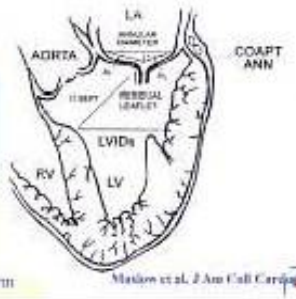
SAM Prediction in Mitral Repair



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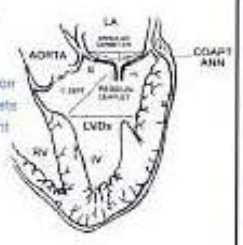
Maslow et al. J Am Coll Cardiol

- LVOTO / SAM / MR seen after AVR & Mitral repair
- May be variants of HOCM
- Similar mechanism
  - asymmetric septal hypertrophy
  - septal bulge
  - small ventricular cavity
  - hyperdynamic LV systolic function
  - excess or redundant mitral leaflets
  - anteriorly placed coaptation point
  - short c-sept



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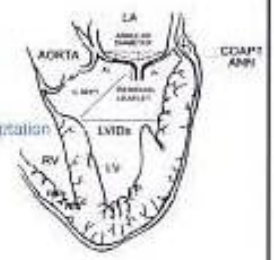
Predictors of LVOTO / SAM / MR

- Long AL >18mm in systole
- AL/PL ratio < 1:3
- C-Sept <25mm
- Anterior placement of the coaptation line of the MV



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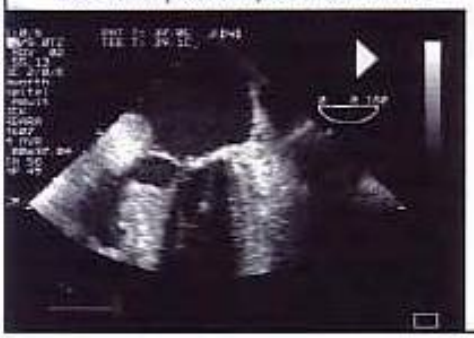
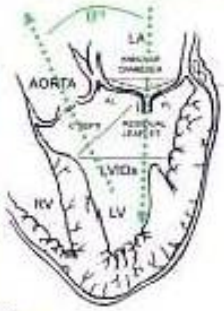
The addition of posterior mitral annular calcification adds to the probability of SAM / LVOTO



SAM Mechanism



θ > more SAM
θ < less SAM

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Wide Inflow / Outflow angle

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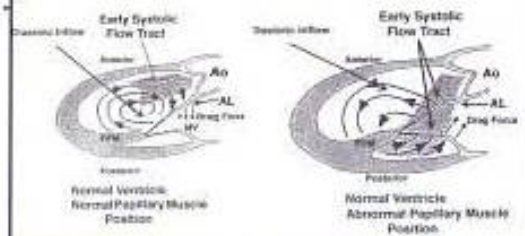
Papillary muscle position



Leichtweve XP et al. Systolic Anterior motion of the Mitral Valve in Hypertrophic Cardiomyopathy. J Heart Valve Dis 1995;4:322

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Making decisions

Intra operativesurgical decisions: a new role for the anaesthesiologist-sonographer

Curling PE et al. Anesthesiology 1988;69:A3

"...the need for surgical (re) intervention can be resolved immediately"

Dangerous stuff – the decision to re-operate is a surgical one. But we are part of a team

We provide data

Patient factors – age, LV & RV function, comorbidities

Papworth rules

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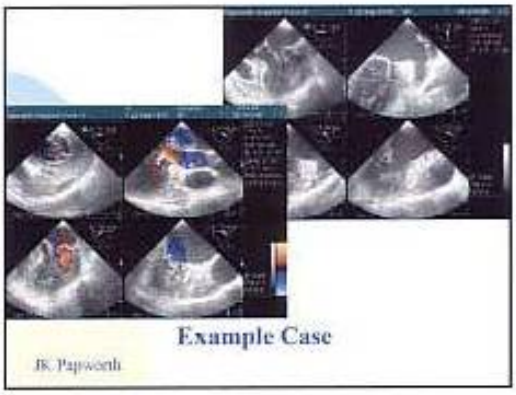
Example Case
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Relate findings to pathology
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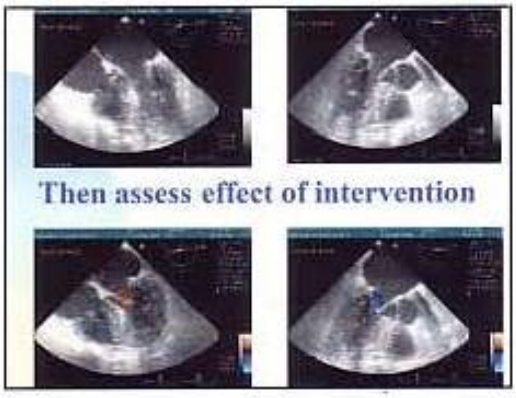
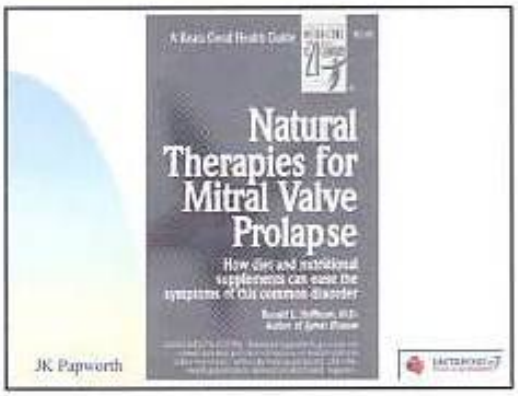

Then assess effect of intervention


Natural Therapies for Mitral Valve Prolapse
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Assessment of prosthetic valves

Christian Hassager, Copenhagen, Denmark

A comprehensive echocardiographic evaluation of a patient with a prosthetic valve often requires both transthoracic (TTE) and transesophageal (TEE) examination. The sewing rings and stents that the valve leaflets are attached to gives shadows that interfere with the ultrasound examination, and alignment of the Doppler wave and the blood flow may be difficult – thus all available ultrasound windows must often be used.

The key parameters that should be addressed by echocardiography in patients with prosthetic valves – depending on the clinical situation – are:
Ventricular systolic function
Gradient across the valve
Valve regurgitation
Masses (thrombi, pannus or vegetations) that may inhibits the valve function
While the first 2 are often best addressed by TTE, the two latter often requires TEE.

The systolic function of the ventricle to which the prosthetic valve is attached, a very important parameter in a patient with a suspected prosthetic valve dysfunction, is evaluated as in all other patients. It is often best evaluated by TTE.

The gradient across the valve is used to evaluate whether there is any obstructive problems with the valve. It is estimated from the blood flow velocity (V) though the valve measured by Continues Wave Doppler (CW) using the modified Bernoulli equation: Pressure Gradient = 4 x V^2. Maximal instantaneous and mean gradients should be reported for aortic valves and mean gradient for mitral valves. A high gradient does not necessarily indicate a stenosis if there is an increased flow across the valve as in valve insufficiency, anaemia or sepsis; conversely, a high normal gradient may indicate a stenotic valve in a patient with a severe ventricular dysfunction. The estimated pressure gradients are "flow volume rate" dependent. A reference flow measurement is therefore needed. This is most often done by measuring the flow velocity in the left outflow tract (LVOT) by Pulsed Wave Doppler (PW). The effective valve area may then be estimated by using the continuity equation:

<u>For aortic valves</u>: Aortic valve area (AVA) = (A_{LVOT} X VTI_{LVOT})/ $VTI_{prostetic\ aortic\ valve}$
where A_{LVOT} is the cross sectional area of the LVOT, and VTI is the velocity time integral (using mean instead of peak values). Another even simpler approach is to use the so-called "Doppler Velosity Index": DVI = VTI_{LVOT}/ $VTI_{prostetic\ aortic\ valve}$, where a value below 0.28 indicates a stenotic valve.

<u>For mital valves</u>: Mitral valve area (MVA) = (A_{LVOT} X VTI_{LVOT})/ $VTI_{prostetic\ mitral\ valve}$
The mitral equation requires that there is no more than mild mitral or aortic insufficiency. The Pressure Half-Time equation was originally derived from native rheumatic mitral stenosis, and has only been validated in a few prosthetic valves with variable results.

Valve insufficiency is evaluated by standard Color Doppler. Mechanical prosthetic valves all have a "built in physiologic leakage", which is believed to prevent thrombus formation by a washing mechanism. This "physiologic" valve leakage is short and narrow, symmetrical and of relatively low velocity compared to a large and wide, asymmetrical high velocity pathological regurgitation. It is important to note whether a regurgitant jet is within the sewing ring or outside the sewing ring and therefore a paravalvular regurgitation. This distinction often requires a TEE.

While determination of the degree of a stenosis can be done accurately and often best by TTE in most patients, the reason for a stenosis often requires TEE. The sensitivity of TTE for detection of various masses attached to a valve prosthesis is too low – TEE performs much better. This is true both for thrombosis and pannus formation. Sometimes when even a TEE does not give the answer, a cinefluoroscopy of a

mechanical valve may be a better and easier way to evaluate opening and closing of the leaflets. Finally TEE is also required if prosthetic valve endocarditis is suspected.

If prosthetic valve dysfunction (other than endocarditis) is suspected a careful auscultation and a TTE should be performed. If this indicates normal prosthetic valve function search for another cause should be initiated. A TEE (and in case of a mechanical valve perhaps a cinefluoroscopy) should be performed, if auscultation and TTE indicates abnormal prosthetic valve function.

TEE for detection of myocardial ischemia

Manfred D. Seeberger, Basel, Switzerland

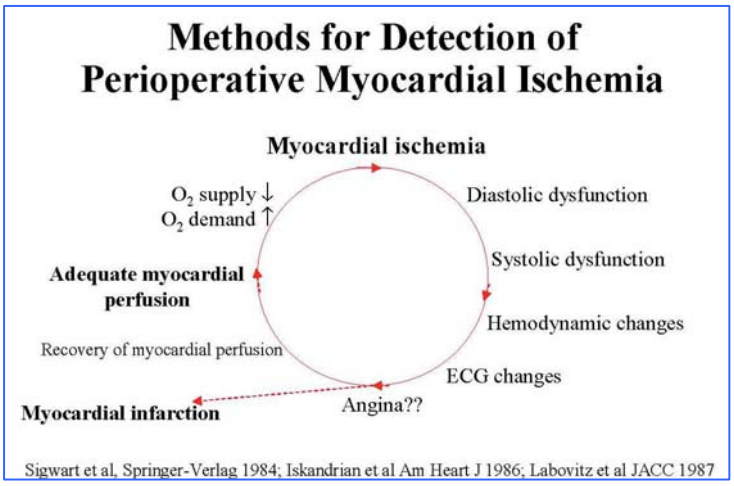
This lecture will discuss the value of echocardiography for detection of myocardial ischemia. It will mainly focus on the established method for echocardiographic detection of ischemia, i.e., analysis of segmental wall motion. Alternative echocardiographic methods that have been proposed for detection of ischemia will be concisely discussed, including stress echocardiography, contrast echocardiography, and Doppler echocardiographic assessment of intramyocardial blood flow. A final topic of the lecture is mechanical complications of myocardial infarction. Typical echocardiographic images of these complications will be presented, including ischemic myocardial regurgitation, papillary muscle rupture, left ventricular aneurysm, left ventricular thrombus, post infarction ventricular septal defect, and pseudaneurysm / contained rupture.

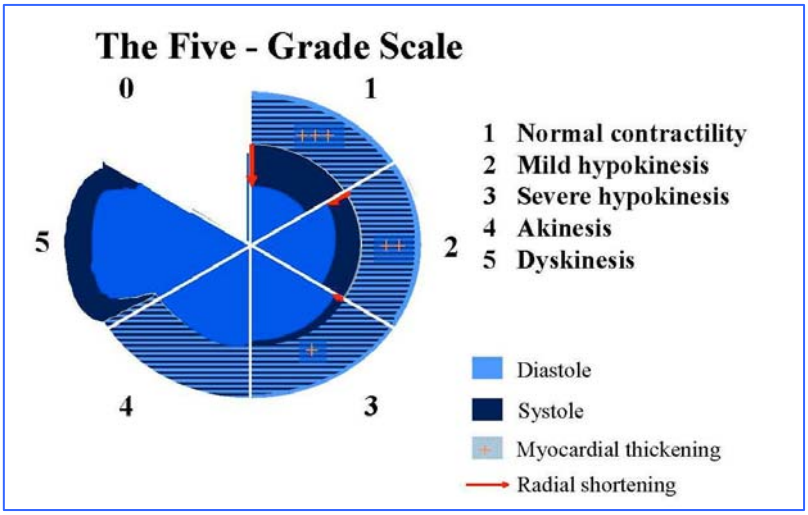
References:

1. Seeberger MD, Skarvan K, Cahalan MK: Myocardial ischaemia, Transoesophageal echocardiography in anaesthesia and intensive care medicine (2nd edition), 2nd Edition. Edited by Skarvan K, Poelaert J. London, BMJ Books, 2004, pp 196-220.

2. Shanewise JS, Cheung AT, Aronson S, Stewart WJ, Weiss RL, Mark JB, Savage RM, Sears-Rogan P, Mathew JP, Quinones MA, Cahalan MK, Savino JS. ASE/SCA guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography examination: recommendations of the American Society of Echocardiography Council for Intraoperative Echocardiography and the Society of Cardiovascular Anesthesiologists Task Force for Certification in Perioperative Transesophageal Echocardiography. Anesth Analg. 1999 Oct;89(4):870-84.

Figures and tables





Evaluation of Segmental Wall Motion

	Radial shortening	*Myocardial thickening*
0 No view / insufficient view		
1 Normal contractility/hyperkinesis	>30%	+++ (+25%)
2 Mild hypokinesis	10-30%	++
3 Severe hypokinesis	>0, <10%	+
4 Akinesis	0	0
5 Dyskinesis	systolic lengthening	systolic thinning

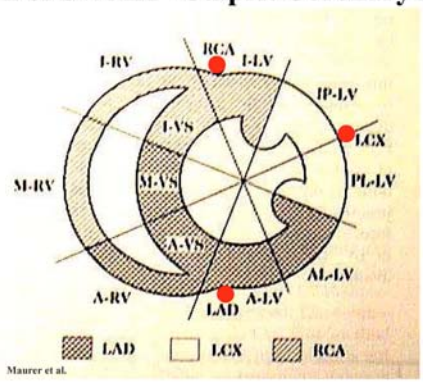
Diagnostic of ischemia if contractility worsens by 2 classes

Evaluation of Right Ventricular Wall Motion

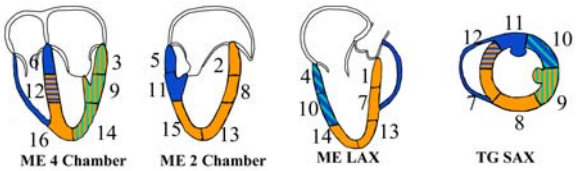
	Radial shortening	*Myocardial thickening*
1 Normal contractility	>30%	+++
2 Hypokinesis	>0 - 30%	+ - ++
3 Akinesis	0	0
4 Dyskinesis	systolic lengthening	systolic thinning

- *Diagnostic of ischemia if contractility worsens by 1 class*
- *No published perioperative studies*

Location of SWMA– Culprit Coronary Artery



16-Segment Model of the LV



	Basal Segments	Mid Segments	Apical Segments
RCA	1 = basal anteroseptal	7 = mid anteroseptal	13 = apical anterior
LAD	2 = basal anterior	8 = mid anterior	14 = apical lateral
LCx	3 = basal lateral	9 = mid lateral	15 = apical inferior
RCA or LCx	4 = basal posterior (inferolateral)	10= mid posterior (inferolateral)	16 = apical septal
LAD or LCx	5 = basal inferior	11= mid inferior	
RCA or LAD	6 = basal septal	12= mid septal	

Adult congenital heart diseases

Keld Sørensen, Aarhus, Denmark

As a result of the success of paediatric cardiology and cardiac surgery over the last three decades, there are now more adults than children with congenital heart disease (CHD). Now, more deaths from CHD occur in adults. This "new population" of patients with CHD does not fit within traditional divisions of training and practice, which have separated adult and paediatric cardiology. Over the last few years, the specialist needs of this growing population have begun to be appreciated, not only within the cardiology community but also among anaesthetists/intensivists who increasingly are exposed to these patients whether admitted to specific cardiothoracic units for cardiovascular problems (e.g. cardiac catheterisations/interventions or cardiac surgery) or admitted to general intensive care units for non-cardiac causes. Anaesthetists must be able provide optimal care for these patients who often have very specific needs which unappreciated may put the patients at high risk.

The disease spectrum covering adult congenital heart disease (ACHD) or grow-up congenital heart disease (GUCH) is unfamiliar to many anaesthetists/intensivists. Cardiac morphology and nomenclature may appear "strange", complex surgical procedures named after the innovative surgeon rather than descriptive and haemodynamics highly variable and commonly very "un-physiological".

Echocardiography remains the single most important investigational technique employed in these patients. Due to chest wall deformities, multiple previous cardiac operations, abnormal position of the cardiac mass and position of the cardiac apex, unusual position of valves, abnormal veno-atrial connections and arterial position, transthoracic echocardiography (TTE) may often prove insufficient in providing the echocardiographer with complete anatomical and functional information. Transesophageal echocardiography (TEE or TOE) usually provides important supplemental information, particularly for assessment of the interatrial septum, atrial situs (atrial arrangement), venous connections, valvular morphology, and intracardiac clots. For percutaneous interventions, particularly ASD and VSD closure and for intraoperative and postoperative assessment, TEE is an essential requirement. 3D and 4D echocardiography may soon provide additional and important morphologic information. Multislice CT and MRI are increasingly used in these patients, as imaging usually is optimal, particularly for extracardiac structures (venous and arterial connections, pulmonary arteries and the aortic arch). Conventional Doppler interrogation remains hugely important for functional evaluation. Tissue Doppler Imaging (TDI) may potentially add useful information, particularly in complex cases (e.g. univentricular hearts) and in patients with abnormal loading conditions.

In paediatric cardiology, anatomical scanning provides a much easier and understandable approach to the highly variable anatomy of congenital heart malformations, whether pre – or postoperatively. Adaptation of this technique, however, remains difficult for people trained with conventional orientation of the various echocardiographic scanning planes, although transducer positions and transducer movements remain exactly the same.

Diagnostic echocardiography is based on "segmental analysis" which is crucial to become oriented with CHD. A simple segmental approach comprises 4 steps. Firstly, the position of the apex is determined (levocardia, mesocardia, dextrocardia) from subcostal scanning. Secondly, the atrial situs is determined either directly from the anatomy of the atrial appendages on TEE or indirectly from the abdominal situs inferred from the relations of the aorta, the inferior vena cava (or vena azygos) and the spine as seen on subcostal transverse scanning. In some patients, both atria have identical appendages and the patient therefore two (morphologic) identical atria. These situations are defined as either right or left atrial isomerism. Thirdly, the atrioventricular relationship is assessed. This requires identification of the ventricular morphology (right ventricle

with the moderator band, the gross apical trabeculations, the septal attachments of the tricuspid valve, the lower insertion of the tricuspid valve compared to the mitral valve and the left ventricle without a moderator band, with smooth apical endocardium, lack of septal attachments of the mitral valve which also has a higher insertion compared with the tricuspid valve). When the morphologic right atrium is connected to the morphologic right ventricle (and similarly for the morphologic left chambers), a concordant atrio-ventricular (AV) connection has been established. If the morphological right atrium is connected to the morphologic left ventricle (and vice versa for the morphologic left atrium), the AV-connection is discordant. A double-inlet connection is similarly present when both atria empty into one ventricle (either the right or the left). Fourthly, the ventriculo-arterial (VA) connection is determined, once the echocardiographer has identified the pulmonary artery and the aorta. The VA-connections are similarly defined as either concordant or discordant. Double outlet from a ventricle is present when more than 50% of both great arteries arise from one ventricle (right or left). Single outlet is present when only one of the great arteries exit directly from the ventricles (i.e. pulmonary atresia). Finally, associated or specific lesions are identified, i.e. a ventricular septal defect (VSD), an aortic coarctation (COA) etc.

Anatomical assessment is based on these principles, but remains difficult for the anaesthetist/intensivist not familiar with the morphologic diversity of the lesions encountered in this specific group of patients. Awareness of abnormalities of the systemic venous drainage such as a left superior vena cava to coronary sinus and blocked superior vena cava must be exhibited.

Functional assessment remains the most challenging aspect of the echocardiographic examination in the ACHD population. Assessment of left sided valvular lesions is usually not different from what is generally applied to the normal adult patient. In contrast, evaluation of the right sided valves is more challenging, in particular for regurgitant lesions, e.g. in patients with Ebsteins anomaly or after Fallot repair. Shunt lesions (e.g. atrial septal defects, partial abnormal venous drainage, ventricular septal defects) can be quantified as the ratio of pulmonary to systemic blood flow (Qp:Qs). Pulmonary pressure is of particular importance in patients with functional univentricular circulations but also in shunt lesions. Assessment of ventricular function is often more complex due to anatomical abnormalities such as small ventricles, systemic right ventricles, "univentricular" hearts, abnormal interventricular interactions. Most (all ?) echocardiographic ventricular performance parameters used in adult patients are heavily load-dependent (ejection fraction, fractional shortening, myocardial performance (Tei) index) and clearly not optimal markers of contractility. In the ACHD patient, loading conditions are often more complex, making assessment of "myocardial function" particularly inaccurate. Reliable estimates of "contractility" are highly in demand. Echocardiographic surrogates for invasive parameters obtained from the pressure-volume curves such as the end-systolic elastance and the end-systolic pressure-volume relationship (ESPVR) are currently not readily available, in particular for the right ventricle. A move towards developing better and less load-dependent performance indices is however underway, some based on TDI, such as the isovolumic acceleration (IVA) and application of the force-frequency relationship (FFR) in clinical practice may further improve assessment of "cardiac function", particularly in the intensive care unit. Finally, ultrasound guided evaluation of the ventricular-vascular coupling will in due course most likely be an integrated part of the assessment of many of these complex patients.

TEE and Endocarditis

Christian Hassager, Copenhagen, Denmark

Echocardiography and blood cultures are the two cornerstones in establishing the clinical diagnosis endocarditis. Endocarditis most often affects the aortic, the mitral and the tricuspidal valve, and only rarely the pulmonic valve. The echocardiographic key findings that indicate a possible endocarditis are:

A vegetation. This is a mobile mass attached to the endocardium that can not be explained by an other reason. It is most often, but not exclusively, attached to a heart valve or its near surroundings.
An abscess. A rare echocardiographic finding because the content of an abscess may have acoustic properties close to that of other soft tissue and thus difficult to discriminate from heart muscle.
A pseudoaneurism, which is thought to be an abscess that has emptied its content into the blood stream, is a much more common finding. It is most commonly seen in association with an artificial aortic valve. Valve perforations may be caused by destruction of the valves involved and fistulas may also form between approaching heart chambers and vessels.
A new paravalvular leakage or perhaps even a loose artificial heart valve may also be seen in conjunction with endocarditis.

Pseudoaneurisms, fistulas etc. does of cause not disappear with adequate medical therapy, but this is often also true regarding vegetations – a sterile remnant of the vegetation may remain after complete clearance of the infection by antibiotics. It is therefore important to underline that echocardiography cannot securely distinguish between an active infection and a former treated endocarditis with a now sterile vegetation.

The sensitivity of transthoracic echocardiography (TTE) regarding the diagnosis endocarditis in patients with native valves is only about 50-60%, while the specificity is rather high – about 90%.

A significantly reduced echocardiographic window in about ¼ of the patients and the fact that this method does not have sufficient resolution to visualise small vegetations are the main causes for the lack of sensitivity. On the other hand transesophageal echocardiography (TEE) has both a high sensitivity and and a high specificity (both above 90%) in native valve endocarditis.

The sensitivity of TTE in prostetic valve endocarditis is as low as 30%, while TEE also in this population has a sensitivity above 90%. Perivalvular expansion of the infection resulting in abscesses, pseudoaneurisms and fistulas, which are found in more that half of the patients with prostetic valve endocarditis at the time of the diagnosis, is especially poorly visualised by TTE.

The data regarding pacemaker endocarditis are sparser, but TEE is also superior to TTE in this setting with a sensitivity of TEE at about 90% and of TTE at about 30%.

The above data are mainly derived from left sided endocarditis. A few reports with very limited cases also indicate that the sensitivity of TEE is superior to TTE regarding endocarditis of the pulmonic valve. TEE and TTE seems only to have comparable sensitivity regarding native tricuspid valve endocarditis – however even in this situation a TEE is often indicated to rule out endocarditis on the other valves.

The sensitivity of even TEE is however never 100%. It is therefore recommended to repeat the TEE a week later in patients where there is still a clinical suspicion of endocarditis even though the initial TEE is negative.

Artefacts and Illusions

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Imaging artefacts occur with all imaging modalities (2D-, M-, Doppler-modes). The most frequent causes of imaging artefacts are:
Ultrasound physics (and insufficient consideration of its laws)
Inappropriate instrument settings
Inappropriate alignment of the ultrasound beam in regard to the structure being investigated
Inappropriate equipment (sector defects of transducers, transducers of improper frequency)

2D echo imaging artefacts

Echo imaging artefacts can be grouped into three major categories:

- *Failure to visualize structures that are present*
- *Extraneous ultrasound signals that mimic structures that are not actually present, at least not in the imaged plane*
- *Image of a structure that differs in size and/or shape from its actual appearance*

Suboptimal image quality

Biological reasons:

- *Interposition of adipose tissue, lung, bone or gastric fluids or air (impedance mismatch) between transducer and cardiac structures*
- *Insufficient filling of cardiac chambers*

Technical reasons:

- *Inadequate penetration due to high frequency*
- *Suboptimal echocardiographic window due to transducer malposition*
- *Wrong gain settings (time and lateral gain controls)*
- *Maladjusted settings of imaging modes, contrast and post processing modes*

Advice: ***First optimize imaging window, then adjust controls (frequency, gain, focus, contrast).***

Motions across the imaging plane

The heart shows a twisting motion in the pericardium during the cardiac cycle. In addition, with respiration the heart is dislocated laterally. Both effects cause the heart to move across or in and out of the imaging plane during the cardiac cycle. The echocardiographer needs to minimize or eliminate this effect by optimal selection of the imaging plane or at least to recognize it.

Acoustic shadowing

At tissue boundaries or structures with significant difference in acoustic impedance, total reflection of ultrasound occurs (i.e. at calcifications, air, prosthetic valves, cannulas or tubes). Ultrasound travel distal to this structure is blocked. This causes a fan shaped shadow devoid of reflected signals, extending distal to the reflecting structure, following the direction of the scan lines. Structures close to the transducer cast large shadows, peripheral ones small shadows.

Advice: ***Chose a different angle of approach to visualize structures which are shadowed out.***

Reverberations

Reverberations are linear high amplitude echo signals originating from two strong specular (mirror) reflectors. Ultrasound is reflected back-and-forth between reflectors before it travels back to the transducer. The resulting increased time delay mimics structures distal to the reflectors extending into the far field. Prominent reverberations can extinguish information from structures in the far field: ⇒ Comet tail artefact

Beam width artefact

Ultrasound beam widens with increasing imaging depth, which causes lateral resolution to decrease with depth. Therefore point targets distant to the transducer appear as lines, and two close neighbouring points appear as one line.
Also the 3D volume of the ultrasound beam is displayed in a single tomographic plane. While the „slice thickness" is little near the transducer, it increases at the opposite site of the transducer (the far field) with increasing penetration depth. Structures in different spatial planes are superimposed in one imaging plane.

Advice: Minimize the beam width artefact by proper adjustment of the focus position.

Side lobe artefact

Strong specular (mirror) reflectors (for example calcifications, prosthetic material, catheters) produce echo signals if they are hit by the side lobes of neighbouring ultrasound beams. Side lobe echoes are depicted lateral to the object in the same distance to the transducer, resulting in arched lines extending laterally beyond the object in equidistance to the transducer.

Refraction artefact

The ultrasound beam is deviated from a straight path (the scan line) by refraction in tissue between transducer and object. The equipment assumes that the reflected beam has originated from the transmitted scan line, and the object is displayed on the image in the wrong location, often as double image next to the correctly displayed object.

Advice: Change the transducer position to eliminate or reduce refraction artefacts.

Range ambiguity artefact - mirror image artefact

In good imaging conditions, with a good window and little interpositioned tissue, little attenuation of the ultrasound signal takes place. Therefore, a strong, specular reflector in the near field sends much ultrasound energy back to the transducer, a part of which is reflected at the transducer or at a second specular reflector close to the transducer. With a depth setting at least twice that distance (resulting in a long listening period), ultrasound travels twice to this reflector, but the equipment assumes reflection has originated from twice as far. The result is a mirror image at double distance from the transducer.

Range ambiguity artefact - doubled image artefact

With a low depth setting and little attenuation, a part of the ultrasound energy travels beyond the depth setting (beyond the extend of the listening period). If deeper structures (below the bottom of the image) act as strong reflectors, ultrasound reflections from an earlier impulse will reach them in the meantime and bounce back to the transducer during the next sampling (listening) cycle. The equipment assumes that the signal has originated from a reflector closer to the transducer within depth range (actual listening time), and the image from the deeper structure is displayed overlying other structures close to the transducer.

Advice: Change of depth settings will alter or eliminate range ambiguity artefacts.

Doppler imaging artefacts

Doppler imaging artefacts, like 2 D artefacts, can be grouped into three major categories:

- *Failure to visualize flow velocity and direction that is present*
- *Extraneous ultrasound signals that mimic flow velocities and directions that are not present, at least not in the imaged plane*
- *Measurements of flow velocities and directions that differ from their absolute values*

Intercept angle artefact

A nonparallel angle between blood flow and ultrasound beam leads to underestimation of the flow velocity. With the use of the formula $V = F_d \times c / 2 \times F_0 \times \cos \alpha$, the equipment assumes that $\cos \alpha$ is 1. An angle α up to 20° can be tolerated, as $\cos \alpha$ is 0,94 at an angle of 20°, which would result in a 6 % measurement error.

Signal aliasing artefact

If the actual velocity exceeds the adjusted Nyquist limit (maximal measurable velocity) in PW- or CF-Doppler, the signal is displayed with inverted +/- signs:

- *With PW Doppler upside down*
- *With CF Doppler with reversed colours*

Advice: Prevent aliasing artefacts in maximising pulse repetition frequency and Nyquist limit
- by decreasing measuring depth in PW Doppler and colour flow area in CF Doppler
- by using the baseline shift to unidirectional double the Nyquist limit.

Range ambiguity artefact

With the PW Doppler sample volume close to the transducer and little attenuation, strong signals from double or triple the distance are recorded in the next receive phase and are misinterpreted as originating from the sample volume depth. This is constructively used in high PRF PW Doppler and always present in CW Doppler. Another form of range ambiguity are flow signals from adjacent structures which are superimposed in one Doppler signal due to the 3 D volume of the ultrasound beam. This artefact is more pronounced with increased sample volume depth and size.

Mirror image artefact

Often appears with spectral Doppler if strong signals are recorded from a low sample volume depth. A symmetric signal of somewhat less intensity is recorded in the opposite direction of the actual flow signal (upside down mirror image).

Advice: Reduce mirror imaging by using less gain and/or power output at the instrument.

Shadowing artefact

Structures being strong reflectors cause total ultrasound reflection, with no signals penetrating to and reflecting from beyond these structures. No velocities and flow directions can be measured in the area of the ultrasound shadow.

Ghosting artefact

Brief large colour patterns overlying anatomic structures with no underlying flow patterns, appearing inconsistent from beat to beat and mostly monochromatic (blue or red), caused by strong moving reflectors.

Gain settings artefact

This is very important with the use of colour flow Doppler. Extensive gain settings cause random background noise, whereas too low gain settings result in smaller flow areas than actually present being displayed.

Electronic interference artefact

These artefacts in the 2D and Doppler modes result from other electric instruments with inadequate shielding, for example with electric cautering and continuous cardiac output devices.

Illusions or Pitfalls

Pitfalls or illusions are normal anatomical structures or variations that are mistaken for pathology. Often they are misinterpreted as foreign bodies, thrombi or tumours.

Common pitfalls in the right atrium:

- *Crista terminalis: muscle ridge running from SVC towards IVC, separating SVC and right atrial appendage. Can be mistaken for a membrane or a catheter.*
- *Eustachian valve: membranous structure at the entrance of the IVC into the RA.*
- *Thebesian valve: fibrous structures at the opening of the coronary sinus.*
- *Chiari network: very mobile, filamentous structures arising from the Eustachian or Thebesian valve reaching to the lateral and superior walls of the RA.*
- *Pectinate muscles: muscle ridges in the RA and atrial appendages imitating thrombi.*
- *Enlarged coronary sinus: could be mistaken for an ASD, atrial aneurysm or cyst.*
- *Invaginated atrial appendage: imitating an atrial mass.*

Common pitfalls in the left atrium:

- *Pectinate muscles and invaginated atrial appendage as above.*
- *Membrane between LA appendage and left upper pulmonary vein (Warfarin ridge): may imitate an atrial mass, especially if fat has increased its thickness.*
- *Left atrial membrane or remnants of it. Seen in partial cor triatriatum. Restriction to blood flow needs to be ruled out.*
- *Double membrane of fossa ovalis, atrial septal aneurysm: can appear as a cyst or additional space. Needs to be checked for a patent foramen ovale.*

Common pitfalls in the right ventricle:

- *Trabeculae: normal muscle ridges mistaken for thrombi.*
- *Moderator band: very prominent trabeculum, stretching from the anterior RV to the interventricular septum.*

Common pitfalls in the left ventricle:

- *False tendons: echogenic structures spanning between walls and papillary muscles, often towards the apex.*
- *Calcified papillary muscles, calcified chordae tendinae: Highly echogenic structures in the ventricular cavity, which move in accordance to wall motion and valve function.*
- *Lobulated papillary muscles: may be confused with masses or thrombi.*

Common pitfalls at valves:

- *Valvular strands: thread-like fibroelastic tissue with endothelial cover, very thin (1 mm, up to 10 mm long) and mobile. Can be attached to all valves, predominantly to aortic valve (Lambl's excrescences) and mitral valve. Can be confused with vegetations.*

Common pitfalls in the pericardial space:

- *Transverse sinus: pericardial fold between aortic and pulmonary root and the left atrium. Can appear as an echo free space if fluid filled, or contain fibrinous tissue, air or parts of the left atrial appendage. Can be confused with the left atrium.*
- *Oblique sinus: pericardial fold be posterior wall of left atrium and pulmonary veins. Fluid collections after cardiac surgery are regularly seen, often with blood clots.*

Advice:	***Use multiple imaging planes to evaluate ominous structures. Follow them from side to side, sort out their anatomical relation to other structures and their moving pattern.***

The EACTA / EAE Accreditation process

Robert Feneck, London, UK

- *Accreditation is run as a service jointly by the European Association of Echocardiography and the European Association of Cardiothoracic Anaesthesiologists and is not necessarily a compulsory or regulatory certificate of competence or excellence.*
- *Applications for accreditation are welcomed from sonographers (technicians) and doctors of all disciplines.*
- *The goals of accreditation are to protect patients from undergoing transesophageal echocardiographic examinations performed by unqualified persons and to set a European standard for competency and excellence in transesophageal echocardiography.*
- *Accredited echocardiographers are expected to be able to perform and report routine transesophageal echocardiographic studies unsupervised.*
- *While European Accreditation is designed to test the competency of an individual to be able to perform, interpret and report routine transesophageal echocardiographic studies unsupervised, the right to report and sign clinical studies in individual countries will be defined by national laws and regulations.*
- *Accreditation in transesophageal echocardiography should bring credibility and professional legitimacy to an individual by demonstrating competency by the successful passage of examinations.*
- *The accreditation process will include a written and a practical component.*
- *The accreditation process will identify qualified practitioners of transesophageal echocardiography and should enhance the professional image of transesophageal echocardiographers. It will also provide statistics and records about transesophageal echocardiography that can be easily accessed.*
- *As echo skills can only be maintained by continued education and practical involvement, accreditation will be granted for a period of five years. After this time a re-accreditation process will be required to maintain accreditation involving demonstration of continued practice and learning in the field of transesophageal echocardiography.*

Further details of the registration process, examination and future consideration of re-accreditation can be accessed through the website. The best way to do this is to access www.eacta.org and then follow the TEE accreditation link. This is much less cumbersome than going through the EAE website.

Haemodynamic Calculations

Marco Ranucci, Milan, Italy

Introduction

The use of the Doppler signal coupled with a bidimensional echocardiographic study provides a quantitative information that is widely used in the standard echo examination and that can be useful in an intraoperative environment. Of course, we should always keep in mind that many complex techniques and calculations have a limited role during an intraoperative TEE examination; conversely, other measurements (i.e. the stroke volume assessment) may be of paramount importance. The present overview is aimed to stress the quantitative assessment that can be useful from the anesthesiologist's point of view, in order to assess the hemodynamic profile of the patient.
The hemodynamic calculations during a TEE examination require (a) the use of the PW and CW Doppler signal, (b) the measurement of areas and lenghts with the 2D echocardiography, and (c) an EKG trace. The following items will be addressed:

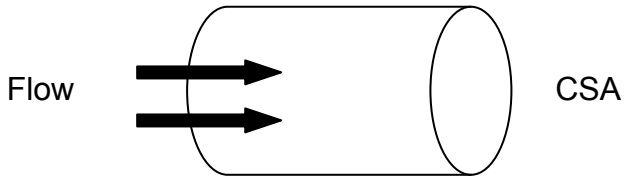
1) Volumetric measurements
   a) Stroke volume (SV)
2) Pressure gradients
   a) Peak gradient
   b) Mean gradient
3) Intracardiac pressures
   a) Pulmonary pressure
   b) Left atrial pressure (LAP)
   c) Left ventricular end diastolic pressure (LVEDP)
   d) dP/dT
4) Preload assessment
   a) The end diastolic area (EDA)
   b) The mitral flow pattern
   c) The aortic peak velocity changes

Of course, many other measurements can be obtained during a TEE examination both in terms of valve function (valve area, regurgitant volumes, effective regurgitant orifice) and diastolic function of the LV (mitral flow pattern, pulmonary venous flow pattern), but their applications will be addressed in other chapters of the present volume.

Basic equations

Three main equations are the basis for the following hemodynamic calculations:

(a) The flow equation states that the flow is the product of the cross section area (CSA) and average velocity of the blood cells passing through that CSA.



Flow = CSA x Velocity

Since volume is the product of flow and time, the above equation can be rewrote:

Volume = CSA x Velocity x Time

Velocity x Time is the velocity time integral (VTI) that is automatically given by all the common echo machines by tracing the profile of a PW or CW Doppler signal (figure 1 and 2). To assess a volume passing through an orifice at any beat, we therefore must apply the equation:

Volume (cm^3) = CSA (cm^2) x VTI (cm) (1)

(b) The Bernoulli equation, in its complete structure, determines the value of a pressure gradient for a flow passing through a restricted orifice. It can be semplified in the following equation:

$$\Delta P = \frac{1}{2} \rho (V_b^2 - V_a^2)$$

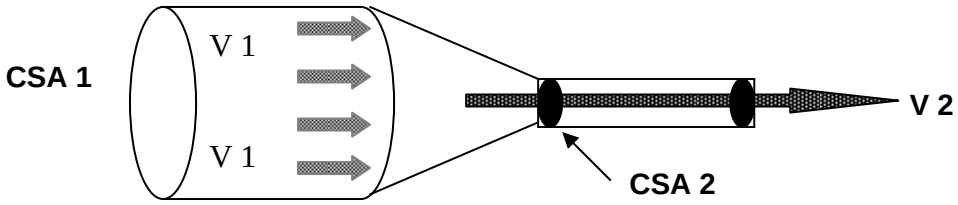
where ρ is the mass density of blood ($1.06 \times 10^3 kg/m^3$)
Vb is the velocity at point b (distal to the restriction)
Va is the velocity at point a (proximal to the restriction)

Since Va is very low, it can be skipped, and therefore the final equation that is commonly applied is

$$\Delta P = 4 \times V^2 \qquad (2)$$

(c) The continuity equation is the Gorlin formula of echocardiography and is applied to calculate the area of a stenotic or regurgitant valve. Basically, it states the "conservation of flow" regardless of the CSA that is met by the flow itself:

Flow (Q) = CSA x Velocity



$$Q_1 = Q_2$$
$$Q_1 = CSA_1 \times V_1 = CSA_2 \times V_2 \qquad (3)$$
$$CSA_2 = Q_1 / V_2$$

1. Volumetric measures

(1a) Stroke volume and cardiac output

To determine the stroke volume the equation (1) is applied. It requires the assessment of the aortic flow VTI and of the CSA. It is assessed with the following steps:
*1. Research of the best parallel orientation with the blood flow at the 2D examination. This is usually obtained using the transgastric views **J** or **K** of the Society of Cardiac Anesthesiologists (SCA) scheme.*
2. PW Doppler signal tracing by placing the sampling volume at the left ventricle outflow tract (LVOT) level, and VTI determination by tracing the outer edge of the dense envelope of the spectral recording.
*3. CSA determination: when using the PW Doppler for VTI determination, the LVOT area is to be considered. It can be assessed by the 2D examination, using the **I** view of the SCA scheme, and by calculating the LVOT diameter. Trace the measurement line at the level of the aortic annulus during systole. From this diameter, and by assuming a circular section, the CSA is determined by the following equation:*

$$CSA = D^2 \times 0.785 \qquad (4)$$

4. Calculate the SV (CSA x VTI) and cardiac output (SV x heart rate)

*A second way to determine SV is based on CW Doppler signal instead than PW. In this case, there is not a well determined sampling volume, and the CW signal reflects the velocity of all the blood cells moving along the path of the sound beam. If we consider the VTI obtained with a CW signal, it is usually larger than the corresponding one obtained with the PW Doppler, and we must refer it not to the CSA at the level of the LVOT, but to the CSA of the aortic valve (that is smaller). In this case, we must use the **H** view at the 2D examination, and obtain the CSA by tracing the edges of the aortic*

valve during systole. This method may be less precise due to possible errors in this last measurement.

*A third way for determining SV is based on transmitral flow instead of aortic flow. In this case the VTI of the mitral flow is assessed using the **A** view of the SCA scheme at the 2D examination. The sampling volume of the PW Doppler should be positioned so that in diastole it is at the level of the annulus. Using the same view the mitral annulus diameter can be measured, tracing a line from the base of the posterior and anterior leaflets during early to mid-diastole. The CSA can be assessed applying the equation (4) although the mitral annulus is not perfectly circular, the approximation can be accepted.*

Whatever method is applied to assess the SV, the main question remains: is a TEE-based SV and cardiac output determination reliable? Of course, the theoretical calculations at the basis of all the equations applied are sound, but the main problem remains that both 2D and Doppler determinations are strongly operator-dependent. To this respect, the ability of the operator to limit the potential sources of error (non-parallel flow determination; incorrect border determination of the Doppler signal; excessive approximation in CSA determination...) is of paramount importance, and limits the applicability of echo-derived SV measurements. Nevertheless, TEE may be useful as a "trend monitor" of SV and cardiac output during cardiac and non-cardiac operations. Given that the CSA is relatively stable in the same patient, the VTI serial changes in time accurately reflect equivalent SV changes. When using a serial monitoring of VTI, the best way is to use the CW Doppler of the aortic flow, that appears to have less variability than the equivalent PW Doppler.

2. Pressure gradients

The pressure gradients across a stenotic structure are assessed by the modified Bernoulli equation (2). A CW Doppler signal is commonly applied when exploring aortic stenotic lesions, while the mitral valve can be explored with a PW Doppler signal too. All the commonly available Echo machines automatically calculate the pressure gradients from the Doppler signal waveform. To obtain the mean pressure gradient it is needed a complete tracing of the waveform profile, while the peak gradient assessment only requires to settle the velocity peak.
*Transaortic pressure gradients calculations need a Doppler signal parallel to the transaortic flow; this is achievable in the transgastric views **J** or **K** of the SCA classification. Transmitral pressure gradients calculations can be obtained in the standard four chamber view **A** .*
*Gradients across the pulmonary valve are more difficult to be obtained due to a difficult parallelism of the Doppler signal with transpulmonary flow. This can be obtained, in expert hands, by a 2D transgastric view or using the **T** view of the SCA classification.*

Since the calculation of pressure gradients relies on the quadratic expression of velocity, little errors in determining this last parameter are amplified. It is therefore very important to find the best parallelism between Doppler signal and blood flow.
Once obtained this, the measure is reliable unless (1) the velocity proximal to the stenosis exceeds 1.5 m/s; (2) there are two stenotic lesions in the blood flow path (subaortic stenosis + aortic valve stenosis); and (3) the stenotic lesion is very long, tunnel-like.

3. Intracardiac pressures

Intracardiac pressures may be calculated with TEE providing that one or more cardiac valves are regurgitant. By measuring the peak velocity of the regurgitant flow, a pressure gradient can be established. The pressure gradient plus the pressure in the chamber receiving the regurgitant flow is the pressure inside the chamber driving the regurgitant flow:

Driving Pressure = Pressure Gradient + Pressure distal to the regurgitant flow

Many pressures can be calculated depending on the presence of regurgitant flows and septal defects. The most commonly measured pressures are:

(3a) Systolic pulmonary artery pressure (sPAP)
Requires a tricuspid valve regurgitation. The peak velocity and the resulting pressure gradient are determined usually using a CW Doppler signal. The pressure distal to the regurgitant flow is the RAP. The equation is:

sPAP = Peak Pressure Gradient + RAP

This equation is true in absence of a pulmonary valve stenosis, assuming that the systolic right ventricular pressure is equal to the sPAP. Considering a sPAP in the range of 20-60 mmHg and a RAP of 10 mmHg, the peak velocity of the tricuspid valve regurgitant flow is in the range of 2-3.5 m/s. A 10% error in its determination (due to inexact parallelism or other causes) will result in an error of 5-10 mmHg in the determination of the sPAP. This margin of error is clinically acceptable, giving an 85% approximation of the sPAP. This measurement is actually frequently used both in the preoperative assessment and in the intraoperative environment.

(3b) Left atrial pressure (LAP)
Requires a mitral valve regurgitation. The peak velocity and the resulting pressure gradient are determined usually using a CW Doppler signal: consider that the mitral regurgitation flow is often characterized by a very high peak velocity. The driving pressure is the systolic systemic arterial pressure (sSAP). The equation is:

LAP = sSAP – Peak Pressure Gradient

This equation is true in absence of an aortic valve stenosis, assuming that the systolic ventricular pressure is equal to the sSAP. The main problem with this calculation is that the mitral regurgitant flow has a very high velocity (in a patient with a sSAP of 130 mmHg and a LAP of 10 mmHg the peak velocity is 5.5 m/s). A 10% error in the determination of the peak velocity gives course to an error of 20 mmHg in the determination of LAP. This level of approximation, for a critical measure ranging between 5 and 25 mmHg is clinically unacceptable. Therefore, the intraoperative measurement of LAP with a Doppler technique is highly arguable and often clinically irrelevant.

(3c) Left Ventricle End Diastolic Pressure (LVEDP).
Requires an aortic valve regurgitation. The end diastolic pressure gradient is assessed using a CW Doppler signal (see figure 6). The driving pressure is the diastolic systemic arterial pressure (dSAP).The equation is:

LVEDP = dSAP – End Diastolic Pressure Gradient

(d) dP/dT
The rate of LV pressure change during the isovolumic contraction phase is an index of myocardial contractility. To be determined, it requires a mitral valve regurgitation. The mitral valve regurgitation velocity changes are proportional to the left ventricle dP/dT. The analysis of the early phase of the mitral valve regurgitation signal (between 1 m/sec and 3 m/sec velocity) allows a calculation of the dP/dT that, in a normal left ventricle, should be 1,200 mmHg/sec or greater.
The same calculations are applied to the tricuspid valve regurgitation for the right ventricle, generally using the time interval between 1 and 2 m/sec velocity.

Both LVEDP and dP/dT evaluations are typical preoperative LV function measurements. Their usefulness and clinical relevance in the intraoperative setting are doubtful.
Preload assessment
In the presence of a low output state during the perioperative period, the ability to detect which patients are more likely to benefit from volume administration while avoiding a

fluid overload is of paramount importance. Many echocardiographic indicators of preload and of responsiveness to "fluid challenge" have been proposed.
The most common way for assessing the LV voulmetric preload is to determine the End Diastolic Area (EDA) with a transgastric view at the mid-papillary level. More recently, Doppler – derived indices of responsiveness to volume administration have been proposed. The mitral flow was extensively studied to this respect: some authors identified that a low VTI E wave/A wave ratio was associated with a large increase of SV after volume administration. A mitral E/A ratio < 1.26 before fluid administration best predicted a 20% increase in SV after fluid administration.
The Aortic flow Doppler signal has been proposed as a preload indicator as well: the respiratory cycle induced aortic blood velocity variations are a good indicator of the preload conditions and of responsiveness to fluid administration. A variation in aortic peak flow velocity of 12% is discriminant between responders and non-responders to fluid administration in patients with septic shock, and in any case of mechanical ventilation the respiratory changes of aortic peak flow velocity may be used to detect biventricular preload dependence and fluid responsiveness.

The use of TEE for preload assessment in mechanically ventilated patients has a major role in ICU more than in the intraoperative setting. Anyway, the heart-lung interactions during mechanical ventilation may be well identified with a TEE 2D and Doppler examination. This information, together with a sPAP determination, may usefully guide the fluid therapy.

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Deformation imaging - new modalities with focus on global and regional function

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Left Ventricular Function – why should we measure and what should we measure?

Systolic function is a substantial determinant of overall hemodynamics and organ function. In the last 100 years we therefore have undertaken great efforts to develop diagnostic tools for imaging heart chambers and cardiac function.
Echocardiography has been proved as a very promising method to qualify and quantify cardiac structures and function. However, evaluation of cardiac function has been shown to be challenging over the last 40 years. Reasons for that were limitations in imaging techniques (from one-dimensional to two-dimensional to three-dimensional), limitations of the shape of the ventricles, and of the complex physiology of blood-circulation with continuous interactions between cardiac performance and loading conditions of pulmonary and systemic circulation.
Over a long period ejection fraction has been the gold standard for quantifying (left-) ventricular systolic function, and ischemia-detection relied on the sensitive eye of the examiner and a qualitative assessment of regional myocardial wall motion.
But what about very re-shaped ventricles especially with regional abnormalities, changes in loading conditions, or an exact differentiation between ischemic wall motion and different reasons (e.g. LBBB) for wall motion abnormalities? What about significant numbers for global and regional function, what about an exact mapping of the regional distribution of regional wall motion to suggest on coronary perfusion deficits?
The needs are well defined – thus, do we already know the answer on: which modalities, which parameters, which limitations? Yes, we do.

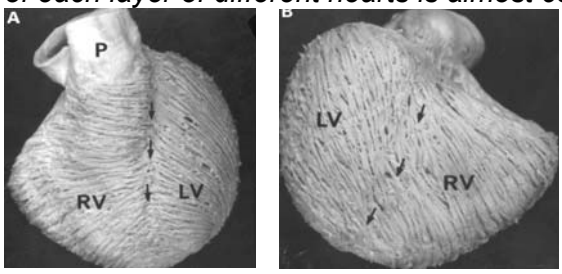
How to quantify Global and Regional Systolic Function?

At first it should be stated that if one relies on the clinical value of the qualitative and quantitative assessment of systolic function it is well studied that an experienced examiner can estimate left ventricular systolic function by visual assessment as good as he can measure it by conventional modalities. McGowan et al[1] showed that LV ejection fraction as estimated by Simpson's rule, wall motion index (WMI), and subjective visual assessment - compared with radionuclide or contrast ventriculography - is neither significantly under- nor overestimated by one of the three methods.
For a discussion on current techniques and methods in assessment of systolic function we have to make a step back to the anatomical and physiological basics to discuss right ventricle (RV) and left ventricle (LV) anatomy and their specific contraction mode.

Myocardial Architecture of the Ventricles[2]

In normal hearts, the ventricular mass is composed of three layers of muscle fibres: superficial (subepicardial), middle, and deep (subendocardial). The three layers can be identified by a different orientation of the muscle fibres. Whereas in the left ventricle all three layers are present the middle layer is missing in the right ventricle.
The superficial layer of the heart (Fig 1, A, B) consists of muscle fibres running from base of the heart to the apex in a oblique manner crossing anterior and posterior interventricular groove. The fibres in the superficial layer of the right ventricle were arranged more circumferentially than in the left ventricle.
The middle layer in left ventricles (Fig 2, A, C) of normal hearts contains circumferential orientated myocardial fibres. This layer is thickest at the equator, thinning out towards both the basal and apical with a small aperture at the apical region through which the superficial muscle fibres invaginate to become subendocardial and a large oval aperture in the basal area. No proper middle layer can be defined in the normal right ventricle.
The deep (subendocardial) layer (Fig. 3, E) is composed of longitudinal arranged fibres which pass through the vortices toward the papillary muscles, to the atrioventricular orifices and the arterial orifices, and to the ventricular septum.

Compared to the total myocardial mass the middle layer takes 53-59%, while the superficial layer occupies 25%, and the rest being the deep layer. The relative thickness of each layer of different hearts is almost constant.



Torrent-Guasp[3] and his group showed from studies on dissected hearts that the ventricular myocardium can successfully be unrolled into a single muscular myocardial band. The band extends from the pulmonary artery to the aorta and in its middle portion suffers a 180° twist (Fig. 3).

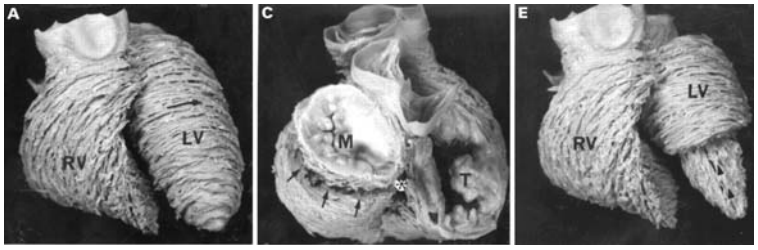


Fig. 1: From „Sanchez-Quintana – Myocardial architecture[2]“
A,B: Subepicardial layer

Fig 2 (second line): A,C: middle layer, E: subendocardial

Although global systolic function (fractional shortening, ejection fraction, cardiac output) does not change significantly with increasing age[4], we can observe a change in the relative amount the different layers contribute to LV-contraction. In young adults contraction is performed with longitudinal muscle fibres. With increasing age the relative amount of longitudinal contraction decreases (up to 20%) accompanied by an increase in circumferential contraction (up to 18%). The changes can be found irrespective to LV-wall thickness, heart rate or sex[5].

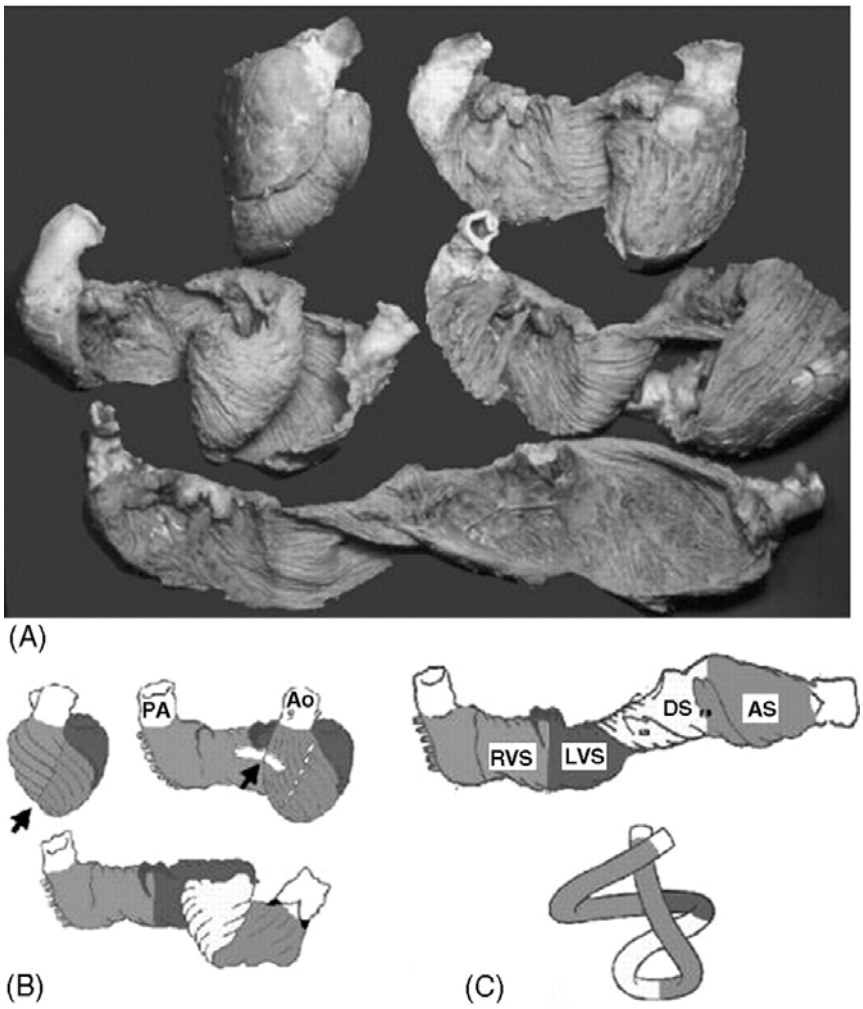


Fig. 3. Unwrapping the heart to a single muscle band from the beginning at the Pulmonary Artery (PA) to the end at the Aorta (AO)[3]

Influences of the Myocardial Architecture on Contraction Pattern

Left Ventricle

Differently orientated muscle fibres generate the global contraction of the left ventricle in a very complex pattern. Of course it is possible to focus on one major direction (e.g. measuring longitudinal function). However, the contraction of each of the 3 layers of the muscle band generates a deformation vector in either longitudinal, radial or circumferential direction. Playing together, deformation during systole occurs as a counterclockwise rotation of the LV-apex (as viewed from the apex), whereas the base rotates clockwise, creating a torsional deformation originating in the dynamic interaction of oppositely wound epicardial and endocardial myocardial fiber helices (Fig. 4)[6,7].

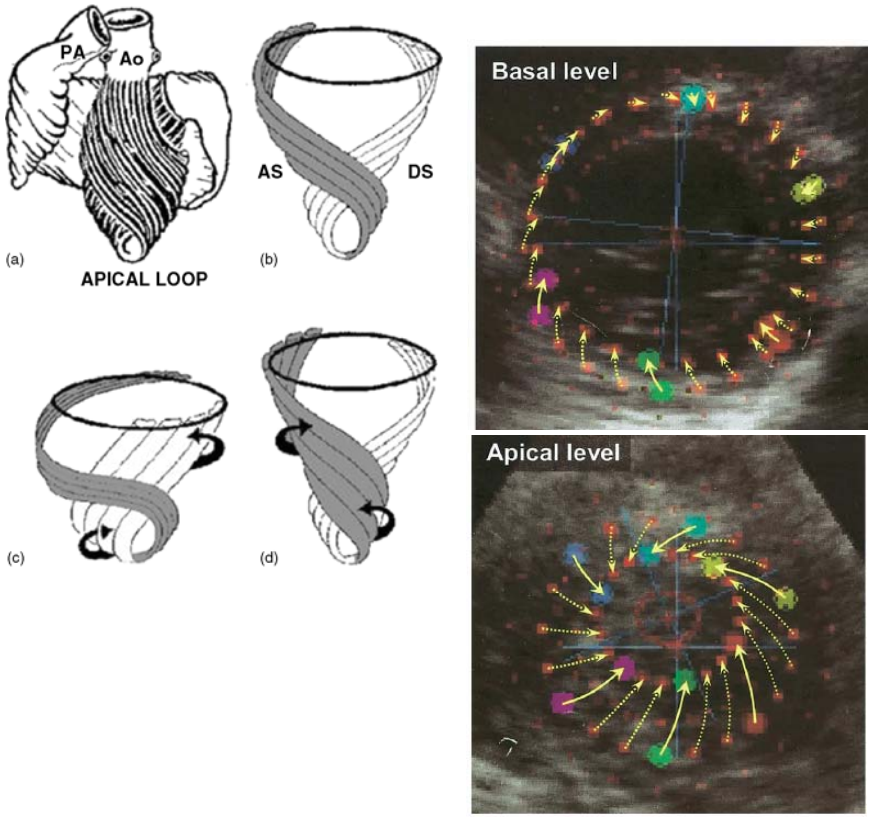


Fig. 4: LV-torsion during systole

The overall motion is a wringing of the LV and shortening in the longitudinal direction. (Fig. 5)

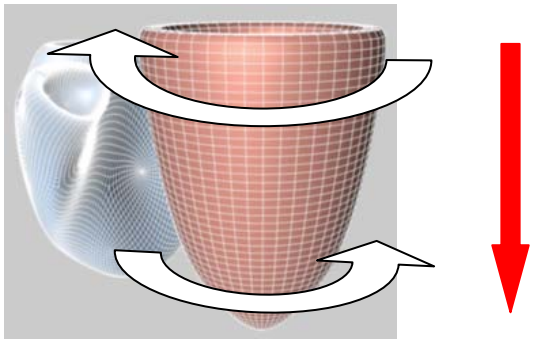


Fig. 5. Clockwise and counter-clockwise rotation and overall shortening in longitudinal direction

Right Ventricle

The right ventricle (RV) has a much more complex conduction and contraction pattern[8]. From a functional point of view the RV can be divided into 3 chambers: the inflow tract, the trabecular (apical) portion and the right ventricular outflow tract (RVOT). For a

regular contraction during systole the 3 chambers have to contract in a serial manner: 1. inflow tract, 2. trabecular portion, 3. RVOT. (Fig 6,7).

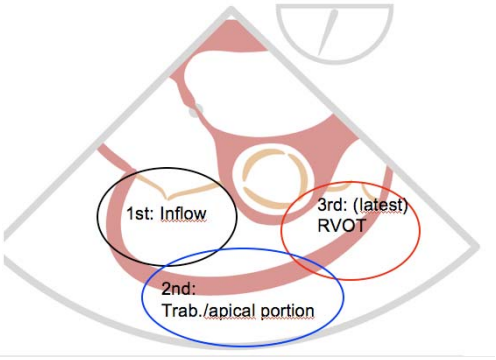


Fig. 6

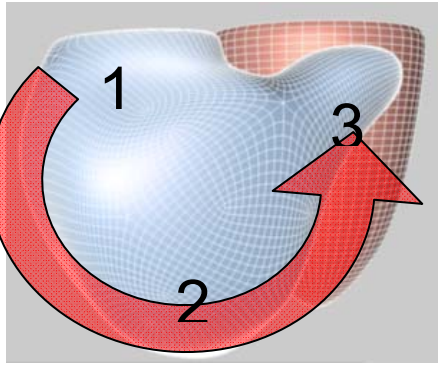


Fig 7

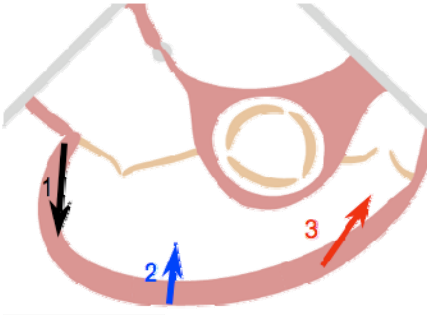


Fig. 8

Fig. 7, 8: Sequence of RV-contraction. Wringing motion from the base to RVOT

Defining motion vectors of the main directions of contraction, the inflow tract contraction follows a longitudinal direction, whereas the apical portion contracts circumferential and thus radial, and the RVOT in an oblique manner. (Fig 8)

Tissue Doppler or „a First Understanding of Myocardial Motion“

Tissue Doppler imaging was the first powerful tool with major contributions to parametric imaging and quantification of myocardial deformation. By measuring myocardial deformation parameters – velocity, strain, strain-rate – it was now possible to quantify myocardial motion or to sensitively detect myocardial events such as myocardial ischemia or infarction by characteristic changing waveforms.

Peak systolic velocity, peak systolic strain and peak systolic strain rate are measures of global and regional systolic function. (Fig. 9, 10, 11).

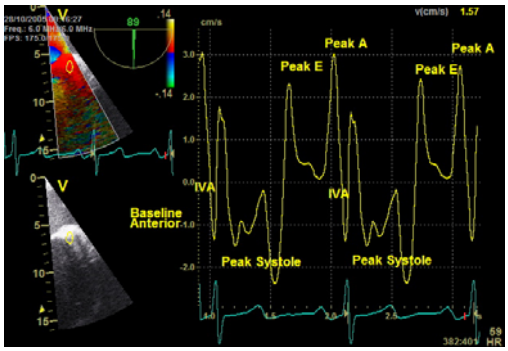


Fig. 9: velocity curve – Peak systolic velocity

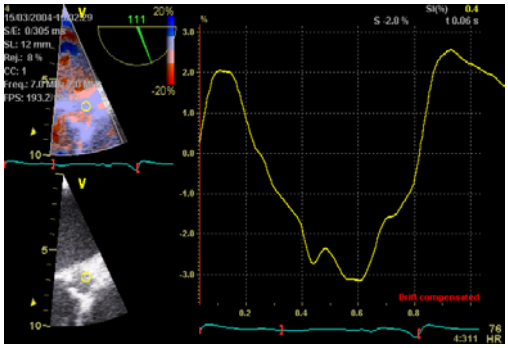


Fig. 10: strain curve – Peak systolic strain

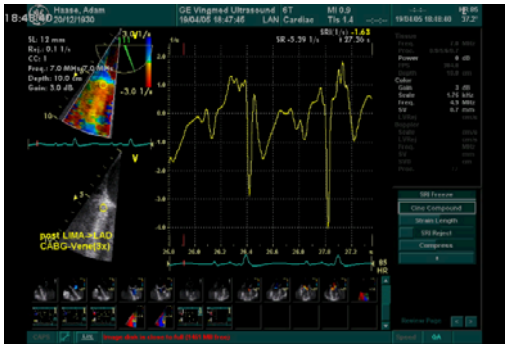


Fig. 11: Strain rate curve – Peak systolic strain rate

Weidemann et al[9] looked on global and regional contractility and how changes in contractility can be quantified by myocardial velocity, strain and strain rate and found those parameters to be sensitive for changes in contractility (Fig. 12).

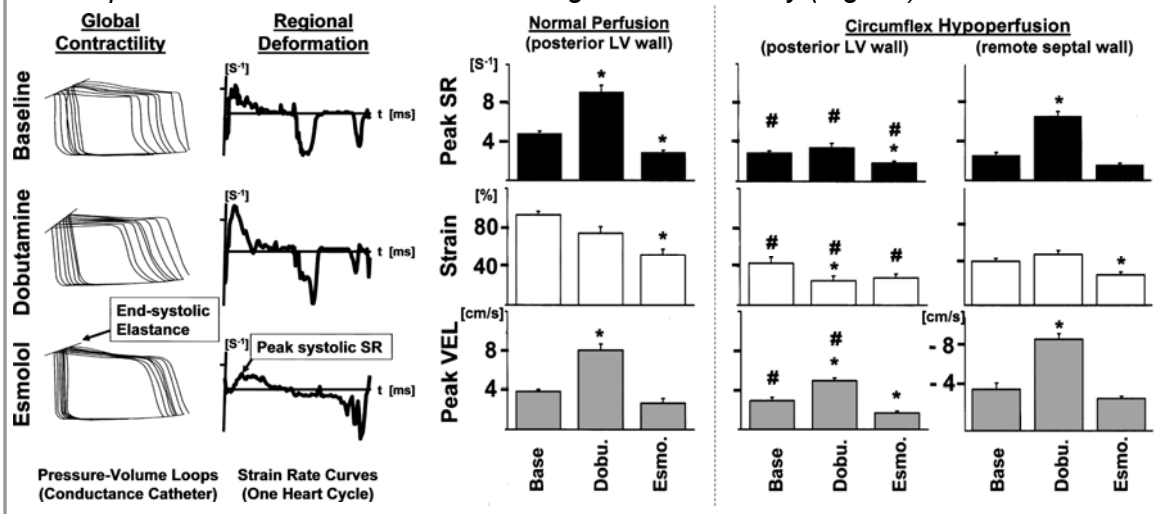
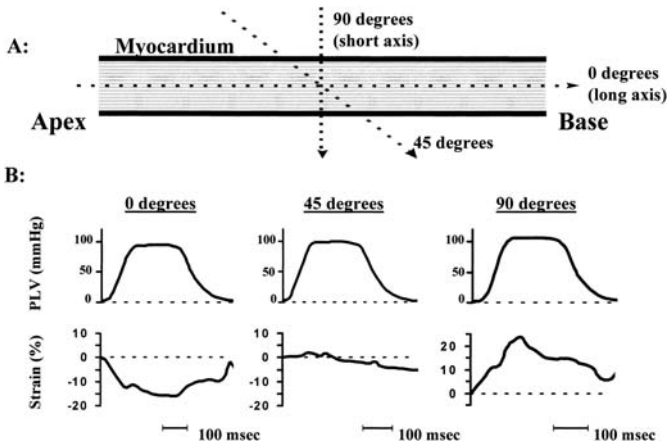


Fig. 12: peak velocity, strain and strain rate: changes due to changes in contractility (dobutamine, esmolol)[17].

Limitations of Tissue Doppler imaging (TDI)

A significant limitation of tissue Doppler imaging is the angle dependency as the same with all other Doppler methods. Urheim et al[10] demonstrated that strain and strain rate curves and values may change from negative to positive dependent on the angle between Doppler beam and the direction of myocardial deformation.(Fig. 13).



Understanding this limitation a correct orientation of the echo beam is critical for the application of this imaging modality. Especially for TEE a proper alignment of the wall in its longitudinal motion direction prior to measurements can sometimes be challenging. Moreover measures of the apical segments will always display incorrect values due to a significant angle of the round shaped apical wall.

Fig. 13: Angle dependency of strain rate – changing from negative to positive values with increasing angles.

Speckle-tracking and Two-dimensional Strain (2D-strain)

Although Tissue Doppler imaging is a powerful tool with major contributions to the quantification of myocardial deformation some limitations (angle dependence) are not easily to overcome.

Therefore significant efforts have been undertaken to extract myocardial tissue information from the 2D-image. Indeed complex analysis of the speckles (the grey-scale information of a myocardial region with its specific pattern) within the myocardium and its motion vectors during the cardiac cycle can now be analyzed to obtain velocity, strain and strain rate.

2D strain - the most recent development – is derived from a standard 2D echo scan and maybe the most versatile method to measure and display myocardial function.

2D-strain (2DS) - the Principle, Measurements and Displays

The basic principle is to determine myocardial wall motion from the movement of specific speckles of the grey-scale image from the myocardial walls.

The grey-scale image of the myocardium consists of speckles of different brightness and shape. By tracing these speckles in their movement throughout the cardiac cycle they can be analyzed to derive their direction and velocity.

Current 2D-strain techniques do not trace specific speckles but regions of myocardial tissue of the size of 20 x 20 pixels that generate a specific pattern of speckles. Using cross-correlation this characteristic pattern can be traced frame by frame throughout a major part of the cycle.

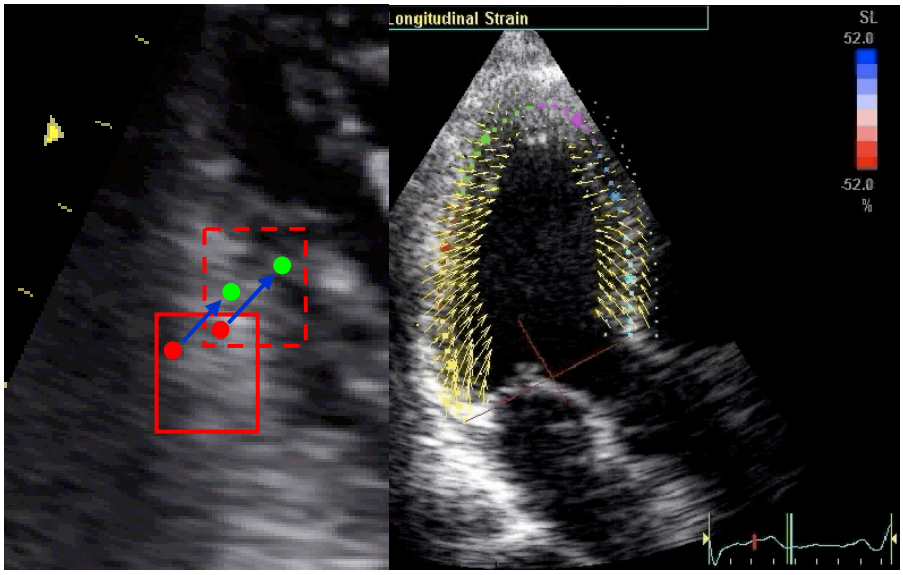


Fig. 14: Speckle tracking and vectorized imaging

By analyzing direction, shift over time (velocity) and spatial relationship between neighboured regions/speckles myocardial deformation can be quantified. (Fig. 14)

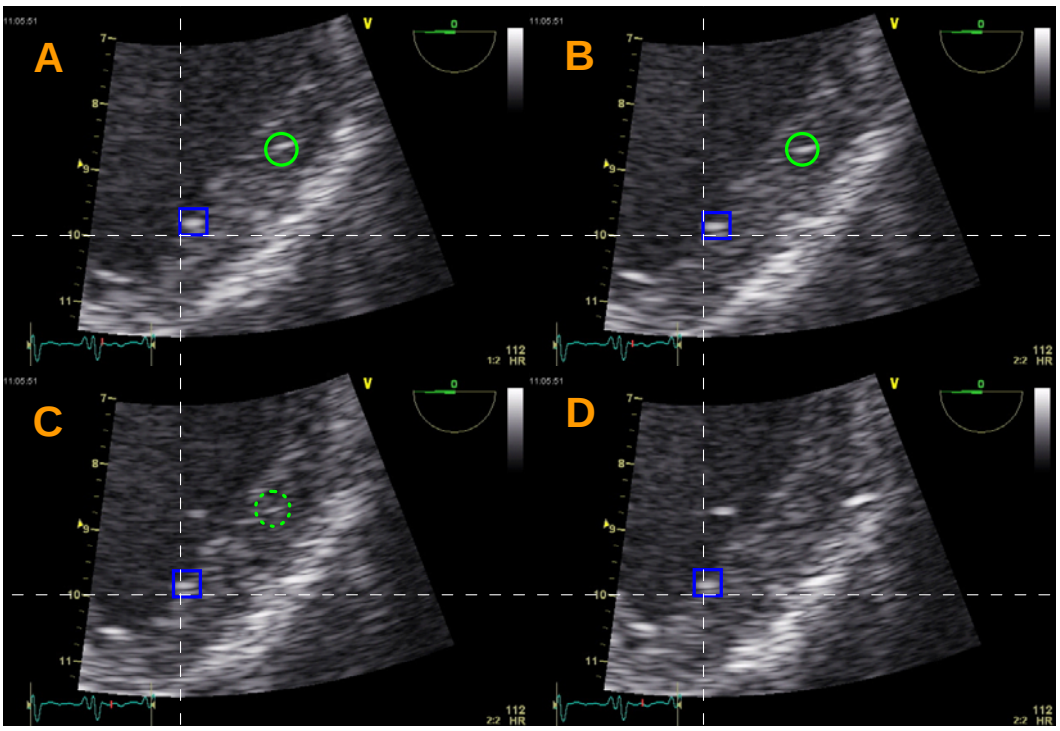


Fig.15: Speckle analysis: speckle tracking throughout the heart cycle

The tracing of characteristic tissue pattern is shown in figure 15. The pattern defined by the blue square can be traced throughout a major part of the systole, moving slightly down and leftward, whereas the pattern surrounded by the green circle vanishes in C and has disappeared in D.
Note that some tissue areas may move out the scan plane and need then to be replaced by their neighbour pattern in order to continuously determine the velocity of all the myocardial tissue on the scan-plane.

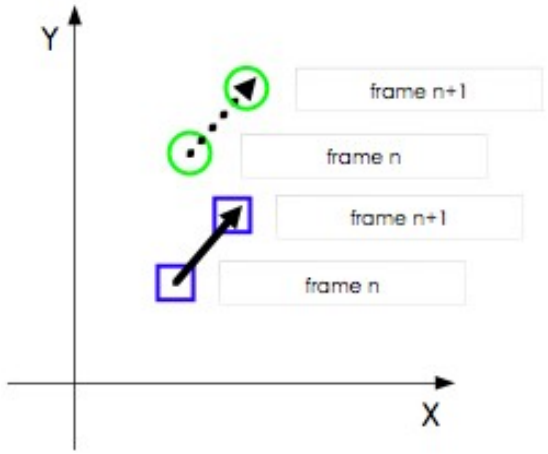


Fig. 16 Speckle tracking: identical velocities

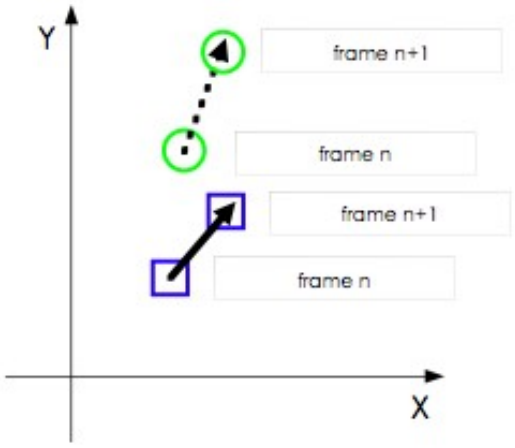


Fig. 17 Speckle tracking: different velocities

The velocities of neighbour tissue areas ("speckles") are used to determine the movement of tissue area relative to each other. In fig. 16 both tissue areas are moving in the same direction with the same velocity, thus the distance between these areas remained unchanged and the myocardium between them is not deformed. In fig. 17 both tissue areas are moving in different directions and with different velocities, resulting in an increasing distance between these areas, means the myocardium is distending. Deformation is the net measure of the tissue movement vectors. Fig. 18 shows deformation of a tissue area between diastole and systole. The deformation shown is related to radial thickening, circumferential shortening and slight twisting.

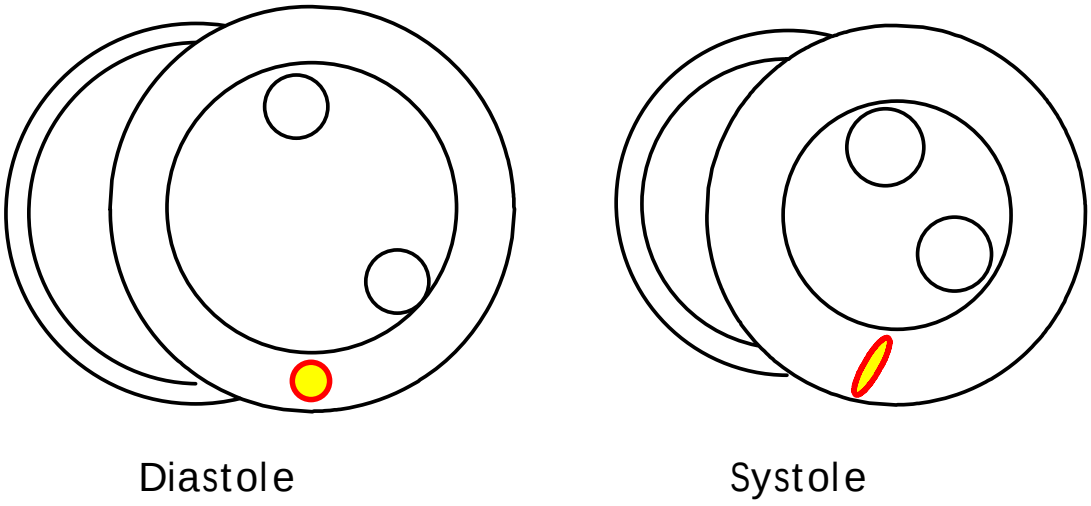


Fig. 18: Tissue deformation: a round tissue area during diastole becomes an ellipse during systole.

2D - Strain: the Measures

Velocity (V):
Velocity is the original measurement and is given in mm.sec-1.

Strain-rate (SR):
Strain rate is determined as SR = ΔV/ ΔL, where L is the initial distance between the given tissue areas and is given in units.sec-1. SR could also be defined as the speed of

deformation. This myocardial deformation parameter requires a high frame rate and is rarely used for interpretation because there are many artefacts on the graphs.

Strain (S):
Strain is obtained by integration of SR over time and is given as units (%). S could also be defined as the amount of deformation.

Displacement (D):
Displacement is obtained by relating deformation to some resting area and is given in mm. The resting area for longitudinal scans is the apex and for radial evaluation the centre of the contraction on the TG short axis view.

Rotation, Rotation-rate and Torsion:
Rotation can be determined from TG short axis views and is given in degrees. The LV-myocardium rotates clockwise in basal and counter-clockwise in the apical segments. Rotation-rate is the temporal derivative of the rotation means the speed of rotation and is given in degrees.sec-1. (Fig. 19, 20)

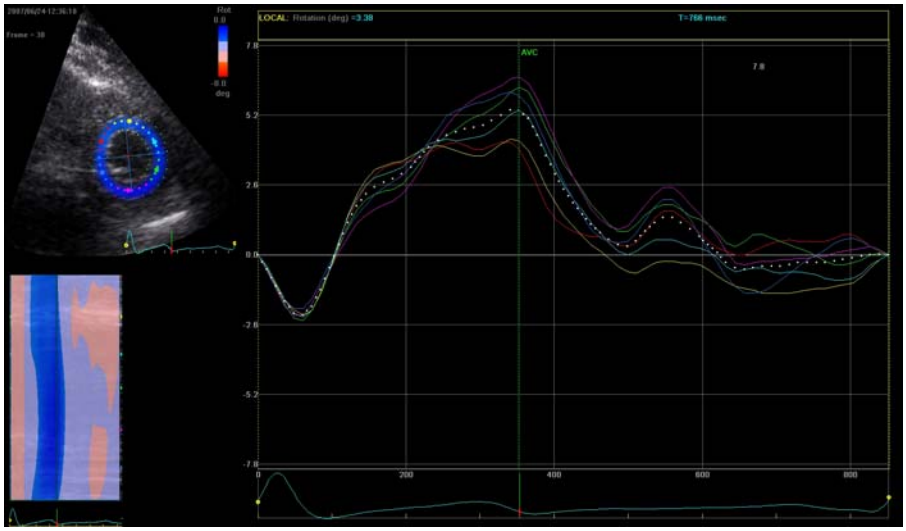


Fig. 19

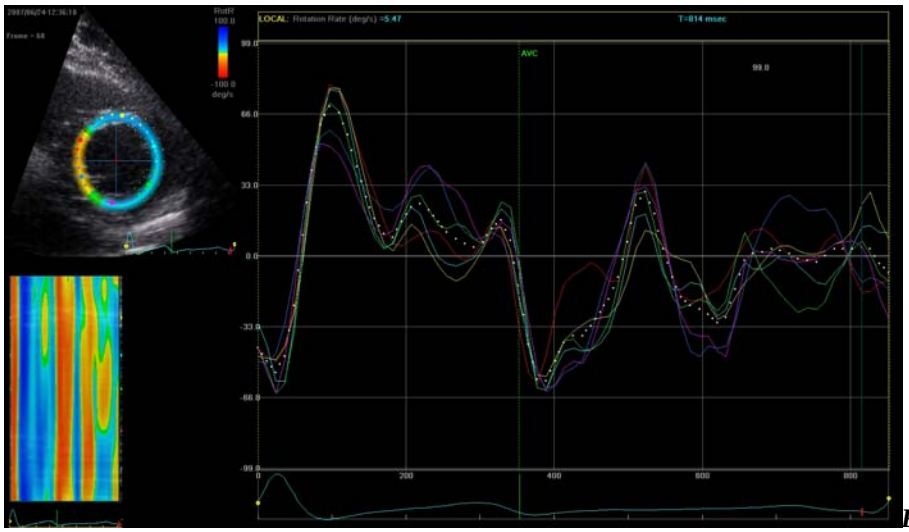


Fig. 20

By analyzing basal and apical SAX-views, torsion is the difference between apical and basal rotation and is given in degrees.sec-1[12]. (Fig. 21)

Indices:
In general the same indices as known from Tissue Doppler Imaging can be derived:
Peak (longitudinal) velocity (S, E, A)
Peak (longitudinal) strain (systolic, postsystolic)
Peak (longitudinal) strain rate (S, E, A)

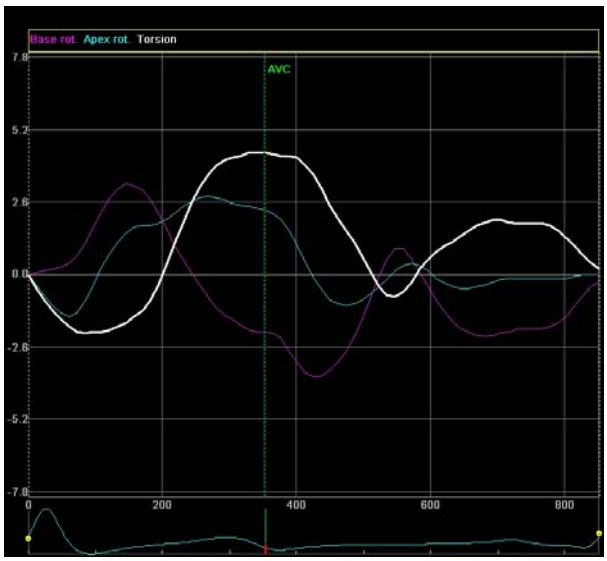


Fig. 21

Peak (longitudinal) displacement
Time to peak (longitudinal) strain
Additionally some indices have been developed on base of the advantages/less limitations respectively capabilities of the new technology. The huge advantage compared with TDI is the lack of angle dependency. Therefore the analysis of all 16 (17) segments of the left ventricle is now possible regardless of the shape of the LV-apex. On base of the angle-independence a complete analysis of LV in short-axis views is possible. Therefore we can now analyze radial and circumferential motion components.

- *Radial/circumferential peak velocity*
- *Radial/circumferential peak strain*
- *Radial/circumferential peak strain-rate*

By measuring the degree of rotation of the LV during heart cycle following indices can be derived:

- *Peak rotation*
- *Peak rotation-rate*

and from the difference of rotation values derived from apical and basal SAX views:

- *Peak torsion*

2D-Strain – Advances and Limitations

Since 2D-strain is no longer angle dependent this technology ideally provides – even by Transesophageal Echocardiography (TEE) - the opportunity to analyze the whole length of the ventricular walls including the apical region. Especially the apical region with a more or less round wall or a dilated cardiomyopathic left ventricle is challenging to analyze as it is impossible to extract longitudinal or radial shortening alone. Measures of myocardial deformation have always been sum of individual components of directions of the moving heart. (Fig. 19)

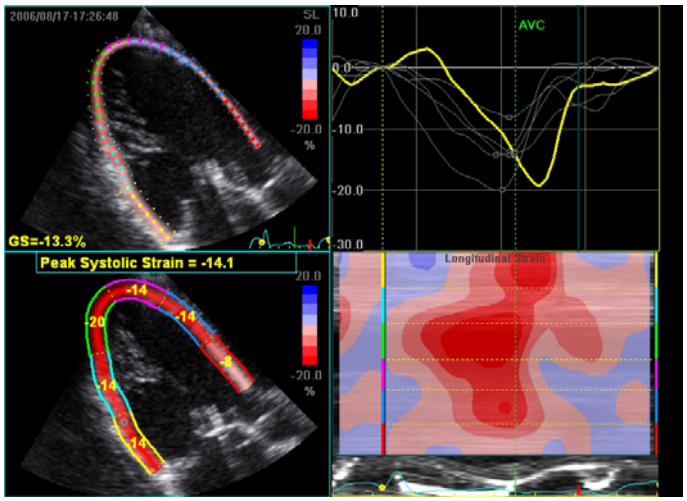


Fig. 19: 2D-strain analysis regardless of the angle between direction of deformation and the scanning plane.

Currently the major limitation of the technique is obviously the temporal resolution and its contribution to the delineation of speckles. When frame rate goes up speckles move a very short distance from frame A to frame B to C. Therefore it is hard to analyze direction and distance of the individual speckles. Even from the 2D-image it can easily be recognized: with upcoming frame rate the myocardium and its grey-scale pattern smoothens and contours and pattern of the speckles get lost. In contrast, a low frame-rate helps to distinguish myocardial structure but information about brief events in the cardiac cycle gets lost. Taking that into account frame-rates of 60-100/s are ideal to analyze.

2D-Strain and Assessment of Global Left Ventricular Systolic Function

*To assess systolic function 2D-strain provides a powerful clinical tool – **global longitudinal peak systolic strain (GLPSS)**. Therefore, both walls of each of the three midoesophageal longitudinal planes – ME 4Ch, ME 2Ch, and ME LAX – are speckle-tracked. Peak strain values are displayed as average-values from both walls of each plane. Global peak systolic strain is obtained by again averaging peak strain values from each of the three planes. The obtained global strain value is an overall measure of left ventricular systolic function[11].*

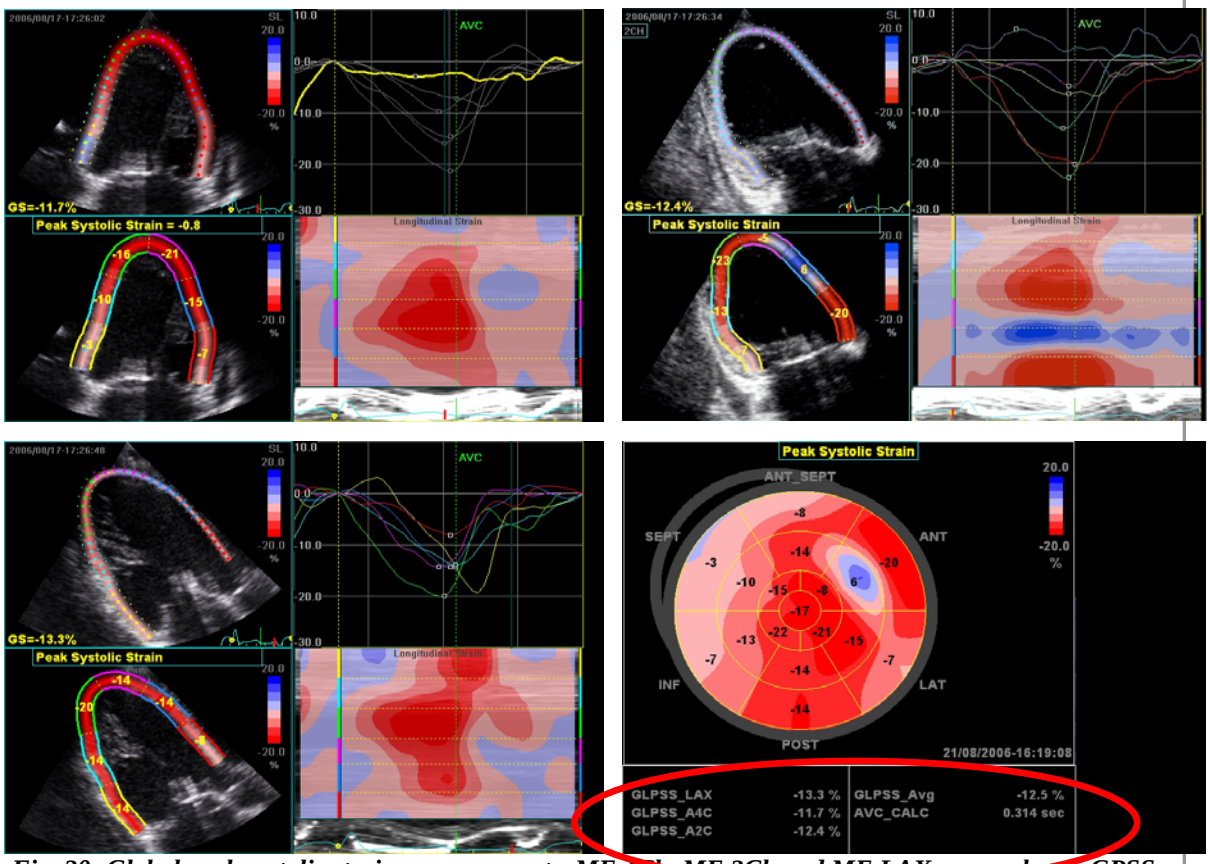


Fig. 20: Global peak systolic strain measurements. ME 4Ch, ME 2Ch and ME LAX are analyzed. GPSS values are obtained from averaging values of all 6 walls of the 3 planes.

Additionally distribution of peak systolic strain is visualized on a colorized bull´s eye that provides contribution to global strain and distribution of regional peak systolic strain. Regional deformation abnormalities are well graphically displayed on this systolic deformation map. (Fig. 20, right bottom; 21)

Right ventricular systolic function is much more difficult due to the complex three-dimensional shape of the right ventricle. However, 2D-strain allows - at least from the ME4Ch – to measure global peak systolic longitudinal strain from the U- shaped length of right ventricular free lateral wall and septum. (Fig. 22)

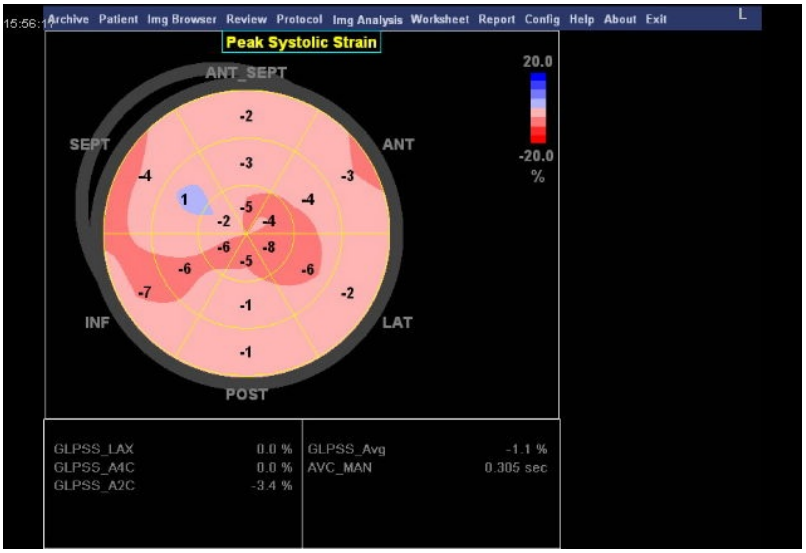


Fig 21: Bull´s eye-view example from a patient with dilative CMP

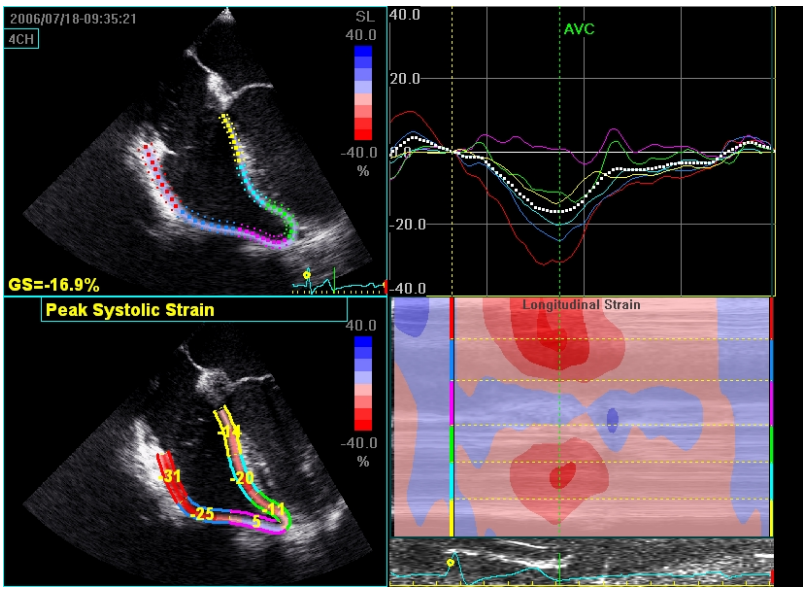


Fig. 22: Longitudinal peak global systolic strain of the right ventricle.

2D-Strain and Assessment of Regional LV-Systolic Function

Regional ventricular function results from normal working myocardial wall that again needs sufficient blood supply to perform with adequate contraction. Over the years wall motion abnormalities due to myocardial ischemia or infarction have only been detected by qualitative visual assessment of the experienced examiner.
Diagnosis of hypokinesia, akinesia, dyskinesia underlied the subjective interpretation and sensitive investigation of the examiner with a huge inter-observer variability. Small changes in regional function on one hand or discrimination between systolic and postsystolic contraction on the other is often challenging.
Since 2D-strain technique has been evolved almost all of our requirements on sensitivity and specificity in detection of ischemia can be fulfilled:

- *Robustness and small inter-observer variability*
- *Analysis of all 16 (17) LV-segments regardless of shapes or angles*
- *Parametric imaging with quantification of regional wall motion*
- *Detection of postsystolic events*
- *Regional distribution mapping*

Regional function analysis is more or less based on strain curve analysis. Interpretation of the curves follows two principles:

- *Normokinesia, hypokinesia or akinesia are defined through their peak strain value. Analysing longitudinal motion contraction is presented by negative values.*

Dyskinesia is displayed through curves with either positive values or no clear direction.

- *Timing is critical for a proper curve interpretation. Normal systolic contraction ends with aortic valve closure (AVC) – relaxation starts with mitral valve opening (MVO) and is displayed through a down-sloping strain curve. During ischemia contraction is delayed and prolonged beyond AVC with a characteristic postsystolic peak. Therefore detection of postsystolic shortening (PSS) is a powerful tool to detect ongoing ischemia. PSS can either be displayed on strain-curves, from anatomical M-mode or in a colorized bull´s eye-view with its regional distribution.*

Postsystolic index (PSI): *To significantly identify acute ischemic myocardium Kukulsi et al[13] described the postsystolic index (PSI) which relates systolic to postsystolic strain:*

*PSI = 100 * (Peak Systolic Strain -Post Systolic Strain) / Post Systolic Strain*

With a new feature PSI is also displayed in bull´s eye-view for a regional distribution mapping of acutely ischemic myocardium. (Fig.23,24)

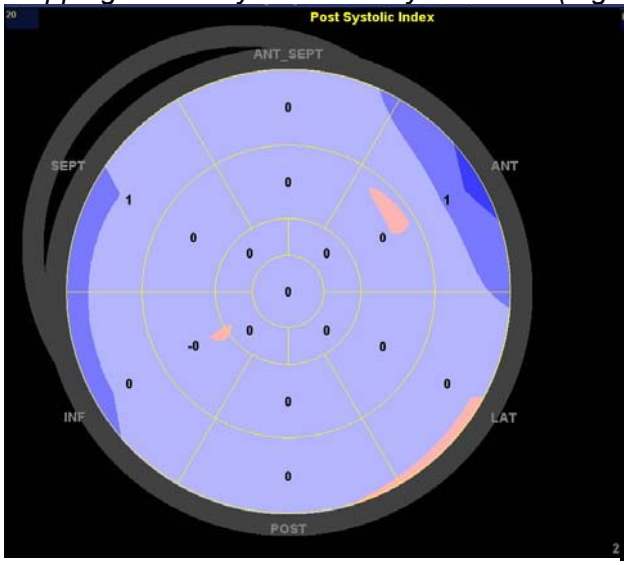


Fig. 23: PSI – normal BE

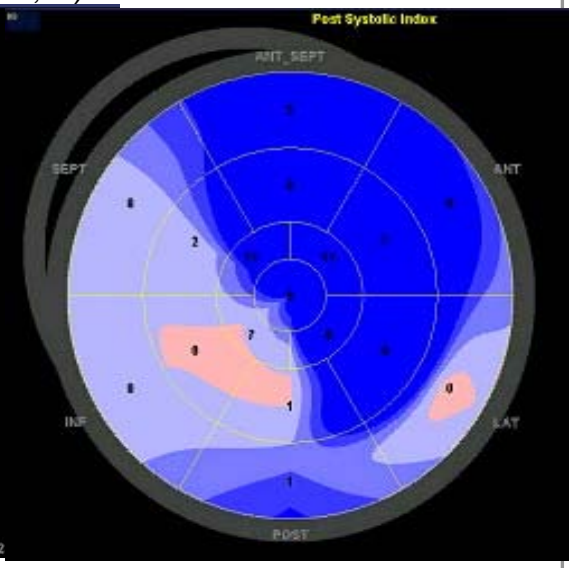


Fig. 24: PSI –BE from ischemia of the LAD

Regional Distribution Mapping and Coronary Artery Disease

By displaying regional differences in systolic contraction or occurring postsystolic shortening in a bull´s eye-view one can easily conclude from the regional distribution perfusion deficits on coronary perfusion. (Fig. 25,26)

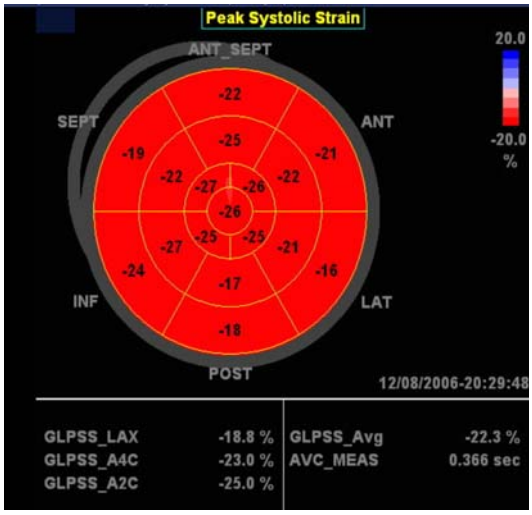


Fig. 25: Normal bull´s eye

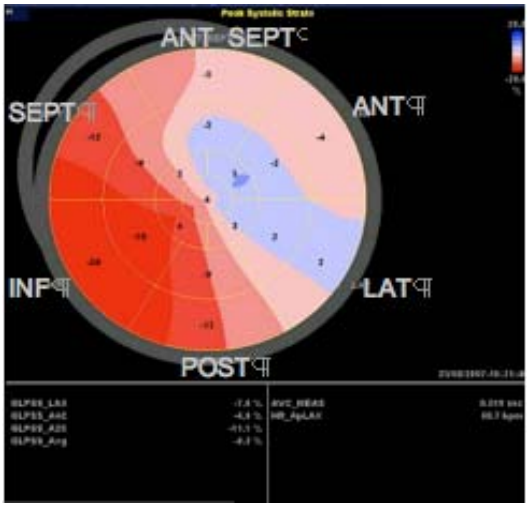


Fig. 26: Ischemia of the LAD

2D-Strain: the Clinical Applications

The clinical application of 2D–Strain echocardiography will gain more and more importance in the following fields:

- Detection of acute myocardial infarction
- Assessment of revascularisation after CABG surgery
- Quantification of dyssynchrony and optimization of settings
- Monitoring of pharmacological interventions
- Quantification of diastolic and systolic effects as a result of drugs and interventions
- Measurement and optimization of LV/RV interaction.

Future aspects

During the next years advanced techniques of tissue tracking and analysis of myocardial deformation on base of 2D-information will evolve. This will lead to the feasibility of acquisition and analysis of high frame-rate images, which again will help to analyze brief events in the cardiac cycle.
The other challenge is to overcome the limitation to analyze only longitudinal or radial or circumferential myocardial deformation. Ventricular contraction is a complex three-dimensional twist of clockwise and counter-clockwise rotation of base and apex leading to net longitudinal shortening. To analyze this ventricular contraction mode there is the substantial need of three-dimensional strain analysis.
While the left ventricle is relatively "easy-shaped" and therefore easily described on base of algorithms the right ventricle with its complex three-dimensional anatomy and functionality is really challenging when tried to put into numbers of systolic contraction. Therefore, it seems that merging parametric three-dimensional imaging and two-dimensional tissue tracking techniques may solve some problems.

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Real-time L3D TOE

Jan Hultman, Stockhom, Sweden

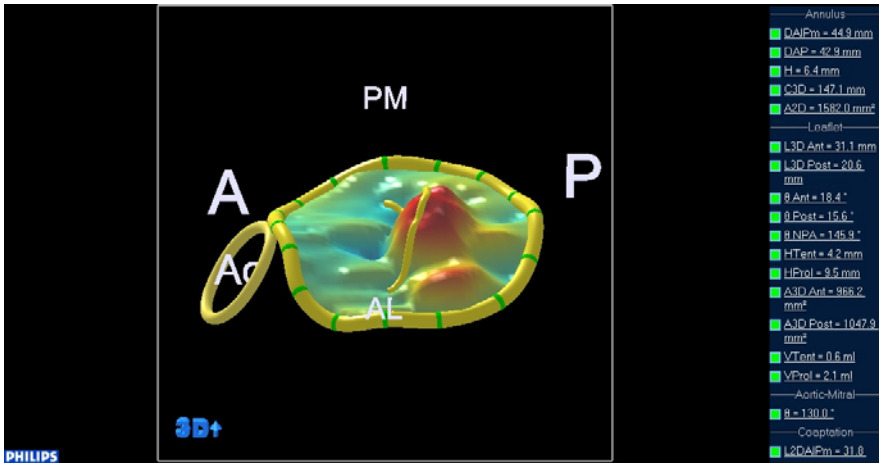
3D has been around for several years but hampered by big probes, poor images, time-consuming and off line assessments. Mitral valve anatomy and function in repair has been a focus of interest in comparing 2D and 3D transthoracic echocardiography (TTE) and 2D and 3D TOE and the benefits of 3D imaging and especially the 3D TOE with a more precise diagnosis are reported (1,2). However, the clinical use in the OR and ICU requires easily accessible techniques and methods of analysis. An important step forward in this process is the introduction of real time 3D TOE by Philips. Because the required investment in money and knowledge it raises questions as how does it contribute or supplement the regular real time 2D imaging, is it user-friendly, and how is it received among users.

My experience is limited to a couple of weeks usage in the open heart surgery setting and my conclusions should be regarded as based on a great experience in regular 2D TOE imaging. The reason that I emphasize this fact is that the benefit from real time TOE imaging is dependent on good knowledge in basic 2D imaging. As with the system from Philips the real time image uses two orthogonal 2D images to set the real time 3D view. Consequently it requires good alignment of two orthogonal 2D planes.

Nevertheless, 3D imaging has come to stay. It adds information on both structural and functional properties in the heart and in the lecture I will focus on two topics namely the mitral valve apparatus in mitral regurgitation (MR) and the left ventricle (LV).

The repair of the regurgitant mitral valve

In regular 2D TOE imaging the user has to construct a 3D image in the mind. Therefore it could be difficult to explain the anatomy and function to the cardiac surgeon. Because a detailed assessment is absolute crucial in mitral valve repair the lack of understanding or poor diagnosis is unacceptable. To my experience, most cardiac surgeons lack enough knowledge to question the report from the echocardiographer. However, with 3D TOE any cardiac surgeon skilled in mitral repair for MR can understand and assess the 3D image. Especially the more detailed morphology and function of the different leaflet scallops is appreciated (1,2). Also in the system from Philips there is mitral valve quantification software which adds important information (Fig. 1)



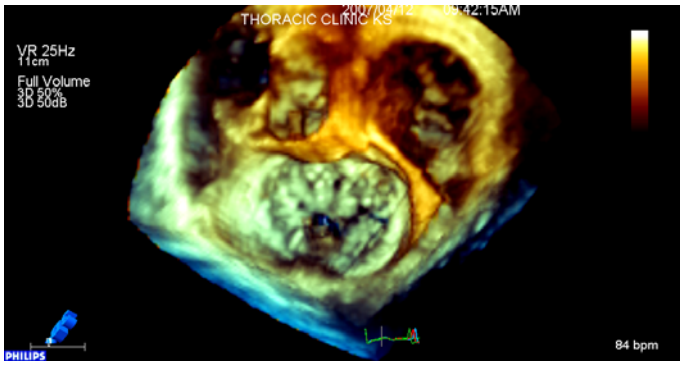
The saddle shape of the annulus is obvious and the clear prolaps (in red colour) of the p2 segment and also some billowing of the middle part of the p1 can be seen.

To the right numbers on leaflet

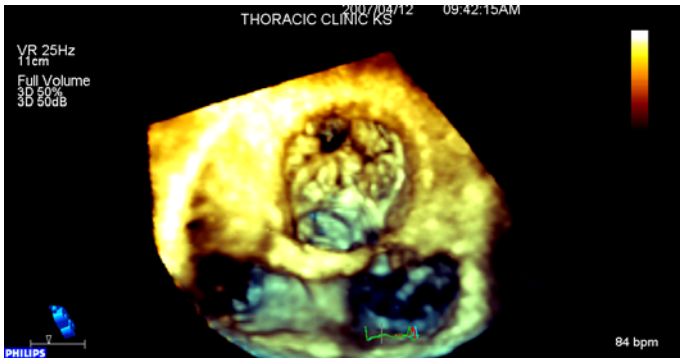
Figure 1
This analysis is performed using Q-lab software from a collected 3D full volume of the heart. A = anterior leaflet, P=posterior leaflet, PM=posteromedial commisure, AL=anterolateral commisure, Ao=aorta.

characteristics and how they are appear with regard to the mitral annulus are shown. The bulky appearance is also evident which actually is one reason for the great success rate in mitral valve repair. There is simply enough tissue for resections and together with the narrowing from the annuloplasty the valve is mostly sufficient after surgery.

Another added feature is the new colour maps which makes it easier to appreciate the depth in the image. Below is a typical example showing an isolated p2 prolaps seen from both the left atrium (LA) and the left ventricle (LV).

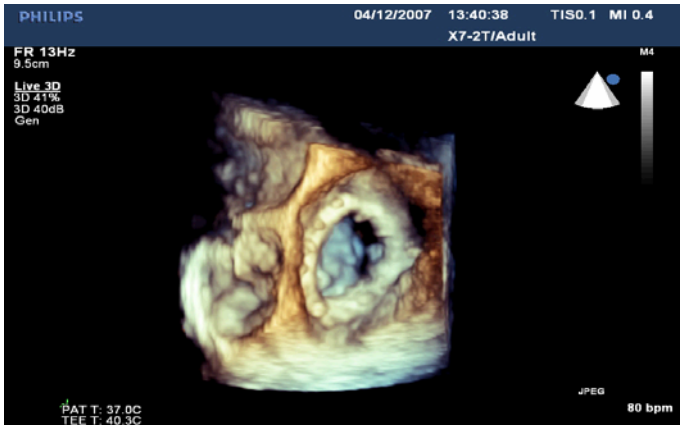


View from the LA.
The mitral valve is seen at the bottom of the picture and how it is view by the operating cardiac surgeon. Close structures are brown and distant blue. It shows a clear prolaps of the p2 scallop. The poor coaptation between the prolapsed p2 and the normal a2 scallop is obvious and there is also some lack of *coaptation between p3 and a3. The aortic valve is seen to the left and the pulmonary valve to the right in the upper part of the image*

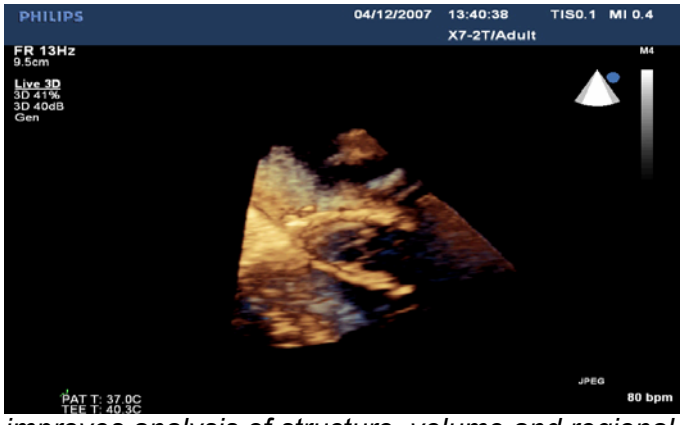


View from LV
To view the mitral valve from the LV we have rotated the same image. Now the posterior leaflet is depicted at the top of the image and the prolaps of the p2 scallop is confirmed.
Images after repair and annuloplasty are also shown.

View from the LA
After quadrangular resection of p2, p1 sutured to p3 and with the annuloplasty (a closed ring) the mitral valve has almost been changed to a monocusp.A little bulk of tissue after suture between p1 and p3 can be appreciated to the right and closed to the ring. The anterior leaflet covers almost the whole area.



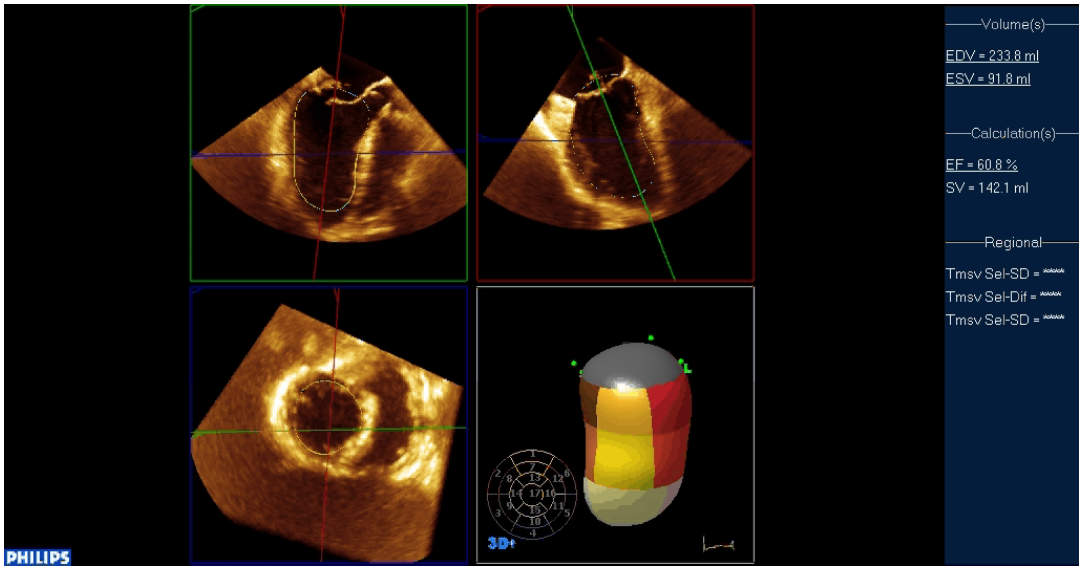
A modified ME 4CH view
The "monocusp" appearance is well seen.
Anatomic and functional study of the preoperative mitral regurgitation (MR) is essential in surgical repair. In the predominantly isolated p2 prolaps the benefit from 3D could be argued However, in the more complex cause for MR a 3D study can complete the diagnosis. Still further research is warranted to establish the true clinical relevance of 3D studies in MR.



The left ventricle

3D offers a great advantage in assessing LV characteristics. It improves analysis of structure, volume and regional function. Still the cine-loops have to

be analyzed with the Q-lab software and this is somewhat time-consuming. A typical layout after collecting a full 3D volume of the heart is shown in figure 6. To start the calculation the planes have to be adjusted to obtain the true long- and short axis. Thereafter numbers on volumes are at hand and a 17segment model is shown.



Top left is the ME LAX view, top right ME 2CH view, bottom left TG SAX view, and bottom right the 17 segment model with the bull's eye presentation.

With these new features assessment of LV morphology and function is improved. Earlier we had to make geometric assumptions for the LV but it is not required in 3D. Comparisons with the golden standard MRI techniques 3D echocardiography have shown accurate and global and regional assessments of LV size and function.(Marwick)

In summary

3D TOE is a promising tool in cardiac surgery. The most obvious benefit is the easier interpretation compared with 2D imaging. So far most studies have favoured 3D compared to 2D and 3D techniques in diagnosis of MR and LV function. Despite the real time 3D TOE feature the images demand a precise gain and compress settings for optimization. Further studies have to be undertaken to fully appreciate the clinical relevance.

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8. *J Am Coll Cardiol 2004;44:878-886*

Three dimensional assessment of the Mitral valve

Andreas Ziegler, Freiburg, Germany

Purpose

Transesophageal echocardiography (TEE) and more recently 3 dimensional transesophageal echocardiography (3D TEE) have found multiple applications in peri-operative care, including the assessment of the morphology and function of the Mitral valve. This presentation is intended to demonstrate the feasibility and efficacy of acquiring and displaying intra-operative 3D images with a TEE transducer in the short time window between the begin of anaesthesia and the engagement of the heart- lung machine. In comparison to 2 dimensional transesophageal echocardiography, additional information can be obtained which can add value to the surgical decision making process.

Methods

The clinical examples presented were acquired using an Acuson CV70 echocardiography system (Siemens, Erlangen) and analysed using a Mitral valve assessment software (TomTec Munich). The 3D volumes are obtained via the reconstruction of 60 to 70 ECG triggered 2D images acquired at known angles of rotation of the multi-plane TEE.

In addition to the assessment of Mitral valve prolapse, various applications as well as pit falls and artefacts are presented, including one particular case in which the speed and accuracy of the method enabled the correction of an unexpected complication during the fourth revision of a mitral valve replacement while the patient was still on the heart-lung machine.

Results

3D TEE is an efficient and effective method of imaging and diagnosing Mitral valve pathology in the very short time window available in the intra-operative environment Further, the acquisition of the required 3D volumes presents less of a challenge with the anesthetised and intubated patients when compared to the sedated and non-intubated patients in the Echo Lab.

Conclusion

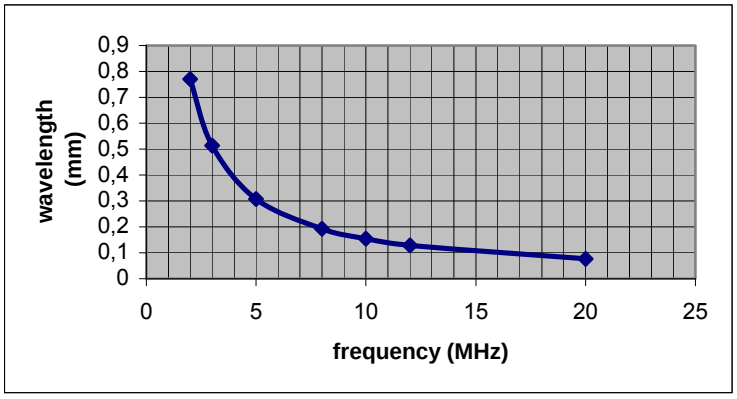
According to the opinion and experience of the author 3D TEE is an important development in the understanding of the broad range of Mitral valve. As the volume acquisition in the intra-operative environment has advantages over that in the Echo lab with respect to patient compliance and therefore image quality, this method should be available in every operating theatre where modern Mitral valve reconstruction is performed. It is clearly much more a “tool" than a "toy".

TTE physics, settings and controls

Frank Steensgaard-Hansen, Copenhagen, Denmark

Sound waves are mechanical vibrations characterised by their wavelength, amplitude (dB) and frequency or Hertz (Hz), which is the number of repetitions per second. Ultrasound imaging, in case echocardiography, uses sound waves at frequencies between 1 MHz and 20 MHz.

Wavelength is mathematically related to frequency and the propagation velocity of ultrasound, which is 1.540.000 mm/sec in human tissue.



$$\lambda = \frac{c}{f}$$

λ = bølgelængde
c = udbredelseshastighed
f = frekvens

The resolution in ultrasound imaging varies directly with the frequency and inversely with the wavelength. With increasing frequency and thus decreasing wavelength the spatial resolution is enhanced, but the depth of tissue penetration of the ultrasound beam decreases. With decreasing frequency the tissue penetration improves but the trade-off is a loss in resolution.

This is very important to remember in clinical imaging. Adjust the transducer frequency depending on the depth of the structures you like to study and the quality of the acoustic window.

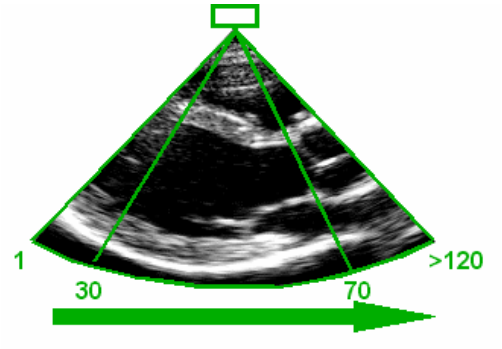
When ultrasound hits tissue interfaces with different acoustic impedance as a consequence of different tissue density, part of the ultrasound energy is reflected back to the transducer. The degree of reflection increases with increasing difference in tissue density.

The magnitude of reflected ultrasound received by the transducer depends upon the size, surface and density of the tissue interface and the angle between the ultrasound beam and the tissue interface.

The angle of incidence is very important. The best possible reflection of ultrasound to the transducer occurs when the ultrasound beam is perpendicular (90^{o}) to the tissue interface or in practice the cardiac structure of interest.

Phased-array transducers, which are the most common in clinical echocardiography, have a large number of ultrasound crystals that can be steered electronically without any moving parts. Each crystal generates an ultrasound wave and after a very brief period of transmission the same crystal receives the reflected ultrasound wave.

Regarding image information the time interval from ultrasound transmission to reception of reflected signals is proportional to the distance of the reflector from the transducer. The amplitude depends on the acoustic impedance of the reflecting structure and the angle of incidence.



In 2D echocardiography each ultrasound crystal forms a scan line in the tomographic slice. By increasing scan line density image quality is enhanced. The trade-off is a lower frame rate due to the increased number of scan lines.

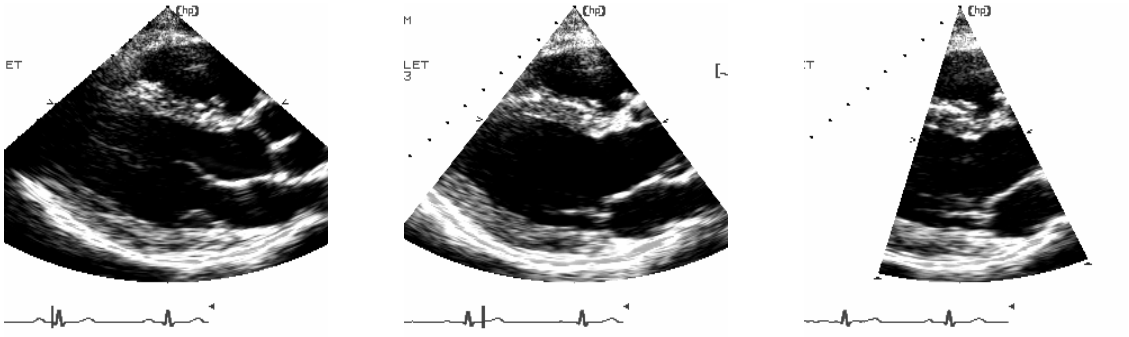
The resolution in an ultrasound image is defined by the ability to separate two closely positioned objects. In 2D echocardiography three types of resolution has to be considered:

- *Axial resolution: Along the length of the ultrasound beam. The smallest resolvable distance between two reflecting objects is 1 wavelength. Thus the axial resolution is enhanced with increasing transducer frequency.*
- *Lateral resolution: In the horizontal plane. It depends on several factors including transducer frequency, beam width and aperture of the transducer. At greater depth the beam width increases with a decrease in lateral resolution as the inevitable consequence.*
- *Elevational resolution: The thickness of the tomographic slice. It varies depending on width and focusing of the ultrasound beam but also transducer design. It varies typically between 3 and 10 mm depending on depth.*

Passing through tissue an ultrasound wave generates harmonics of the original or fundamental frequency. Broadband transducers can receive double the transmitted frequency thus creating second harmonic imaging in which the fundamental signals are filtered out.

Second harmonic imaging enhances the visibility and definition of structures like the endocardium and offers better signal-noise ratio. The trade-off is a reduction in resolution, but this is not much of a problem in the newest versions of second harmonic imaging. In general, second harmonic imaging is used default in adult TTE.

Most important 2D echocardiographic settings and controls in a logical sequence:
***Transducer**: select best suitable*
***Preset:** choose factory preset for TTE (adult or pediatric) or if preferred a customised TTE preset*
***Modality:** select 2D*
***Second harmonic imaging**: usually default in adult TTE*

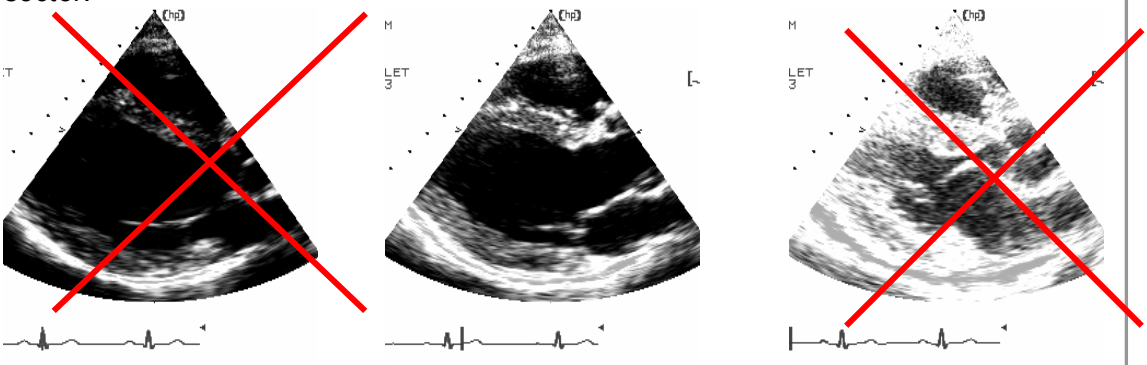


For each 2D projection optimize:

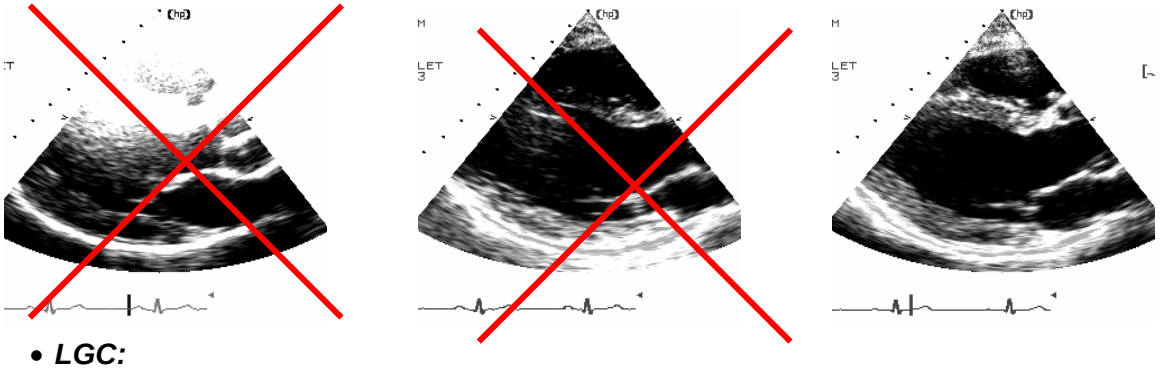
- ***Frequency:***
  increase for improved resolution, trade-off reduced depth of penetration
  decrease for improved depth of penetration and in case of poor visualisation and definition of structures, trade-off reduced resolution

- ***Depth***

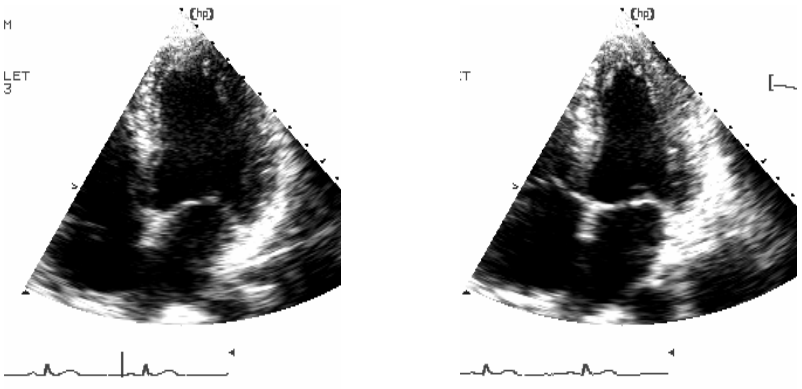
- ***Sector width (angle):***
  90^{o} provides best overview at the expense of image quality and frame rate for most standard recordings about 60^{o} is preferable between 30^{o} and 40^{o} for recordings in great detail of smaller structures (valves etc.)
- ***Focus:***
  repositions the acoustic depth of the focal zone
- ***Overall 2D gain:***
  adjusts the amplification of received signals. Generally, select a medium position about 50%. Then adjust regional gain (TGC and – if available – LGC) until gain is satisfactory in all areas of the 2D sector.



- ***TGC:***
  Time Gain Compensation. Each of the usually 8 TGC-sliders adjusts the amplification of returning signals at a specific acoustic depth.



- ***LGC:***
  Lateral Gain Control. Each of the usually 8 LGC-sliders adjusts the amplification of returning signals within a specific lateral area of the 2D sector. LGC is not available in all machines.



without LGC *with LGC*

How to make a TTE

Frank Steensgaard-Hansen, Copenhagen, Denmark

2D projections

If at all possible place the patient as shown in fig. 1.

Figure 1

Transducer locations in TTE are shown in fig. 2.

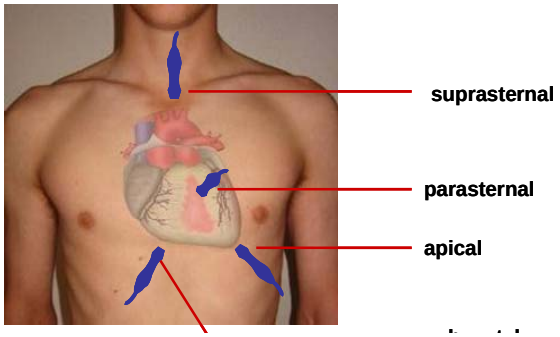


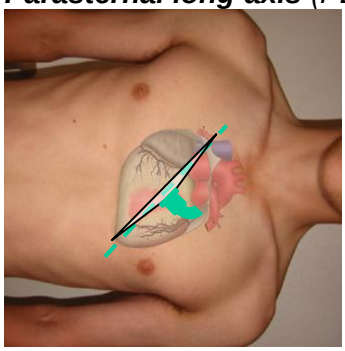
Figure 2

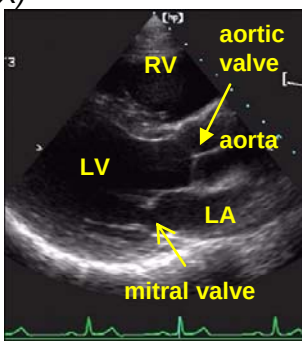
A complete TTE in the hands of a cardiologist is a very comprehensive and time consuming examination. For cardiothoracic anaesthesiologists the most relevant 2D projections are the following:

- *Parasternal long-axis (PLAX)*
- *Parasternal short-axis (SAX)*
- *Apical four-chamber view (AP4CH)*
- *Apical two-chamber view (AP2CH)*
- *Apical long-axis (APLAX)*
- *Subcostal or subxiphoid four-chamber view (SUBXIPH4CH)*
- *Subcostal or subxiphoid short-axis (SUBXIPHSAX)*
- *Vena cava inferior and right atrium (SUBXIPH VCI-RA)*

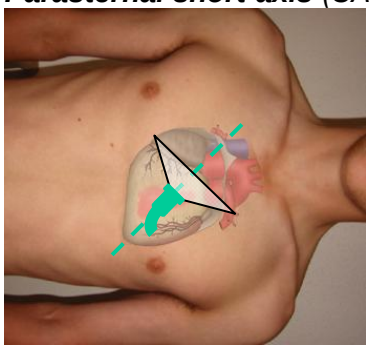
These 2D projections should not be made on a trial-and-error basis but following systematic and highly standardised procedures. These procedures will be presented with live demonstrations Monday 24th 09.00-10.00.

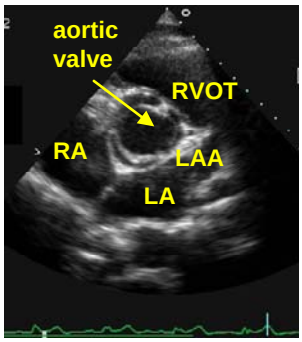
Parasternal long-axis *(PLAX)*



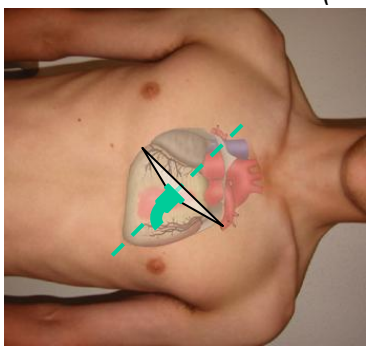


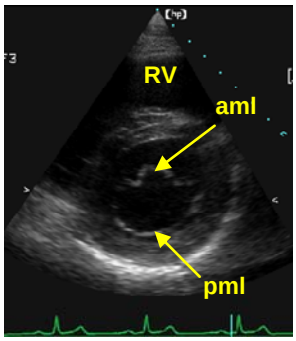
Parasternal short-axis** (SAX) **- aortic level



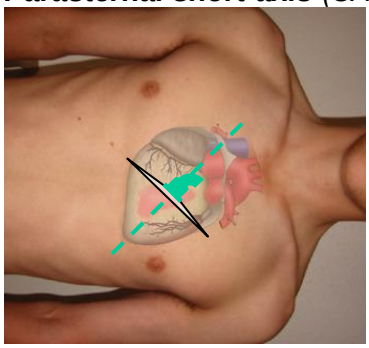


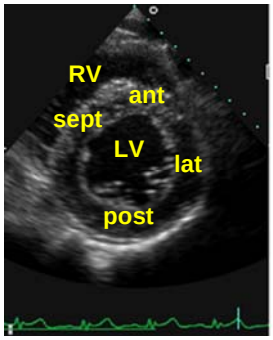
Parasternal short-axis** (SAX) **– mitral level



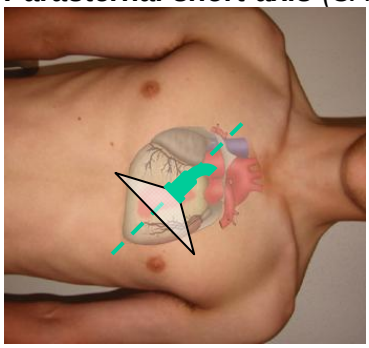


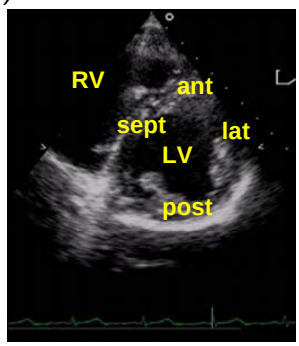
Parasternal short-axis** (SAX) **– mitral chordae level



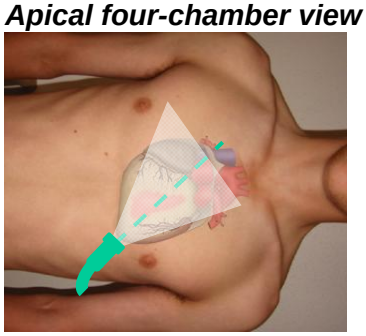


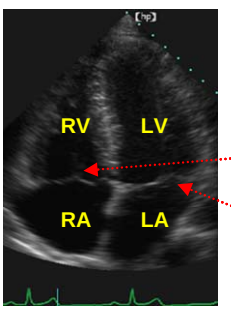
Parasternal short-axis** (SAX) **– mid ventricular level



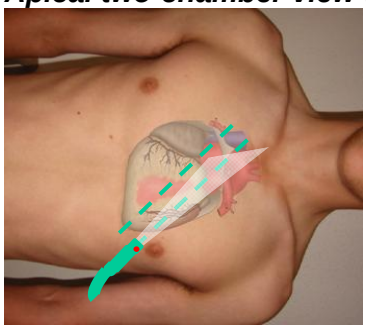


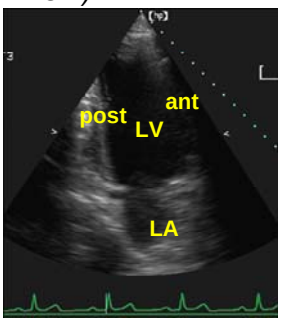
***Apical four-chamber view** (AP4CH)*



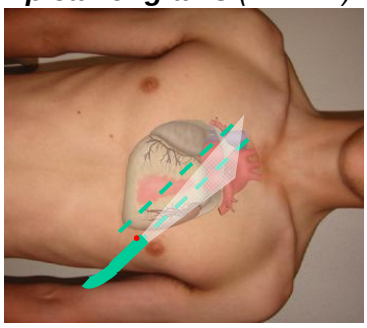


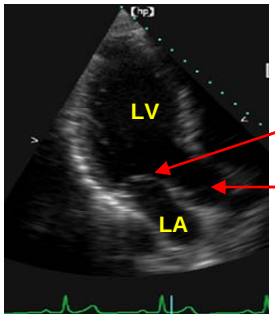
Apical two-chamber view *(AP2CH)*



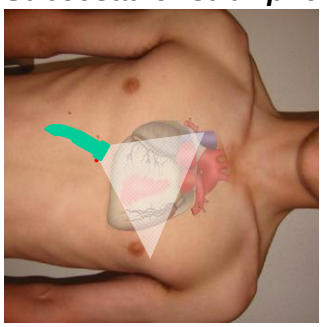


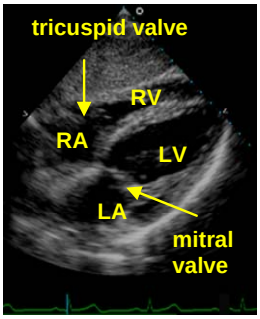
Apical long-axis *(APLAX)*



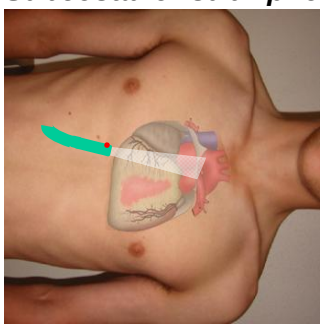


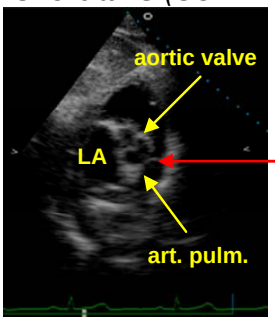
Subcostal or subxiphoid four-chamber view *(SUBXIPH4CH)*



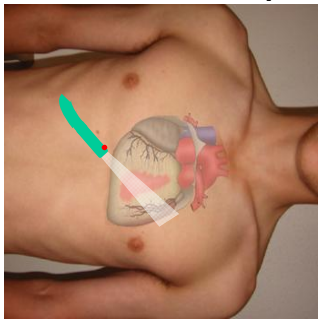


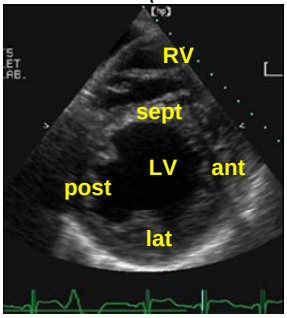
Subcostal or subxiphoid short-axis *(SUBXIPHSAX)* ***– aortic level***



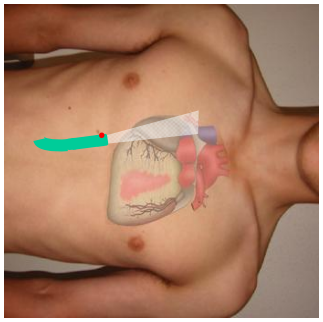


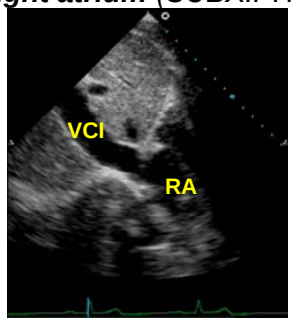
Subcostal or subxiphoid short-axis *(SUBXIPHSAX)* ***– mid ventricular level***





Vena cava inferior and right atrium *(SUBXIPH VCI-RA)*





Recommendations for basic chamber quantification:
Measurements on 2D frames are superior to M-mode measurements regarding accuracy and reproducibility.

Interpretation of values: ASE guidelines, Chamber Quantification (www.asecho.org)

Left ventricular anatomy:

- *PLAX, end diastolic frame, chordae level, perpendicular to LV long axis*
- *measure IVS_D (= SWT_D), $LVID_D$ and PWT_D*
- *in case of increased IVS_D or PWT_D check SAX mitral chordae level in order to avoid erroneous measurements including trabecula septomarginalis (IVS_D) or mitral chordae*
- *for more advanced scientific purposes: volumetric measurements (modified Simpson) or 3D*

Left ventricular systolic function:

- *<u>homogenous</u> LV geometry and regional systolic function: Fractional Shortening (FS)*
- *PLAX, end systolic frame, chordae level, perpendicular to LV long axis*
- *measure $LVID_S$*
- *calculate FS: $FS = (LVID_D – LVID_S) \div LVID_D$*
- *<u>heterogenous</u> LV regional systolic function and geometry (aneurism etc.): systolic Mitral Annulus Displacement (MAD)*
- *using M-mode the systolic MAD is measured in 4 sections of the mitral annulus: anterior and posterior (AP2CH), lateral and septal (AP4CH)*
- *calculate the mean value of MAD based on the four measurements*
- *estimated LVEF = (5,5 x MAD) – 5*
- *for more advanced scientific purposes: volumetric measurements (modified Simpson) or 3D*

Left atrium and right atrium:

- *PLAX, end systolic frame*
- *measure LA anterior-posterior diameter*
- *AP4CH, end systolic frame*
- *trace LA area and RA area*

Right ventricle:

- *RV diameter in PLAX is not recommendable*
- *RV size: AP4CH, end diastolic frame, measure mid RV diameter*
- *RV hypertrophy: measure RV wall thickness in SAX chordae level, SAX mid ventricular level, SUBXIPH4CH or SUBXIPHSAX chordae or mid ventricular level*
- *RV systolic function: using M-mode the systolic Tricuspid Annulus Displacement (TAD or TAPSE) is measured in AP4CH*

Echocardiography as a tool in haemodynamic optimization

Erik Sloth, Aarhus, Denmark

TOE is well established as a diagnostic and monitoring tool in the operating room but like Transthoracic echocardiography (TTE) it has hitherto only been sporadically used for hemodynamic optimization in the ICU and critical care. However, recognition of a complex interaction between the right and left ventricle and the importance of an exhaustive knowledge of the physiological and patho physiological determinants of haemodynamic instability/shock (Figure 1), abbreviated echoprotocols are rapidly spreading in all areas of emergency medicine (1-3). Several reviews, which are highly recommended reading, have also been published recently emphasising the growing interest (4,5).

Echocardiography is, at present, the only method which can provide bedside real-time and dynamic imaging of the heart and pleura. Recent studies indicate respiratory changes in the inferior vena cava diameter as a marker of volume responsiveness and thus, making echo even more attractive as a monitoring tool (6,7). The introduction of small and easy portable systems with superior image quality and full range Doppler tools is of secondary importance but facilitates the implementation.

Most important haemodynamic determinants.

Right and left side physiological determinants
Systolic
Preload
Afterload
Contractility
Heart rate
Diastolic
Compliance
Relaxsation
Heart rate
Patophysiological determinants
Specific post surgical complications
Bleeding
Endocarditis
Perikardial effusion
Pleural effusion/pneumothorax
Valvular dysfunction
Aortadissektion
Pulmonary embolism
Hypoxemia
Post myocardial infarction VSD

WHERE ARE WE RIGHT NOW?

A Medline search confirmed that TOE is very little used and TTE almost never used in the ICU for hemodynamic screening and monitoring purposes until very recently. Mechanical ventilation, subcostal drainage and an unfavorable supine position with limited mobility have been claimed as the major reasons for limited value of TTE in ICU-patients (8). However, technical refinements including second harmonic imaging, have dramatically improved imaging capabilities of TTE and we have recently shown that the abbreviated echo protocol called FATE (Focus Assessed Transthoracic Echocardiography) provided images of sufficient quality to answer the immediate question in 97% of mixed ICU patients (1). Others have reported adequate image quality in 99% of 100 consecutive patients in shock (9). The impact of an echo examination can be dramatic and In our FATE study from 2004 carried out in 220 mixed ICU patients it was proved that FATE changed the treatment in 60% of patients (1).

WHY ECHOCARDIOGRAPHY FOR HEMODYNAMIC OPTIMIZATION?

The cardiac contribution to the entire circulation and therefore to the evaluation of patients in shock is very complex with the major determinants shown figure 1.
These determinants are constantly and dynamically changing with time and therapy and must therefore be thoroughly controlled. If not, improper treatment and interventions may be the consequence.

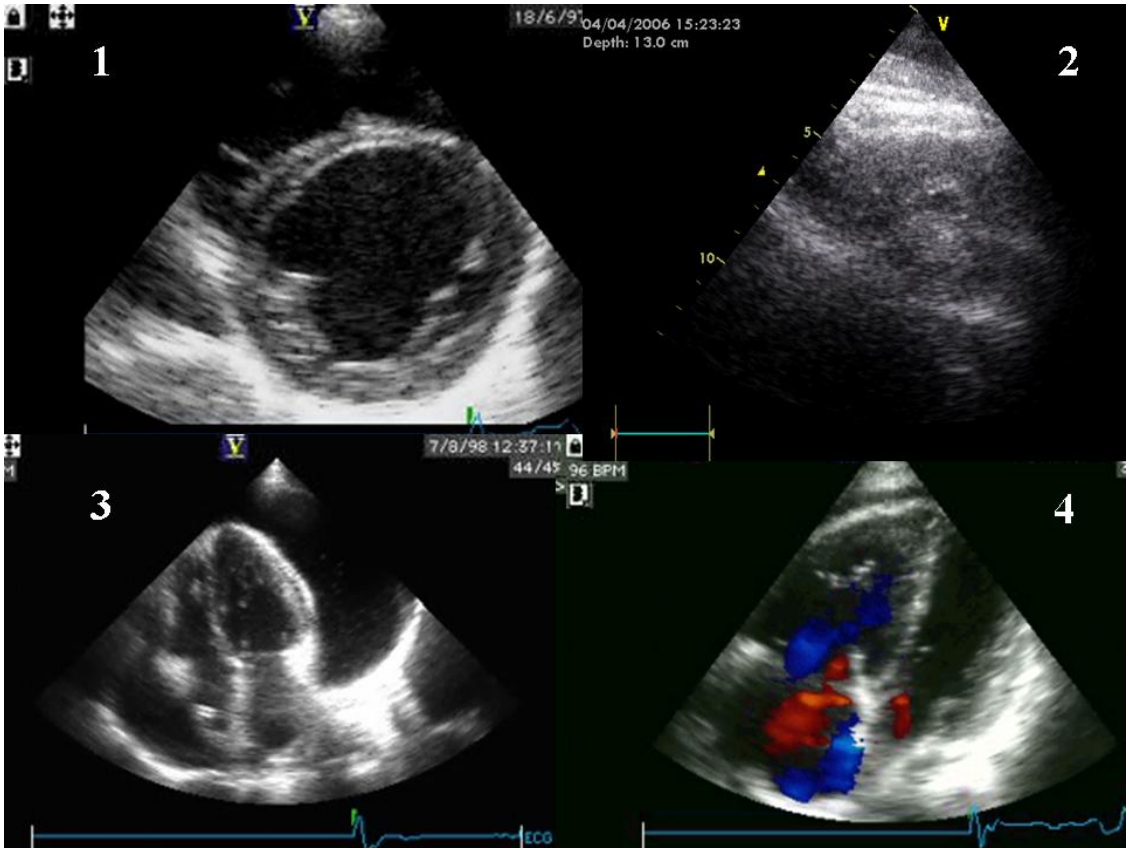


Figure showing the four most important causes of shock. 1) dilated and dysfunctioning left ventricle, 2) severe left ventricular hypovolemia (poor but sufficiently image quality in an emergency case), 3) pericardial effusion and right ventricle pressure overload (pulmonary embolus). These cases are extremely difficult to differentiate between but very easy to recognise echocardiographically.

We have, in many hemodynamically unstable patients, aggressively stopped the administration of up to 3 different inortropic or vasoactive drugs and first thereby fully restored the circulation. Such controversial approach is only possible if the majority of the hemodynamic determinants can be extensively monitored and interpreted. This requires cardiac imaging capabilities and echocardiography provides this opportunity. Quite often drainage of pleural effusion, which can easily be diagnosed by ultrasound, may improve both pulmonary and cardiac function likewise a present pneumothorax can be diagnosed.

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The FATE protocol

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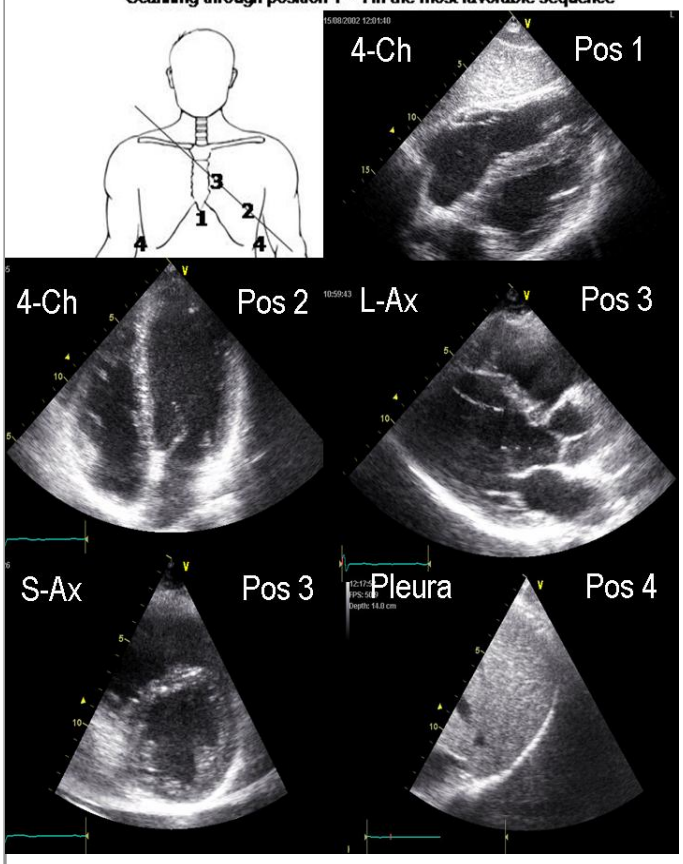
Focus assessed transthoracic echocardiography (FATE)

After almost 20 years experience with transthoracic ultrasound, we have proposed FATE as a rapid and systematic protocol for cardio-pulmonary screening and monitoring. It is designed to be carried out without the knowledge required to fulfil a thorough and much more time consuming cardiologic diagnostic procedure, from which it should be clearly distinguished. FATE should be considered as a supplement to the clinical evaluation.

FATE is a focus assessed ultrasound examination through position 1 – 4 (fig. 1) in a rapid and most favourable sequence depending on patient condition and includes the following steps:

1. *Excluding obvious pathology.*
2. *Assessing wall thickness and dimensions of chambers.*
3. *Assessing contractility.*
4. *Imaging pleura on both sides.*
5. *Relating the information to the clinical context.*

Fig. 1 **Focus Assessed Transthoracic Echo (FATE)**



Generally an over all impression is sufficient; in selected cases however, more accurate quantitative measurements of dimensions and contractility may be applied. All ultrasound-Doppler modalities available, can be applied at any stage during the FATE examination e.g. for pressure measurement, assessment of cardiac output, evaluation of valve pathology, myocardial defects and assessment of inferior vena cava distensibility - and of course to achieve additionally imaging planes to complete a full standard TTE examination.

In principal, FATE may be interrupted as soon as the clinical problem/question has been solved. However, it is recommended to fulfil all imaging positions to exclude competing disorders, which would otherwise be missed. In addition, a specific finding may be better evaluated from a combination of different views.

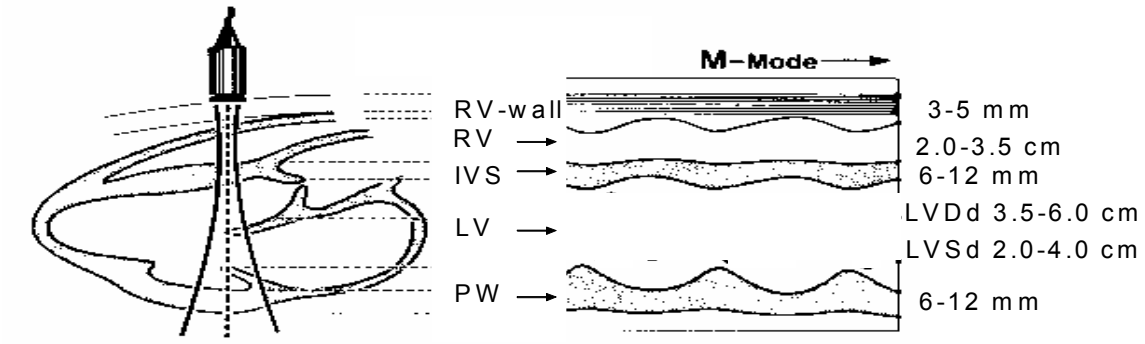
DIMENSIONS, LOAD AND CONTRACTILITY

Dimensions of the ventricles originate from early TTE studies. Cardiac dimensions are important for assessment of volume load since the concept of both preload and afterload imply this knowledge. Conventionally, dimensions are obtained from m-mode scanning in the parasternal long axis view guided by simultaneously 2D-imaging (fig. 2).

In the parasternal long axis view the RV diameter measures approximately 2.0-3.5 cm in diastole. The normal left ventricular end-diastolic diameter (LVDd) is within 3.5-6.0 cm and left ventricular end-systolic diameter (LVSd) between 2.0-4.0 cm (fig. 2). From these measures fractional shortening (FS) is given by LVDd - LVSd x 100/LVDd. The normal range is between 25% and 40%. A rough measure of ejection fraction (EF) is given as 2 x FS which consequently is in the range of 50% to 80%. Dimensions now become a measure of contractility.
In the parasternal long axis view the RV diastolic wall thickness measures approximately 3-5 mm, the interventricular septum (IVS) and posterior wall (PW) 6-12 mm (fig. 2). Finally, by convention, the mitral-septal distance (MSD) is evaluated here and is normally ≤10 mm.
EF is considered the "golden measure" of systolic ventricular function/contractility. In daily routine, many experts agree that it is sufficient to eyeball EF. For eyeballing one should make use of all possible imaging planes. In particular for TOE the LV is evaluated in the transgastric short and long axis view at mid papillary muscle level. In this projection a m-mode recording can be performed quite accurately. In cross-sectional view automated endocardial wall detection, which is available on many echo machines, can guide the assessment of area shortening and provide algorithms for assessment of EF.
A four level grading for EF is very useful in the daily routine: Normal EF (>55%), slightly reduced EF (40-55%), moderate reduced EF (30-40%) and severe reduced EF (<25-30%). It should be noticed that EF is highly influenced by changes in dimensions so increasing LV dimension causes a reduction in EF when the myocardial motion amplitude is constant.

Figure 2

m-mode scanning



From a haemodynamic point of view it is not important whether the echocardiographic information is based on TOE or TTE. Peroperatively, TOE is the method of choice since this can be performed without interfering with the surgical field. TTE is quicker, cheaper and completely non-invasive without contraindications and allows us therefore to offer it to any patient. In a few percent a combination of TOE and TTE is necessary for full evaluation of the cardiac function.

FUTURE

New methods derived from tissue velocity imaging (TVI) e.g. strain-rate, strain and tissue tracking and speckle tracking are promising tools for quantitative assessment myocardial contractility. Together with 4-dimensional and contrast echocardiography they may prove reliable and easy applicable for cardio-pulmonary screening and

monitoring and therefore important in the ICU. Older echo machines are available to a much lover cost than just few years ago. Having an echomachine as a part of ICU monitoring equipment is no longer unattainable. Hand held echocardiographic equipment of extremely good quality has been introduced with success and further increased the great expectations to TTE. So called "pocket ultrasound machines" or "FATE-machines" has been advertised from the manufactures and will, without any doubt, set up a new standard in cardio-pulmonary optimization. Reports indicate that TTE will find its way to every place where patients suffer from hemodynamic instability including shock – no matter which location.

Thus, FATE should be recommend as the first choice of hemodynamic instabillity instead of the fading patients last chance.

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TTE Doppler Echo

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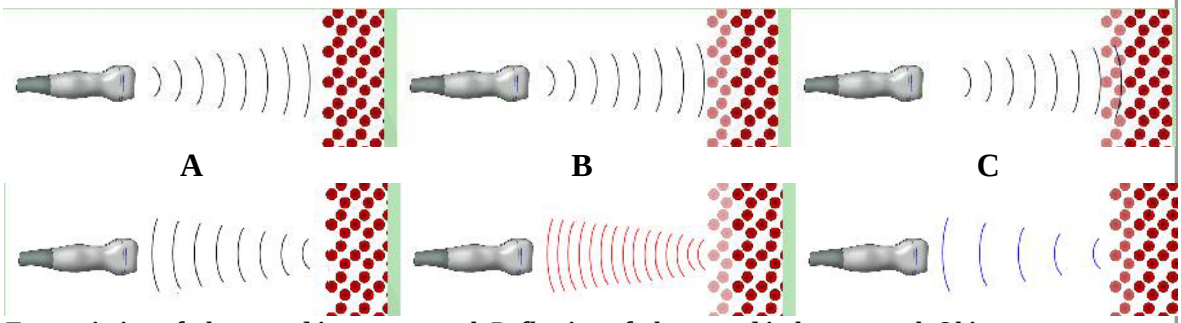
Doppler echocardiography utilizes ultrasound to visualize, record and measure blood flow within the cardiovascular system.

Doppler echocardiography is based on frequency analysis of returning ultrasound signals from small moving objects, i.e., erythrocytes. When a moving object is intercepted by the ultrasound beam the frequency of the reflected ultrasound (F_R) received by the transducer will differ from the frequency of the originally transmitted ultrasound (F_T). This difference in frequency is called the Doppler shift.

$$\text{Doppler shift } (F_D) = F_R - F_T$$

If an object is moving towards the transducer the frequency of the reflected ultrasound is higher than the frequency of the transmitted ultrasound leading to a positive Doppler shift.

If an object is moving away from the transducer the frequency of the reflected ultrasound is lower than the frequency of the transmitted ultrasound leading to a negative Doppler shift.



Transmission of ultrasound in upper panel. Reflection of ultrasound in lower panel. Objects are: not moving (A), moving towards transducer (B) or away from transducer (C)

Blood flow velocity (V) is related to the Doppler shift by the propagation velocity of ultrasound in blood (C) and the angle between the ultrasound beam and the direction of flow (θ).

$$V = \frac{(F_r - F_t) \cdot C}{2 \cdot F_t \cdot \cos(\theta)}$$

This is very important to remember in clinical Doppler echocardiography. Before activating quantitative Doppler (PW or CW), adjust the 2D projection guided by anatomy and Color Doppler in order to minimize the angle between the ultrasound beam (curser) and the direction of flow. Angles exceeding 20° will result in progressively significant and clinical important underestimation of flow velocity and derivates like pressure gradients.

Using, or from a physicist point of view misusing, the Bernouilli equation, pressure differences between cardiac chambers or across valves can be estimated. Generally, a highly simplified version of the Bernouilli equation is used, in which the viscous losses and the velocity proximal to the point of measurement (for instance the velocity in LVOT in aortic stenosis) are neglected.

$$\Delta P = (0{,}5 \cdot p \cdot (V_2^2 - V_1^2)) + (p \cdot (\frac{dv}{dt})) + R_v \qquad \textit{or super simplified:} \quad \Delta P \text{ (mmHg)} = 4 \cdot V_2^2$$

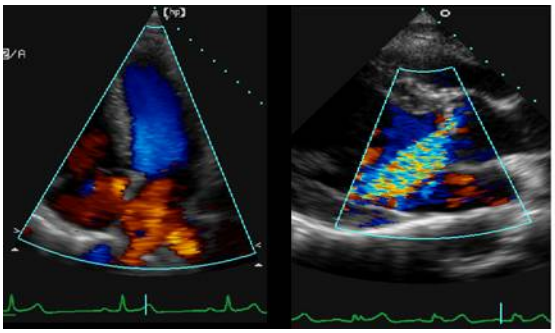
Color Doppler imaging:
Color Doppler is based on the principle of pulsed wave Doppler (se below). Backscattered signals are received from multiple gates or sample volumes along each ultrasound line in the "colorbox". The frame rate is limited and depth dependent due the pulsed Doppler technique.

Laminar blood flow towards the transducer is displayed in red and blood flow away from the transducer in blue. Turbulent flow is displayed in a color-mosaic.

Color Doppler is very helpful in alignment of quantitative Doppler ((PW and CW) and the screening and assessment of a number of cardiac diseases including regurgitant flows and intracardiac shunts.

Procedure:

- *optimize 2D projection*
- *reduce overall gain below normal level in 2D echo*
- *activate Color Doppler*
- *adjust the position and size of the "colorbox" . Avoid excessive width of the box in order to maintain reasonable Color Doppler frame rate*
- *eliminate excessive 2D depth in order to maintain reasonable Color Doppler frame rate*
- *Color Doppler frame rate ought to be at least 20 fps in case of high velocity jets*
- *adjust scale:*
  low velocity flow: 40-50 cm/sec
  normal and high velocity flow: 60-70 cm/s in adult echo and 70-90 cm/s in paediatric echo
- *optimize Color Doppler gain*



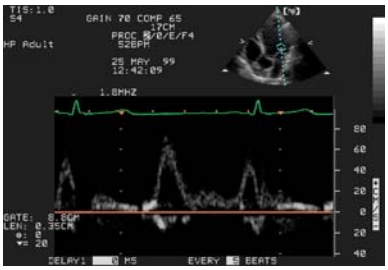
Pulsed Wave Doppler (PW Doppler):
PW Doppler permits sampling of blood flow velocities from a specific region or sample volume. The position of the sample volume is optional but confined to the 2D tomographic slice.

An ultrasound impulse is transmitted and the receiver is open during a subsequent time interval corresponding to the distance between the transducer and the sample volume. The sequence is repeated at an interval called the pulse repetition frequency (PRF). Thus, the PRF is depth dependent and lower in deeper regions. A decrease in PRF causes a decrease in the maximal velocity that can be displayed without aliasing. Depending on depth, velocities up to 1,5 – 2,0 m/sec can be displayed with traditional PW Doppler.

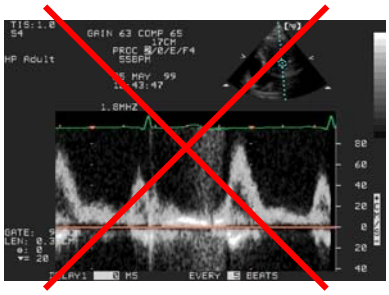
PW Doppler is very helpful in many situations including assessment of transmitral and transtricuspid blood flow, pulmonary venous flow, left atrial appendage flow, estimation of stroke volume and shunt fraction, evaluation of valvular lesions etc.

Procedure:

- *optimize 2D projection guided by anatomy and Color Doppler*
- *position curser and sample volume*
- *activate PW Doppler*
- *optimize PW Doppler gain*
- *optimize scale*
- *optimize filter (usually 100-200 Hz)*
- *horizontal sweep 100 mm/sec or 150 mm/sec in case of rapid heart rate*

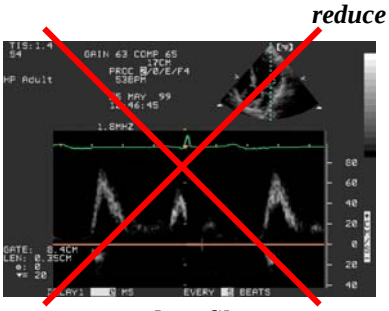


correct



reduce gain

reduce scale



reduce filter

increase horizontal sweep

Continuous Wave Doppler (CW Doppler):
CW Doppler employs two dedicated ultrasound crystals, one for continuous transmission and another one for continuous reception.

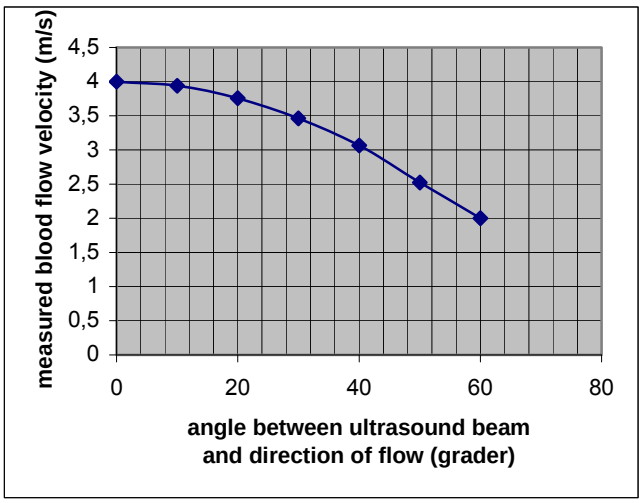
This permits measurement of extremely high blood flow velocities. The trade-off is that all blood flow velocities along the entire length of the ultrasound beam are received and displayed.

CW Doppler is very helpful in many situations including quantification of aortic and pulmonic stenosis, pressure gradients across ventricular septal defects and estimation of right ventricular and pulmonary arterial pressure.

Procedure:

- *optimize 2D projection guided by anatomy and Color Doppler*
- *position curser*
- *activate CW Doppler*
- *optimize CW Doppler gain*
- *optimize scale*
- *optimize filter*
- *horizontal sweep usually 100 mm/sec or 150 mm/sec in case of rapid heart rate*

Please do not forget the importance of the angle between the ultrasound beam and the direction of blood flow. Optimize 2D projection guided by anatomy and Color Doppler in order to obtain good alignment. Do not use angle correction, which is usable in vascular ultrasound but hazardous in echocardiography.



Is V underestimated by 10%, 15% or 20% the derived pressure gradient will be underestimated by 19%, 28% or 36% !

Principle of myocardial velocity and deformation imaging

Fabio Guarracino, Pisa, Italy

Introduction.

Traditionally, myocardial function has been investigated with ultrasounds by evaluating firstly endocardial motion, and then changes in both motion and thickening of wall segments during systole on M-mode and two dimensional echo in order to evaluate the degree of myocardial dysfunction in several disease states, as ischemia and infarction. On this basis, a whole evaluation of the segmental model of the left ventricle allows to determine a wall motion score index and to grade regional myocardial function in patients with coronary artery disease or other myocardial disease states.

This M-mode and two-dimensional analysis of the moving heart provides qualitative but not quantitative motion information. Therefore, the assessment of myocardial motion from echocardiogram is routinely accomplished by visual interpretation and manual evaluation. This leads to subjective and semi-quantitative diagnoses, which suffer from a significant intra- and inter-observer variability.

In the last years the introduction of Tissue Doppler Echocardiography (TDE), an emerging ultrasound modality capable of measuring myocardial velocities during both systole and diastole by measuring high-amplitude, low-frequency Doppler shifts of ultrasound signal reflected by tissue, has opened a new way of evaluating myocardial performance. Myocardial motion presents with low velocity and high amplitude signals, a physic profile very different from intracardiac flow velocities, which requires narrow image sector to optimise the frame rate during data acquisition. Different TDE modalities have been used to study myocardial velocities. Spectral pulsed wave Doppler allows direct online measurement of velocities and time intervals both from transthoracic and transoesophageal approach. According to the different point of view the velocities have different directions, following the Doppler rule that velocities moving towards the transducer are displayed positive and velocities moving away from the transducer are displayed negative.

Colour Doppler TDE represents myocardial motion towards the transducer with red scale, which are the positive velocities, and myocardial velocities away from the transducer with blue scale, which are the negative velocities. This modality has the advantage of allowing measurement of peak and mean velocities, and time velocity integral in each myocardial segment and in all phases of the cardiac cycle. On the other hand, at this moment it still requires off-line analysis, which needs data acquisition and storage.

The analysis of myocardial velocities allows investigation of both longitudinal and radial myocardial function, so providing interesting insight into different aspects of myocardial motion. Under normal conditions, systolic movement of myocardium consists of longitudinal shortening, radial contraction and rotation. These different patterns of motion depend on myocardial fibers orientation. The TDE evaluation of velocities within the cardiac tissue, both systolic and diastolic, radial and longitudinal, entirely depends on the position and the alignment of the transducer, that means the angle at which the region is imaged, either transthoracic or transoesophageal. This implies that velocity profiles can be affected by rotation and translation of the heart, and also by traction and tethering effect from adjacent myocardial segments.

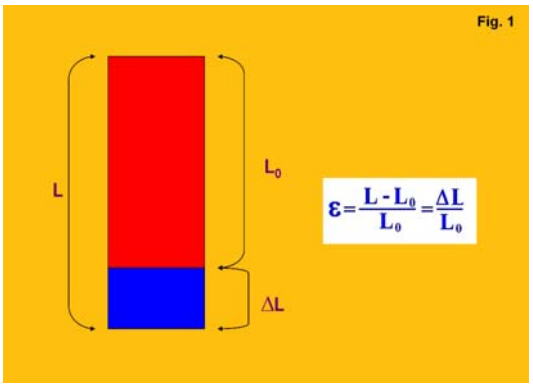
To overcome these limitations new echo modalities have been introduced into clinical practice recently.

Strain and strain rate.

The drawbacks reported for myocardial velocities analysis can be neutralized by approaching myocardial evaluation from a different way of looking at it. The most recent concept that has been introduced is that of evaluating myocardial function by imaging myocardial deformation during cardiac cycle.

To introduce this topic it is crucial to distinguish between motion and deformation. Motion is well represented by displacement and velocity. Deformation is expressed by strain and strain rate. An object does not undergo deformation while moving if every part

of the object moves with the same velocity. This moving object can be described as having translational velocity, but its shape remains unchanged. Over time, the object will change its position in the space, and this can be referred to as displacement. On the other hand, if different parts of the object move with different velocities, the object necessarily has to change its shape. So, when describing a moving object we can 1) describe the motion of the different parts by their velocity and displacement, 2) describe the whole object as undergoing deformation.
Strain, in daily language means, "stretching". In scientific language it means "deformation".
Strain defines the amount of deformation of an object referred to its initial dimension, caused by an applied force. For any object with one possible deformation, for example elongation and shortening, the one-dimensional strain, the so-called Lagrangian strain ε is defined as the relative elongation with respect to the initial length L_0: $\varepsilon = L - L_0 / L_0$. (Fig. 1)



Thus, strain is a dimensionless quantity which describes the myocardial deformation during cardiac cycle. It expresses the relative change of segmental length occurring between the reference state (L_0) (end-diastole) and the state of deformation (L) (end-systole) expressed in percentage of end-diastolic length. From the formula above, it is evident that positive strain is lengthening or stretching, negative strain is shortening or compression, in relation to the original length.

Strain rate (SR) can be interpreted as the speed at which tissue deformation occurs.
SR= $(L - L_0 / L_0) / T$ (where T is time)
It is measured in (cm/s)/cm = 1/s units. The SR is negative during shortening, while positive during elongation, and is more suited for diastolic deformation, i.e. the rate of elongation or thinning will be positive during diastole.
SR can also be described as the difference in velocities at both ends of an object with initial length L_0. In other words, SR expresses the spatial gradient in velocities.
ε and SR are reported to be uniform along heart walls and are much less influenced by global and regional cardiac motion.
Current approaches to calculate myocardial strain rate are based on tissue colour Doppler imaging, where the axial strain rate component is computed as the spatial derivative of the Doppler velocities. Strain is calculated by temporal integration of SR. The technique of raw data storage and off-line processing permits the measurement of tissue velocity, peak systolic strain rate, peak early and late diastolic strain rate, and peak systolic strain from the same sample volume within the same cardiac cycle.

Curve interpretation.
Systolic strain curve is positive when the explored segment expands. This is thickening in short-axis view (parasternal on transthoracic and midoesophageal SAX on TEE), and lengthening in apical views (Fig.2-3). Systolic strain curve is negative when the explored segment shows compression, which is thinning in short-axis view and shortening in apical views. Infarcted myocardial tissue does not demonstrate shortening or lengthening activity and shows no or minimal systolic strain rate or strain.

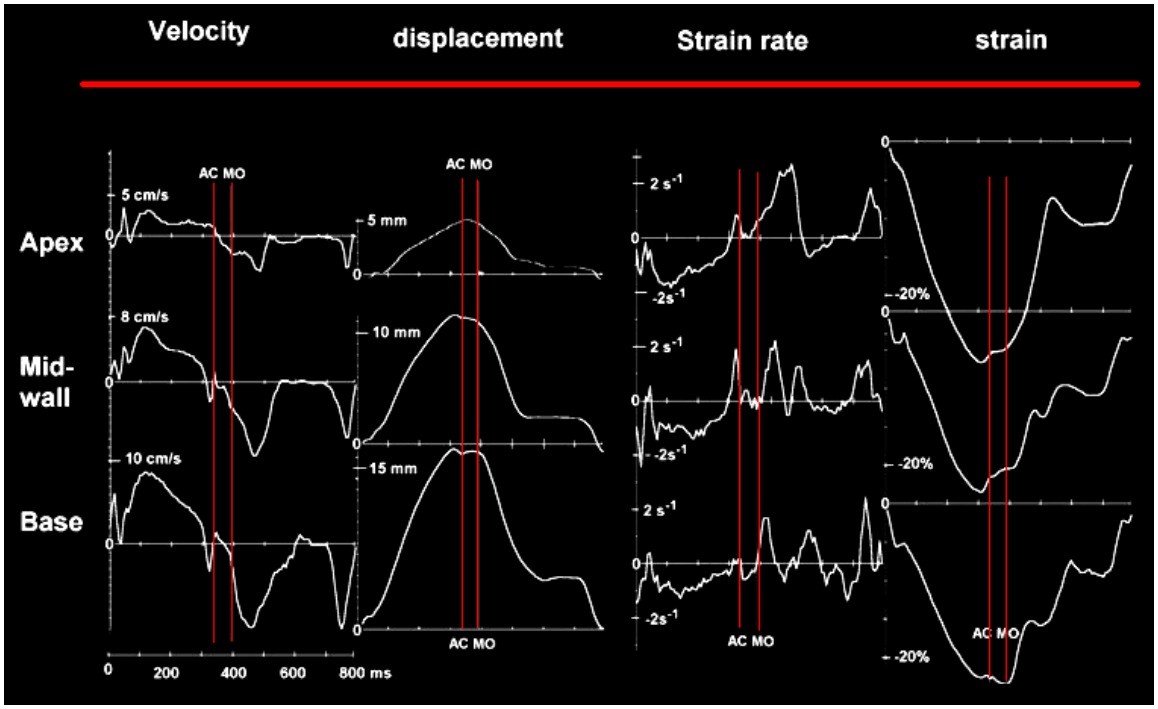


Fig. 2 Velocity, displacement, strain rate and strain from apical, mid and basal interventricular septum in longitudinal approach. Displacement is obtained by temporal integration of velocity. Strain rate is obtained by spatial derivation of velocity, and by temporal integration of strain rate one obtains strain. These curves all represent the same data set. It is evident that motion (velocity and deformation) increases from apex to base, showing a gradient, while deformation (strain rate and strain) is more constant, in fact a direct measure of the motion gradient.

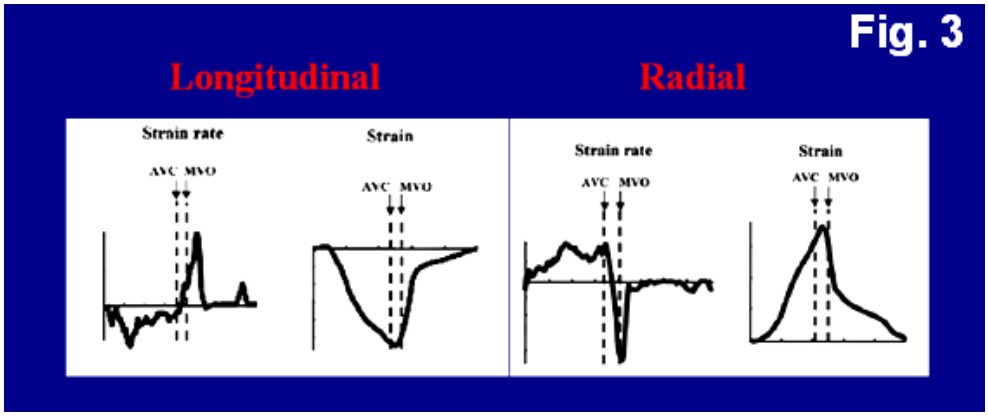


Fig. 3 Strain and SR in longitudinal and radial approach.

Preventing pitfalls: technical issues.

Some practical points and stringent procedures have to be kept in mind to obtain optimal image quality when registration of data for strain and SR analysis is performed.
Real-time 2D color Doppler myocardial imaging data are recorded from the LV and RV using standard TTE and TEE views. An appropriate velocity scale has to be chosen to avoid data aliasing. It is very important to align the Doppler beam with the vector of motion of the explored segment. For short axis views, careful attention is needed to keep the explored LV wall perpendicular to the ultrasound beam so that it would align at zero degrees to radial motion. For apical views, each explored wall has to be kept in the center of the ultrasound sector to be certain of alignment as near to zero degrees as possible to long-axis motion.
The pulse repetition frequency should be optimised. The sector size and depth should be adjusted in order to increase the frame rate, which has to be at least as high as 140 frame per sec. The region of interest should be tracked to remain within the tissue under investigation.

A ECG line displayed on echo screen is necessary for timing. Three to five consecutive heart cycles are acquired in sinus rhythm and averaged.
Recordings are performed from long axis view for longitudinal strain and SR (4 chamber view on both TT and TEE echo), and short axis view for radial parameters (parasternal from TT approach; midpapillary SAX from TEE).

Limitations.
As with flow Doppler, TDE velocity, strain rate, and strain measurements are angle dependent. With the currently available technology, strain rate has a relatively poor signal-to-noise ratio and seems to be load sensitive. Strain measurements are made along a single ultrasound scan line. Limitations related to complex fibre architecture and to translation and rotation of the heart apply to all imaging modalities.
Off-line analysis is required for colour images, and this is time consuming.

Implications.
An advantage of ultrasound-based SR/ε imaging is that such indices can be derived both at the bedside and in the intensive care unit, but also in the operation theatre by using transoesophageal echocardiography.
A relevant clinical implication of SR/ε relies on the possibility to study myocardial ischemia and infarction, and to recognise different patterns of myocardial dysfunction related to ischemia. This is based on the different wall deformation that can be detected in stunned and hibernating myocardium, and also in viable and non viable myocardium. The SR/ε evaluation can be used in the setting of stress echocardiography too, in order to better distinguish different ischemic substrates, specially in patients with conduction abnormalities and/or rhythm abnormalities, in whom it may be particularly useful.
The evaluation of deformation indices could also play a role in the evaluation of diastolic function and right ventricular function, of cardiomyopathy and transplanted hearts, but further studies are needed for better understanding of the clinical implications.

Conclusions.
The study of myocardial velocities and of myocardial deformation, meant as gradient of velocities within a part of myocardial wall has lead to a new fascinating and intriguing way of evaluating myocardial function. Clinical experience and literature show that SR/ε imaging is a practical and useful clinical tool for the calculation of regional longitudinal and radial function in both the cardiac ventricles. The combined use of regional SR/ε indices and the timing of specific regional systolic or diastolic events offers a new non-invasive approach to the quantification and monitoring of myocardial dysfunction.

Aortic dissections – ascending and descending

Justiaan Swanevelder, Leicester, UK

Aortic dissection is a rare but potentially fatal event resulting in separation of the layers of the tunica media by ingress of blood, producing a false lumen with variable proximal and distal extension. Ascending aortic dissection is the most common catastrophe of the aorta, 2-3 times more common than rupture of the abdominal aorta. Mortality of untreated acute dissection involving the ascending aorta is about 1 to 2% per hour during the first 48 hrs. The first documented famous case was King George II.

Classification

Several different classifications have been advocated to describe aortic dissection. The classification systems in common use are either based on the duration of onset of symptoms prior to presentation or the anatomy of the dissection. Aortic dissection is acute if the diagnosis is made within 2 weeks following the initial onset of symptoms, and chronic if present for more than 2 weeks. Aortic dissection is classically classified using the DeBakey or Stanford classification (fig 1).

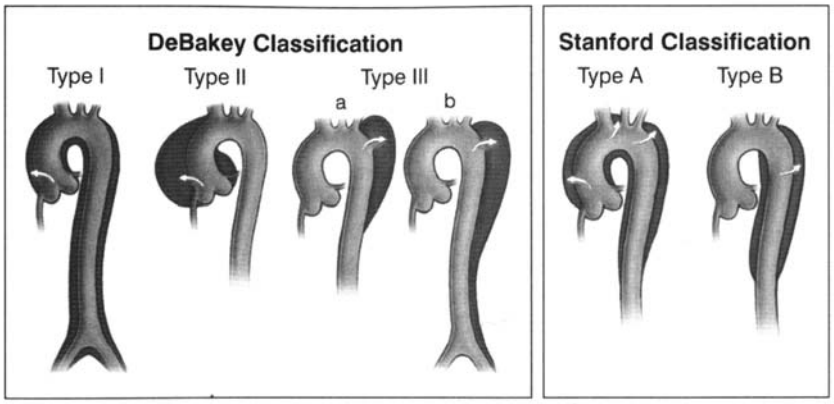
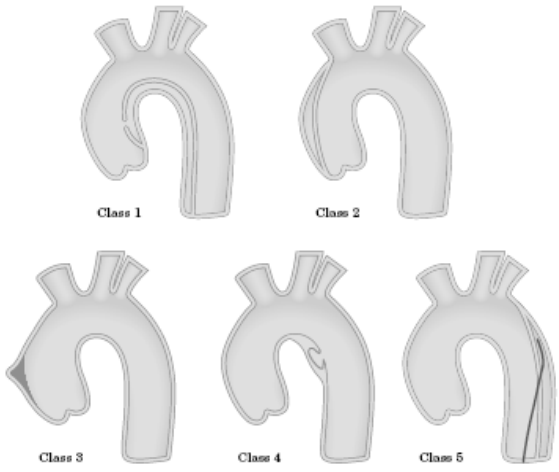


Fig 1 Anatomical classification of aortic dissection

The most commonly used is the Stanford classification, which is based on involvement of the ascending aorta. In Stanford type A the ascending aorta is always involved. In Stanford type B the dissection is distal to the origin of the left subclavian artery. The Stanford system also helps to delineate two distinct risk groups for management. Usually, type A dissections require surgery, whilst type B dissections are under most conditions best managed conservatively with medical treatment. Recently the European Society of Cardiology Task Force on Aortic Dissection has come up with a more comprehensive etiological classification (fig 2).



Class 1	Classical aortic dissection
Class 2	Intramural haematoma/haemorrhage
Class 3	Subtle-discrete aortic dissection
Class 4	Plaque rupture/ulceration
Class 5	Traumatic/iatrogenic aortic dissection

Fig 2 European Society of Cardiology Classification (Erbel et al)

Thoracic aortic dissections should be distinguished from aneurysms (localized abnormal dilatation of the aorta) and transections, which are caused most commonly by high-energy trauma. Advanced imaging technology has demonstrated that intramural haemorrhage, intramural haematoma and aortic ulcers may be signs of evolving dissections or dissection subtypes. All these are grouped under acute aortic syndromes.

Pathophysiology

Aortic dissection is more common in males with a peak incidence between 50-70 yrs of age. Aortic dissection can result either from a tear in the intima and propagation of blood into the media or else from intramural haemorrhage and haematoma formation in the media followed by perforation of intima. The former is more common. These are commonly preceded by medial wall degeneration or cystic medial necrosis. Blood may re-enter the true lumen at any point, thus making it a communicating dissection.

An intimal tear can occur in the regions of the aorta that are subjected to the greatest stress and pressure fluctuations. Because mechanical stress in the aortic wall is proportional to intramural pressure and vessel diameter, hypertension and aortic dilatation are known risk factors for dissections. Integral wall abnormalities such as Marfan's syndrome may also predispose to dissection. While no single disorder is responsible, several risk factors have been identified that can damage the aortic wall and lead to dissection (Table 1).

Most aortic dissections occur with an initial transverse tear along the greater curvature of the aorta, usually within 10 cm of the aortic valve. The aortic root motion has a direct impact on the mechanical stresses acting on the aorta (Beller et al). The next most common site is the descending thoraci c aorta immediately distal to the origin of the left subclavian artery.

Long standing arterial hypertension
- Advanced age, smoking, dyslipidemia, cocaine/crack

Connective tissue disorders
- Hereditary fibrillinopathies - Marfan's, Ehlers-Danlos, Turner's syndrome
- Hereditary vascular diseases - Bicuspid aortic valve, Coartaction
- Vascular inflammation - Giant cell arteritis, Takayasu arteritis, Syphilis

Aortic aneurysm

Pregnancy

Deceleration trauma
- Accident, Fall from height

Iatrogenic factors
- Catheter/Instrument intervention
- Aor tic surgery - cross clamp or side clamp, graft anastomosis, cannulation site

Table 1 Risk factors for aortic dissection (Adapted from Tsai et al)

Clinical features and diagnosis

Clinically aortic dissection presents as a two-step process. The first event is the interruption of the intima which is associated with severe pain and loss of pulse volume. The second event sets in when the pressure exceeds a critical limit and rupture occurs. A high index of suspicion is important in patients with predisposing risk factors (Table 1).

Abrupt sharp high intensity chest pain at the onset is its most specific characteristic. It has been described as stabbing, tearing or ripping in nature. Analysis of the International Registry of Acute Dissection (IRAD), noted that severe chest pain is more common with type A dissections, whereas back pain and abdominal pain are more common in type B dissection. The pain may be migratory and follow the path of propagation of the dissection. The clinical manifestations are diverse and overlap. Physical examination may reveal tachycardia, usually accompanied by hypertension in the setting of baseline primary hypertension and increased catecholamine levels from anxiety and pain. Tachycardia and hypotension result from aortic rupture, pericardial tamponade, acute aortic valve regurgitation or acute myocardial ischemia. Differential or absent pulses in the extremities and a diastolic murmur of aortic regurgitation may also be present. Syncope, stroke and other neurological manifestations secondary to malperfusion syndrome may develop.

In all patients immediate ECG must be done to exclude acute myocardial infarction. For primary myocardial infarction the treatment will be very different and may involve thrombolysis. About 20% of patients with type A dissection have ischemic changes on ECG due to extension of the dissection into a coronary ostium. In such patients further imaging should be done before thrombolysis or revascularisation procedures are attempted. Biochemical markers of myocardial damage may help in the diagnosis (Khan et al). The most promising biochemical marker to date for diagnosing acute aortic dissection is an elevated circulating smooth muscle myosin heavy chain protein, released from damaged aortic medial smooth muscle (Tsai et al).

Imaging studies

Multiple modalities (CT, MRI scanning and echocardiography) can be used to complement each other to facilitate diagnosis depending upon availability. The overall diagnostic accuracy of these different modalities is similar (Erbel et al). Table 2 outlines the diagnostic goals.

Confirm diagnosis
Classify the dissection/delineate the extent
Differentiate true & false lumens
Localise intimal tear; intimal flap, entry sites
Distinguish between communicating & non-communicating dissection
Assess side branch involvement (ie. coronary, carotid, subclavian, coelic & renal arteries)
Detect & grade aortic regurgitation
Detect extravasations (peri-aortic or mediastinal haematoma, pleural or pericardial effusion, tamponade)

Table 2 Diagnostic goals (Adapted from Erbel et al)

On chest X-ray (CXR) the aortic knuckle changes may be observed, with intimal calcification separated more than 6mm from the edge. A widened mediastinum, cardiomegaly (due to pericardial effusion) and loss of costo-phrenic angle secondary to the presence of a haemothorax may be noted.

Aortography is the historical "gold standard" for diagnosis. This distinguishes the origin of branch arteries from true or false lumens. This is not appropriate in the unstable patient. With the availability of advanced non-invasive imaging techniques, aortography is now rarely performed.

A CT-scan is relatively rapid and non-invasive and with contrast image enhancement the extent of the dissection along with the true and false lumens can be identified. This technique is inappropriate if the patient is haemodynamically unstable. MRI gives high-resolution images without contrast dye, but can be time consuming. It is also not advocated in haemodynamically unstable patients. Transthoracic echocardiography (TTE) is easily available and the ascending aorta and aortic arch can be visualised well. In obese or chest trauma patients image quality may be inadequate due to poor echo windows. Intravascular ultrasound (IVUS) is a catheter based imaging study with high accuracy, which provides dynamic imaging of the aortic wall and intimal flap.

Transoesophageal echocardiography (TOE) has become more popular as experience and availability increase, and is highly diagnostic. It is useful peri-operatively in the haemodynamically unstable patient. Although oesophageal intubation with the probe is a strong stimulus, TOE can be performed at the bedside and in the operating theatre with immediate results. Care must be exercised while inserting a TOE probe in a patient with known dilation of the thoracic aorta. When resistance to the TOE probe in the oesophagus is encountered, it may be better to wait until sternotomy before advancing the probe any further. This is especially important in cases where a preoperative history of dysphagia, hoarseness, or stridor is present.

TOE images the entire thoracic aorta except for the most distal ascending aorta and a part of the arch obscured by the trachea or right main bronchus. Views of the thoracic aorta are usually obtained at an imaging depth of 5 to 7 cm due to its proximity to the oesophagus. The 6 views of the thoracic aorta standardized by the Society of Cardiovascular Anesthesiologists/American Society of Echocardiography guidelines, provide a comprehensive evaluation of most aortic pathology (Shanewise et al). Diagnosis is confirmed when two lumens are seen separated by an intimal flap within the aorta (fig 3). Multiplane scanning provides information

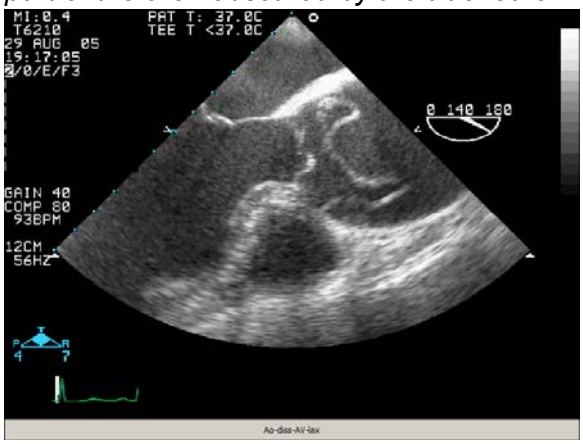


Fig 3 TOE of Type A aortic dissection with characteristic intimal flap

concerning extension of dissection into the innominate artery, left common carotid, or left subclavian artery. Involvement of the coeliac or renal arteries may also be detected. Tears and entry sites can be identified, and differentiation between true and false lumen can be achieved. The true lumen is often compressed by the false lumen and demonstrates systolic expansion and systolic forward flow (Erbel et al). It is close to the inner curvature of the aortic arch. It is common to see not only one, but multiple entry sites and tears. Multidirectional flow jets can often be observed. In acute communicating dissections the intimal flap may display strong excursion and phasic motion during the cardiac cycle. When no communication is present such movement and flow may be reduced or absent. Depending on the degree of communication, spontaneous contrast or thrombus formation may be observed in the false lumen. The presence of a raised pulse pressure and diastolic murmur may indicate aortic insufficiency. Further assessment can be accomplished with M-mode, colour flow, pulsed, and continuous wave Doppler.

Ventricular function and filling can be evaluated throughout the procedure. Ventricular wall motion abnormalities due to myocardial ischaemia can be spotted and managed early. With TOE the ostia of both coronary arteries can be visualized. Fluid extra-vasation into the pericardium, pleural space or mediastinum is an indicator of urgency. A pericardial effusion as little as 30 ml can be detected by TOE. Fluid around the aorta is a sign of ongoing penetration or perforation, often combined with intramural hae-morrhage which eventually results in rupture. Pleural effusions are usually left sided.

Reverberation artefacts within the lumen of the ascending aorta can be misleading and should be differentiated from the intimal flap by examining the pathology in several image planes. Care must be taken to avoid false positives, because mirror images ("double-barrel aorta") are common in the descending aorta. Together with dissections, intramural hematomas and atherosclerotic penetrating ulcers can also be diagnosed with high sensitivity (99%) and specificity (89%). Positive predictive accuracy is 89% and negative predictive accuracy is 99% (Erbel et al).

Patient management

Acute type A aortic dissections are operated upon without delay, as rupture can be imminent. Possible contraindications include paraplegia and severe incurable comorbidities. Neurological involvement, metabolic acidosis and acute renal impairment are associated with a poor prognosis. The anaesthetist is involved in resuscitation and stabilisation, pain relief, sedation for TOE, transfer, anaesthesia and peri-operative care of aortic dissection patients. The anaesthetist's role may also include diagnostic peri-operative TOE to aid surgical decision making.

Several surgical approaches are described. The goals of surgical therapy are to prevent extension, excise the intimal tear and replace the segment of aorta susceptible to rupture with an interposition synthetic graft (elephant trunk technique). Combined aortic valve and ascending aorta replacement with re-implantation of coronary arteries using a composite graft is performed if the aortic valve is not salvageable. During aortic valve sparing procedures the use of intraoperative TOE is of utmost importance to guide successful outcome.

Cardiopulmonary bypass (CPB) is established using various cannulation sites depending on the anatomy and urgency. Arterial cannulation for antegrade perfusion is accomplished either via the distal aortic arch if not involved, right subclavian artery, innominate artery, or true lumen of the dissected ascending aorta. An alternative cannulation site for antegrade perfusion is through the left ventricular apex and aortic valve. Cannulation of either femoral artery will provide retrograde aortic perfusion with potential extension of the dissection area. Venous cannulation is most often through the right atrium using a two-stage venous cannula. Femoral or bicaval venous cannulation is other options. If the aortic valve is incompetent a left ventricular vent is necessary to prevent LV distension and subsequent subendocardial ischemia. TOE is very useful in guiding various cannulation manoeuvres.

In acute type B aortic dissections surgical intervention is only indicated if there is persistent or recurrent intractable pain, aneurysm expansion, peripheral ischemic complications and rupture. Medical management is preferred for uncomplicated descending aortic dissections. This is because surgical repair has no proven superiority over nonsurgical treatment in stable type B dissection patients. The primary focus is to reduce blood pressure and hence prevent extension of the dissection. Adequate pain relief is provided as required.

Interventional management

Endovascular interventions are gaining popularity in type B aortic dissections especially in patients at high risk for thoracotomy because of severe coexisting cardiopulmonary abnormalities or other medical problems. The advent of percutaneous stenting and/or fenestration technology provides an alternative to open surgery for selected patients. Aims of the treatment include reconstruction of the thoracic aortic segment containing the entry tear, induction of thrombosis of the false lumen, and reestablishment of the true lumen and side branch flow (Tsai, et al). TOE is a valuable tool for identifying aortic pathology, confirming the guidewire position in the true lumen, and guiding stent graft positioning (Swaminathan et al). It also supplements angiography for detecting endoleaks. Although endovascular techniques do not involve extended periods of aortic occlusion, TOE can reliably determine whether myocardial function has been compromised as result of hemodynamic disturbances.

Prognosis and follow up

According to the International Registry of Acute Aortic Dissection (IRAD) (Hagan et al), current overall in-hospital mortality rates for type A dissections is 26% for surgically managed, and 58% for medically managed (because of advanced age and comorbidities) patients. Mortality rates for type B dissection are 31.4% and 10.7% respectively. The overall 10 year mortality is about 55% in treated patients. In-hospital mortality remains high despite recent advances. Survival rates are improved by prevention, prompt diagnosis and timely management.

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Congenital diseases

Bent Østergaard Kristensen, Aarhus, Denmark

Congenital Heart Diseases (CHD) are the most common congenital defects, the incidence being about 0.8 %. The list is long, and this one is not complete:

Aortic stenosis (AS)
Atrial septal defect (ASD)
Atrioventricular (A-V) canal defect
Bicuspid aortic valve
Coarctation of the aorta ("Coarct")
Ebstein's anomaly
Hypoplastic left heart syndrome
Patent ductus arteriosus (PDA)
Univentricular Hearts
Pulmonary atresia
Pulmonary stenosis (PS)
Subaortic stenosis
Tetralogy of Fallot
Total anomalous pulmonary venous (P-V) connection
Transposition of the great arteries
Tricuspid atresia
Truncus arteriosus
Ventricular septal defect (VSD)

Today most CHD are diagnosed by fetal echocardiography. In experienced hands less than 5 % of CHD are missed at scanning between 18. and 21st gestational week. Recent reports show rather surprisingly, however, that a quite a number of babies with ductus dependent pulmonary or systemic circulation are discharged from the newborn nurseries undiagnosed. This emphasizes the great variety of symptoms in newborns.

***Pediatric echocardiography** requires great knowledge about patoanatomy and – physiology, e.g.: A volume loaded left ventricle can as in adults cover over an aortic or mitral valve incompetence, but also a VSD, PDA, Truncus arteriosus and an aortic-pulmonary window will cause overloading of the left ventricle. A hypertrophic right ventricle in a newborn is seen in left-sided obstructive diseases: Subaotic stenosis, AS and Coarct. One – two weeks later the strain has swifted over to the left heart side as in adults.*

An experienced pediatric cardiologist can diagnose most congenital heart malformations quickly and accurately with the help of the established echo modalities: M-mode, 2D echocardiography, pulsed- and continue wave and color Doppler. However, 2D fails whenever detailed information about spatial relationship of cardiac structures is necessary. Most patients with CDH are sent to surgery or catheter-interventions based on echocardiography. In a number of cases more information about complex intracardiac structures such as abnormal atrioventricular valves, an abnormal left ventricular outflow tract, size and exact location and relation to adjacent structures of intracardiac shunts is desirable. The introduction of the matrix technology has made it possible to obtain 3D data set of the heart within a few seconds, in which cross-sectional reconstructions as the "surgeon view" can be performed prior to surgery. Recent reports seem to show that 3D real time echocardiography enhances the ability to identify and pinpoint the exact locations of abnormalities. The scanplanes used in pediatric cardiology are similar to those used in adults, supplied with subcostal and suprasternal views. See www.ekkokardiografi.dk/kongenit.

ACYANOTIC CHDs WITH SYMPTOMS IN THE NEONATAL PERIOD:
Obstruction defects
Critial Pulmonary stenosis (PS), Aortic stenosis (AS), interrupted aortic arch (IAA), coarctation (Coarct), hypoplastic left heart (HLH) and others — , In these malformations treatment is needed soon after birth. A fast and accurate diagnosis made by echocardiography is mandatory, using parasternal short axis, Subcostal and suprasternal views. PS and AS may be treated with balloon valvuloplasty, otherwise surgery is needed. Prognosis is dependent on the size and function of the feeding ventricle.

Shunts
Patent ductus arteriosus (PDA) Normally this closes within a few hours of birth. A ductus that doesn't close is quite common in premature infants. In some children symptoms

may not occur until after the first weeks or months of life. Most PDA today are closed by catheter technique (Coils, Device) in babies above 6 kilograms. If surgery is needed, the surgeon can close the ductus arteriosus by tying it, without opening the heart.

Septal defects
***Atrial septal defect (ASD** secundum- or sinus venosus type) — Many children with ASD have few, if any, symptoms. About half of the secumdum types ADSs can be closed by catheter technique.*

***Ventricular septal defect (VSD)** — If the opening is small, it doesn't strain the heart. In that case, the only abnormal finding is a loud murmur. But if the opening is large, open-heart surgery is recommended to close the hole and prevent serious problems. Babies with VSD may develop severe symptoms or high blood pressure in their lungs. Repairing a ventricular septal defect with surgery usually restores normal blood circulation. The long-term outlook is good, but long-term follow-up is required.*

***Atrioventricular (A-V) canal defect** (also called endocardial cushion defect or atrioventricular septal defect) — A large hole in the center of the heart exists with a single large a-v valve. Because of the large amount of blood flowing to the lungs, high blood pressure may occur there and damage the blood vessels. In some babies the common valve between the upper and lower chambers doesn't close properly. Then regurgitation can occur on the right side, left side or both sides of the heart. In babies with severe symptoms or high blood pressure in the lungs, surgery usually must be done in infancy between 3 and 6 months of age. The surgeon closes the large hole with one or two patches and divides the single valve. Surgical repair of an atrioventri-cular canal usually restores the blood circulation to normal. Rarely, the defect may be too complex to repair in infancy. In this casea pulmonary artery banding to reduce the blood flow and high pressure in the lungs is necessary. When a child is older, the band is removed and corrective surgery is done. More medical or surgical treatment is sometimes needed.*

CYANOTIC CHDs WITH SYMPTOMS IN THE NEONATAL PERIOD
***Transposition of the great arteries** — The aorta is connected to the right ventricle, so most of the blood returning to the heart from the body is pumped back. The pulmonary artery is connected to the left ventricle, so most of the blood returning from the lungs goes back to the lungs again. Infants born with transposition survive only if they have one or more connections that let oxygen-rich blood reach the body: ASD, VSD, PDA. Most babies with transposition of the great arteries are extremely cyanotic soon after birth because these connections are inadequate. To improve the body's oxygen supply, a balloon atrial septostomy is performed. Two general types of surgery may be used to help fix the transposition. One is an arterial switch. Another is a venous switch or intra-atrial baffle procedure that creates a tunnel inside the atria: Mustard/Senning. After surgery, the long-term outlook varies. Lifelong follow-up is needed.*

***Tricuspid atresia** In this condition, there's no tricuspid valve. That means no blood can flow from the right atrium to the right ventricle. As a result, the right ventricle is small and not fully developed. The child's survival depends on there being an ASD and or a VSD. Often a surgical shunting procedure is needed to increase blood flow to the lungs. Some children with tricuspid atresia have too much blood flowing to the lungs. They may need a pulmonary artery banding. Other children with tricuspid atresia may have a more functional repair (Fontan procedure). Children with tricuspid atresia require lifelong follow-up by a cardiologist.*

***Pulmonary atresia** No pulmonary valve exists, so blood can't flow from the right ventricle into the pulmonary artery and on to the lungs. The right ventricle acts as a blind pouch that may stay small and not well developed. The tricuspid valve is often poorly developed, too.*

An opening in the atrial septum lets blood exit the right atrium, so venous (bluish) blood mixes with the oxygen-rich (red) blood in the left atrium. The baby appears cyanotic. The only source of lung blood flow is the PDA. Early treatment includes prostaglandins to keep the PDA from closing. A shunt between the aorta and the pulmonary artery to help increase blood flow to the lungs. A more complete repair depends on the size of the pulmonary artery and right ventricle. If they're very small, it may not be possible to correct the defect with surgery. In cases where the pulmonary artery and right ventricle are a more normal size, open-heart surgery may produce a good improvement. If the right ventricle stays too small to be a good pumping chamber, the surgeon can compensate by connecting the right atrium directly to the pulmonary artery. The atrial defect also can be closed to relieve the cyanosis. This is called a ***Fontan procedure.*** *Children with tricuspid atresia require lifelong follow-up by a cardiologist.*

Tetralogy of Fallot *has four components. The two major ones are a ventricular septal defect, that lets blood pass from the right to the left ventricle without going through the lungs; and a stenosis at or just beneath the pulmonary valve. The other two components are: the right ventricle is more muscular than normal; and the aorta lies directly over the ventricular septal defect. This results in cyanosis which may appear soon after birth, in infancy or later in childhood.*

Some infants with severe tetralogy of Fallot may need a shunt. This is done by making a connection between the aorta and the pulmonary artery. Most children with tetralogy of Fallot have open-heart surgery between 6 and 9 months of age. The operation involves closing the ventricular septal defect and removing the obstructing muscle. After surgery the long-term outlook varies, depending largely on how severe the defects were before surgery. Lifelong medical follow-up is needed.

Truncus arteriosus*— This is a complex malformation where only one artery arises from the heart and forms the aorta and pulmonary artery. Surgery for this condition usually is required early in life. It includes closing a large ventricular septal defect within the heart, detaching the pulmonary arteries from the large common artery, and connecting the pulmonary arteries to the right ventricle with a tube graft. Children with truncus arteriosus need lifelong follow-up to see how well the heart and valves are working.*

Total anomalous pulmonary venous (P-V) connection *—The pulmonary veins aren't connected to the left atrium. Instead, the pulmonary veins drain through abnormal connections to the right atrium. A supra-, infra- and cardiac form exist. Part of blood passes through the atrial septum into the left atrium. This defect must be surgically repaired in early infancy. The pulmonary veins are reconnected to the left atrium and the atrial septal defect is closed. When surgical repair is done in early infancy, the long-term outlook is very good. Still, lifelong follow-up is needed to make sure that any remaining problems, such as an obstruction in the pulmonary veins or irregularities in heart rhythm, are treated properly. It's important to make certain that a blockage doesn't develop in the pulmonary veins or where they're attached to the left atrium. Arrhythmias also may occur at any time after surgery.*

Hypoplastic left heart syndrome *In hypoplastic left heart syndrome, the left side of the heart is underdeveloped – including the aorta, aortic valve, left ventricle and mitral valve. The right ventricle pumps the blood into the pulmonary artery, and blood reaches the aorta through a patent ductus arteriosus. Duct dependent systemic circulation. Oxygenated blood passes from left to right atria through an ASD. The baby often seems normal at birth, but will come to medical attention within a few days as the ductus closes. This heart defect is usually fatal within the first days or months of life without treatment. Some babies can be treated with a series of operations or with a heart transplant. Until an operation is performed, the ductus is kept open by Prostaglandins. Surgery will be done in several stages. The first stage, called the* ***Norwood procedure****, allows the right ventricle to pump blood to both the lungs and the body. It must be*

performed soon after birth. The final stage(s) has many names ***including bi-directional Glenn, Fontan operation and lateral tunnel****. These operations create a connection between the veins returning blue blood to the heart and the pulmonary artery. The overall goal is to allow the right ventricle to pump only oxygenated blood to the body and to prevent or reduce mixing of the red and blue blood. Less than 30% of the babies survive the three stages. Children with hypoplastic left heart syndrome require lifelong follow-up by a cardiologist for repeated checks of how their heart is working.*

CHD detected later in life is dealt with in the lecture on CHD in Adults by Keld Soerensen.

For more detailed information about the echocardiographic feature of the different congenital heart diseases there is a lot of excellent websites. Just search on the net. The lecture will concentrate on selected cases with 3D presentations included.

The Echo Report

Heinz Tschernich, Vienna, Austria

The first and most burning question mostly been asked is: do we need to write an Echo-report?

The answer is yes, because

- *from a legal point a diagnostic examination requires a written report*
- *from a medical point a written report is needed for proper documentation to provide everyone of the patient´s medical team with the patient´s medical diagnosis*
- *unnecessarily repeated examinations can be avoided*

Should a written Echo report follow a standardized form?

Of course there are many – more or less – similar reporting forms available and for some instances the decision-making for the one or other alternative can meet subjective taste and interests. However, a reporting form should follow some important quality standards.

Which points should be listed, is there a hierarchy needed?

A hierarchy of a standard report form should list following things:
Patient´s personal data (first and last name, date of birth, patient ID, maybe height and weight for calculate BSA and to be able to index some measurements)

1. *Date and site of the Echo-examination*
2. *The referring question respectively reason, and the operation the patient is scheduled for*
3. *Type of echocardiography (TTE, TOE, epicardial scan,..)*
4. *Image-quality*
5. *Probe-insertion – adverse events (TOE)*
6. *Special procedures (e.g. Echo contrast)*
7. *Size and configuration of the cardiac chambers (visual assessment and measurements)*
8. *Presence of ventricular hypertrophy*
9. *Global systolic and diastolic function (RV, LV): visual assessment and quantification (fractional shortening, biplane Simpson´s rule method)*
10. *Regional ventricular function (wall motion abnormalities): RV, LV, which wall, which segment (basal, mid, apical), grading (normo-, hypo-, dys-, akinesia)*
11. *Valvular function: TV, MV, AV, PV(?), morphology, motion, grading of regurgitation, stenosis (visual), quantification (velocities, gradients)*
12. *Aorta: atheromatosis, dissection, diameter*
13. *Pericardial effusion: extent and location, hemodynamic impairment?*
14. *Other findings*
15. *Date of report*
16. *Reporting physician*
17. *Validating physician*

Paper work or software?

Current standard Echo-report forms are printout versions, however, there are several disadvantages, a software can solve: lack of clarity, no opportunities to be an educational tool, no data-base in background, therefore loss in information, no report-archive and no information that is fed in the hospital-PDMS.

Echo-reporting software – OpTEEmizer®

To overcome these limitations we have developed an Echo-reporting software (OpTEEmizer®) which fulfills following criteria:

- *Relational data-base*
- *Reporting archive*
- *Graphically based user-interface*

- *Self-explaining*
- *Educational- and training tool features*
- *Reporting either in the order of standard echo investigations (comprehensive or abbreviated- fig. 2) or in the conventional reporting hierarchy*
- *Displayed history and trends*
- *Automated translation from graphic user interface to a verbal Report printout form*
- *Export to a the hospital – PDMS*
- *Single-user or server version*

Heart-chambers – size and configuration

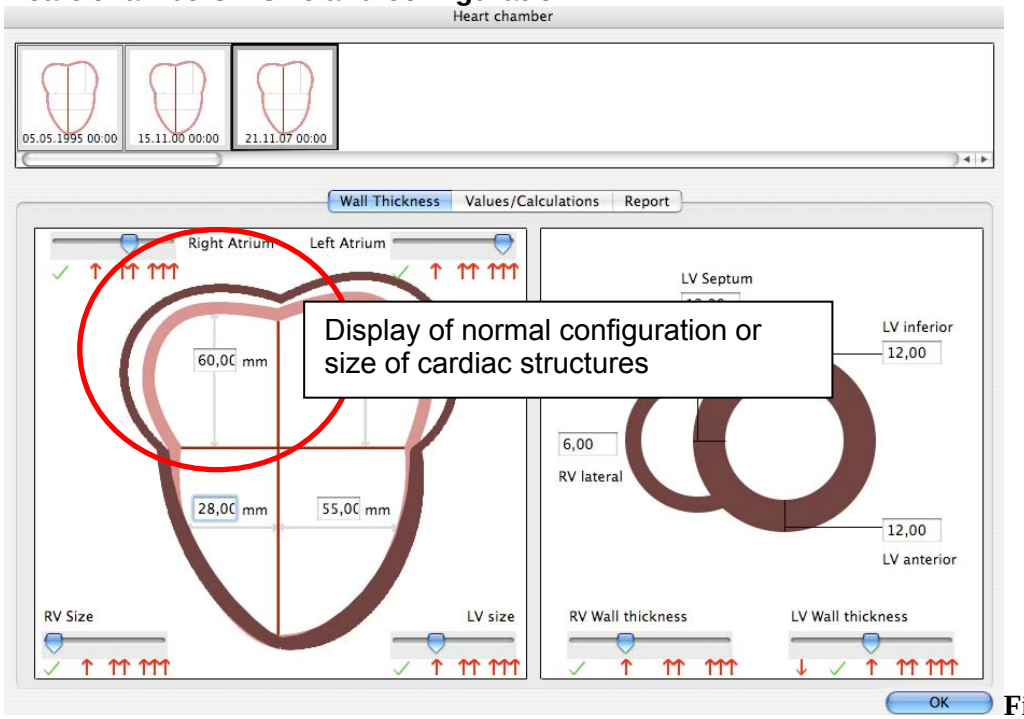


Fig. 1

Reporting by TEE-planes – ME 4CH

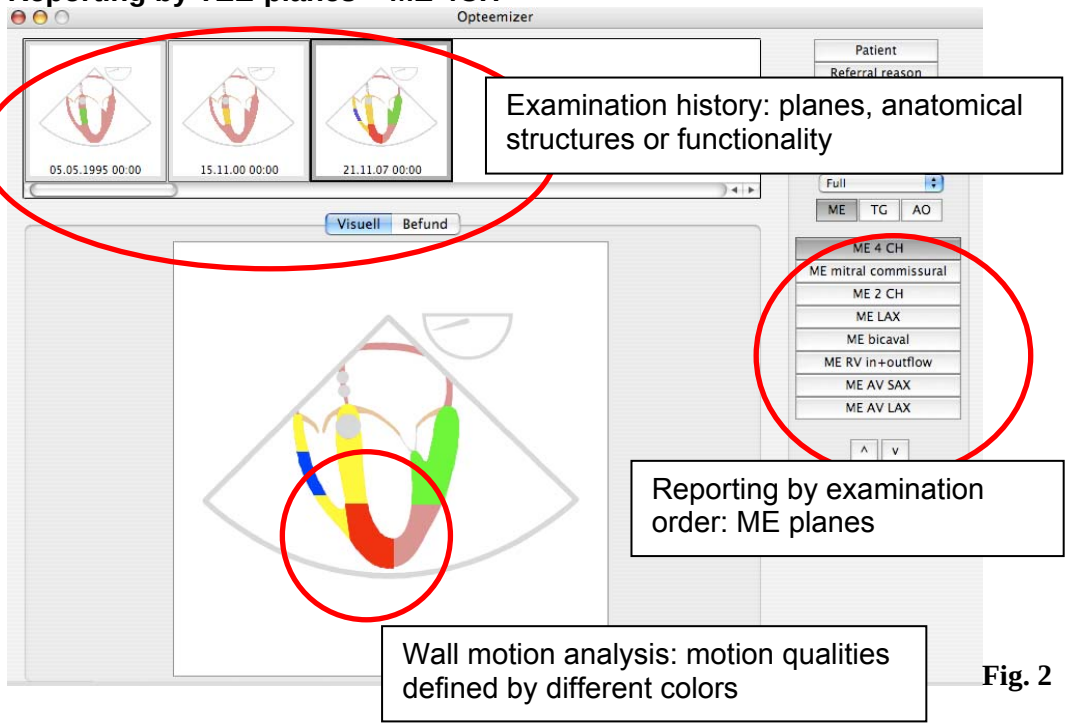
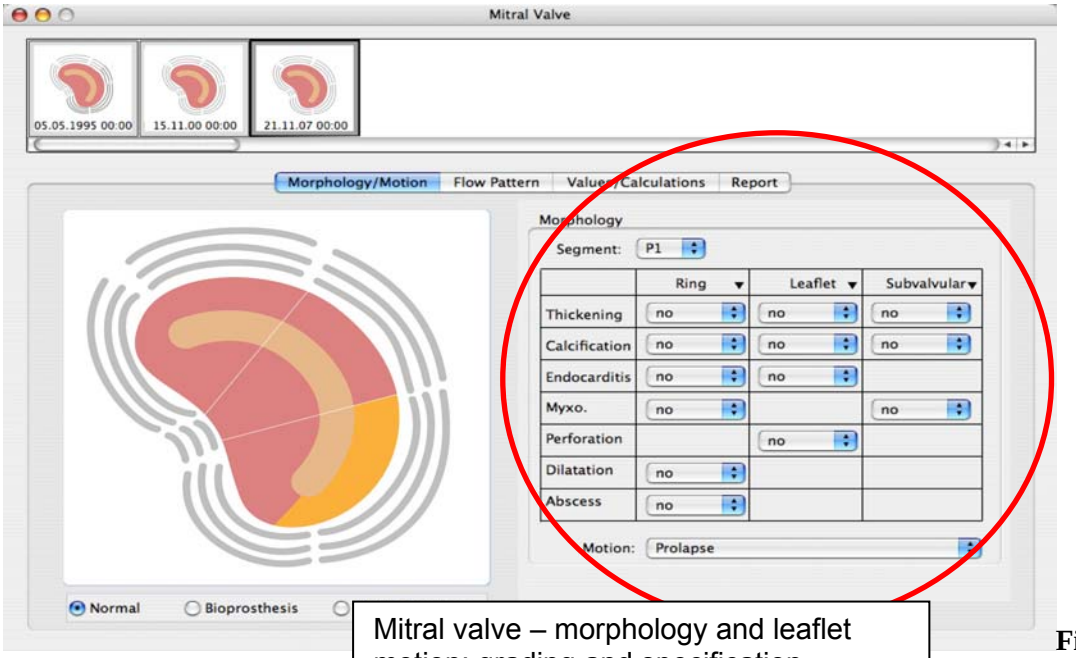


Fig. 2

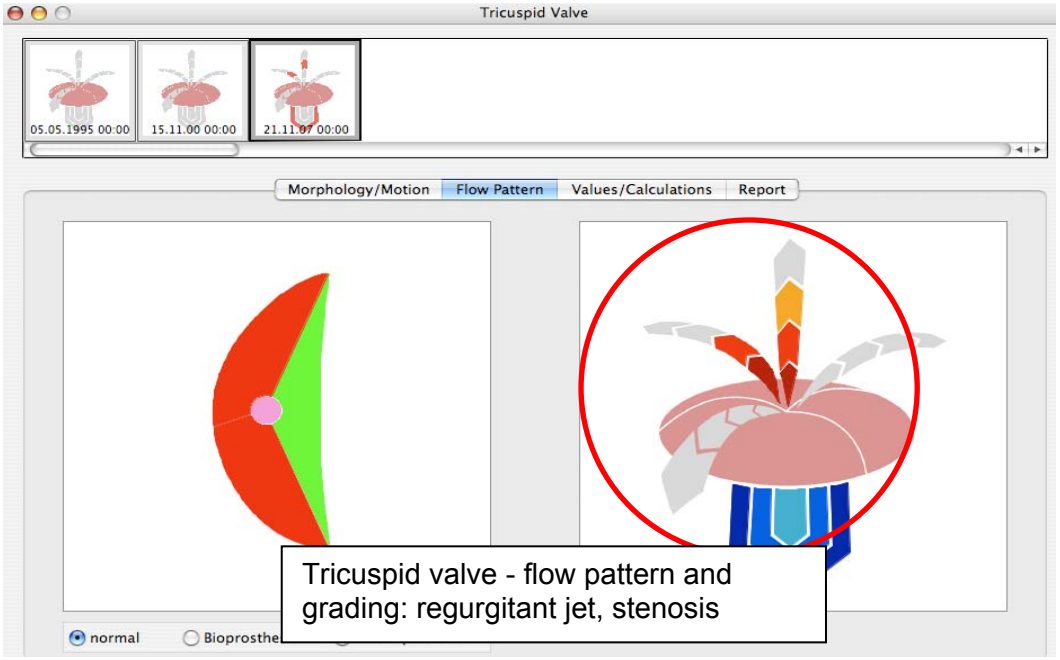
Mitral valve



Mitral valve – morphology and leaflet motion: grading and specification

Fig. 3

Tricuspid Valve



Tricuspid valve - flow pattern and grading: regurgitant jet, stenosis

Fig. 4

Aorta

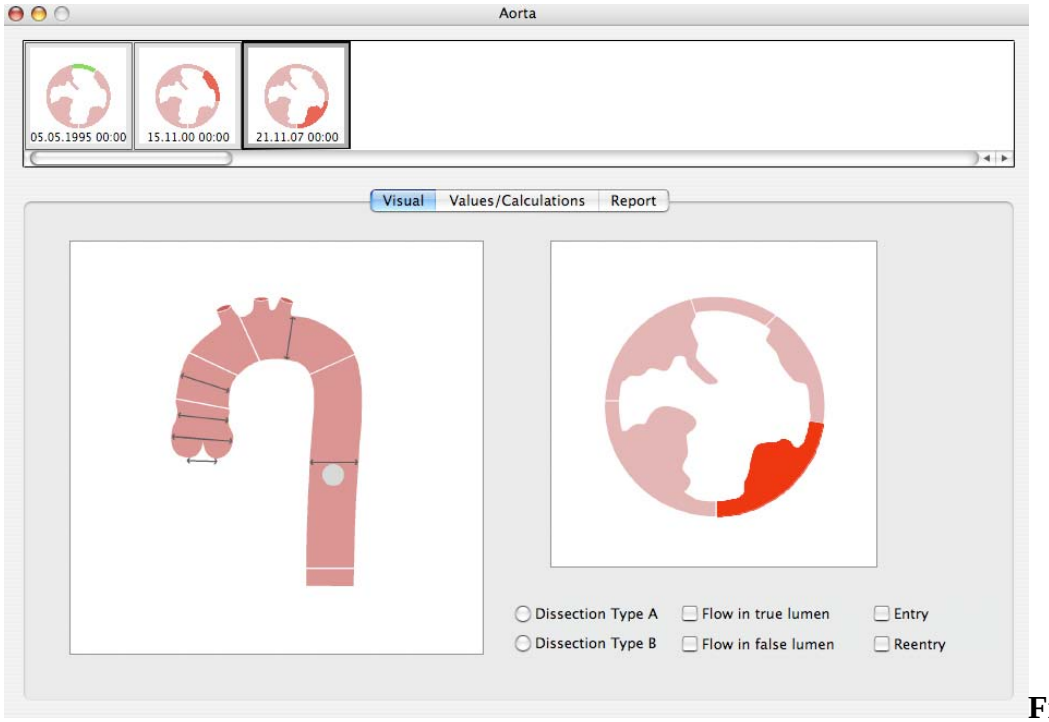


Fig. 5

Notes:

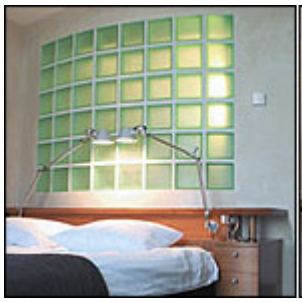
Abstracts by author

Radisson SAS Scandinavia Hotel, Copenhagen



Located only a few minutes away from Copenhagen's bustling city centre, our hotel is close to many of Copenhagen's attractions. The Copenhagen International Airport is only fifteen minutes away and the main railway station is also close by.

A good way to explore Copenhagen is by the pedestrian streets, or to take a guided canal boat tour, which passes castles, churches and several famous sights such as the Little Mermaid. Copenhagen is famous for its blend of metropolitan excitement and relaxed atmosphere. It offers an abundance of history, museums and beautiful sights and is also well known for its large selection of Danish design and fashion.



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INFORMATION ON GRANTS

EACTA has established an educational fellowship and/or a Research Grant funded to the level of 25000 Euros/year.
Research grant and clinical fellowship are open to application each year.
The EACTA Scientific Committee will evaluate applications provided they arrive to the Scientific Secretary (scisec@eacta.org)
no later than October 31.
EACTA Scientific Committee will score and rank applications (see below). The final decision comes under the EACTA directory board on January according to the Scientific Committee advice and possible splitting of the annual grant.
See more on www.eacta.org

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Location

Denmark is located in northern Europe between the North Sea and the Baltic Sea. It is the southernmost of the three Scandinavian countries and consists of a mainland peninsula, Jutland, and 406 islands.

The capital, Copenhagen, is situated on the island of Sealand and is the largest city in Scandinavia.

In total, Denmark covers an area of about 44.000 square km/17.000 square miles

Transfer from Copenhagen Airport

The hotel is located just 15 minutes from the Airport. There is a regular bus service directly to the hotel just outside Terminal 3.

Danish Currency

The Danish currency is the krone (DKK) which is divided into 100 oere.
Coins are circulated in the following denominations:
25 øre, 50 øre (copper)
1 krone, 2 kroner, 5 kroner (silver, each with a hole in the centre)
10 and 20 kroner (both brass)
Bank notes are issued in denominations of:
DKK 50, DKK 100, DKK 200, DKK 500 and DKK 1.000

Health Insurance

As a citizen of an EU country visiting Denmark, you are covered by public health insurance within the limits agreed upon between your own country and the Danish authorities.

Also, as a temporary foreign visitor, you are entitled to free medical treatment in hospitals and emergency wards if you are taken ill or have an accident, provided that you have not travelled to Denmark with the intention of obtaining treatment and are physically unable to return to your own country.

If you are a citizen of a non-EU country, you should ensure that you have adequate health insurance. Check with your travel agency or your insurance company.

Tipping

Service is normally included in restaurant, hotel and taxi bills, so any further tip should only be given for exceptionally good service.

It is not uncommon, however, to round up the bill.

Opening hours

Opening hours are at the discretion of proprietors. But certain rules must be followed:
Monday-Friday from 09.30/10.00 to 17.30/18.00/19.00 (9.30/10 am - 5.30/6/7 pm)
Saturday from 09.00 to 15.00/16.00 (9 am - 3/4 pm)
Sunday only bakeries, florists and souvenir shops are open.

Office hours
are usually 09.00 to 16.00/16.30 from Monday to Friday (9 a.m. - 4/4.30 p.m.)

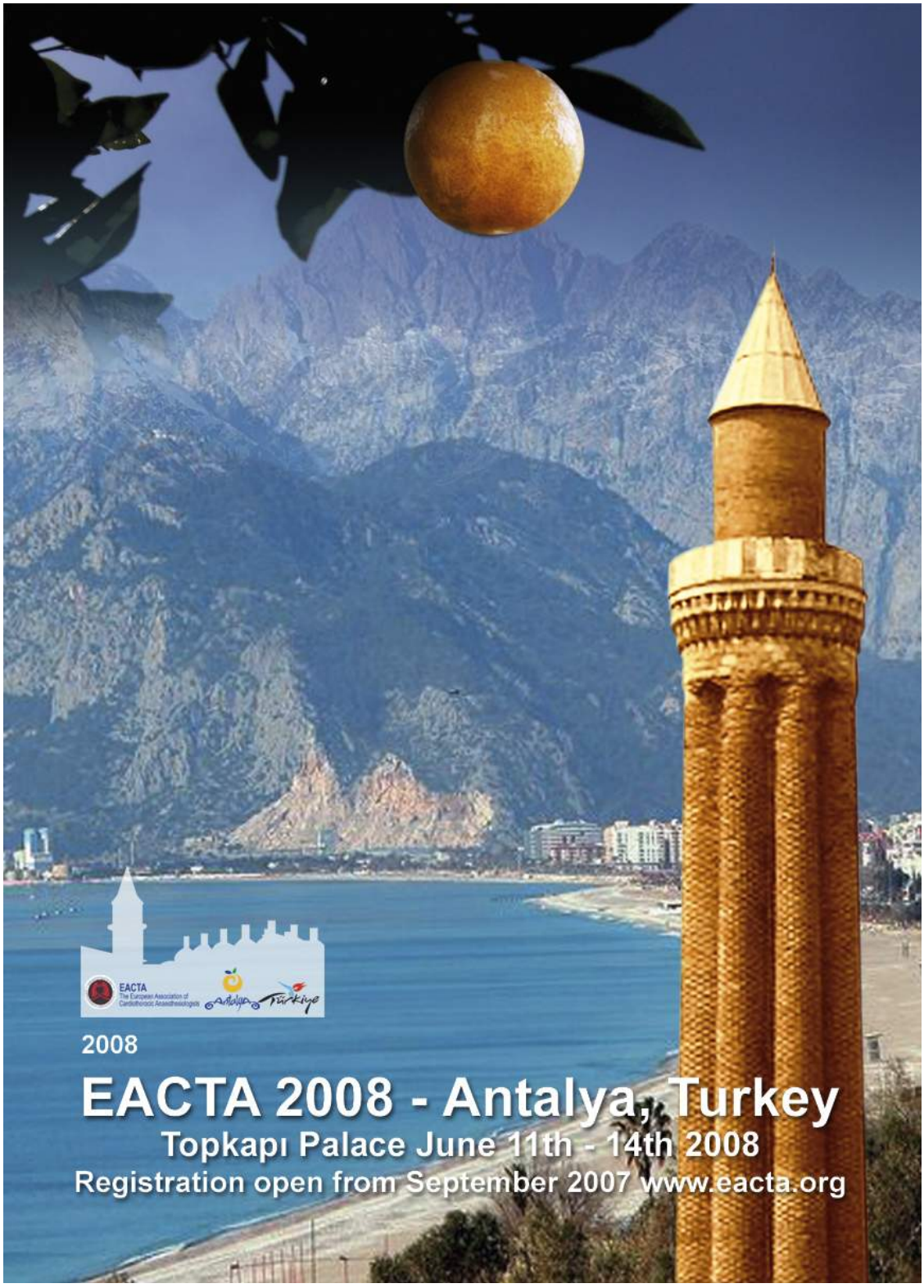
Transportation

In the Greater Copenhagen area, you can transfer freely between buses, Metro, trains and the yellow "harbour bus" using the same tickets and discount cards.

Ordinary basic tickets can be purchased from vending machines at stations, ticket offices and on buses from the driver.

Alternatively, you can purchase a CPHCARD which is valid for any travel within the Greater Copenhagen area. There are two types: valid 24 hours or 72 hours.

EACTA 2008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