EACTA ECHO'08
European Association of Cardiothoracic Anaesthesiologists
Focus on accreditation
Rome, 27-30 September 2008

SYLLABUS EACTAECHO 2008 Rome, Italy

EBAC®

21 h CME external credits

Edited by
Fabio Guarracino, Pisa, Italy
Copyright©EACTA, Dublin, Ireland

Dear Collegues,

it is a great pleasure for us to have you all at the 7th EACTAECHO Annual Course In Rome.

For the first time since the first EACTAECHO in 2002, we will have a course run by EACTA and EAE together. The joint faculty will provide a course on perioperative transoesophageal echocardiography strongly oriented to prepare for the TOE Accreditation Exam. The course will be didactic for those less experienced with TOE, but also new developments will be addressed. The programme is divided into two parts: on the first two days all the topics on TOE will be covered, while on day three and four some more "anaesthesiological-ICU" aspects of echocardiography will be addressed. Extensive opportunity for hands-on TTE will be provided with participants divided in small groups.

With this course EACTA and EAE are pleased to be able to highlight their commitment to provide 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 EACTA and EAE.

We really hope this course will meet your expectations and, last but not least, give you the opportunity to visit "the eternal city"!

Fabio Guarracino
Chairman EACTA-ECHO' 08

Marco Ranucci
President EACTA

Petros Nihoyannopoulos
President EAE

Rob Feneck
Chairman, EACTA ECHO Committee

Faculty

Beldekos D, Greece
Dominique Bettex, Switzerland
Emanuele Catena, Italy
Pio Caso, Italy
Joachim Erb, Germany
Robert Feneck, UK
Fabio Guarracino, Italy
Gilbert Habib, France
George Karatasakis, Greece
John Kneeshaw, UK
Luca Lorini, Italy
Isabelle Michaux, Belgium
Owen Miller, UK
Petros Nihoyannopoulos, UK
Mauro Pepi, Italy
Bogdan A. Popescu, Romania
Marco Ranucci, Italy
Maanfred D. Seeberger-Stucky, Switzerland
Erik Sloth, Denmark
Justiaan Swanevelder, UK
Heinz Tschernich, Austria
Patrick Wouters, Belgium
Andrea Ziegler, Germany

"The event "*EACTAECHO 2008*" is accredited by the

European Board for Accreditation in Cardiology (EBAC)

for "*21*" hours of External CME credits.

"In compliance with EBAC/ EACCME guidelines, all speakers/ chairpersons participating in this programme have disclosed potential conflicts of interest that might cause a bias in the presentations. The Organising Committee is responsible for ensuring that all potential conflicts of interest relevant to the programme are declared to the audience prior to the CME activities."

We are grateful to our Sponsors for their support

Table of Contents

Physics of Ultrasound and Knobology

Dr. med. Joachim M. Erb, DEAA,
Universitätsklinik für Anästhesiologie mit Schwerpunkt op. Intensivmedizin,
Campus Charité Mitte, 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: $\mathbf{c = \lambda \bullet f}$, 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 $\Rightarrow$ 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 $m \cdot s^{-1} \Rightarrow$ 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 approx. 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

Indications, Contraindications , Safety and Complications of TEE

Dr Rob Feneck,
Guys and St Thomas' Hospitals, London UK

INDICATIONS FOR TEE EXAMINATION

The first report on the indications for perioperative TEE was published in 1996 by the American Society of Anesthesiologists (ASA) and the Society of Cardiovascular Anesthesiologists (SCA). [1]. In these guidelines, the authors addressed the indications for TEE based on the level of evidence supporting their use. These levels of evidence were divided into 3 categories;

- Class 1 evidence: conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective.
- Class IIa evidence: where the weight of evidence/opinion is in favor of usefulness/efficacy.
- Class IIb evidence:
- Class III evidence

The indications for perioperative TEE have been subject to a more recent report, and recommendations from the same body were published in 2003. [2] Using a similar system of analysis, the authors identified publications supporting the use of TEE in the perioperative period, and ranked the evidence according to the three categories described. The authors identified a total of 706 publications that have contributed to the evidence base on intraoperative TOE. After due analysis, they have identified Class 1 evidence for seven perioperative indications and Class IIa evidence for 3 perioperative indications. Given that some of the conditions considered are pathophysiological states, for example the. risk of ischemia and/or myocardial infarction, rather than specific diagnoses, these 10 indications actually cover the majority of cardiac surgery in adults and paediatrics.

Notwithstanding the clear authority of these guidelines, other important contributions include the following;

- Savage et al [3] demonstrated that in high risk coronary patients the routine use of intraoperative TEE resulted in major changes in surgery in 33% of the patients, and in major changes in the haemodynamic management in 51% of the patients.
- Mishra et al [4] examined 5,025 patients, including 3,660 coronary bypass (CABG) surgeries and 1,365 valve surgeries. Routine TEE examination before cardiopulmonary bypass (CPB) led to a major change in surgical planning in 5 % of the coronary cases and in 3.5 % of the valve cases. Following cardiopulmonary bypass (CPB) , haemodynamic interventions were introduced on the basis of the TEE – derived information in 26 % of the CABG patients and in 10.5% of the valve patients.
- Click et al [5] reported unexpected findings before CPB in 15% of the patients undergoing routine TEE during cardiac surgery. In 95% of the cases, these new findings resulted in major changes in the planned surgery. In the same series, 4% of the patients required major changes in surgery including a second run on CPB, or complex haemodynamic management after CPB.
- Schmidlin et al [6] reported a series of 2,296 cardiac operations with the routine use of TEE: 9.6% of the patients received additional surgery and 49% required changes in the haemodynamic management based on TEE assessment and monitoring.

The incidence of new intraoperative findings during a TEE examination has been identified as a reason for routine TEE examination during cardiac surgery, but also as a cause for concern. Clearly, a full preoperative examination and work-up should remove the possibility of unexpected and new findings on intraoperative TEE examination. However, the demonstration of new information is a common finding in many series of patients, and from major institutions . This does not necessarily imply an inadequate pre-operative study, but may be due to the higher definition of a TEE examination in patients with poor transthoracic acoustic windows, the non-routine use of preoperative TEE in the majority of institutions, and recent changes that may have occurred in the

clinical status of the patient. The presence of a new finding on perioperative TEE has frequently led to an important change in surgical planning (from 7% to 14%).
Routine intraoperative use of TEE has been shown to lead to an improvement in both the surgical and anaesthesiological management. In high-risk coronary surgery it has been demonstrated that the routine use of TEE leads to a significant decrease in both perioperative myocardial infarction rate and mortality . Clearly, for TEE to be routinely available during cardiac surgery there needs to be both adequate equipment and sufficient trained operators, both of which may be a major limitation to the provision of a comprehensive service. In such a situation, the perioperative TEE service may need to be limited to Class I indications.

Safety issues
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%. (7.8) 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.
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 (9). In contrast, others have shown an increased incidence of dysphagia (10) and a further study identified age, duration of endotracheal intubation, and use of TEE as significant predictors of postoperative dysphagia.(11)
7. Airway obstruction may occur, particularly in paediatric patients. An incidence of 1-2% has been reported (12), 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 (13), and 0.18% of anaesthetised and ventilated adult patients.(14)
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.

References

1. Cheitlin MD et al : J Am Coll Cardiol. 1997 ;29(4):862-79.
2. Cheitlin MD et al Circulation. 2003 ;108(9):1146-62.
3. Savage RM et al Ann Thorac Surg 1997;64:368-73
4. Mishra M et al. J Cardiothorac Vasc Anesth 1998:12:625-32
5. Click RL et al : Mayo Clin Proc. 2000 ;75(3):241-7.
6. Schmidlin D et al Br J Anaesth 2001,86:497-505
7. Daniel WG et al. Circulation 1991:83:817-821
8. Silvis SE et al. JAMA 1976 ;235 :928-930
9. Messina AG et al. Am J Cardiol 1991;67:313-314
10. Rousou JA et al. Ann Thorac Surg 2000;69:486-489
11. Hogue CW et al. J thorac Cardiovasc Surg 1995;110:517-522
12. Stevenson JG. J Am Soc Echocardiogr 1999;12:527-532
13. Green MA et al Chest 1999;116;1247-1250
14. Kallmeyer I et al. Anesth Analg 2001;92:1126-1130

Comprehensive TEE Examination

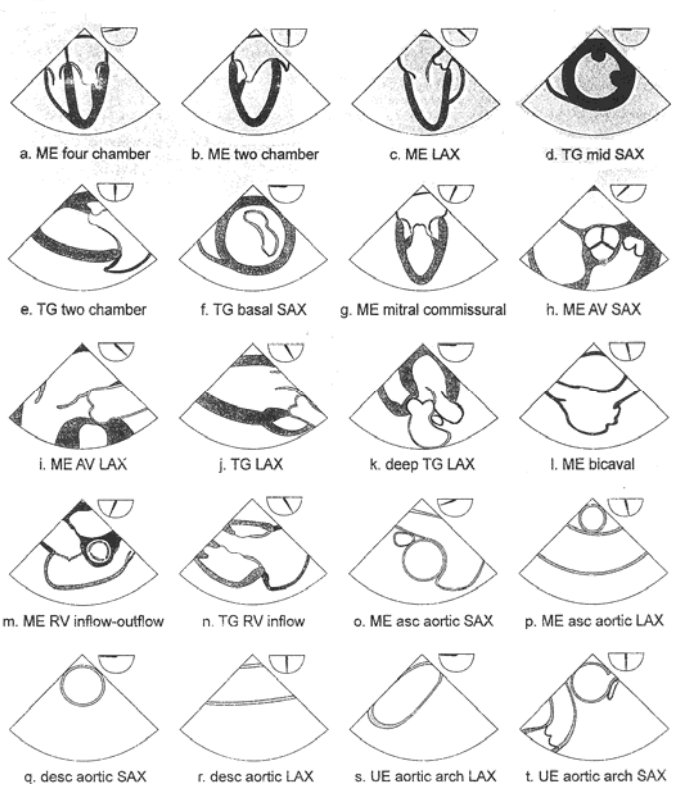
Justiaan Swanevelder,
Glenfield Hospital, University Hospitals Leicester, UK

History

Frazin, a cardiologist from Loyola University in Illinois, USA, published the first report of transoesophageal echocardiography in 1976. Over the past twenty years technology has improved tremendously and intra-operative TOE has become the gold standard of intra-operative cardiac diagnosis and monitoring in many cardiac procedures, e.g. mitral valve repair, congenital heart surgery, etc.

Basic examination

In 1999 an American Society of Echocardiography/Society of Cardiothoracic Anesthesiologists Task Force published guidelines for performing a comprehensive intra-operative TOE examination.[1] They recommend 20 basic views.



This was followed by similar guidelines from an echocardiography working group of the European Society of Cardiologists.[2] Some of the more commonly used views are:

- Upper oesophageal basal short axis view (0-20 degrees) of the pulmonary artery bifurcation and the ascending aorta: This “inverted trousers” image is good for PA investigation and cardiac output measurements.

- Upper oesophageal basal short axis view through the LA (0 degrees). Visualisation of the LA appendage and pulmonary veins is possible.

- On the coronary artery view (0- 40 degrees) it is possible to see the coronary arteries and the RA appendage. The pulmonary vein can also sometimes be visualised.

- When the probe is advanced further, the mid-oesophageal short-axis view of the aortic valve (40 degrees) is visible, and the different cusps can be examined.

- If the angle of the transducer is changed to 140 degrees the mid-oesophageal long-axis view of the left ventricular outflow tract, aortic valve and ascending aorta is visible.

- Further advancement of the multiplane probe at 0 degrees will show the mid-oesophageal 5-chamber view of the heart. The "5^{th}" chamber is the LV outflow tract. The aortic valve, mitral valve leaflets, papillary muscles and parts of the RV and RA can be examined in this view.

- At this point or a little more down with mild retroflection of the probe the classical mid-oesophageal 4-chamber view (0 degrees) of the atria and ventricles is visible. Extreme retroflection will show the coronary sinus.

- Advancement of the probe into the stomach will show the transgastric short axis basal view of the left ventricle (0- 20 degrees). This is good for evaluation of the mitral valve ("fish mouth" view). A little bit further down LV function and possible wall motion abnormalities (transgastric midpapillary short-axis view of the LV) can be assessed. When rotating the angle of the transducer to 90 degrees al these structures can be examined in the long-axis (transgastric mid-papillary long-axis and transgastric basal long-axis views of the LV).

- Further advancement of the probe with extreme flexion will display the deep transgastric view. It is possible to measure the pressure gradient across the aortic valve in this view (0- 20 degrees).

- From the 4-chamber view the probe can be turned to the left to visualise the descending aorta in the short-axis (0 degrees). This structure can be followed down to where it become the abdominal aorta. On withdrawal of the probe the aorta can be evaluated up to the point where the descending arch starts. Unfortunately the arch itself cannot be examined due to the trachea blocking the view. If the plane is rotated to 90 degrees, a long-axis view of the descending aorta will be visualised. The superior end will be on the right hand side, while the inferior side will be on the left side of the screen. This view is ideal for diagnosing descending aortic dissections.

At the level of the 4-chamber view, the biplane probe can be rotated to 90 degrees (vertical plane). The vertical plane anatomy is examined by left and right manipulation of the probe, with little flexion-extension movement. The top of the video screen is the posterior side of the patient in close relationship to the oesophagus. The bottom part of the screen will be anterior. The left and right sides of the screen will be the inferior and superior sides of the patient respectively.

- The mid-oesophageal 2-chamber view (90 degrees) provides good visualisation of the mitral valve, LA and LV. The LA appendage and left upper pulmonary vein can also be examined.

- If the transducer is turned to the right, the RV inflow-outflow view (90 degrees) can be seen. This view is good for examination of the pulmonary and tricuspid valves, right ventricular outflow tract and pulmonary artery.

- The bi-caval or bi-atrial view (90 degrees) is obtained by more clockwise rotation of the probe. The SVC is on the right side of the screen, while the IVC will be on the left side. Both the LA and RA are visible. This view is ideal to diagnose an ASD or other inter-atrial septum pathology.

There may be a variable appearance of the different views between different patients. When specific pathology is expected, a targeted but complete examination must be done. The introduction of multiplane technology has made many more examination windows possible, but it does take more time and practice to get use to the different multiplane views. In every view, colour-flow Doppler, pulse-wave Doppler and continuous-wave Doppler should be applied across valves, vessels and shunts as necessary, to measure and confirm flow, flow velocities and turbulence.

In recent years real-time 3D echocardiography (RT 3DE) has been an exciting addition to our armamentarium. It's availability may significantly reduce the number of steps required to complete a standard echocardiographic examination, thus reducing the examination time. It has also been shown to be feasible with potentially a significant impact on clinical decision-making. RT 3DE appears to be particularly valuable in patients with congenital heart disease and mitral valve disease.[3] Automated online measurement of the ventricles will also provide a much more objective assessment of volumes and function. However, there is a learning curve for the echocardiographer and suboptimal image data remains a problem in some patients. As 3D image quality improves, a single volume acquisition may be sufficient to provide unique 3D views and allow comprehensive 2D evaluation in any plane extracted from this 3D dataset.[4] I suspect that the integration of the 3D modality in 2D transoesophageal echocardiography transducers will lead to a combined 2DE and 3DE examination becoming routine in clinical practice.

Training and quality assurance

Intra-operative TOE is a new addition to the anaesthetist's skills. Special dedicated training is therefore needed. This can be obtained with a dedicated training period consisting of in-theatre training, books, videotapes, courses offered by specialist units, computer learning programmes and a close working relationship with your local cardiology/radiology department. There must always be proper backup available by an experienced echocardiographer. The National Board of Echocardiography in the USA developed the first formal examination in perioperative TOE in 1998 that can be taken to prove competence and forms part of a certification process. The Association of Cardiothoracic Anaesthetists together with the British Society of Echocardiography (ACTA/BSE) followed with a similar accreditation process in 2003. Since then the European Association of Cardiothoracic Anaesthesiologists (EACTA) and the European Association of Echocardiography (EAE), and the Japanese Society of Cardiovascular Anesthesiologists launched their own processes respectively in 2005 and 2004.

There is potential for individuals who are not fulltime echocardiographers to make the wrong diagnosis.[5] Serious commitment of time and effort is necessary by anaesthetists who want to perform these procedures. Every TOE study should be recorded in an appropriate fashion e.g. a written report in the patient's medical records, a video tape or a digital image and a data base entry. Some of this data should be routinely sampled and examined by an experienced echocardiographer to make certain it is of a high standard. Intra-operative TOE is here to stay. Technology is improving at a tremendous pace and will probably become more user friendly in years to come. For any cardiac anaesthetist (and others) it will become important to conquer this skill at least at a basic level.

References

1. Shanewise JS, Cheung AT, Aronson S, et al. ASE/SCA guidelines for performing a comprehensive intra-operative multiplane transesophageal echocardiographic examination: recommendations of the ASE and SCA. Anesth Analg 1999;89:870-84
2. Flachskampf FA, Decoodt P, Fraser AG, et al. Guidelines from the working group: Recommendations for performing transoeophageal echocardiography. Eur J Echocardiography 2001;2:8-21
3. Hare JL, Jenkins C, Nakatani S, et al. Feasibility and clinical decisionmaking with 3D echocardiography in routine practice. Heart 2008;94:440-45
4. Suseng L, Mor-Avi V, Lang RM. Three-dimensional echocardiography: coming of age. Heart 2008;94:23-25
5. Miller JP, Lambert AS, Shapiro WA, et al. The Adequacy of Basic Intraoperative Transesophageal Echocardiography performed by experienced anaesthesiologists. Anes Analg 2001;92:1103-10

Left ventricular systolic function.

Fabio Guarracino
Department of Cardiothoracic Anaesthesia and ICU,
University Hospital of Pisa, Italy.

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%.

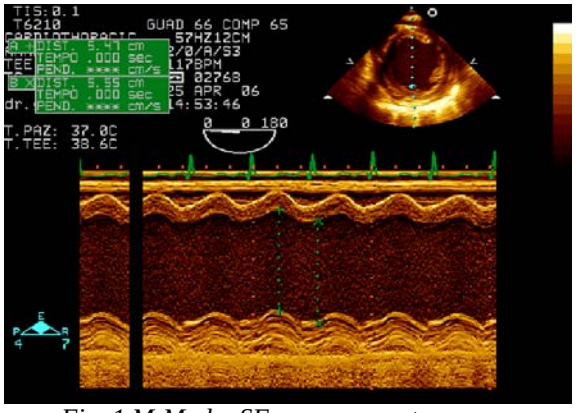


Fig. 1 M-Mode: SF measurement

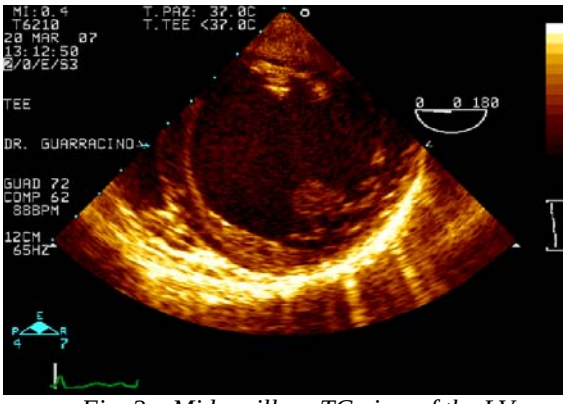


Fig. 2 a Midpapillary TG view of the LV

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, 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.

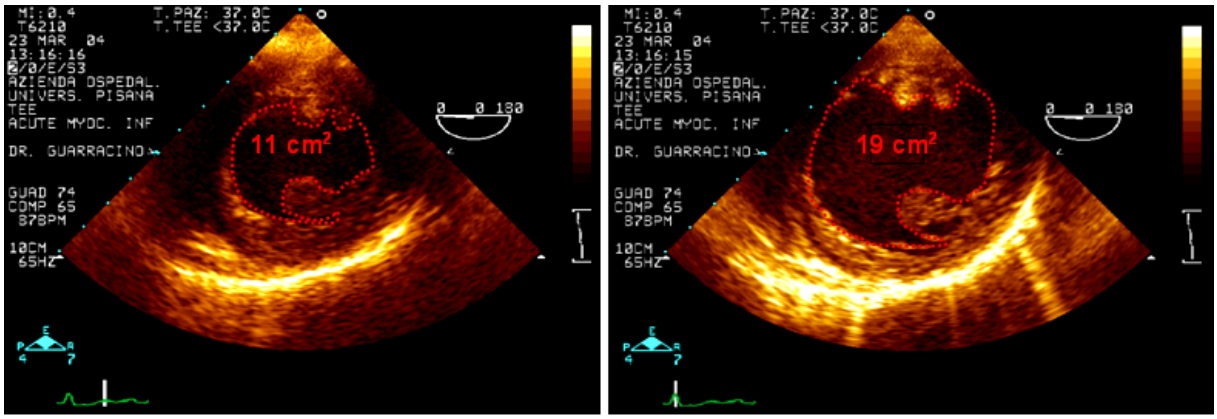


Fig. 2 b Measurement of LV end-diastolic and end-systolic areas.

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).

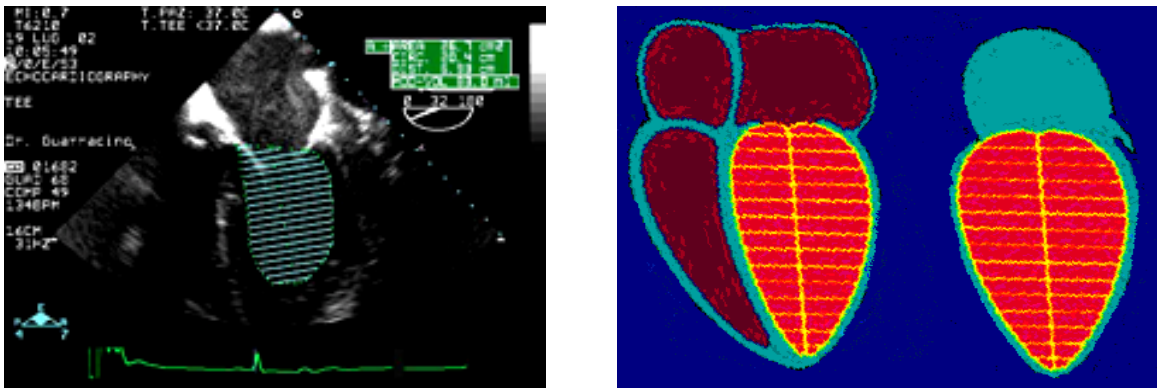


Fig. 3 Long-axis four chamber view of LV for EF measurement.

The normal value 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:

$CSA = \pi \bullet (diameter / 2)^2$ (fig. 4 a).

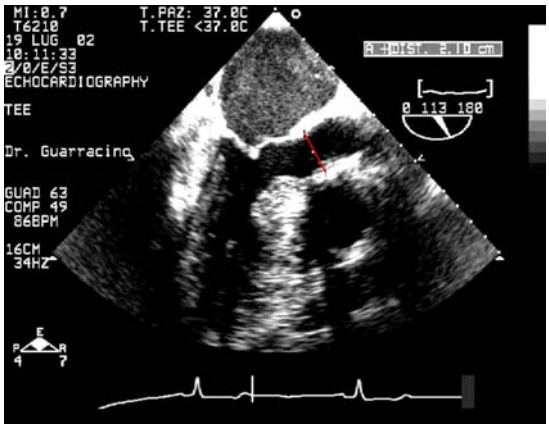


Fig. 4 a LAX at 120°: measurement of CSA

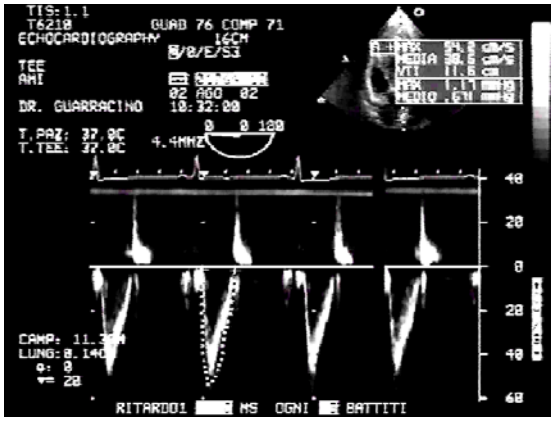


Fig. 4 b VTI measurement by Doppler

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.

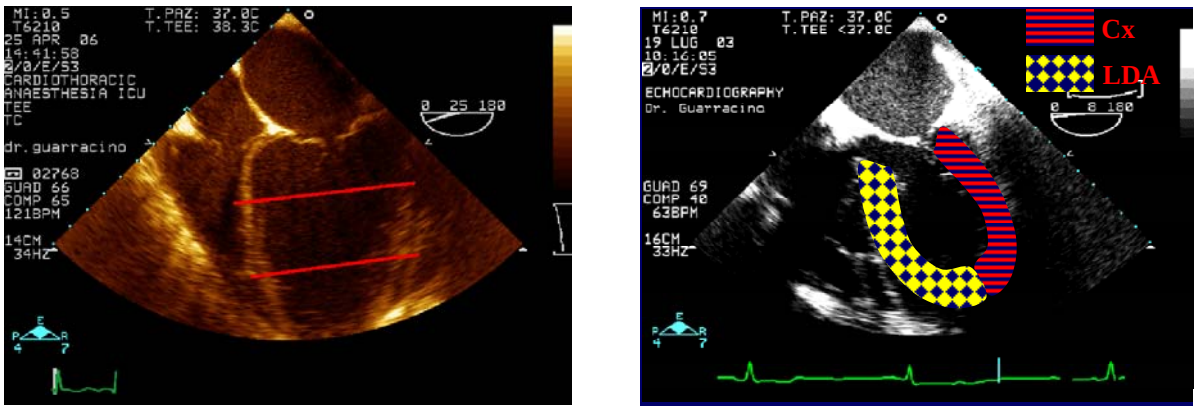


Fig. 5 LV segmentation for regional wall motion analysis

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.

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.

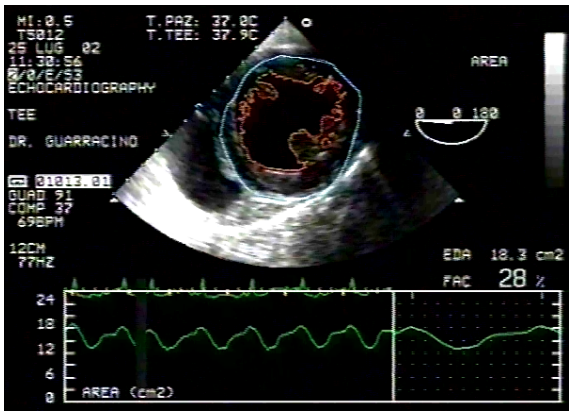


Fig. 6 Acoustic quantification of LV

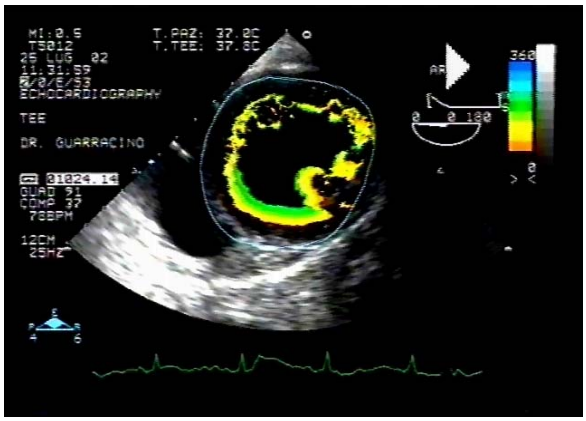


Fig. 7 Colour Kinesis of LV

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.

Echocardiographic assessment of LV Diastolic Function

Patrick F Wouters MD, PhD
Department of Anesthesia
University of Ghent, Belgium

The diagnosis of diastolic heart failure requires three conditions: (1) the presence of signs/symptoms of heart failure; (2) a normal or slightly reduced LV systolic function (LVEF > 50% and LVend-diastolic volume index (LVEDVI) < 97 mL/m2) and (3) evidence of diastolic dysfunction. It is also referred to as heart failure with normal ejection fraction (HFNEF). Large-scale epidemiological studies show that the incidence of primary diastolic heart failure is rather high, particularly in the elderly, and accounts for half 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 for normal or sometimes even reduced end-diastolic volumes. The higher filling pressures ultimately lead to pulmonary congestion – particularly when heart rate increases e.g. in response to exercise or stress. The prognosis of diastolic heart failure is similar to that associated with systolic failure and morbidity rates are even higher.

There is no specific therapy to selectively improve LV diastolic function in a direct fashion. Treatment of the underlying disease (myocardial ischemia, arterial hypertension, aortic valve stenosis) is often still the most effective therapeutic approach, although recent data suggest that calcium channel blockers, beta-blockers, ACE-inhibitors and AT2-blockers as well as nitric oxide donors may be particularly useful.

Invasive assessment of diastolic function includes the determination of the time constant of relaxation (tau) from the exponential pressure decay during isovolumetric relaxation, and the passive elastic properties of the heart from the slope of the diastolic pressure–volume (constant of chamber stiffness) and stress–strain relationship (constant of myocardial stiffness). Doppler Echocardiography has become a primary non-invasive clinical 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 established diastolic dysfunction and adapt our hemodynamic management accordingly. The difficulty in studying diastolic function in surgical patients is related to the continuously changing loading conditions because preload changes have an independent effect on almost all echocardiographic variables used to quantify diastolic performance.

Basically there are four techniques to evaluate diastolic performance, i.e. analysis of

1) Transmitral flow patterns using pulsed wave Doppler
2) Pulmonary venous flow patterns with pulsed wave Doppler
3) Mitral annular motion using tissue Doppler imaging (either with pulsed or color wave Doppler)
4) LV inflow propagation velocity using color Doppler M-mode

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

The transmitral flow pattern is easy to acquire and rapidly classifies patients with normal diastolic function, delayed relaxation, or a restrictive filling pattern. It is important to 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.(Figure 1) Signal gain needs adjustment to obtain a clear spectral envelope and the ECG tracing must be displayed on the screen to properly relate the signal components to distinct 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) and the subsequent rise in LV pressure subsequent to filling. In healthy adults peak E velocities are typically higher than the second component of the mitral inflow signal which relates to atrial contraction (A-wave) and the filling pressures in the LV. 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 a reduction of peak E velocities, a lower ratio between peak-E and peak-velocities and a prolonged deceleration time of the E wave component. (Figure 2) The natural adaptive response to this condition is an elevation of atrial pressures which restores the early transmitral pressure gradient. As a result, transmitral flow variables (i.e. E and A velocities, E/A ratio and E deceleration time) will paradoxically return to near-normal levels, despite a further progression of disease. This stage is referred to as 'pseudonormalization' because the mitral inflow profile cannot be distinguished from the normal pattern - except for the fact that it is associated with elevated atrial pressures (the compensatory response) (Figure 3). At this stage, additional measurements are required – either by invasive assessment of filling pressures or by using one of the other echo-techniques described below which offer clues to discern normal from pseudonormal transmitral flow patterns. 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 all of which indicate restrictive filling.(Figure 2)

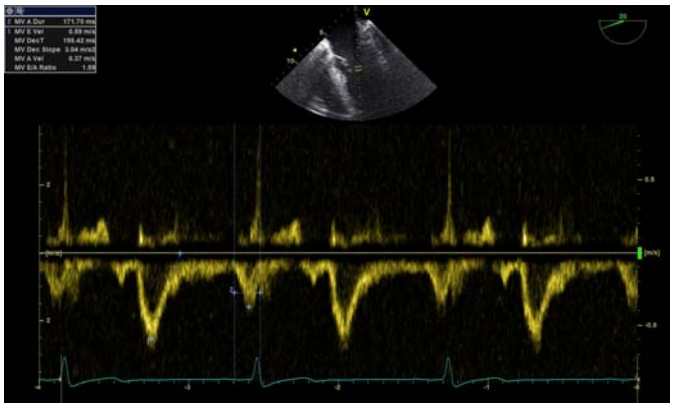


Fig 1

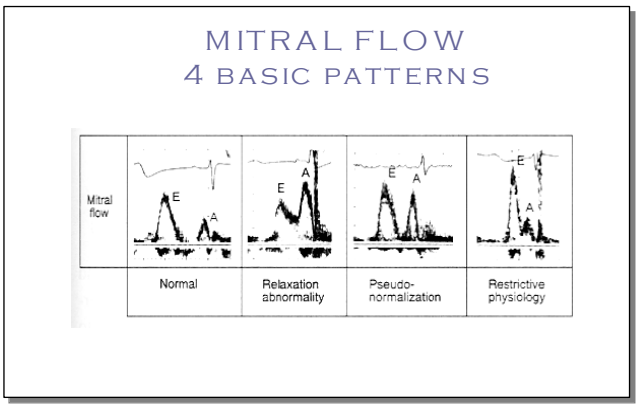
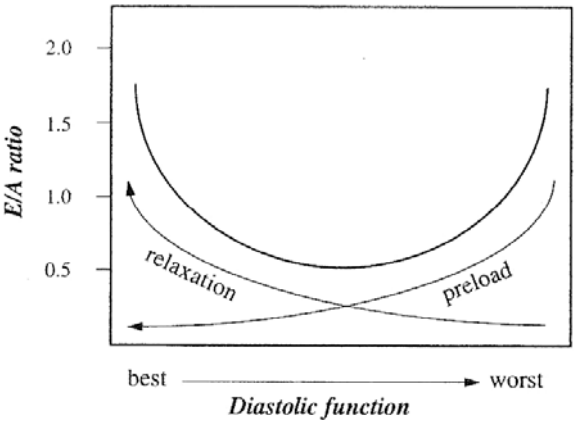


Fig 2

Pulmonary venous (PV) flow velocities reflect the instantaneous pressure differences between the pulmonary veins and the left atrium. The flow pattern consist of a systolic (Spv-often biphasic), a diastolic (Dpv) and an atrial (Apv) component. The diagnostic utility of the diastolic Dpv-wave is comparable with that of the mitral E-wave (Figure 4) The atrial component, however, increases 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 Mitral atrial component is directly related to the prevailing atrial pressure (Rossvoll and Hatle JACC 1993). Hence, this variable adds useful information to the study of transmitral flow profiles in differentiating a normal from a pseudonormal MV flow signal. However, A-wave characteristics are also affected by atrial function (e.g. atrial stunning) and are unreliable in patients with non-sinus rhythm.

Two more recently developed techniques (described below) are claimed to have a higher diagnostic potential for the analysis of diastolic function because the measurement variables are less affected by loading conditions hence more directly related to the severity of diastolic disease.



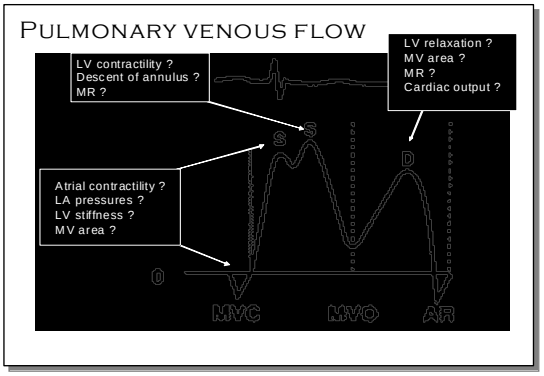


Fig. 3

Fig 4 Garcia et al. JACC 1998; 32:865-75

Tissue Doppler imaging is used to analyze the velocity of the MV annulus as it reflects global long axis LV performance. The velocity tracing 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 5). The maximum E' velocity was demonstrated to be inversely related to the time constant of isovolumetric relaxation tau (Am J Cardiol 1997;79:928) and less dependent on loading conditions than the corresponding transmitral E flow component. Consequently, the ratio between E (flow) and E' (annulus) velocities corresponds directly with LV filling pressures. In addition, E' max velocities show a almost linear decrease with the progress of diastolic disease (ie does not show pseudonormalization) and remains useful in patients with atrial fibrillation. An important limitation of Doppler-based measurements, however, is the measurement error associated with improper alignment of the interrogating Doppler beam. This is a particular problem for transesophageal probes where space constraints may render parallel alignment of the Doppler beam with respect to MV annular motion nearly impossible.

Tissue velocity imaging can be performed with either the pulsed Doppler or 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. The velocity scales should be adjusted as well since tissue velocities are about $1/10^{th}$ of flow velocities. Color Doppler tissue imaging requires specific software modules. In general, absolute values obtained with the color Doppler technique are lower because they represent an average of velocities within a sample area. The location of the sampling site is also important : peak velocities obtained at the lateral part of the mitral annulus are always higher than at the septal site.

A recent study showed that of all echocardiographic parameters investigated, the LV filling index E/E'lateral was identified as the best index to detect diastolic dysfunction in HFNEF, in which the diagnosis of diastolic dysfunction was confirmed by conductance catheter analysis. Figure 6 (*Circulation* 2007;116:637-647) A consensus document by the Heart Failure and Echocardiography Associations of the European Society of Cardiology included the ratio of E to E' as an essential tool for noninvasive diagnostics of diastolic function in patients with HFNEF. Figure 7 (Eur Heart J. 2007 Oct;28(20):2539-50)

Fig 5

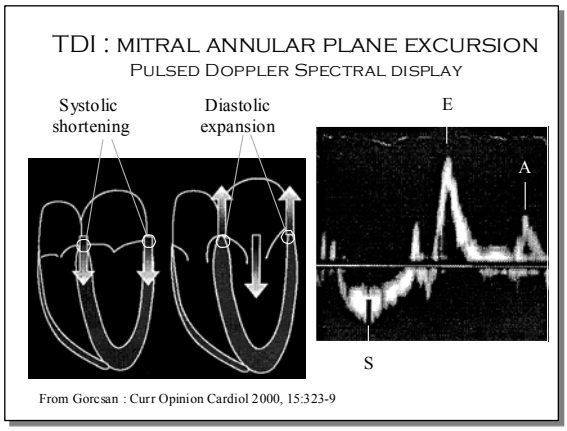
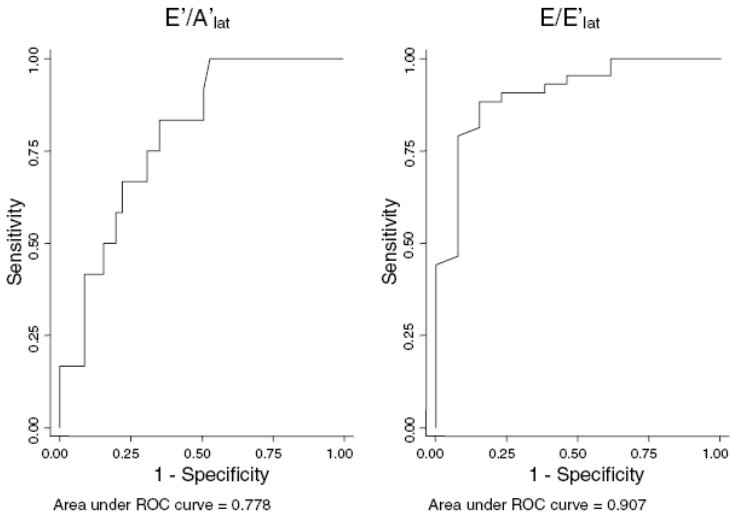


Fig 6



***Circulation*. 2007;116:637-647**

The propagation velocity of early mitral inflow (VpE) has also 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 8) 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 recent addition to the modes described above, is the analysis of LV long-axis deformation using 2D-speckle tracking. (Figure 9 and 10) Global LV strain rate during the isovolumetric relaxation phase was shown to strongly depend on relaxation. (***Circulation*. 2007;115:1376-1383)** These observations certainly need to be confirmed in the near future because tissue deformation during isovolumic relaxation is a very short-lived phenomenon that may not be accurately quantified by 2D speckle tracking – a technique with a notoriously low temporal resolution.
- Another interesting new approach to the analysis of diastolic function is the study of LV torsion. In animal studies, LV untwisting rate was shown to correlate with the time constant of LV pressure decay tau. (Am J Physiol Heart Circ Physiol 2008; 294: H505–H513)

A combination of methods is advocated to assess diastolic performance in the clinical setting – JASE 1999; 12:609-17. (Figure 11) A complete study of mitral flow, mitral annular motion and pulmonary venous flow can be performed in a few minutes by any trained echocardiographer. However, a fair understanding of the pathophysiology of diastolic dysfunction and much attention to detail for the proper application of these techniques remains essential.

Fig 7

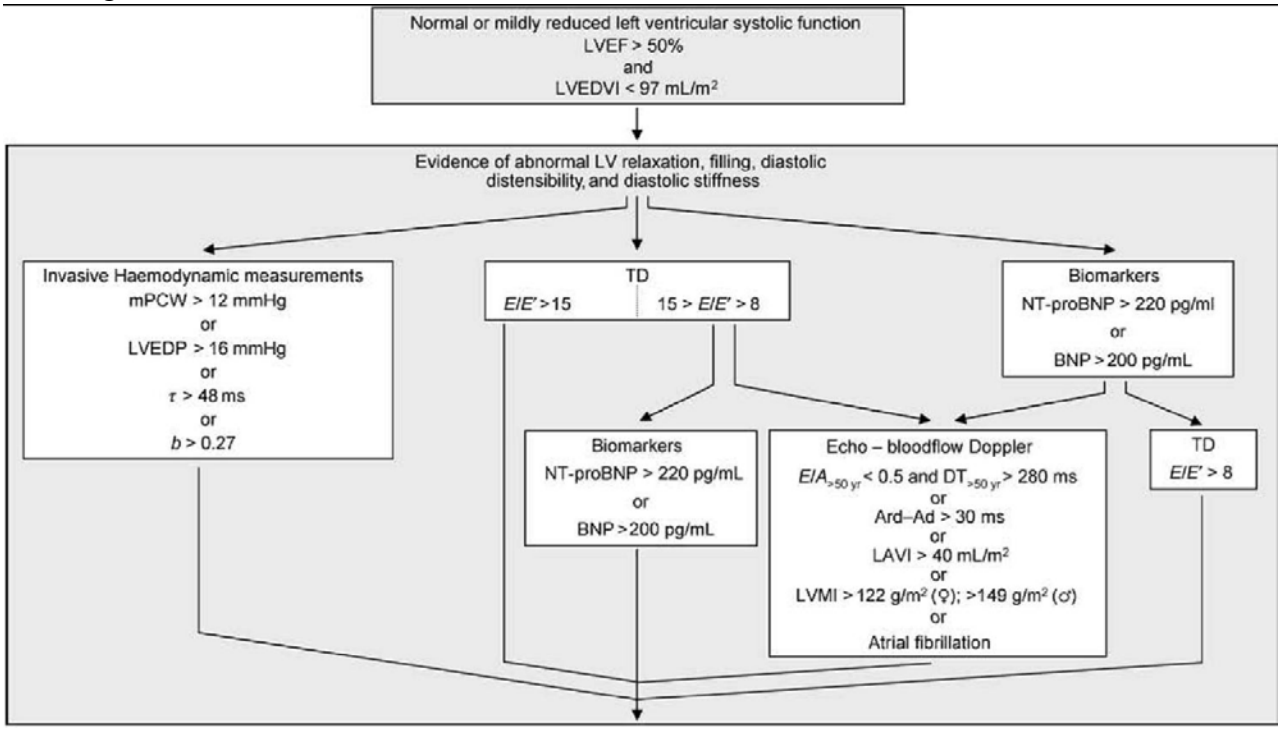


Fig 8

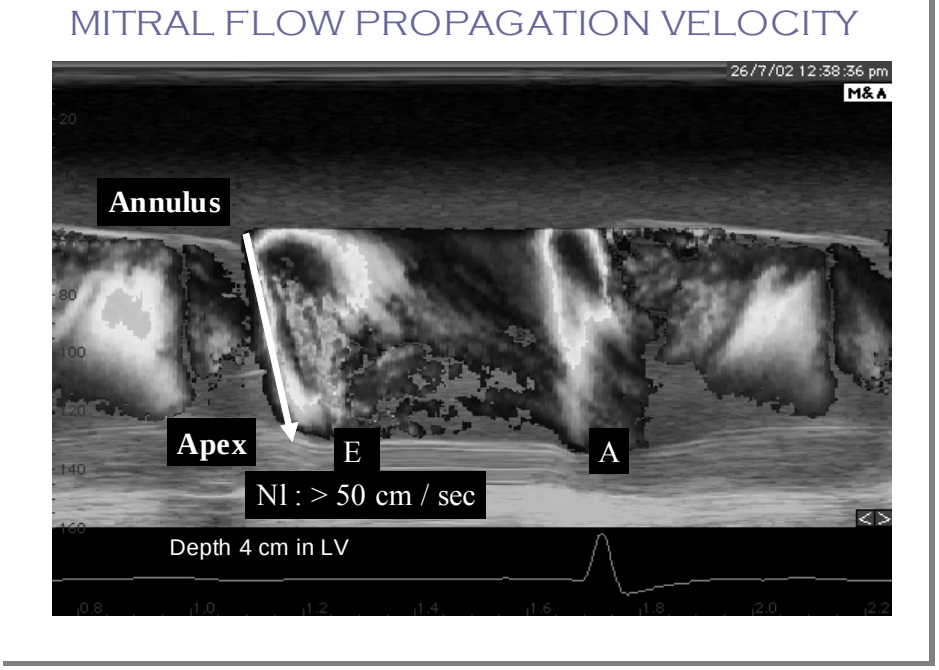


Fig 9 *Circulation* 2007;115;1376-1383

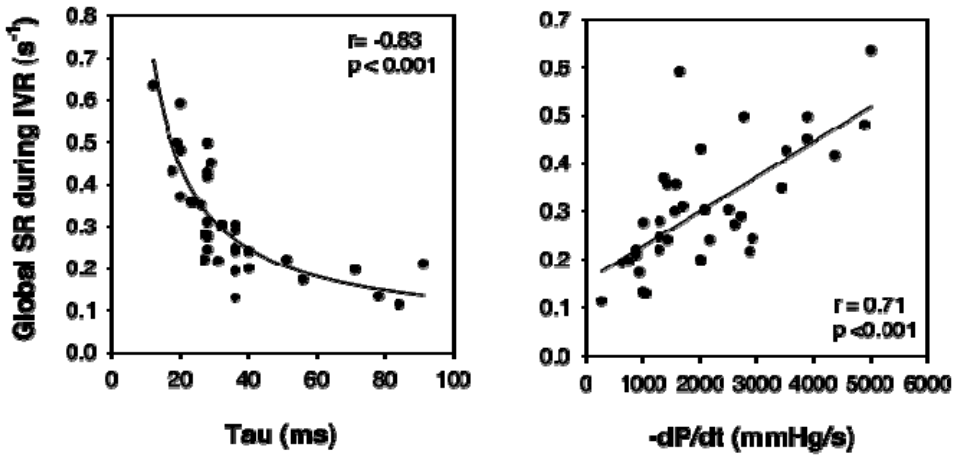
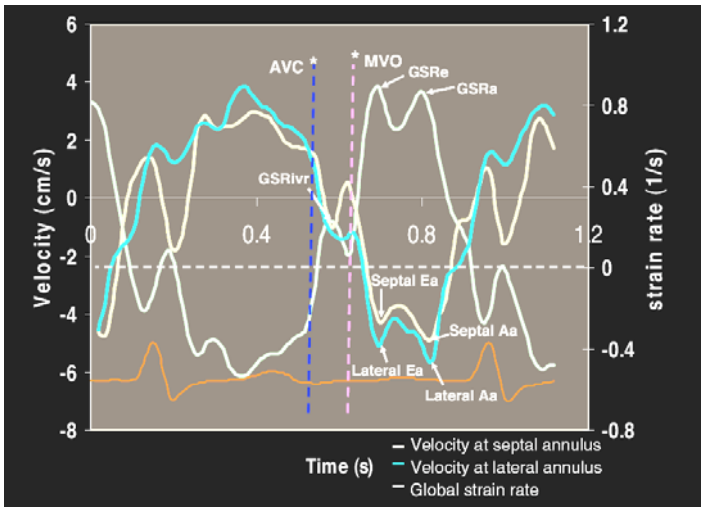
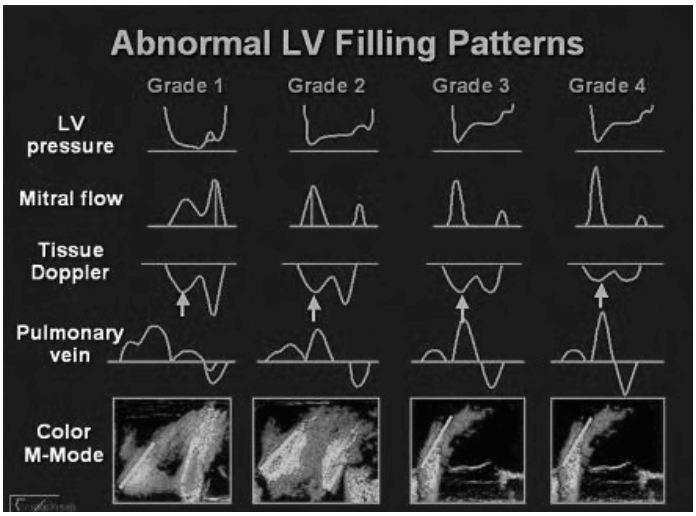


Fig 10

Fig 11





Assessment of the right ventricular function and the pulmonary circulation.

Isabelle Michaux.
Department of Intensive Care Medicine, University Hospital UCL Mont-Godinne, Yvoir, Belgium.

Introduction

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 (RVIT) 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 tricuspid valve (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 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 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.
The peak RV pressure is reached early during ejection which, in contrast to left ventricular (LV) ejection, continues even during the late systolic decline of the RV pressure. Therefore, the isovolumic relaxation period is also short and often absent.

TOE evaluation of the right heart

1. Two-dimensional examination of the right heart

Please see the illustrations at the end of this abstract.

Right ventricle

The right ventricle can be visualised by transoesophageal echocardiography (TOE) in four main views and by transthoracic echocardiography (TTE) in 3 main views. They include the *mid-oesophageal* (*MOE) 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 either the anterior or posterior part of the free wall, depending on the degree of TOE probe retroflexion, 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 transgastric (*TG) SAX view* of the right ventricle (*TG RV SAX*) that allows for evaluation of the basal or midventricular 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 (basal and apical segments at the lower part of the display).
In TTE, the right ventricle can be observed in the *apical 4-chamber view*. The RV free wall is displayed to the left of the screen, with the anterior or posterior part of the free wall, depending on the degree of TOE probe retroflexion. The *parasternal long-axis view* shows the anterior part of the RV free wall on the upper part of the display. The *parasternal short-axis view of the aortic valve*, obtained from the parasternal long-axis view by turning the probe by 90° (clockwise), shows the inferobasal (left part of the display) and anterobasal (upper part of the display) segments of the RV free wall and the RVOT on the right.

Right atrium and Vena Cava

The RA and the atrial septum can be studied in the *MOE 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, the right atrial appendage on the right (just beneath the SVC) and the IVC on the left. The SVC can also be seen in the *upper-oesophageal (UOE) SAX view* of the ascending aorta (at 0°).
In TTE, the IVC is observed in the *subcostal view*. The utility of this subcostal view of the IVC is in measuring the diameter of the IVC and its index of collapsibility as indicator of preload in spontaneously breathing patient.

Hepatic veins

From the *MOE 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.
In TTE, the hepatic veins are observed in the *subcostal view*.

Right ventricular inflow tract

The RV inflow tract consists of the tricuspid valve, the chordae tendinae and the papillary muscles. The RVIT can be visualised by TOE in 3 main views that are the *MOE 4-chamber view* (or the modified 4-chamber view), the *MOE RV inflow-outflow view* and the *TG RV inflow view*.

In TTE, we analyse the RVIT in the *apical 4-chamber view*, the *parasternal inflow view* of the RV and *parasternal SAX view of the aortic valve*, and finally in the *sub-costal view*.

Right ventricular outflow tract and the pulmonary artery

The RVOT can be observed by TOE in the *MOE RV inflow-outflow view* as well as in 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.

In TTE, the RVOT and the beginning of the PA are observed in the *parasternal SAX view of the aortic valve.*

2. 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 (FS)* expresses the change of the RV diameter 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 (free wall to septum). The normal value of RV FS% is >30%.

The area of the right ventricle can be measured by planimetry in the MOE (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 > 12 cm/s

separates normal from reduced RV function[7, 8] 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.[7] Wang et al. showed that a cut-off value of Sa at 8.8 cm/s is predictive of a RV EF < 45% (measured by MRI) with a sensitivity of 80% and a specificity of 79%.[9] TOE reference values for Sa have never been validated in ventilated and anaesthetised patients. In patients with a normal RV function and scheduled for elective coronary artery bypass surgery, our group recorded the tricuspid TDI in the TG RV inflow view, just after induction of general anaesthesia and mechanical ventilation, and observed a mean systolic velocity > 7 cm/s.[10] In the same kind of patients and using the same TG view, David et al observed a mean systolic velocity > 5 cm/s.[11]

Myocardial acceleration during isovolumic contraction (IVA) is a new TDI parameter of RV myocardial contractile function. It is calculated by dividing the isovolumic peak velocity by the time to peak velocity. In an animal model, Vogel et al showed that IVA is less affected by preload and afterload than Sa.[12]

The *PA flow:* The pulmonary artery VTI is used for calculation of stroke volume and cardiac output. In ventilated and anaesthetised patients, by rule of thumb, pulmonary artery VTI > 15 cm suggests a normal stroke volume.[13]

b) Diastolic function

RV diastole is composed by 4 phases that are the isovolumic relaxation that begins by the closure of the PV and ends with the opening of the TV, the early filling, the diastase and the contraction of the RA.

The recording of the *tricuspid inflow* velocities through the TV analyses the last 3 phases of the diastole. This tricuspid inflow can be recorded with PW-Doppler from the MOE modified 4-chamber view or from the TG RV inflow view. The velocities across the TV are lower than those across the mitral valve. As for the mitral inflow, the tricuspid inflow shows early and late diastolic filling (during atrial contraction) waves. The measurable variables are the early peak diastolic velocity (E) and late peak diastolic velocity (A) and the E wave deceleration time, i.e. the slope of the deceleration of the E wave. From these measurements, the E/A ratio can be calculated as the peak velocity of the E wave divided by the peak velocity of the A wave.

The tricuspid inflow changes with increasing age as does the mitral inflow: the E wave decreases and the A wave increases, leading to a gradual decrease in the E/A ratio and an increase of the E wave deceleration time.[14] A normal E/A ratio is higher than 1 and remains above 1 even after 50 years of age.[14] The E/A ratio increases when there is elevation of RA pressure, severe tricuspid regurgitation and RV dysfunction (RV infarction or advanced restrictive myocardial disease).[15]

Based on the classification of LV diastolic dysfunction, one can distinguish between an abnormal RV relaxation (E/A <1 with deceleration time of the E wave >240 ms and a RV IVRT >100 ms) or a restrictive RV filling (E/A >2 with a deceleration time of the E wave <140 ms and a RV IVRT <70 ms).[16] Based on the E/A ratio, RV diastolic dysfunction was described in patients with chronic pulmonary disease, pulmonary embolism,[17] systolic LV failure[16] and systemic sclerosis.[18] However, interpretation based only on the tricuspid E/A ratio as an indicator of global RV diastolic function is difficult as the changes in E/A ratio are probably U-shaped during transition from normal to abnormal diastolic function, as observed in LV diastolic dysfunction.[19] There are no studies that focus on the transition from a normal to an abnormal RV diastolic function.

Hepatic venous flow

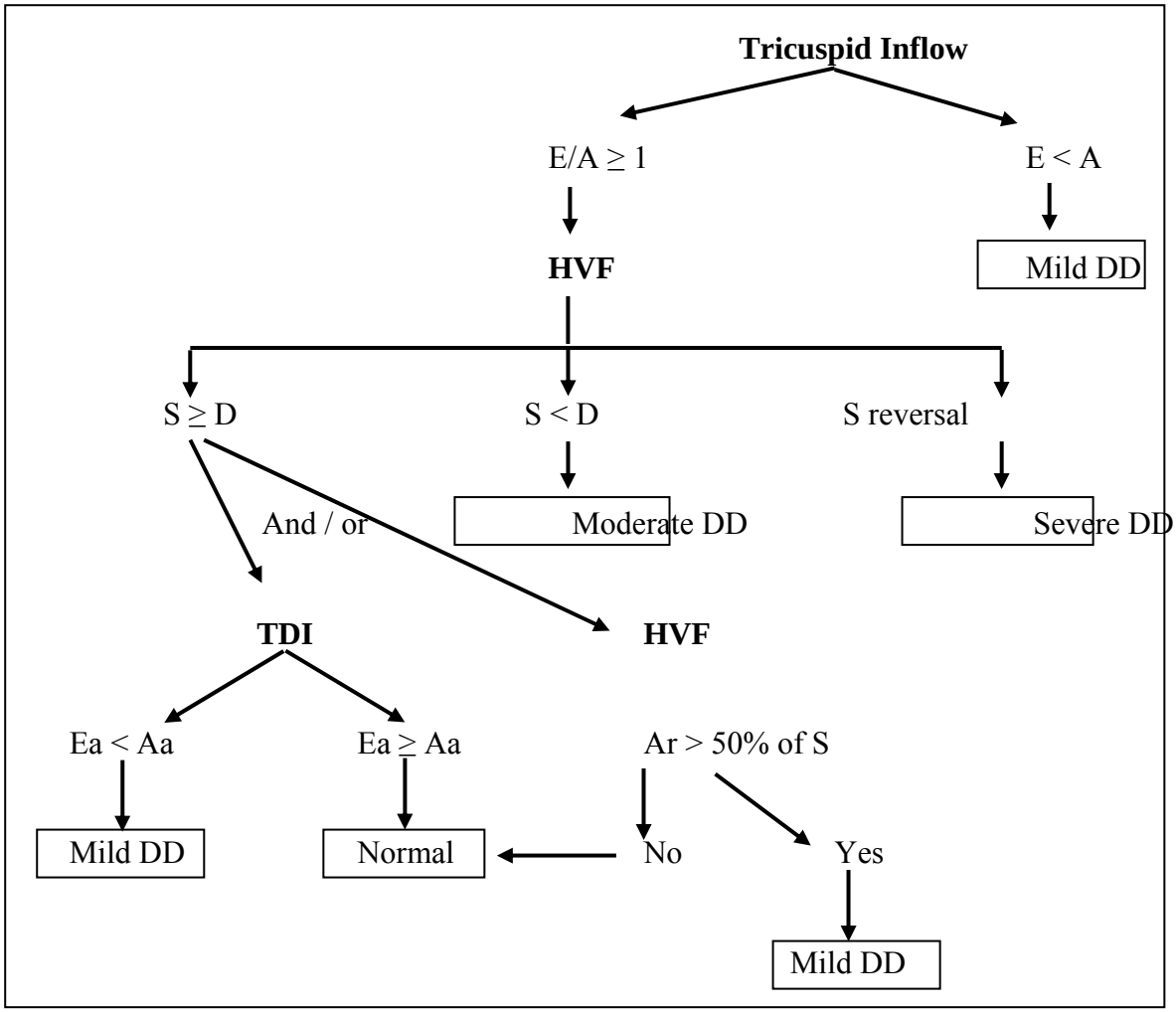
Placing the sampling volume of the pulsed Doppler in one of the three hepatic veins, with a filter reduced to 50 Hz, allows for recording the hepatic venous flow (HVF). This HVF flow pattern has four components: a biphasic forward flow with a systolic and a diastolic wave and two reverse flows, one occurring during the atrial contraction and the other one in late systole. The systolic wave (s) is produced by the RV contraction with an increase in right atrial volume because of the descent of the tricuspid annulus to the apex. The diastolic (d) component occurs with the opening of the tricuspid valve and RV filling. The atrial reverse wave (a) is a backflow in the hepatic veins due to the atrial contraction. Finally, the late systolic reverse flow called V wave (v) can be explained by atrial overfilling. The normal hepatic pattern is a biphasic forward flow with systolic dominance

and two small reverse waves. The systolic forward velocity decreases with elevation of the central venous pressure,[20] in the presence of tricuspid regurgitation or inferior myocardial infarction.[21] In the case of severe tricuspid regurgitation, a systolic flow reversal can be observed.

Tissue Doppler imaging

The diastolic function indices derived from TDI of the tricuspid lateral annulus are the early peak diastolic velocity (Ea), the late peak diastolic velocity of the tricuspid annulus (Aa), the ratio of Ea to Aa and IVRT. From the transthoracic literature, we know that a normal Ea should be higher than 12 cm/s,[22] while an Ea <8.9 cm/s is predictive for cardiac morbidity.[23] The Ea/Aa ratio decreases with age, in cases of inferior myocardial infarction, pulmonary and systemic hypertension.[24] IVRT can also be measured from the TDI signal: it is the time from the end of the Sa to the beginning of the Ea wave.

The evaluation of the RV diastolic function is not clearly defined in literature. Some authors tried to evaluate it based on the combination of tricuspid E/A ratio, hepatic S/D ratio and the TDI Ea/Aa. Based on an algorithm proposed by Denault et al,[25] RV diastolic function can be classified as normal, mild, moderate or severe dysfunction.



Algorithm used by Denault et al to classify RV diastolic dysfunction (DD).[25]

Wall motion reading

Although no segmental model of the RV has yet been validated, the RV free wall is usually divided into four segments: anterobasal, inferobasal, anteroapical, and inferoapical.[26] In addition, the wall motion has to be evaluated in the RVOT and in the ventricular septum. Whereas the anterior or inferior segments (basal and apical) can be best evaluated in the MOE (modified) 4-chamber view (0º), the inferobasal and anterobasal segments and the outflow tract are interrogated in the MOE RV inflow-outflow view. Basal or midventricular segments can also be evaluated in the TG SAX view

of the right ventricle. The TG RV inflow view allows for evaluation of the 4 segments of the free wall.
Because of asymmetric geometry, smaller degree of systolic shortening, and thinner wall, the RV wall motion abnormalities are more difficult to detect than those of the LV wall. Therefore, the grading scale for visual assessment of wall motion of the free wall are scored on a scale of 1 to 4 (1 = normal; 2 = hypokinetic; 3 = akinetic; and 4 = dyskinetic).

Measurement of pulmonary artery pressure

Echocardiography offers non-invasive access to the pressures in the right heart and eliminates the use of a PA 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.[27] 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 left ventricle (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 left ventricle in end-diastole. In diastole, in the TG SAX view, the right ventricle assumes a circular shape, whereas the left ventricle becomes crescentic. These changes are more pronounced if the pericardium is closed. During systole, when the pressure generated by the left ventricle exceeds the RV pressure, the septum moves swiftly towards the right ventricle and the left ventricle 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 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 left ventricle also persists throughout systole.

References

1. Practice guidelines for perioperative transesophageal echocardiography. A report by the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists Task Force on Transesophageal Echocardiography. *Anesthesiology.* 1996;84(4):986-1006.

2. Mansencal N, Joseph T, Vieillard-Baron A, Qanadli SD, Jondeau G, Lacombe P, Jardin F, Dubourg O. Comparison of different echocardiographic indexes secondary to right ventricular obstruction in acute pulmonary embolism. *The American journal of cardiology.* 2003;92(1):116-119.

3. Jardin F, Dubourg O, Bourdarias JP. Echocardiographic pattern of acute cor pulmonale. *Chest.* 1997;111(1):209-217.

4. Maslow AD, Regan MM, Panzica P, Heindel S, Mashikian J, Comunale ME. Precardiopulmonary bypass right ventricular function is associated with poor outcome after coronary artery bypass grafting in patients with severe left ventricular systolic dysfunction. *Anesth Analg.* 2002;95(6):1507-1518.

5. Jardin F. *L'évaluation hémodynamique non invasive au lit du patient par échocardiographie-Doppler*. Paris: Masson; 1995:44.

6. Ghio S, Recusani F, Klersy C, Sebastiani R, Laudisa ML, Campana C, Gavazzi A, Tavazzi L. Prognostic usefulness of the tricuspid annular plane systolic excursion in patients with congestive heart failure secondary to idiopathic or ischemic dilated cardiomyopathy. *The American journal of cardiology.* 2000;85(7):837-842.

7. Meluzin J, Spinarova L, Bakala J, Toman J, Krejci J, Hude P, Kara T, Soucek M. Pulsed Doppler tissue imaging of the velocity of tricuspid annular systolic motion; a new, rapid, and non-invasive method of evaluating right ventricular systolic function. *Eur Heart J.* 2001;22(4):340-348.

8. Haddad F, Hunt SA, Rosenthal DN, Murphy DJ. Right ventricular function in cardiovascular disease, part I: Anatomy, physiology, aging, and functional assessment of the right ventricle. *Circulation.* 2008;117(11):1436-1448.

9. Wang J, Prakasa K, Bomma C, Tandri H, Dalal D, James C, Tichnell C, Corretti M, Bluemke D, Calkins H, Abraham TP. Comparison of novel echocardiographic parameters of right ventricular function with ejection fraction by cardiac magnetic resonance. *J Am Soc Echocardiogr.* 2007;20(9):1058-1064.

10. Michaux I, Filipovic M, Skarvan K, Schneiter S, Schumann R, Zerkowski HR, Bernet F, Seeberger MD. Effects of on-pump versus off-pump coronary artery bypass graft surgery on right ventricular function. *J Thorac Cardiovasc Surg.* 2006;131(6):1281-1288.

11. David JS, Tousignant CP, Bowry R. Tricuspid annular velocity in patients undergoing cardiac operation using transesophageal echocardiography. *J Am Soc Echocardiogr.* 2006;19(3):329-334.

12. Vogel M, Schmidt MR, Kristiansen SB, Cheung M, White PA, Sorensen K, Redington AN. Validation of myocardial acceleration during isovolumic contraction as a novel noninvasive index of right ventricular contractility: comparison with ventricular pressure-volume relations in an animal model. *Circulation.* 2002;105(14):1693-1699.

13. Lambert-Litner A. Referenzwerte für intraoperative transösophageale Echokardiographie. Inauguraldissertation. In: Basel MFdU, ed. Basel; 1997.

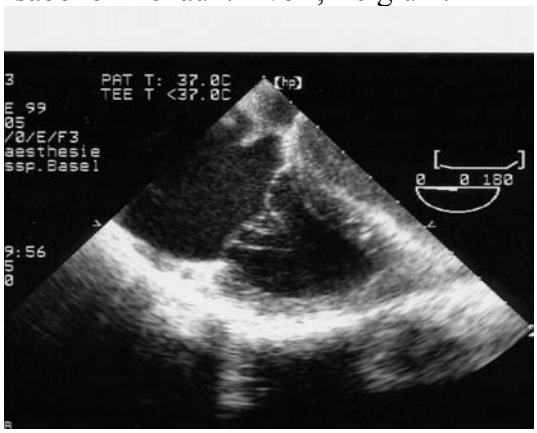
14. Klein AL, Leung DY, Murray RD, Urban LH, Bailey KR, Tajik AJ. Effects of age and physiologic variables on right ventricular filling dynamics in normal subjects. *Am J Cardiol.* 1999;84(4):440-448.

15. Klein AL, Hatle LK, Burstow DJ, Taliercio CP, Seward JB, Kyle RA, Bailey KR, Gertz MA, Tajik AJ. Comprehensive Doppler assessment of right ventricular diastolic function in cardiac amyloidosis. *J Am Coll Cardiol.* 1990;15(1):99-108.

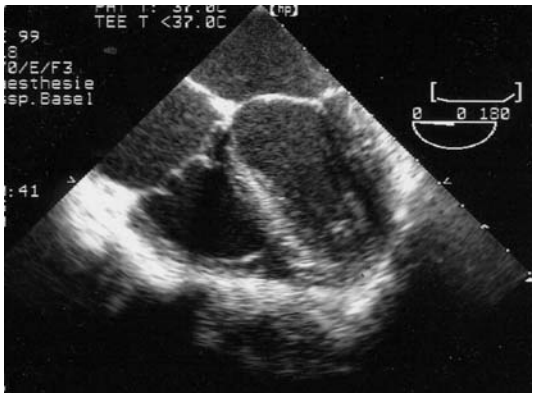
16. Yu CM, Sanderson JE, Chan S, Yeung L, Hung YT, Woo KS. Right ventricular diastolic dysfunction in heart failure. *Circulation.* 1996;93(8):1509-1514.

17. Nakamura K, Miyahara Y, Ikeda S, Naito T. Assessment of right ventricular diastolic function by pulsed Doppler echocardiography in chronic pulmonary disease and pulmonary thromboembolism. *Respiration.* 1995;62(5):237-243.
18. Giunta A, Tirri E, Maione S, Cangianiello S, Mele A, De Luca A, Valentini G. Right ventricular diastolic abnormalities in systemic sclerosis. Relation to left ventricular involvement and pulmonary hypertension
19. Garcia MJ, Thomas JD, Klein AL. New Doppler echocardiographic applications for the study of diastolic function. *J Am Coll Cardiol.* 1998;32(4):865-875.
20. Nagueh SF, Kopelen HA, Zoghbi WA. Relation of mean right atrial pressure to echocardiographic and Doppler parameters of right atrial and right ventricular function. *Circulation.* 1996;93(6):1160-1169.
21. Mishra M, Swaminathan M, Malhotra R, Mishra A, Trehan N. Evaluation of right ventricular function during CABG: transesophageal echocardiographic assessment of hepatic venous flow versus conventional right ventricular performance indices. *Echocardiography (Mount Kisco, N.Y.* 1998;15(1):51-58.
22. Kukulski T, Voigt JU, Wilkenshoff UM, Strotmann JM, Wranne B, Hatle L, Sutherland GR. A comparison of regional myocardial velocity information derived by pulsed and color Doppler techniques: an in vitro and in vivo study. *Echocardiography (Mount Kisco, N.Y.* 2000;17(7):639-651.
23. Meluzin J, Spinarova L, Hude P, Krejci J, Kincl V, Panovsky R, Dusek L. Prognostic importance of various echocardiographic right ventricular functional parameters in patients with symptomatic heart failure. *J Am Soc Echocardiogr.* 2005;18(5):435-444.
24. Galderisi M, Severino S, Cicala S, Caso P. The usefulness of pulsed tissue Doppler for the clinical assessment of right ventricular function. *Ital Heart J.* 2002;3(4):241-247.
25. Denault AY, Couture P, Buithieu J, Haddad F, Carrier M, Babin D, Levesque S, Tardif JC. Left and right ventricular diastolic dysfunction as predictors of difficult separation from cardiopulmonary bypass: [La dysfonction ventriculaire diastolique gauche et droite comme predicteur des difficultes de sevrage de la circulation extracorporelle]. *Can J Anesth.* 2006;53(10):1020-1029.
26. 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;89(4):870-884.
27. Prakash R, Matsukubo H. Usefulness of echocardiographic right ventricular measurements in estimating right ventricular hypertrophy and right ventricular systolic pressure. *The American journal of cardiology.* 1983;51(6):1036-1040.

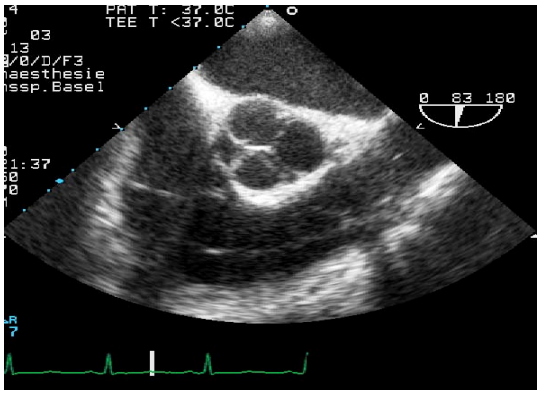
Illustrations - Two-dimensional TOE examination of the right cavities.
Isabelle Michaux. Yvoir, Belgium.



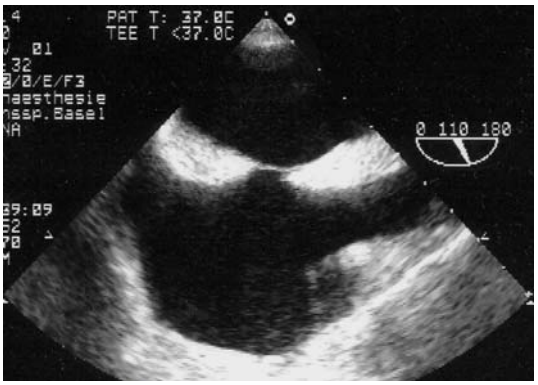
Modified MOE 4-chamber view



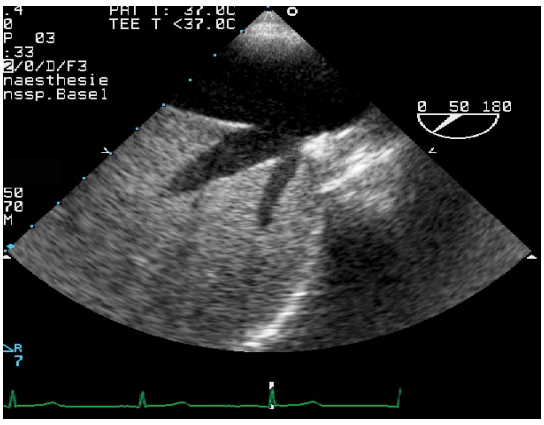
MOE 4-chamber view



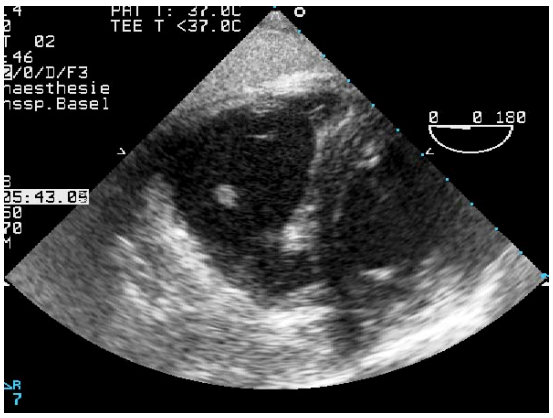
MOE RV inflow-outflow view



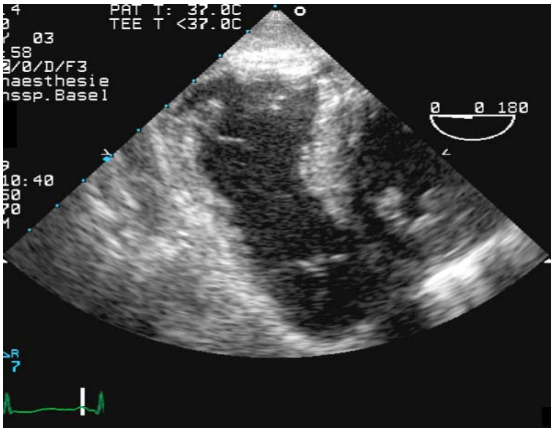
MOE Bicaval view



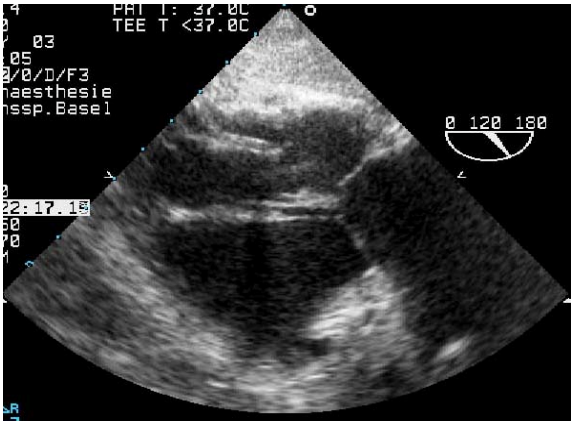
Inferior vena cava and hepatic veins



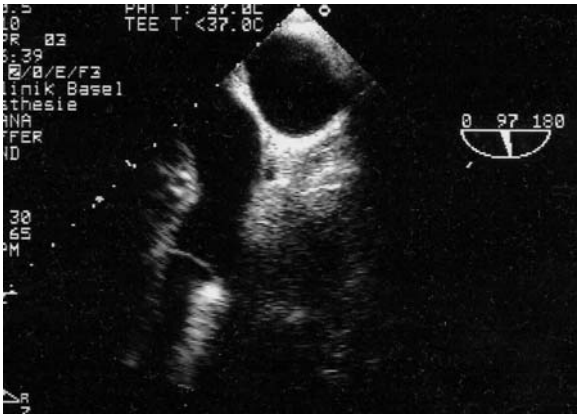
TG RV SAX view



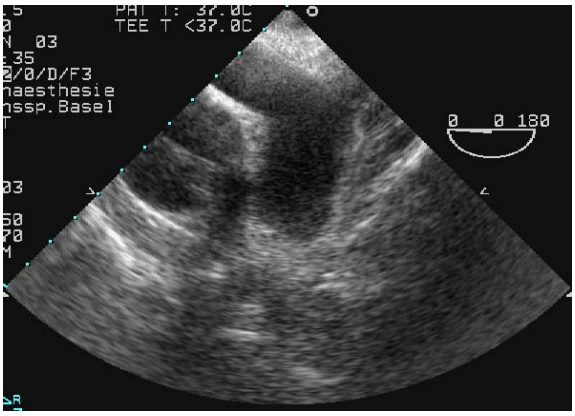
Deep TG RV outflow view



TG RV inflow view



UOE aortic arch SAX view



UOE ascending aortic SAX view

PULMONARY AND TRICUSPID VALVE

G. Karatasakis MD, FESC

TRICUPID VALVE

The tricuspid valve can be easily visualized by transthoracic echocardiography because of its anterior position, very close to the chest wall. The tricuspid valve can be visualized from the parasternal window in the RV inflow view, from the apical four chamber view, and the subcostal four chamber and short axis view. By multiplane transesophageal echocardiography can be visualized in the mid-esophageal 4-chambers view at 0^o, in the mid –esophageal aortic valve short axis view at 45^o, in the mid-esophageal RV inflow- outflow view at 60^o and the transgastric RV inflow view at 135^o. The tricuspid valve is situated 1 cm below the level of the mitral valve. Its anatomy is complex when compared to the other 3 valves. The tricuspid annulus and the 3 leaflets are of irregular shape. The tricuspid valve area is the greatest when compared to the rest of the cardiac valves. The reason for this, is to accommodate cardiac output through the valve with the low right heart driving pressures. The anterior tricuspid leaflet is the biggest, the septal leaflet the smallest, while the posterior is usually of intermediate size. The septal leaflet is placed lower than the anterior and the posterior

TRICUSPID STENOSIS

Tricuspid stenosis is uncommon in adults. Rheumatic fever is the leading cause of tricuspid stenosis. Rheumatic heart disease involves the tricuspid leaflets in 20% of the cases leading to stenosis or to regurgitation. Carcinoid heart disease, thrombi, large vegetations and right atrial tumors that may occlude right ventricular inflow, represent extremely rare causes of tricuspid stenosis.

The echocardiographic characteristics of tricuspid stenosis include thickening and shortening of all three tricuspid leaflets, commissural fusion, reduced excursion of the valve and “doming” during diastole. There is increased tricuspid diastolic velocity, as well as peak and mean gradient. Turbulent flow by color Doppler and prolonged diastolic pressure half time by CW are also characteristics of tricuspid stenosis.

TRICUSPID REGURGITATION

Mild tricuspid regurgitation is seen in a large proportion of normal subjects. Clinically significant tricuspid regurgitation can be schematically divided in organic and functional, although pure functional tricuspid regurgitation does not exist. This is because annular dilatation or pulmonary hypertension, who are considered as the leading causes of “functional” TR, may produce pathologic changes of the tricuspid leaflets, that may end up to severe organic tricuspid regurgitation. “Functional” tricuspid regurgitation is the most frequent clinical problem related to the tricuspid valve. Clinically significant tricuspid regurgitation, can be found in up to 28% of patients undergoing mitral valve replacement or repair. Abnormalities of the tricuspid valve leaflets may be due to rheumatic involvement as is the case in tricuspid stenosis. Rheumatic tricuspid regurgitation does not exist as an isolated lesion. There is almost always involvement of other cardiac valves. Carcinoid heart disease is very rare. It is characterized by thickened, highly echogenic and immobile tricuspiud valve leaflets. Ebstein’s anomaly is the displacement of one or more of the tricuspid leaflets towards the right ventricular apex

PULMONIC VALVE

The pulmonic valve is situated at the end of the left ventricular outflow tract, marking the origin of the pulmonary artery, in a plane perpendicular to the aortic valve. Because of its orientation the pulmonary valve is not visualized in short axis views. Especially in adults the pulmonary valve is poorly visualized. There are three pulmonic leaflets, but usually one or at most two of them are adequately seen by transthoracic or transesophageal echocardiography. The pulmonic valve, can be visualized by transthoracic echocardiography from the parasternal window in the short axis view at the aortic valve level, from the parsternal right ventricular outflow view and the subcostal short axis view at the aortic valve level. By multiplane transesophageal echocardiography the pulmonic valve can be visualized in the mid –esophageal aortic valve short axis view at 45^{o}, and in the mid-esophageal RV inflow- outflow view at 60^{o}

PULMONIC STENOSIS

The echocardiographic findings of pulmonic stenosis include pulmonic leaflet thickening with systolic “hooking”. There is also increased systolic velocity through the pulmonic valve by continuous wave Doppler leading to pressure gradient across the valve. Differential diagnosis of the site of stenosis (subvalvular –infudibular- or supravalvular) should be made. Postenotic pulmonary artery dilatation is associated with valvular stenosis and can be used to distinguish this form of the disease from infudibular or supravalvular stenosis.

PULMONIC REGURGITATION.

Pulmonic regurgitation is rarely a clinically significant finding, In some cases can be the result of congenital heart disease and sometimes residual pulmonary regurgitation after surgery or balloon pulmonary valvuloplasty. Acquired significant pulmonary regurgitation is extremely rare. It can be seen as the result of endocarditis, or carcinoid syndrome. Thickened deformed and immobile leaflets are common echocardiographic findings of congenital disease. The characteristics lesions of endocarditis are vegetations or perivalvular abscesses while in carcinoid syndrome the valve appears thickened with shortened leaflets and highly echogenic.

AORTIC VALVE STENOSIS

Mauro Pepi, MD, FESC
Centro Cardiologico Monzino, IRCCS, Institute of Cardiology, Milan, Italy.

Transthoracic two-dimensional echocardiography (2DTT) and Doppler echocardiography are principal non-invasive tools used to obtain quantitative information concerning the aortic valve. However in selected patients and in specific settings transoesophageal echocardiography (2DTOE) provides a better acoustic window enabling direct aortic valve area planimetry and other important anatomical details.

From the upper esophagus at 2DTOE the aortic valve , left ventricular outflow tract, aortic root and ascending aorta may be easily evaluated. Generally the short axis of the valve is obtained by steering the array from 30° to 60° and the long axis from 110° to140°. In the deep transgastric long axis view gradients across the valve may be calculated.

In patients with aortic valve stenosis several studies showed that planimetry of the valve may be obtained in the majority of patients and this method has been validated. This method is also excellent for determining the morphology of abnormal aortic valves. TOE allows a very detailed visualization of the cusps facilitating the diagnosis of bicuspid aortic valve. It mat also supply new and unique information in patients with discrete fibro-muscular obstruction or subaortic tunnel in the left ventricular outflow tract. Therefore, even though aortic stenosis or left ventricular outflow tract obstructions do not constitute an indication for TOE as a first approach (a complete TTE and Doppler study should always precede a TOE examination) , TOE may be indicated in selected patients particularly when the severity of the lesions with the standard transthoracic examination is not clear or discrepancies exist between gradients and morphology of the valve (including cases in whom the presence of a subaortic membrane is suspected by TTE, but not clearly defined).

TOE facilitates also measurements of the aortic root, sino-tubular junction and ascending aorta diameters allowing, in cases with post stenotic dilatation, a very accurate study of aortic aneurysms.

One potential application is the guidance and monitoring of aortic valvuloplasty and/or percutaneous aortic valve implantation in cases with aortic valve stenosis. In these cases TOE has an important role in case selection, in guiding device placement and in detecting complications of percutaneous valve implanatation.

Intraoperative TOE may be also indicated in selected cases with aortic stenosis , left ventricular outflow gradients, aortic valve repair techniques and congenital heart defects involving the aortic valve. TOE plays several roles with respect to valve surgery. In particular in aortic valve stenosis may assess LV systolic and diastolic function in the pre- and posperative period as well as complications of valve repair (rarely performed in aortic stenosis) and replacement. Prosthetic valve dysfunction or paraprostehetic regurgitant jets may easily detected by TOE. A final area of interest (a part from the new surgical procedures such as percutaneous or transapical prosthetic implantation) in which TOE is used for intraoperative decision making is the placement of newer bioprostheses including cryopreserved homografts and new stentless porcine prostheses.

As concerns rare complication the dynamic outflow tract obstruction may occur after aortic valve replacement. TOE easily demonstrates this complication showing a small hyperdynamic LV (while hypotension is present) with dynamic outflow obstruction associated with or without mitral regurgitation. Rarely in other cases especially if a concurrent myomectomey or anular enlargement procedures have been performed a creation of a ventricular septal defect may occur.

New TOE techniques

Three-dimensional echocardiography offers uniques views of cardiac structures including the aortic valve. The introduction of real-time 3DTOE probes may facilitate the quantification of aortic valve area. In fact previous studies with rotational 3DTOE showed that 3D methods were accurate in the measurements of valve areas (even though time consuming in the acquisition and reconstruction phases) and therefore real-time 3DTOE has potentially similar advantages without significant limitations. However no data on this new method have been yet published concerning quantification of aortic stenosis. Similarly in the pre- and postoperative period 3DTOE may further simplify the

recognition of congenital and acquired aortic and outflow tract diseases and recognition of operative complications.

References

- Hoffman R, Flachskampf FA, Hanrath P: Planimetry of orifice area in aortic stenosis using multiplane transesophageal echocardiography. J Am Coll Cardiol 1993; 22: 529-534.
- Hofman T, Kasper W, Meinert T, Spiller G, Sclasser V, Just H: Determination of aortic valve area ij aortic stenosis by two-dimensional transesophageal echocardiography. Am J Cardiol 1987; 59: 330-335.
- Pepi M, Barbier P, Doria E, Bortone F, Tamborini G: Intraoperative multiplane vs biplane transesophageal echocardiography for the assessment of cardiac surgery. Chest 1996; 109: 305-311.
- Tamborini G, Barbier P, Doria E, Galli C, Maltagliati A, Ossoli D, Susini G, Pepi M: Influences of aortic pressure gradient and ventricular septal thickness with systolic coronary flow in aortic valve stenosis. Am J Cardiol 1996; 78: 1303-1306.
- Kasprzak J, Nosir Y, Dall'Agata A, Elhendy A, Taams M, Ten Cate F, Roelandt J: Quantification of the aortic valve area in three-dimensional echocardiographis data sets. Analysis of orifice overestimation resulting from suboptimal cut-plane selection. Am Heart J 1998; 135: 995-1003.

TOE assessment of Aortic Valve Regurgitation

Justiaan Swanevelder, MB ChB MMed(Anes) FCA(SA) FRCA,
Department of Anaesthesia, Glenfield Hospital, University Hospitals of Leicester, Leicester, UK
E-mail: justiaan.swanevelder@uhl-tr.nhs.uk

Introduction

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

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 an annulus and three similar semi lunar 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 thickenings seen at the central portion of the normal leaflets are called the nodules of Arantius. These tend to enlarge with age. 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. After years of function the leaflets may develop thickening of the edges, as well as filamentous strands on them. These small filamentous strands (up to 5mm in length) may appear in the left ventricular outflow tract during diastole connected to the aortic valve, or on the aortic side during systole. They are referred to as Lambl's excrescences and may be misinterpreted as vegetations. They are usually an incidental finding in elderly patients who are otherwise well.

The sinotubular junction connects the root to the proximal ascending aorta. The upper limit of the aortic annulus diameter is 2.6cm and the sinotubular junction is 3.4cm. 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 2-8mmHg, 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) 2. 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.

Abnormalities of any of the components of the AV or the adjacent structures can affect the valve function.

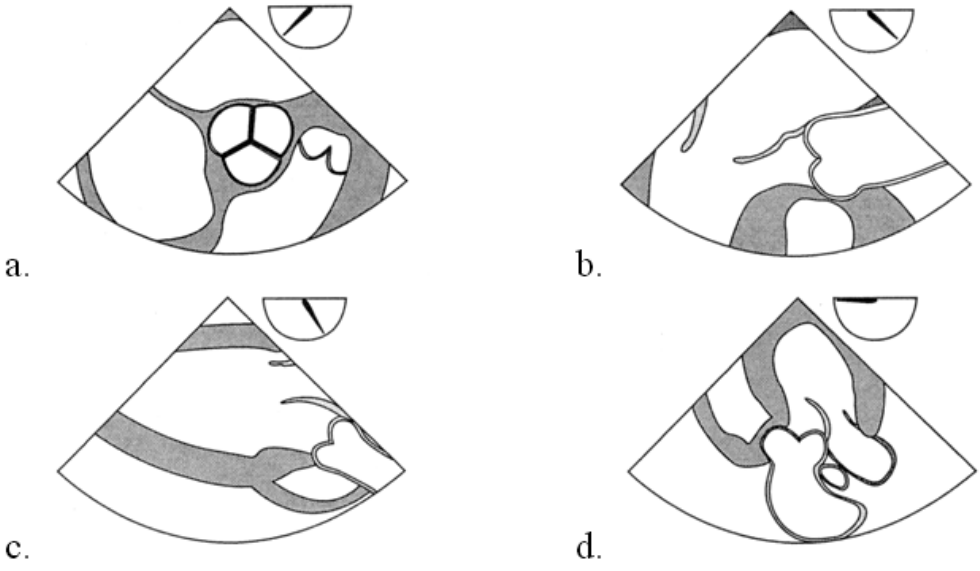
TOE examination of the Aortic valve

Evaluation of the AV should include two-dimensional (2D) images from several views and angles together with colour flow Doppler (CFD) and spectral Doppler displays. By assessing the AV in the short-axis and long-axis all the different components of the valve and root can be examined.

Essentially the four views most often used are

a. midoesophageal short axis view of the aortic valve (ME AV SAX)
b. midoesophageal long axis view of the aortic valve (ME AV LAX)
c. transgastric long axis view (TG LAX)
d. deep transgastric long axis view (deep TG LAX)

Fig 1. Four basic views used to assess aortic valve



The midoesophageal short axis, long axis and five chamber views cannot be used for Doppler measurements of flow velocity, because the ultrasound wave is perpendicular to the blood flow. In the two transgastric views the Doppler wave is almost parallel to blood flow through the AV and flow velocity can usually be measured (using one or both of the views) to calculate the pressure gradient (Bernoulli equation). When evaluating the aortic valve for AR, it is essential to always assess the contractile reserve of the left ventricle (LV).

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. The congenitally bicuspid aortic valve predisposes to infective

endocarditis, which can lead to its destruction with subsequent AR. Infective endocarditis of the AV typically presents with a mobile vegetation connected to a cusp. This is best visible on the ventricular side of the valve when prolapsing into the LVOT during diastole. 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.

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. Movsowitz et al. describes five potential mechanisms of AR in a patient with acute type A aortic dissection.

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.

Qualitative diagnosis of AR with TOE is relatively easy, but quantitative evaluation is much more difficult. It is therefore important to examine multiple imaging planes. The problem to grade severity will probably be overcome in future by three-dimensional (3D) echo.

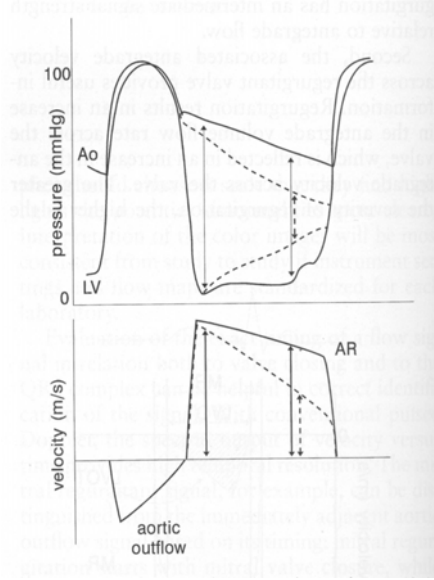
Assessment of AR starts with an accurate 2D examination in the ME SAX and LAX views of the AV. This includes an evaluation of the cusps, LVOT, aortic root and ascending aorta. Poor coaptation, vegetations, cusp prolapse or perforations, and annular dilatation should be noted. The regurgitant jet may impair opening of the anterior mitral valve leaflet ("reverse doming"), or lead to its early closure before the onset of systole. This can even lead to diastolic mitral regurgitation (demonstrated with CFD). On M-mode a high frequency fluttering of the anterior MV leaflet may be seen during diastole as result of this jet. The LV should be carefully assessed for function and any dilatation as result of the chronic volume overload. If the LV end-systolic diameter is less than 5.5cm, the patient has a better long-term prognosis (Bonow).

This 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). The vena contracta (VC) is the narrowest portion of the jet located at or just distal to its orifice (Baumgarten), and is independent of flow rate. It 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. 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. Although this is a semiquantitative index, it became the clinical standard for echocardiographic grading of AR. When the jet width takes up more than 60% of the LVOT diameter it is severe. The jet width is superior to jet length or jet area and is accurate in the setting of eccentric jets (Ishii). The VC has also been shown to correlate well with direct measurements of the regurgitant volume and fraction by aortic flow probe (Willett). 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. Colour M-mode can also be used to determine the jet width-LVOT ratio.

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 250 msec will confirm that fact. The LV compliance and systemic vascular resistance affect these measurements.

Fig 2. **Otto CM. Textbook of Clinical Echocardiography**



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 50% of the stroke volume 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 more than 55%, 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.

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.

Conclusion

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. Patients with severe left-sided regurgitant lesions can remain relatively asymptomatic while the left ventricle dilates and develops irreversible functional impairment. Left ventricular dysfunction continues to predict a very poor outcome in spite of technically successful valve surgery. In an era of modern echocardiography it is no longer appropriate to wait for a change in symptoms to guide management (Wilkens).

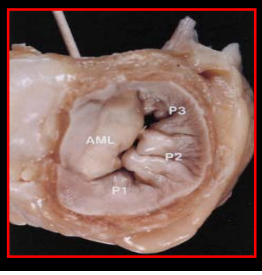
According to the ASA/SCA Task Force Guidelines of 1996 valve replacement is a Category II indication for the use of TOE. That means expert opinion and research studies shows that TOE may possibly influence the outcome of these procedures. In stentless AV and homograft surgery intra-operative TOE plays a very valuable role and these procedures may move to Category I in the near future. Aortic valve repair procedures are complex and should also not be attempted without the support of intraoperative TOE.

References

1. Robicsek F, Thubrikar MJ. Role of sinus wall compliance in aortic leaflet function. Am J Cardiol 1999;84:944-6
2. Sripathi VC, Krishna Kumar R, Balakrishnan KR. Further insights into normal aortic valve function: Role of a compliant aortic root on leaflet opening and valve orifice area. Ann Thorac Surg 2004;77:844-51
3. Shanewise JS, Cheung AT, Aronson S, et al. ASE/SCA guidelines for performing a comprehensive intra-operative multiplane transesophageal echocardiographic examination: recommendations of the ASE and SCA. Anesth Analg 1999;89:870-84
4. Practice guidelines for peri-operative transesophageal echocardiography: a report by the ASA and SCA task force on transesophageal echocardiography. Anesthesiology 1996;84:986-1006
5. Oh JK, Seward JB, Tajik AJ. The Echo Manual, 2nd edition, Lippencott-Raven 1999
6. Shanewise JS. Transoesophageal Echocardiographic Evaluation of the Aortic Valve in *Transoesophageal Echocardiography in Anaesthesia*. Poelaert J & Skarvan K eds. BMJ Books 2000, pp 89-100
7. Wilkens GT. Valvular heart disease: putting guidelines into practice. BMJ 17 May 1997;314:1428-9
8. Roberts WC. The congenitally bicuspid aortic valve. A study of 85 autopsy cases. Am J Cardiol 1970;26:72-83
9. Carabello, BA, Crawford, FA. Valvular Heart Disease. New Engl J of Med 1997;337:32-40
10. Movsowitz HD, Levine RA, Hilgenberg AD, et al. Transesophageal Echocardiographic Description of the Mechanisms of Aortic Regurgitation in Acute Type A Aortic Dissection: Implications for Aortic Valve Repair. J Am Coll Cardiol 2000;36:884-890
11. Willett D L, Hall SA, Jessen ME, et al. Assessment of Aortic Regurgitation by Transesophageal Color Doppler Imaging of the Vena Contracta: Validation against an Intraoperative Aortic Flow Probe. J Am Coll Cardiol 2001;37:1450-1455
12. Baumgarten H, Schima H, Kuhn P. Value and limitations of proximal jet dimensions for the quantitation of valvular regurgitation: an in vitro study using Doppler flow imaging. J Am Soc Echocardiogr 1991;4:57-66
13. Enriquez-Sarano M, Seward JB, Bailey KR, et al. Effective regurgitant orifice area: a noninvasive Doppler development of an old hemodynamic concept. J Am Coll Cardiol 1994;23:443-451
14. Perry GJ, Helmcke F, Nanda N, et al. Evaluation of aortic insufficiency by Doppler color flow mapping. J Am Coll Cardiol 1987;9:952-9
15. Ishii M, Jones M, Shiota T, et al. Quantifying aortic regurgitation by using the color Doppler-imaged vena contracta: a chronic animal model study. Circulation 1997;96:2009-2015
16. Bonow RO, Lakatos E, Maron BJ, et al. Serial long term assessment of the natural history of asymptomatic patients with chronic aortic regurgitation and normal left ventricle systolic function. Circulation 1991;84:1625-1635
17. Otto CM. Textbook of Clinical Echocardiography, 2nd Edition 2000. WB Saunders Company, Philadelphia.
18. Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. J Am Soc Echocardiogr 2003;16:777-802
19. Quinones MA, Otto CM, Stoddard M, et al. Recommendations for quantification of Doppler echocardiography: A report of the nomenclature and standards committee of the American Society of Echocardiography. J Am Soc Echocardiogr 2002;15:167-84

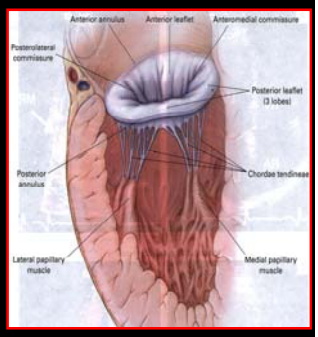
MITRAL VALVE
Anatomy and TEE exam

PD Dr D. Bettex
University Hospital Zurich



MITRAL VALVE

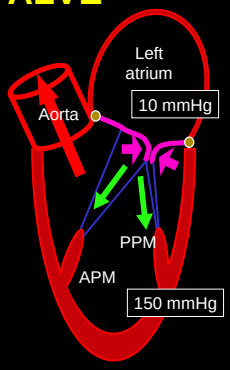
- Leaflets
- Chordae tendinae
- Papillary muscles
- Saddle-shaped annulus
- Fibrous skeleton of the heart
- Left ventricular walls



NEJM 2001

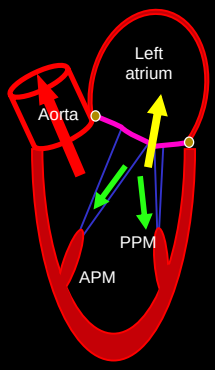
MITRAL VALVE

- Contraction of the left ventricle
- Intracavitary pressure ⇑
- Tightness of mitral valve depending upon
  - Occluding forces
  - Tethering forces



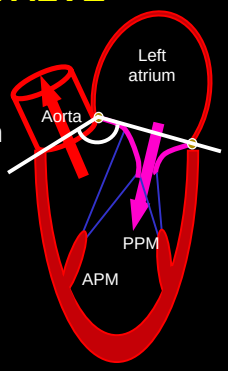
MITRAL VALVE

- Contraction of the left ventricle
- Intracavitary pressure ⇑
- Tightness of mitral valve depending upon
  - Occluding forces
  - Tethering forces
- Leak if edge to edge

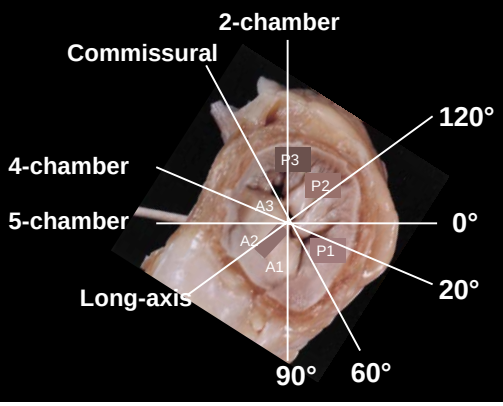


MITRAL VALVE

- Inflow-outflow planes angle
  - Small angle: admission chamber too close to LVOT -> risk of SAM and LVOTO
- Better filling-ejection if inflow-outflow almost parallel

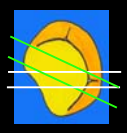


MITRAL VALVE (Carpentier)

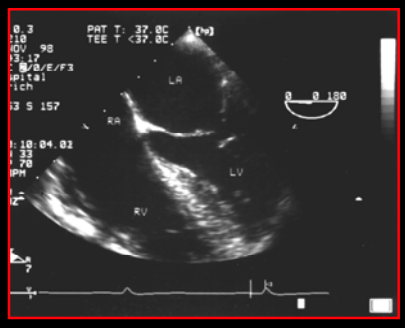


MITRAL VALVE four-chamber view

Mitral annulus
Ant. + post. leaflets
A3 (A2) + P1(P2)
Ant. commissure



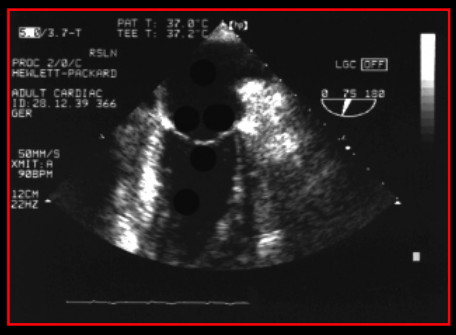
FOUR-CHAMBER VIEW



MITRAL VALVE commissural view

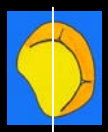
Ant. + post. leaflets
A2 (P2), P1 + P3
Commissures

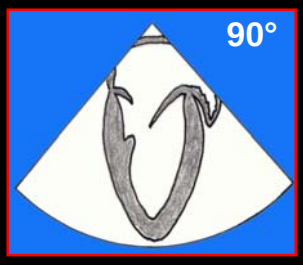
COMMISSURAL VIEW



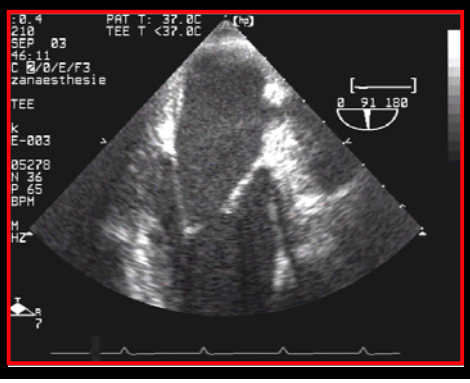
MITRAL VALVE two-chamber view

Mitral annulus
Ant + post leaflets
A1 + P3
Post commissure



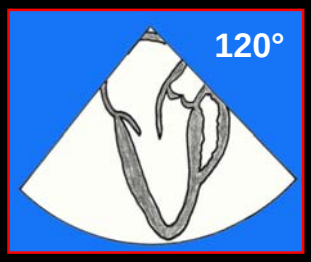


TWO-CHAMBER VIEW

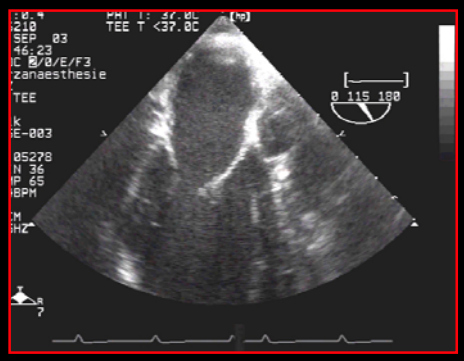


MITRALVALVE long-axis view

Ant + post leaflets
A2 + P2
Annulus highest level



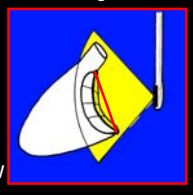
LONG-AXIS VIEW



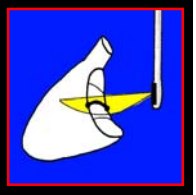
SADDLE-SHAPED ANNULUS

- Coaptation point
  - 4-chamber view
  - Long-axis view
- Mitral valve prolapse

120° long-axis

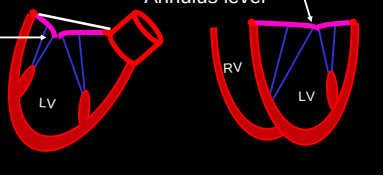


0-20° 4-chamber

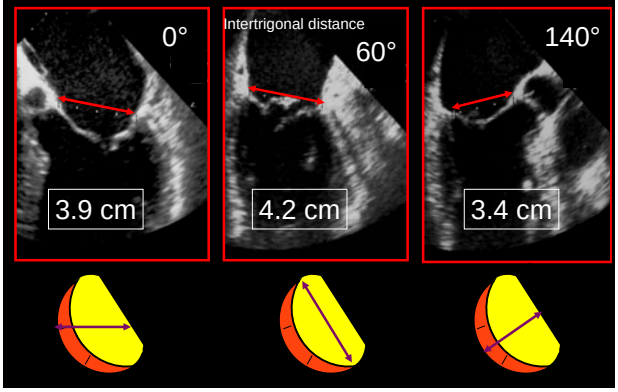


Coaptation on ventricular side

Coaptation on Annulus level

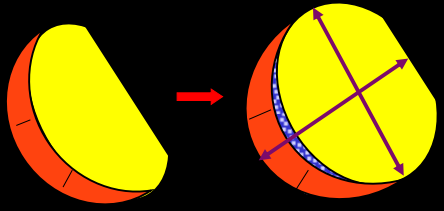


MITRAL DIAMETER



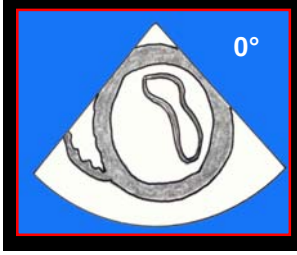
MITRAL DIAMETER

- Mitral ring dilatation: circular shape
- Equalization of diameters
- Medial gap

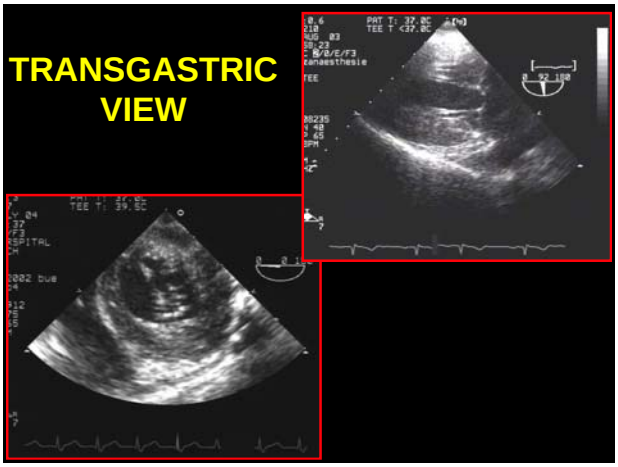


TRANSGASTRIC SHORT-AXIS VIEW

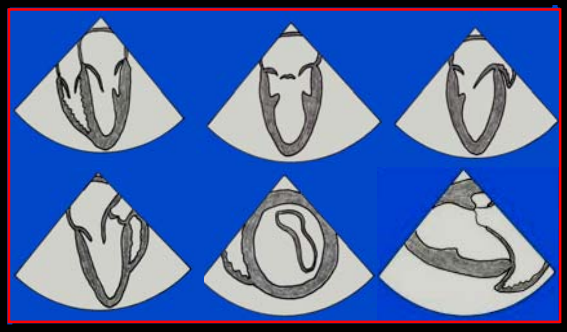
Ant + post leaflet
Ventricular wall
Chordae tendinae
Papillary muscles



TRANSGASTRIC VIEW

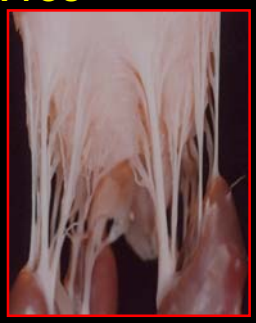


2D SCANNING OF THE MV



THANK YOU

La Sagrada Familia, Barcelona
Antoni Gaudi (1852-1920)



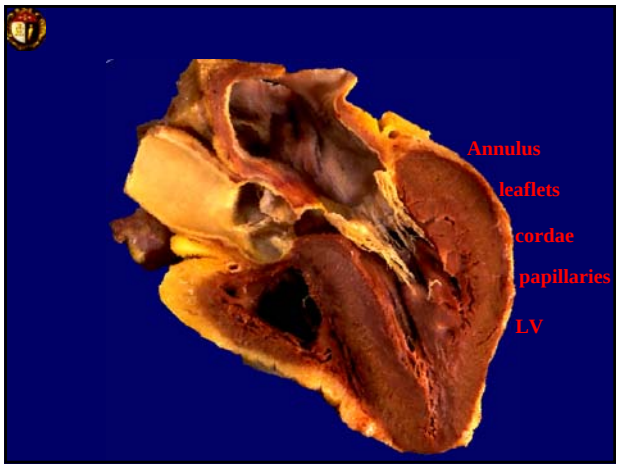
Mitral subvalvular apparatus
Ventricular view

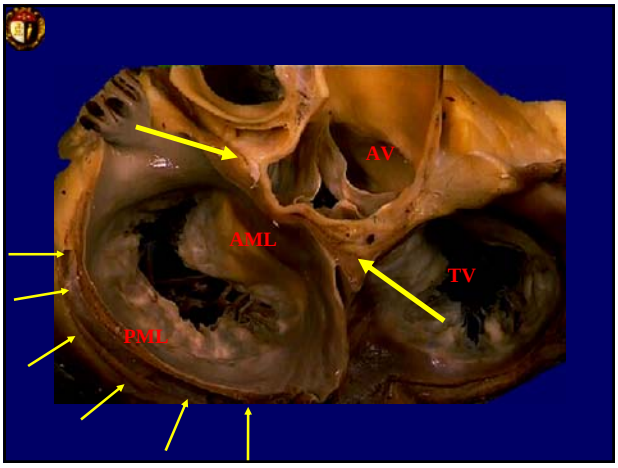
Mitral Regurgitation

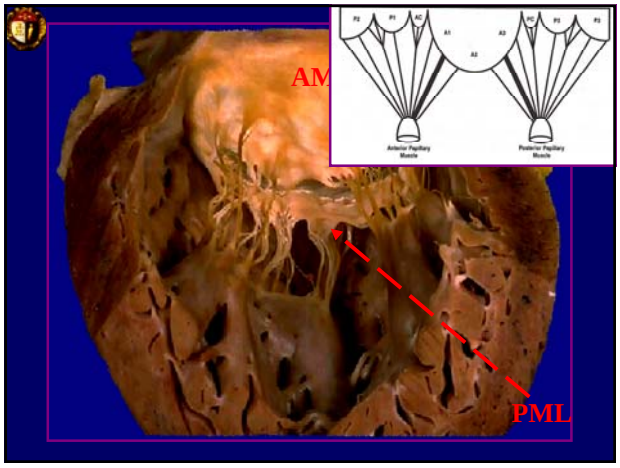
Fabio Guarracino

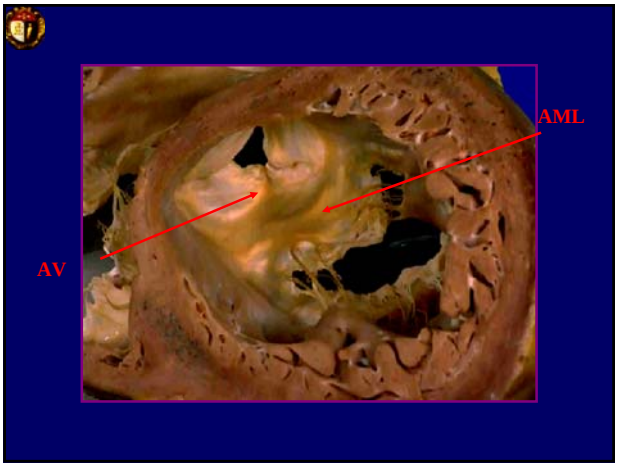
Department of Cardiothoracic Anaesthesia and ICU

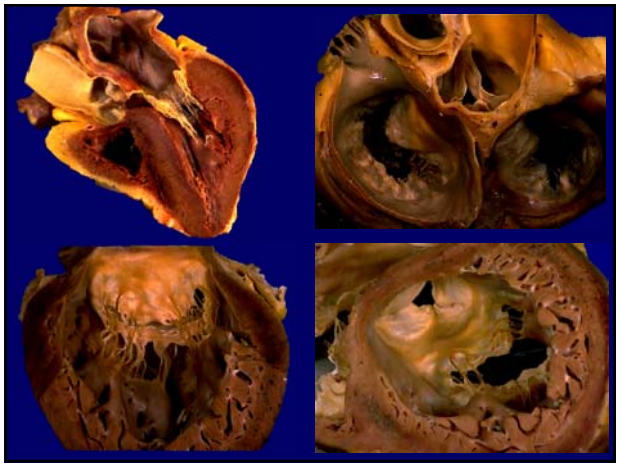
University Hospital of Pisa, Italy

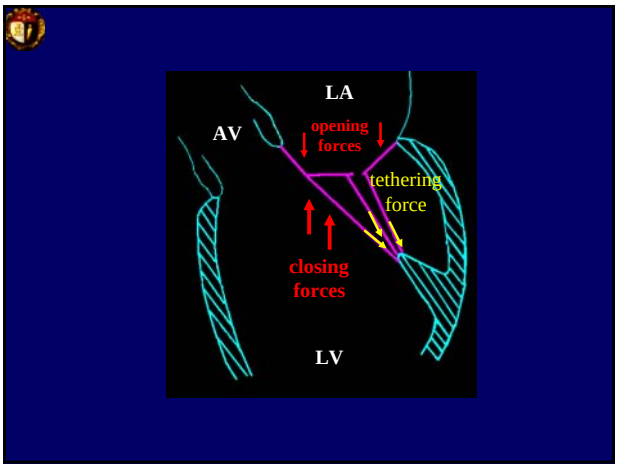


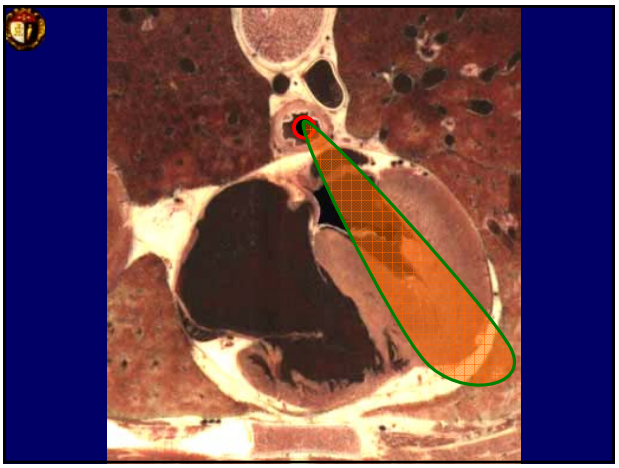


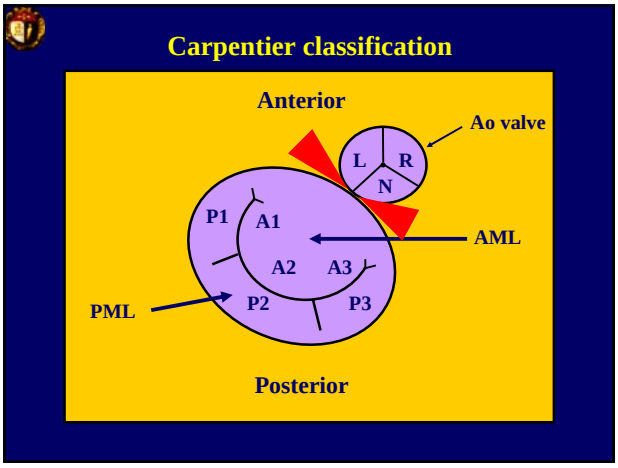


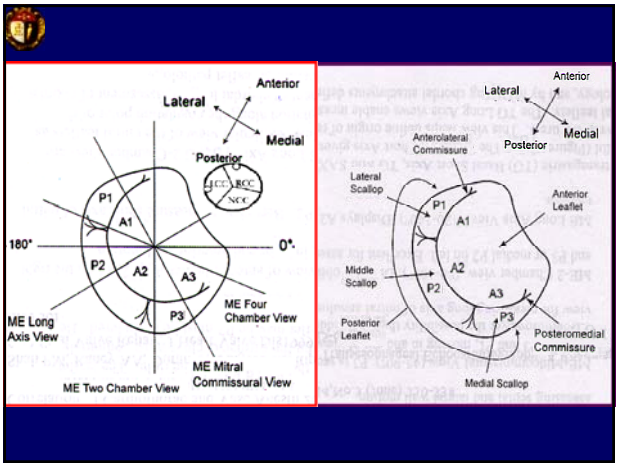


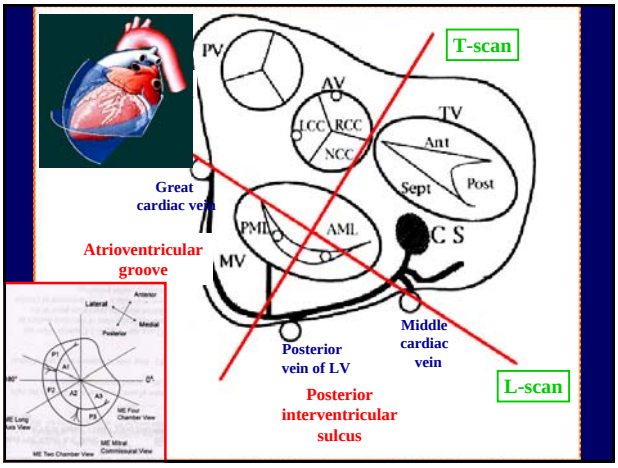


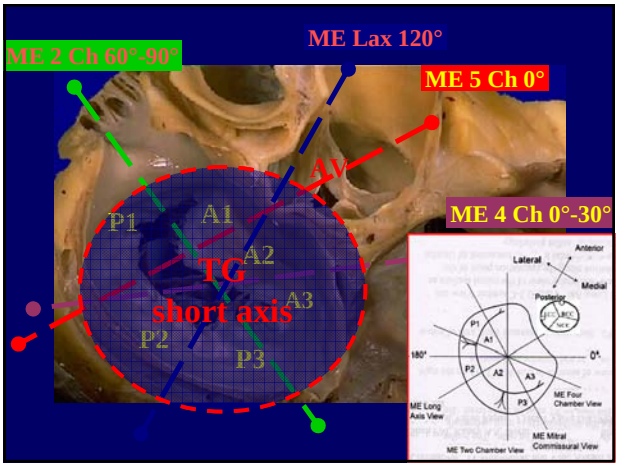

LA
AV
opening forces
tethering force
closing forces
LV

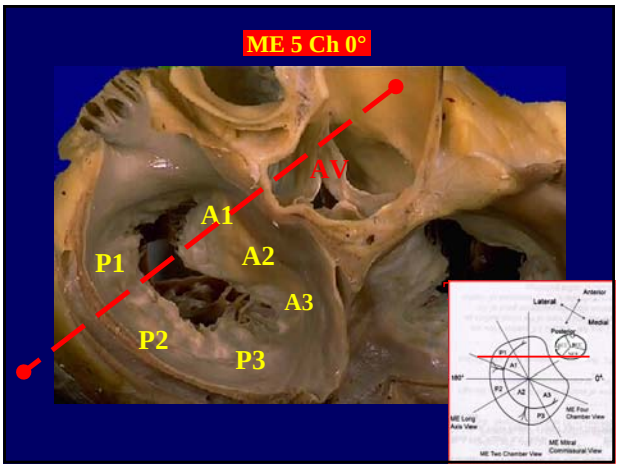


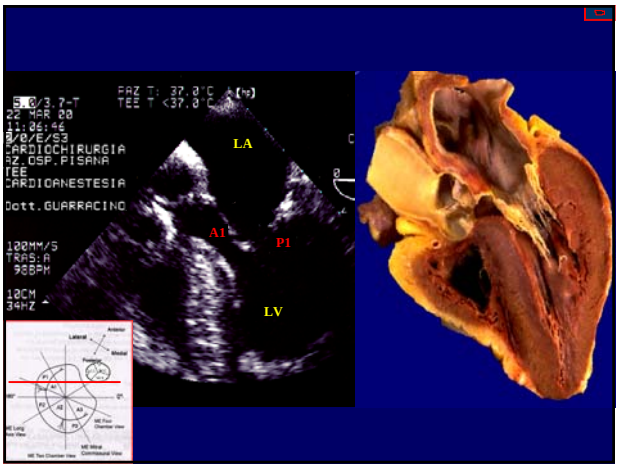

Carpentier classification
Anterior
Ao valve
L
R
N
P1
A1
A2
A3
AML
PML
P2
P3
Posterior

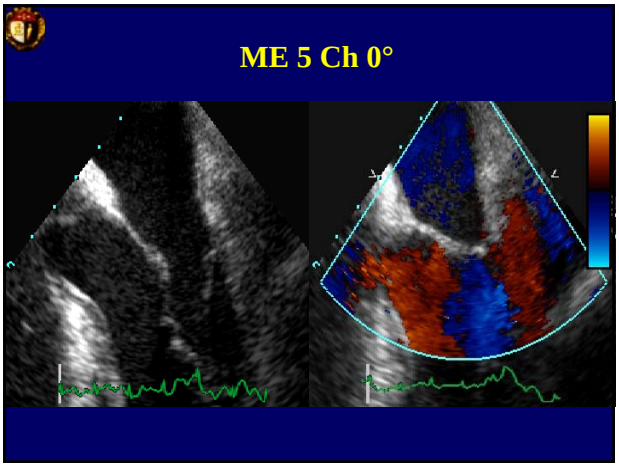

Anterior
Lateral
Medial
Posterior
P1
A1
180°
0°
P2
A2
A3
P3
ME Long Axis View
ME Four Chamber View
ME Two Chamber View
ME Mitral Commissural View
Lateral
Anterior
Medial
Posterior
Anterolateral Commissure
Lateral Scallop
Anterior Leaflet
Middle Scallop
Posterior Leaflet
Posteromedial Commissure
Medial Scallop

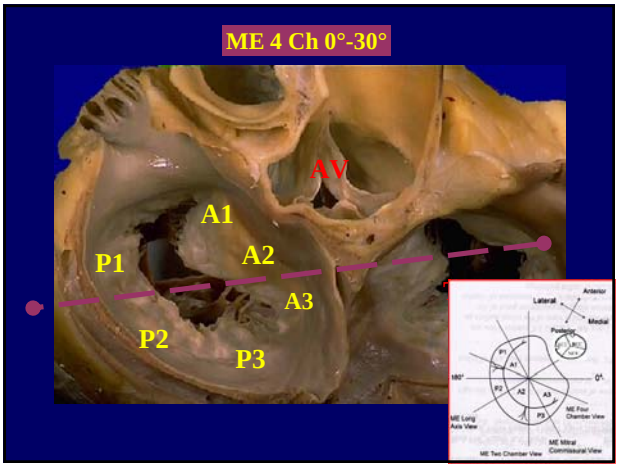

T-scan
PV
AV
LCC
RCC
NCC
TV
Ant
Sept
Post
Great cardiac vein
PML
AML
C S
MV
Atrioventricular groove
Posterior vein of LV
Middle cardiac vein
Posterior interventricular sulcus
L-scan

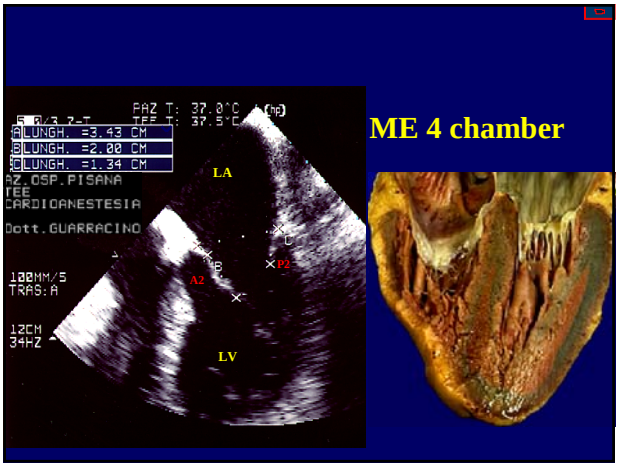

ME Lax 120°
ME 2 Ch 60°-90°
ME 5 Ch 0°
AV
ME 4 Ch 0°-30°
P1
A1
A2
TG
short axis
A3
P2
P3

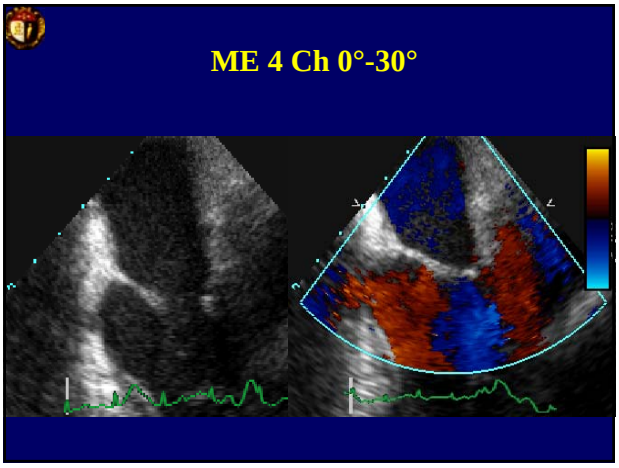

ME 5 Ch 0°
AV
A1
A2
A3
P1
P2
P3

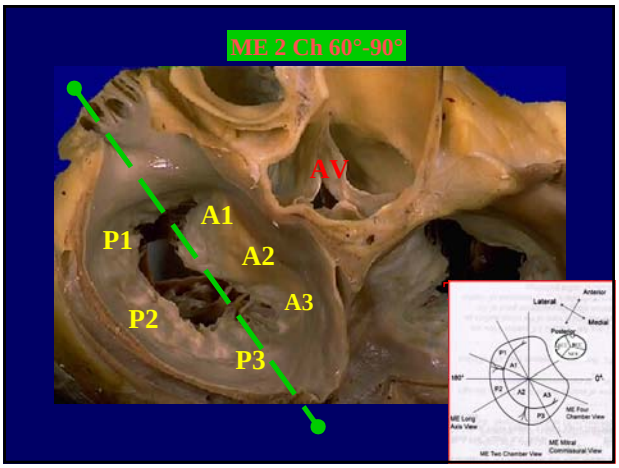

LA
A1
P1
LV

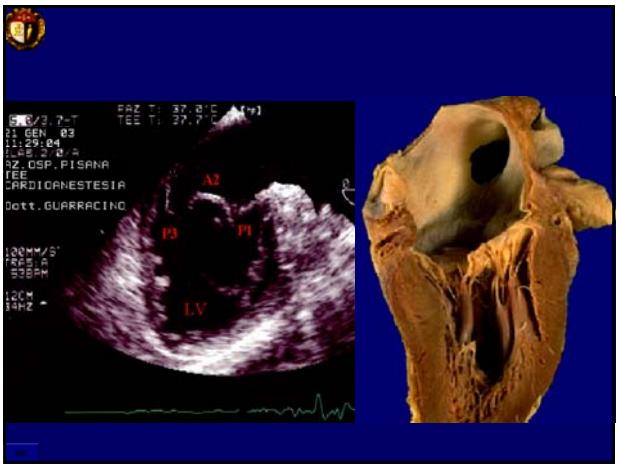

ME 5 Ch 0°

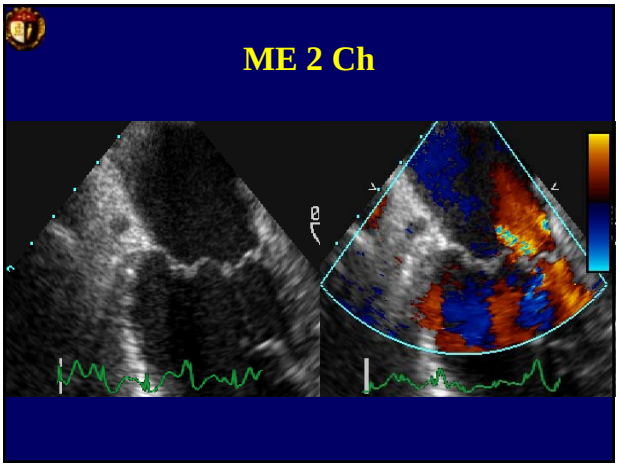

ME 4 Ch 0°-30°
AV
A1
A2
A3
P1
P2
P3

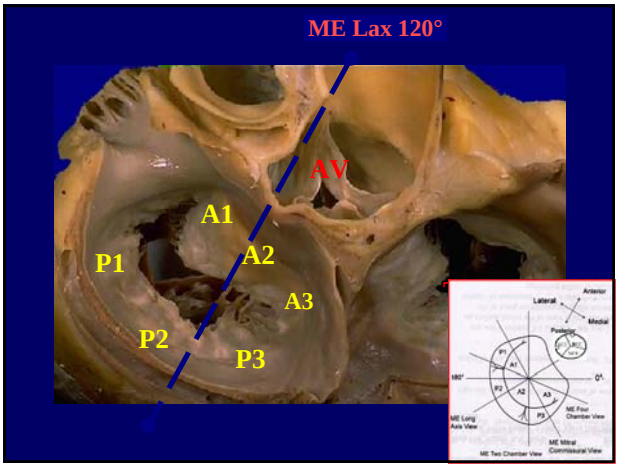

ME 4 chamber
LA
LV

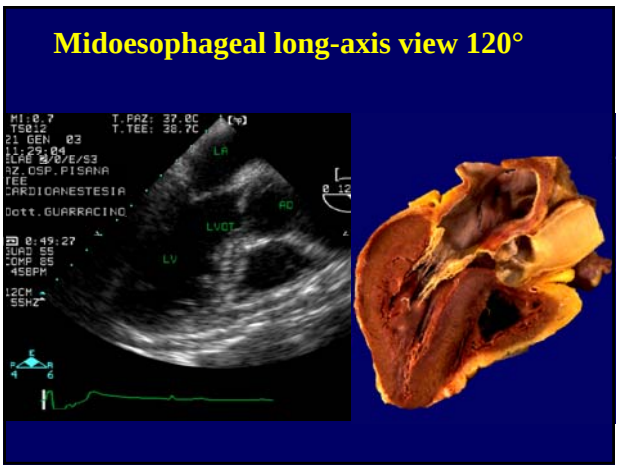

ME 4 Ch 0°-30°

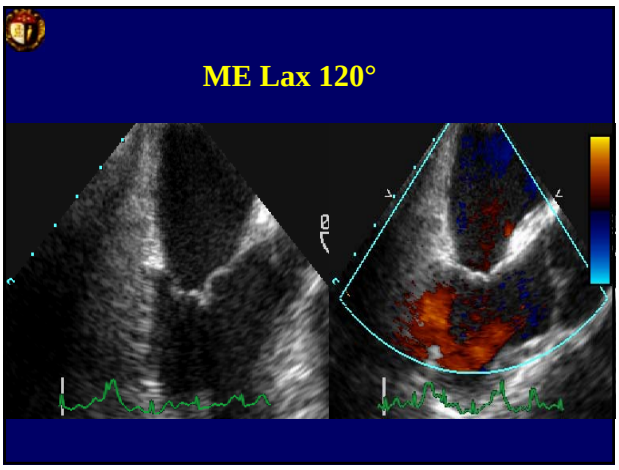

ME 2 Ch 60°-90°
AV
A1
P1
A2
A3
P2
P3


A2
P3
P1
LV

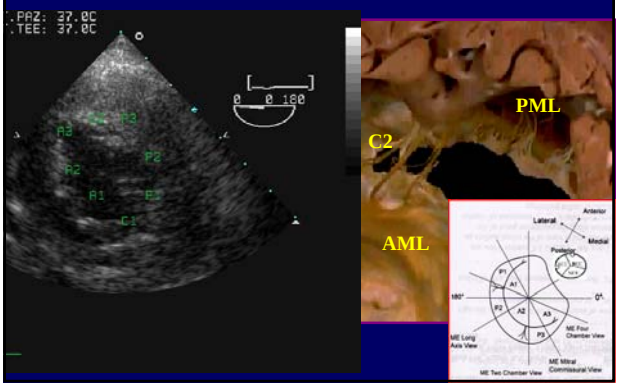

ME 2 Ch


ME Lax 120°
AV
A1
P1
A2
A3
P2
P3

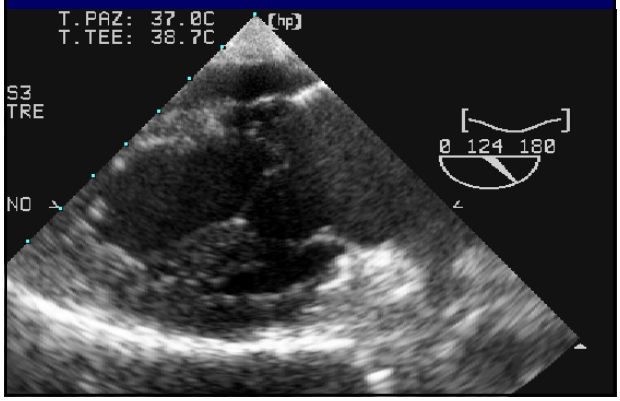

Midoesophageal long-axis view 120°


ME Lax 120°

TG short axis 0°



TG long axis 120°

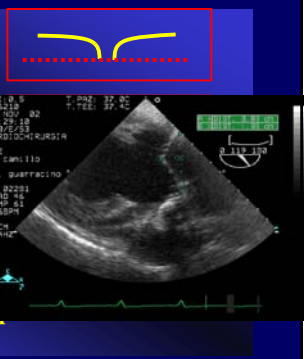


Functional evaluation

- leaflets
- annulus dinamics
- subvalvolar apparatus
- annulus/apex
- LV: shape and function

Leaflets

- Apposition
- Coaptation



Mitral leaflets movement

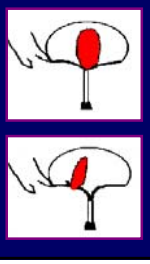
The Carpentier classification

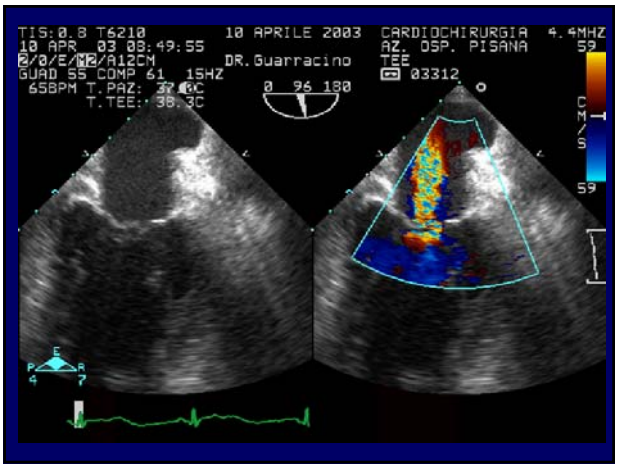
- Normal → type I
- Excessive → type II
- Restricted → type III
  a) b)

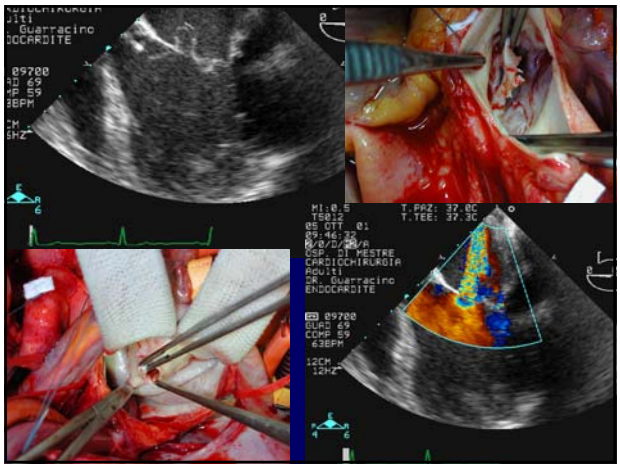
Mitral leaflets movement

- Normal

- Dilatation
- Endocarditis
- Ischaemic



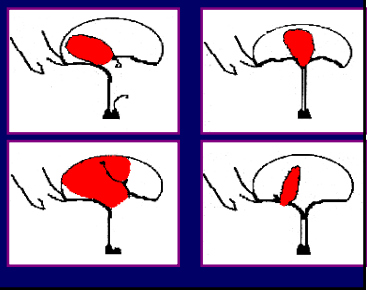


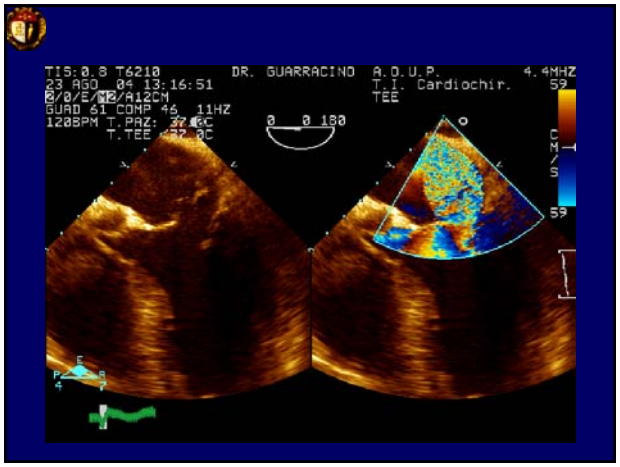


Mitral leaflets movement

- Excessive

- Degenerative
- Mixomatous
- Endocarditis
- Ischaemic





Mitral leaflets movement

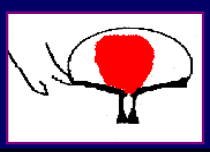
- Restricted

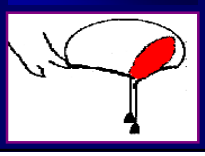
type A diastolic

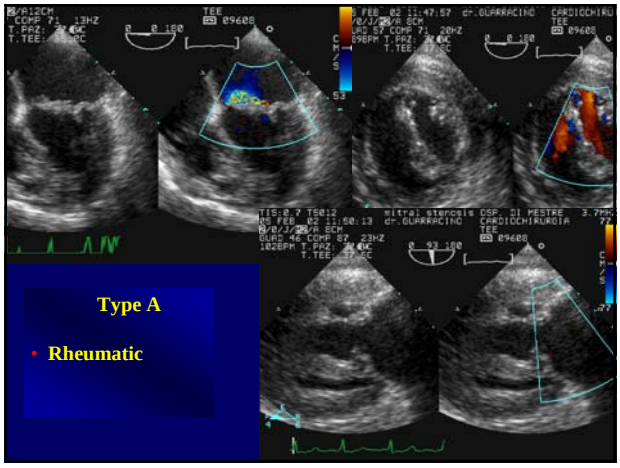
- Rheumatic

type B systolic

- Ischaemic
- Functional







Type A

- Rheumatic

Type B

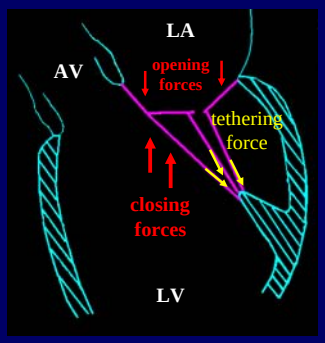
- Ischaemic
- Functional

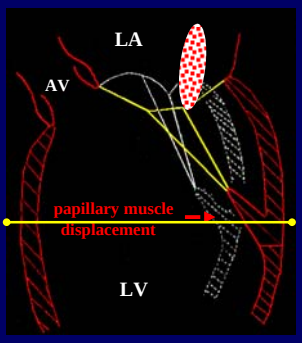
Ischaemic MVR

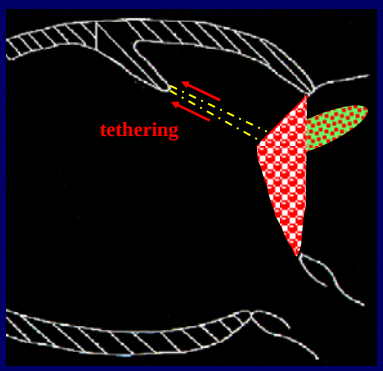
- MR after AMI
- CAD with at least one stenosis > 75 %
- No intrinsic MV pathology
- Incidence 20% post AMI

Functional MVR

- MR in the context of LV dysfunction
- No intrinsic MV pathology



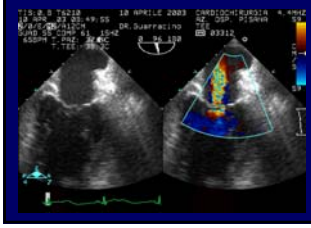


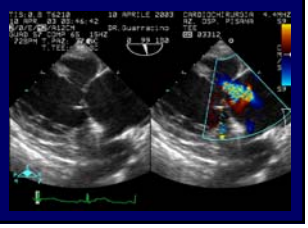


Ischemic MR

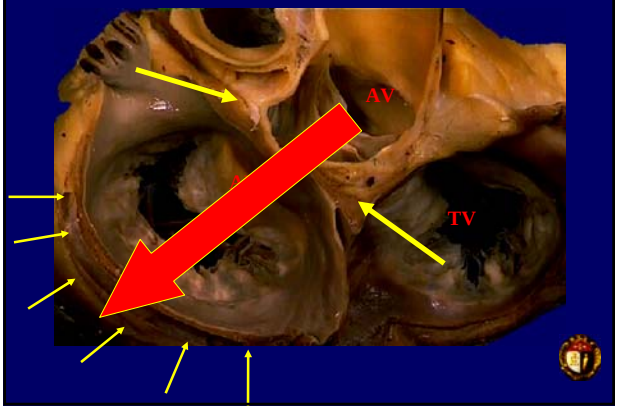
basal geometry

mid-distal geometry

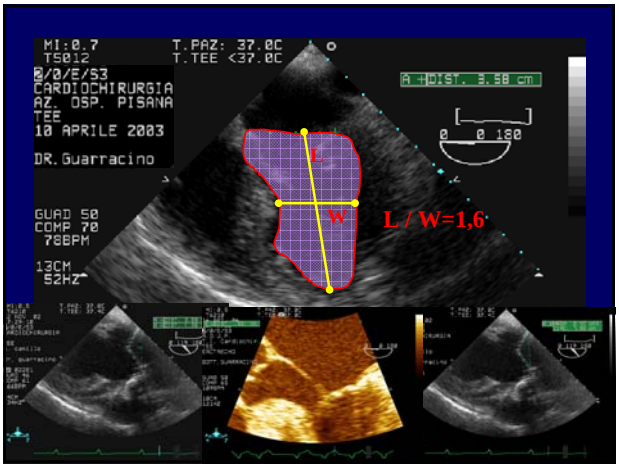




Annulus



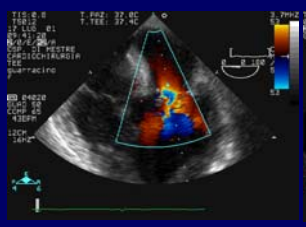
Annulus

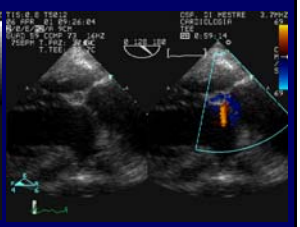


Color Doppler: jet direction

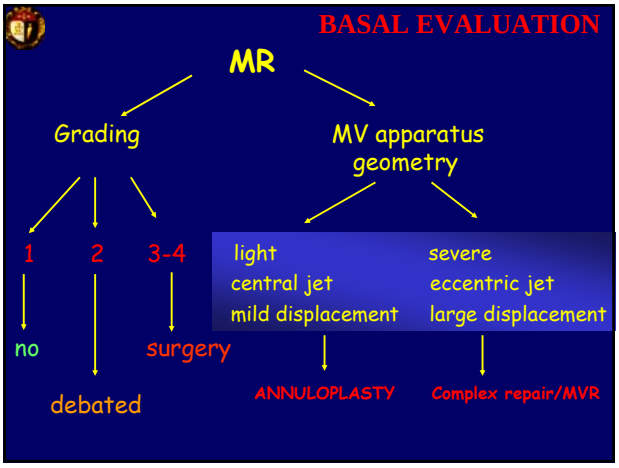
Type II
Opposite to involved leaflet

Type III b
To involved leaflet





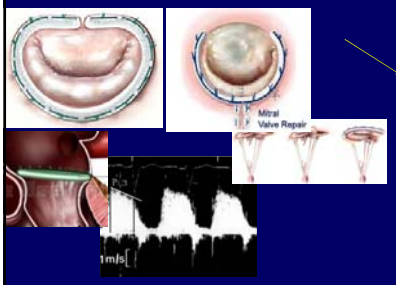
BASAL EVALUATION



Evaluation of MV repair post CPB

- Systematic approach
- Optimize load conditions (EDA/LAP/WP)
- Check LV function
- Complications

MV repair: what to look at post-op



Complex MV repair

RESIDUAL RIGURGITATION ?

Mean Δp < 3 mm Hg

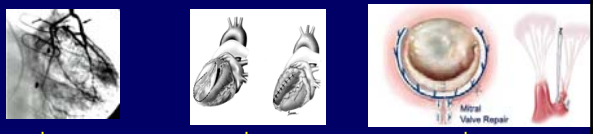
Residual rigurgitation

MV repair: what to look at post-op

CABG → WMA

LV restoring → GLOBAL FUNCTION

MV repair → Residual gradient/MR



Decision making after CPB weaning

Assess efficacy of repair

- NO MR → OK
- Mild → ACCEPTABLE
- Moderate or more → 2nd PUMP RUN

Grading MR

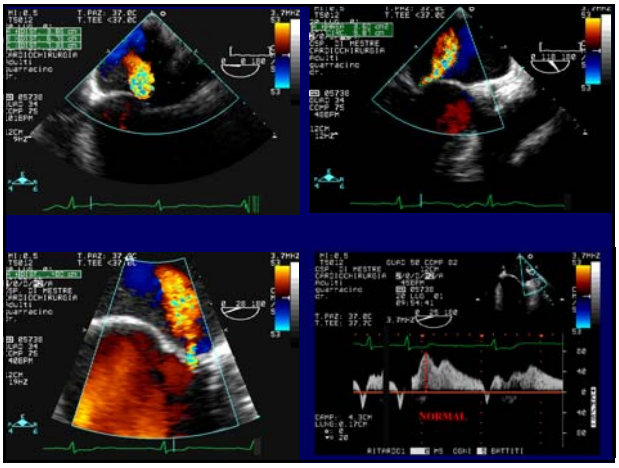
Semiquantitative methods

Color Doppler based:
- jet extension
- jet area
- vena contracta

Pulmonary flow Doppler

Quantitative methods

- Regurgitant Volume
- Regurgitant Fraction
- PISA
- EROA



AMERICAN SOCIETY OF ECHOCARDIOGRAPHY REPORT

J Am Soc Echocardiogr 2003;16:777-802.

Recommendations for Evaluation of the Severity of Native Valvular Regurgitation with Two-dimensional and Doppler Echocardiography

Table 1 Qualitative and quantitative parameters useful in grading mitral regurgitation severity

	Mild	Moderate	Severe
Structural parameters			
LA size	Normal*	Normal or dilated	Usually dilated**
LV size	Normal*	Normal or dilated	Usually dilated**
Mitral leaflets or support apparatus	Normal or abnormal	Normal or abnormal	Abnormal/ Flail leaflet/ Ruptured papillary muscle
Doppler parameters			
Color flow jet area‡	Small, central jet (usually < 4 cm^2 or < 20% of LA area)	Variable	Large central jet (usually > 10 cm^2 or > 40% of LA area) or variable size wall-impinging jet swirling in LA
Mitral inflow –PW	A wave dominant§	Variable	E wave dominant§ (E usually 1.2 m/s)
Jet density –CW	Incomplete or faint	Dense	Dense
Jet contour –CW	Parabolic	Usually parabolic	Early peaking–triangular
Pulmonary vein flow	Systolic dominance§	Systolic blunting§	Systolic flow reversal†
Quantitative parameters¶			
VC width (cm)	< 0.3	0.3-0.69	≥ 0.7
R Vol (ml/beat)	< 30	30-44 45-59	≥ 60
RF (%)	< 30	30-39 40-49	≥ 50
EROA (cm^2)	< 0.20	0.20-0.29 0.30-0.39	≥ 0.40

J Am Soc Echocardiogr 2003;16:777-802.

Table 2 Echocardiographic and Doppler parameters used in the evaluation of mitral regurgitation severity: Utility, advantages, and limitations

	Utility/advantages	Limitations
Structural parameters		
LA and LV Size	Enlargement sensitive for chronic significant MR, important for outcomes. Normal size virtually excludes significant chronic MR.	Enlargement seen in other conditions. May be normal in acute significant MR
MV leaflet/support apparatus	Flail valve and ruptured papillary muscle specific for significant MR	Other anomalities do not imply significant MR
Doppler parameters		
Jet area–Color Flow	Simple, quick screen for mild or severe central MR; evaluates spatial orientation of jet	Subject to technical, hemodynamic variation; significantly underestimates severity in wall-impinging jets
Vena contracta width	Simple, quantitative, good at identifying mild or severe MR	Not useful for multiple MR jets; intermediate values require confirmation. Small values; thus small error leads to large % error
PISA method	Quantitative; Presence of flow convergence at Nyquist limit of 50–60 cm/s alerts to significant MR. Provides both, lesion severity (EROA) and volume overload (R Vol)	Less accurate in eccentric jets; not valid in multiple jets. provides peak flow and maximal EROA.
Flow quantitation–PW	Quantitative, valid in multiple jets and eccentric jets. Provides both lesion severity (EROA, RF) and volume overload (R Vol)	Measurement of flow at MV annulus less reliable in calcific MV and/or annulus. Not valid with concomitant significant AR unless pulmonic site is used.
Jet profile–CW	Simple, readily available	Qualitative; complementary data.
Peak mitral E velocity	Simple, readily available, A-wave dominance excludes severe MR	Influenced by LA pressure, LV relaxation, MV area, and atrial fibrillation. Complementary data only, does not quantify MR severity
Pulmonary vein flow	Simple, Systolic flow reversal is specific for severe MR	Influenced by LA pressure, atrial fibrillation. Not accurate if MR jet directed into the sampled vein

Table 3 Application of specific and supportive signs, and quantitative parameters in the grading of mitral regurgitation severity

	Mild	Moderate	Severe
Specific signs of severity	• Small central jet < 4 cm^2 or < 20% of LA area# • Vena contracta width <0.3 cm • No or minimal flow convergence‡	Signs of MR>mild present, but criteria for severe MR	• Vena contracta width ≥ 0.7cm *with* large central MR jet (area > 40% of LA) or *with* a wall-impinging jet of any size, swirling in LA# • Large flow convergence‡ • Systolic reversal in pulmonary veins • Prominent flail MV leaflet or ruptured papillary muscle
Supportive signs	• Systolic dominant flow in pulmonary veins • A-wave dominant mitral inflow§ • Soft density, parabolic CW Doppler MR signal • Normal LV size*	Intermediate signs/findings	• Dense, triangular CW Doppler MR jet • E-wave dominant mitral inflow (E >1.2 m/s)§ Enlarged LV and LA size**, (particularly when normal LV function is present).
Quantitative parameters¶			
R Vol (ml/beat)	< 30	30-44 45-59	≥ 60
RF (%)	< 30	30-39 40-49	≥ 50
EROA (cm^2)	< 0.20	0.20-0.29 0.30-0.39	≥ 0.40

TEE study of the mitral valve

- ♥ study of functional anatomy
- ♥ understanding mechanisms of native MV dysfunction
- ♥ grading MV dysfunction
- ♥ evaluation of feasibility and result of MV repair

Mitral stenosis

P Caso, S Comenale, L Nunziata, R Ancona, M Macrino, AR Martiniello , S Severino, R Calabrò
Non Invasive Cardiology, Department of Cardiology, Monaldi Hospital, Naples,Italy

Transesophageal echocardiography (TEE) has some advantages over Transthoracic echocardiography (TTE) when it used to analyse mitral valve. These advantages are due to the proximity of the oesophagus to the structure of mitral valve and the use of a higher frequency ultrasound probe giving better definition.
Despite of its advantages, transesophageal echocardiography is not the ideal technique for evaluating rheumatic mitral stenosis. In rheumatic mitral stenosis 2D echo allows detailed evaluation of mitral valve morphology,very important for percutaneous mitral commisurotomy (PMC). Mobility, thickening, calcification of the leaflets, and sometimes the subvalvular apparatus, can usually be well evaluated during a transthoracic echocardiography in parasternal and apical view. Also the dimension of leaf atrium and mitral valve area can be studied better in transthoracic view. The mitral valve area in 2D echo can be studied in parasternal short axis view with direct planimetry as the smallest orifice in short axis in diastole in the more restricted mitral inflow view in the time.
The commissures antero-lateral and postero-medial can be evaluated in parasternal short axis view discovering the presence of the calcium on one or both commissures (contraindication PMC), and as symmetrical or non symmetrical configuration.
Mobility, calcification, thickening and subvalvular apparatus are component of the Wilkins' Score for mitral valve morphology that gives a score from 1 to 4 to each of four morphology component. The ideal valve score for predicting better outcome after percutaneous mitral commissurotomy is a valve with score < 8.

Wilkins et al, Br Heart J 1988;60:299-308.

Also the Doppler interrogation of flow across the mitral orifice is best performed from the apical four chambers view. This position allows us to evaluate:
1) Doppler derived area either with PHT or PISA methods;
2) mean mitral valve gradient (too volume dependent);
3) mitral regurgitation grade (<2 is important for selection of mitral percutaneous valvuloplasty);
4) right ventricular pressure.
The role of TEE in patients with mitral stenosis is to supplement TTE, for example when it is necessary to choose as operative treatment between balloon mitral valvuloplasty or surgery to repair or change mitral valve. TEE is being considered the most accurate method of establishing whether or not there is thrombus in the left atrium, for example in patients who are being assessed for balloon valvulotomy and in those with atrial fibrillation who are considered for cardioversion. Thrombus may be restricted to the left atrial appendage, or it may be laminar and closely applied to the wall of the left atrium so that it is only appreciated on detailed imaging in several planes.
In patients with mitral stenosis, TEE also demonstrates spontaneous contrast within the atria, much more often than TTE. This is a risk factor for embolism. In some circumstances (i.e. if the valve is heavily calcified) TEE could be better than TTE in evaluation morphology of subvalvular apparatus and the leaflets.
It is possible to define the anatomy of the MV apparatus using the degree of rotation presented by Foster et al . The mid oesophageal (30-40 cm from incisors) four-chambers view displays the larger appearing anterior leaflet mitral valve ALMV (A3) to the left of the posterior leaflet mitral valve PLMV (P1). Antiflexing the probe provides imaging of the anterior aspect of the MV, while gradual advancement of the probe and retroflexion shifts the image plane to the posterior aspect of the MV. Maintaining the probe at this depth and rotating the multiplane angle forward to 60-70° develops a

mitral commissural view in which A2 is surrounded by P1 on the right and P3 on the left, giving A2 the appearance of a "trap door" as it moves in and out of the imaging plane throughout the cardiac cycle. Further forward rotation of probe to 80-100° develops the two chamber view, at this depth, revealing P3 to the left and A1 on the right. Final forward probe rotation to 120-160° reveals the long axis view at this depth, which images P2 on the leaft and A2 on the right.
The transgastric basal short axis view enables visualization of both MV leaflets ("fish mouth view") if the probe is the anteflexed and withdrawn slightly from the mid papillary level of the left ventricle. In this view, the posteromedial, commissure is in the upper left, the anterolateral, commissure to the lower right, the PLMV is to the right, and the ALMV to the left of the displayed image. Rotation of the probe to 80-100° develops the transgastric two-chamber view, which is particularly useful for evaluating the chordae tendinae and corresponding papillary muscles.

TEE can be used, during valvuloplasty in patients that cannot take X-ray:
1) to guide percutaneous mitral valvuloplasty as puncture of atrial septum and mitral inflow entrance;
2) to assess the degree mitral regurgitation before and after the intervention;
3) to evaluate the result;
4) to depict the site and size of the atrial septal defect caused by trans-septal puncture.
The most part of Authors prefers follow with TTE alone patients during valvuloplasty showing the result as better mobility, change in area and in Doppler (PHT, not sure after PMC) and color Doppler data (color M-mode).
Particularly population in which PMC could be favourite are women in pregnancy, population with mitral valve score > 10, patients with pulmonary hypertension, older patients > 70 years and patients with restenosis after PMC.
Mitral valve stenosis is the most common valve lesion in pregnancy. In spite of an optimized clinical treatment and a favourable valve anatomy according to Wilkins, in symptomatic patients, percutaneous intervention is shown to be of great importance. In these patients, avoiding x-ray exposure as much as possible is recommended so as to protect the fetus from the deleterious effects of ionizing radiation.
For this reason, valvuloplasty was performed using only transesophageal echocardiography, which made the procedure easy and safe. This option should be encouraged in pregnant patients, particularly with the use of the Inoue technique, because of its greater simplicity, and performed by teams experienced in the percutaneous treatment of mitral stenosis.
In patients with normal left atrium and sinus rhythm, Pollick & Taylor observed at the transesophageal echocardiography that the effective opening of the stenosed mitral orifice resulting from the percutaneous balloon valvulotomy determined an improvement of the flow pattern of the left atrial appendage, which can potentially contribute for the reduction of the embolic risk.

The TEE, nevertheless, has some disadvantages compared with TTE. In particular, a transducer can't allow a complete study of the zone of coaptation of the mitral valve, some transesophageal transducers don't incorporate the facility for continuous wave Doppler and so recording of the complete waveform of regurgitant jets, and analysis of pressure gradients in systole are not possible. In absence of calcification, grading of regurgitation through the atrioventricular valves using colour flow mapping is probably also performed best with TTE.
Complications of PMC is the rupture of mitral leaflets, chordae attachment that can be studied with TEE.

Indications for Symptomatic patients in Class II NYHA:
a) if Mitral valve area is > 1.5 cm2, exercise pulmonary artery systolic pressure >60 mmHg, Wilkins score < 8 there is indication for PMC(to see mitral regurgitation <2,and no thrombi with TEE) (Class IIb);
b) if stenosis is severe <1.5 cm2 area, score of Wilkins < 8 PMC (Class I); while if the valve is not

ideal ,but there is pulmonary hypertension as PA > 60 mmhg, it is better to consider for surgery commissurotomy o valve replacement (Class IIa).

Indications for symptomatic patients in III-IV class NYHA
mitral valve area> 1,5 cm2,
exercise pulmonary artery systolic pressure > 60 mmHg,
Wilkins score < 8 there is indication for PMC,

Class IIb; if area is < 1.5 cm,
Wilkins score < 8 there is indication for PMC (Class I);
otherwise in patients with Wilkins' score > 8 there is indication for mitral valve replacement or mitral valve repair(Class 1).
In patients that are to high risk surgical candidate there is indication for PCM also (Class IIa)

Contraindications to percutaneous mitral commissurotomy are:
mitral valve area >1,5 cm2, left atrial thrombus, more than mild mitral regurgitation,
severe or bicommissural calcification,
absence of commissural fusion,
severe concomitant aortic valve disease or severe combined tricuspid stenosis and tricuspid regurgitation ,
concomitant coronary artery disease requiring bypass surgery.

References

Herrmann HC, Wilkins GT, Abascal VM, et al. Percutaneous balloon mitral valvotomy for patients with mitral stenosis. Analysis of factors influencing early results. J Thorac Cardiovasc Surg. 1988; 96:33-8.
Post J, Feldman T, Isner J, et al. Balloon mitral valvotomy in patients with severe valvular and subvalvular deformity. J Am Coll Cardiol. 1995; 25:1129-36.
Hameed A, Karaalp IS, Tummala PP, et al. The effect of valvular heart disease on maternal and fetal outcome of pregnancy. J Am Coll Cardiol. 2001 Mar 1; 37(3):893-9.
Mangione JA, Bavaresco Cristovao SA, Souto MaiorGI, Abensue A. Percutaneous mitral valvuloplasty in a pregnant woman guided only by transesophageal echocardiography. ArqBras Cardiol 2007; 88:e60-63.
Trehan V, Mukhopadhyay S, Nigam. Mitral Valvuloplasty by Inoue balloon under transthoracic Echocardiographic guidance. J Am Soc Echo 2005; 18:964-969.
Karakaia O, Turkmen M, Bitigen A. Effect of percutaneous mitral balloon valvuloplasty on left atrial appendage function: a Doppler Tissue study. J Am Soc Echo 2006; 19:434-7.
Vahanian A, Baumgartner H ,Bax J et al. Mitral stenosis. Guidelines on management of valvular heart disease. Eur Heart Journal 2007; 28:247-251
Bonow RO,Carabello BA,Chatterjee k et al. ACC/AHA 2006 Guidelines for the mangementof patients with valvular heart disease. J Am Coll Cardiol 2006;48,e1-e148
Fraser A G. Disease of mitral and tricuspid Valve In Transesopageal echocardiography in clinical practice. Ed Sutherland GR ,Roelandt JRTC, Fraser AG, Anderson RH. Gower Medical Publishing, London 1991 Stumper O and Sutherland Transesophageal echocardiography in congenital heart disease. Ed Arnold E ,London 1994

TEE for detection of myocardial ischemia

Manfred D. Seeberger, Department of Anesthesia, University Hospital Basel, Switzerland

mseeberger@uhbs.ch

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.

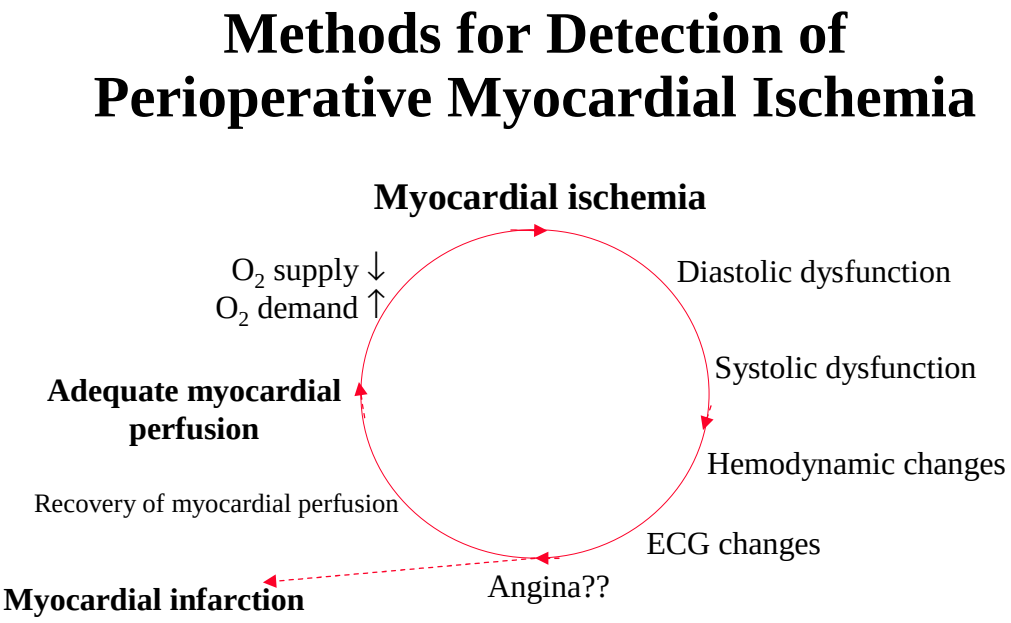
A final topic of the lecture is mechanical complications of myocardial infarction. Typical echocardiographic images of these complications will be presented, including ischemic myocardial regurgitation, papillary muscle rupture, left ventricular aneurysm, left ventricular thrombus, post infarction ventricular septal defect, and pseudaneurysm / contained rupture.

References:

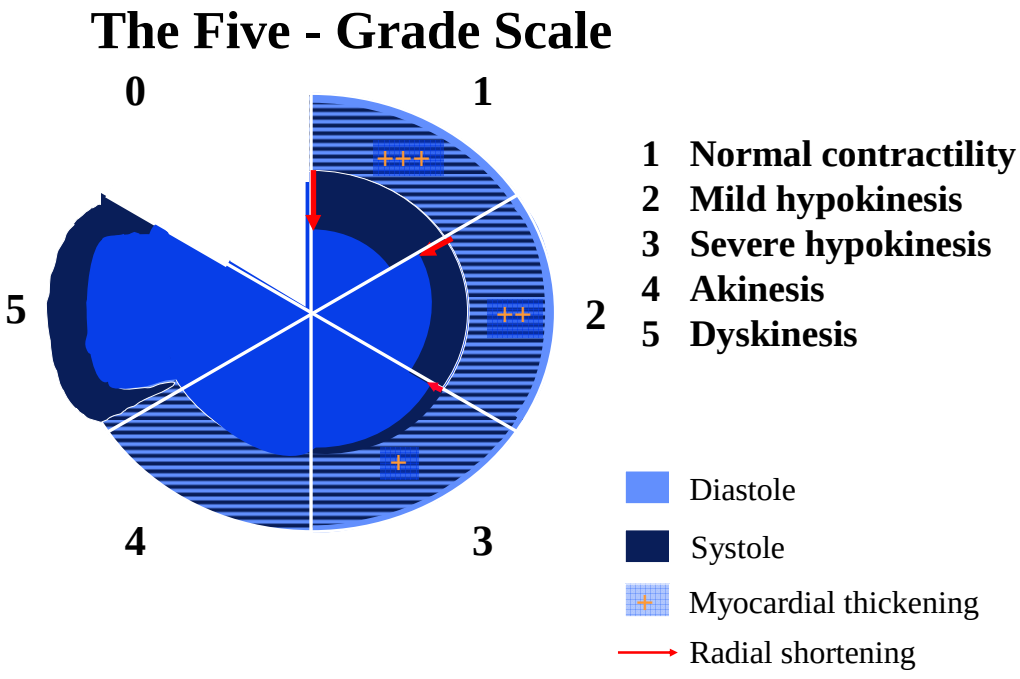
Seeberger MD, Skarvan K, Cahalan MK: Myocardial ischaemia, Transoesophageal echocardiography in anaesthesia and intensive care medicine (2nd edition), 2nd Edition. Edited by Skarvan K, Poelaert J. London, BMJ Books, 2004, pp 196-220.

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



Sigwart et al, Springer-Verlag 1984; Iskandrian et al Am Heart J 1986; Labovitz et al JACC 1987



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**

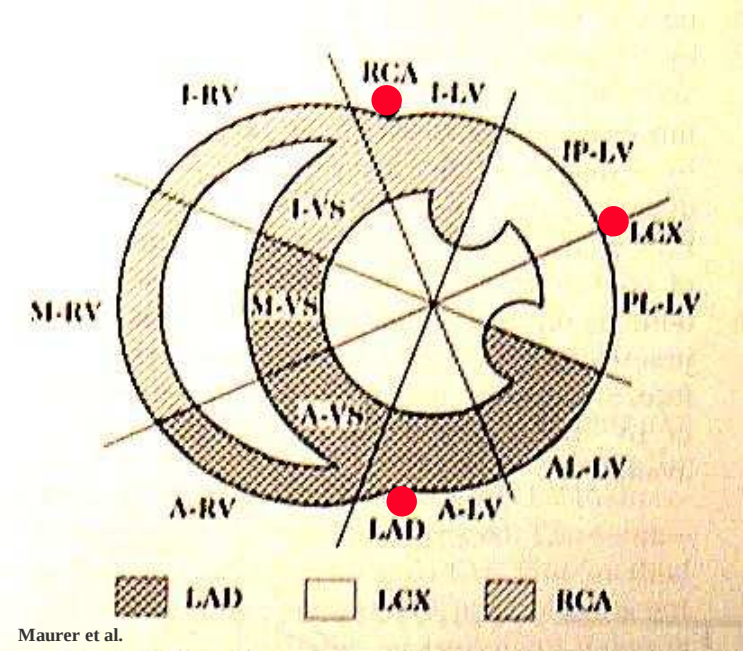
Diagnostic of ischemia if contractility worsens by 2 classes

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**

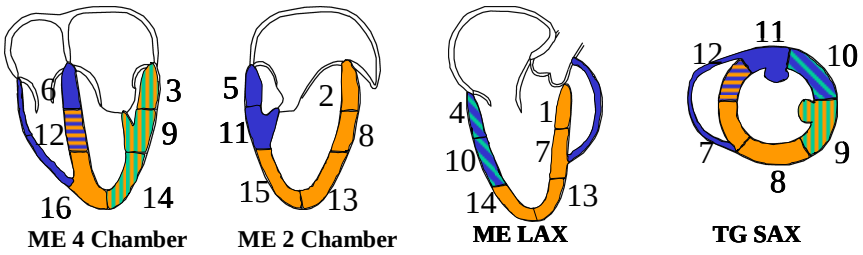
- ***Diagnostic of ischemia if contractility worsens by 1 class***
- ***No published perioperative studies***

Location of SWMA– Culprit Coronary Artery



Maurer et al.

16-Segment Model of the LV



ME 4 Chamber | ME 2 Chamber | ME LAX | TG SAX

- RCA
- LAD
- LCx
- RCA or LCx
- LAD or LCx
- RCA or LAD

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

Transoesophageal Echo in Congenital Heart Disease

Dr Owen I Miller FRACP
Consultant in Paediatric and Fetal Cardiology, Evelina Children's Hospital, Guy's & St Thomas' Hospitals, London. U.K.

Transoesophageal echocardiography (TOE) is a "value added" procedure in congenital heart disease either to add further clarity to an already known morphological diagnosis, to guide catheter or surgical intervention or provide real-time functional data before, during and after congenital heart surgery or catheter intervention.

The procedure is individualised and defined by the requirements of the surgeon or interventionalist based on the underlying morphology. A step by step approach is described for various congenital heart lesions including:

- Morphological assessment of secundum atrial septal defects (ASD) and ventricular septal defects (VSD), including their closure by device.
- Assessment of Atrio-ventricular valves before and after congenital heart surgery
- Preoperative assessment of the aortic valve and subaortic region.

TOE examples of common congenital heart lesion and procedures will be demonstrated.

Although TOE protocols may be useful to avoid missing important information, the TOE operator needs to be in constant dialogue with the surgeon or catheter interventionalist to maximise information transfer and to be guided by the individual requirements of the surgeon, interventionalist and patient.

Guy's and St Thomas' NHS
NHS Foundation Trust

Assessment of prosthetic valves

D. Beldekos

Transesophageal echocardiography (TEE) is very useful in the evaluation of the structure, function, and integrity of prosthetic valves. Because of the proximity of the esophagus to the heart and absence of interference with lungs and ribs, a detailed image of the atrial side of a mitral prosthesis and the posterior part of an aortic prosthesis can be obtained with TEE, but acoustic shadowing from prosthetic material often obscures the anterior part of the aortic prosthetic valve. The most common indications for TEE in the setting of prosthetic valve assessment are suspected dysfunction of a prosthesis (obstruction or regurgitation) and fever in the patient with prosthetic intracardiac material.

Prosthetic valves can be classified as mechanical or bioprosthetic depending on the predominant material of composition. Mechanical valves consist of a sewing ring and an occluding device. The type of the occluding device is used to further sub-classify the prostheses into one of three major categories: ball-in-cage, single-tilting disk, and bileaflet valves. Tissue valves are comprised of a mix of synthetic and natural materials (stented bioprostheses), whereas others contain no synthetic structural elements (stentless bioprostheses). Transesophageal echocardiography usually recognizes mechanical and stented tissue valves, but distinguishing stentless bioprostheses from native valves can prove challenging at times.

Once the type of the prosthetic valve is identified, the echocardiographer must answer the following questions: whether the valve is “well seated”; whether the occluders move normally; what are the features of antegrade and retrograde flow; and whether there are any masses attached to the valve. All this important information can be obtained with TEE. In mechanical valves, an impaired disk excursion or a stuck leaflet can be visualized. Prosthetic valve dehiscence is characterized by a rocking motion of the entire prosthesis. An annular abscess may be recognized as an echo-lucent, irregularly shaped area, adjacent to the sewing ring of the prosthesis. For bioprostheses, evidence of leaflet degeneration can be recognized as leaflet thickening or calcification. Abnormal echoes that may be found in patients with prosthetic valves are vegetations, thrombus, pannus, strands, sutures, spontaneous echo contrast, and microbubbles. In most cases, vegetations**,** thrombi and pannus cannot be distinguished by their echocardiographic characteristics alone. The differential diagnosis between these masses mainly depends on the full clinical picture: they may be interpreted as vegetations in a febrile patient and as thrombi in a patient with inadequate anticoagulation. Strands are thin, filamentous structures that are several millimeters long and move independently from the prosthesis. They are often visible intermittently during the cardiac cycle and they are usually located at the inflow side of the prosthetic valve. Sutures are linear, usually immobile echoes, seen at the periphery of the sewing ring of the prosthetic valve. Spontaneous echo contrast is observed in cases of mitral prosthesis obstruction, whereas microbubbles are characterized by a discontinuous stream of rounded, fast-moving echoes.

After the evaluation of the 2D and M-mode characteristics of a prosthetic valve, the assessment of flow characteristics must follow. All normally functioning mechanical valves cause some obstruction to forward blood flow, as well as closure backflow (necessary to close the valve) and leakage backflow (after valve closure, intended by the manufacturer to prevent thrombosis). The extent of “normal” obstruction and leakage of prosthetic valves depends on valve design and for a proper interpretation of Doppler data in the individual patient, one needs to know prosthesis size, heart rate, and Body Surface Area (BSA). For all Doppler measurements, it is important to align the ultrasound beam as parallel as possible to the transprosthetic flow. Transprosthetic gradient is easy to determine with TEE across a mitral prosthetic valve, but more difficult in the aortic position, where one should use transgastric views. A high transprosthetic gradient may be caused by high stroke volume (slow heart rate or paravalvular regurgitation), patient-prosthesis mismatch, or obstruction by thrombus, pannus, or vegetation. Sometimes it is difficult to determine pathologic obstruction in an aortic prosthetic valve, because gradients differ widely among patients, even with the same valve type and size. In any case, care must be taken not to misdiagnose a dynamic gradient

because of systolic obliteration of the left ventricle as a high gradient in an aortic prosthetic valve. Typical features of such a signal can help in some cases. Pathologic obstruction in an aortic prosthesis must be suspected if there is an increase of the mean gradient by at least 20 mmHg between two Doppler examinations in the same patient. In a mitral prosthesis, a mean gradient of 10 mmHg or more may signify pathologic obstruction (if the heart rate is 70-100 beats /min). A symptomatic patient with a prosthetic valve in the mitral position has always a pathologic obstruction when the mean gradient is >16 mmHg and Pressure Half-Time (PHT) is >160 msec. When the mean gradient is 10-16 mmHg and PHT >160 mmHg, one must consider surgery, but when PHT is <130 msec, a mitral regurgitation is possible and treatment depends on mitral regurgitation severity. Obviously, if one uses TEE, valve leakage from a mitral prosthesis is visualized much easier than one from an aortic prosthesis, because of the position of the mitral prosthesis in relation to the esophagus. Pathologic regurgitation can be distinguished from normal backflow by the color-Doppler appearance of regurgitant jets. Normal closure and leakage backflow jets are specific for each valve type and generally appear as low-velocity, non-aliasing jets with a homogeneous color. In contrast, pathologic jets are more turbulent and extensive; they are often eccentric and adherent to the wall of the left atrium. Pathologic regurgitation in mechanical valves may be caused by valve dehiscence (eccentric jet) or by interference of structures such as thrombus or vegetation with disc closure (central jets). Pathologic regurgitation is categorized as paravalvular and valvular. Anatomic landmarks for localization of a paravalvular leak in the mitral position and for communication with the surgeon are the aorta and the left atrial appendage.

As regards to the Doppler assessment of pathologic leakage of prosthetic valves, the severity of regurgitation in the aortic position is difficult to determine by TEE. A jet width / LVOT diameter ratio of > 40% and a vena contracta diameter of over 0.6 cm are signs of severe aortic regurgitation. In cases of suspected prosthetic mitral valve regurgitation, a transthoracic study (TTE) must always be the first step. It is well known that when regurgitation is moderate, a great volume of blood passes through the mitral prosthesis. It has been shown that 3 parameters can exclude moderate or severe regurgitation:

- **a)** Maximum forward velocity > 1.9 m/sec (sensitivity 90%, specificity 89%);
- **b)** Mean gradient > 5 mmHg (sensitivity 90%, but specificity 70%);
- **c)** Ratio of velocity-time integral (VTI) of mitral inflow to VTI at LVOT > 2.5 (sensitivity 90%, specificity 90%).

When combined with a PHT appropriate to a non-stenotic prosthesis (<130 msec), these parameters effectively screen out patients with only minimal amounts of mitral insufficiency. When no clinical suspicion of regurgitation exists, TTE is enough. However, if these parameters are pathological or clinical suspicion of mitral prosthetic regurgitation exists, then TEE is strongly recommended, due to the fact that although these indexes help to overcome issues of acoustic shadowing, they lack the ability to distinguish valvular from paravalvular regurgitant flow. Therefore, TEE is still necessary to determine the type, location, severity and anatomic correlates of prosthetic valve regurgitation. Recent advances in three-dimensional TEE have allowed better localization of the exact origin of paravalvular leaks. In terms of regurgitation severity assessment, vena contracta and proximal flow convergence area calculation have been found to be superior to regurgitant jet area and systolic flow reversal in the pulmonary veins.

Prosthetic valve thrombosis (PVT) is a rare but serious complication with an incidence of 0.3-1.3% pt-years for obstructive thrombosis, whereas non-obstructive thrombosis reaches an incidence of 10% and can cause peripheral embolism. Right-sided prostheses are obstructed by thrombus approximately 20 times more frequently than left-sided ones because of low flows and pressures in the right heart. Mitral PVT is 2-3 times more often than in the aortic position. The main risk factor for PVT is poor anticoagulation due to warfarin discontinuation prior to non-cardiac surgery or pregnancy. Although PVT can present acutely with a fresh thrombus, it is most often a subacute or chronic phenomenon. Recent surgical studies showed the high prevalence of fibrous pannus formation, which is also associated with a risk of thrombosis. Pannus has a long history of symptoms and is found mostly in patients with adequate anticoagulation. It can be located on both

sides of the prosthesis, mainly on the inflow side (when observed on mitral prostheses, it most often occurs on their atrial side), and restricts valve opening but not leaflet closure. Pannus formation is more frequent on aortic than on mitral prostheses. It appears as a very dense, immobile echo, which is usually observed in proximity to the suture site of the prosthesis. Thrombus has usually a short history of increasing symptoms in a patient with inadequate anticoagulation (INR < 2.5). It seems like a mobile mass, usually attached to the occluder of the prosthesis. Small thrombi have to be differentiated from strands or sutures. An increased mean gradient (> 50mmHg in aortic and >10mmHg in mitral prostheses) is a predictor of thrombus. The presence of thrombus and immobility or reduced leaflet mobility are direct signs of PVT. Indirect TEE signs of PVT are the disappearance of the physiological prosthesis regurgitant flow, the presence of central prosthesis regurgitation and pronounced SEC in the left atrium.

Transesophageal echocardiography has an important role in guiding therapeutic strategy in cases with PVT. For left-sided obstructive PVT, surgery is better in the presence of a large thrombus, as fibrinolysis carries a significant embolic risk. In cases of non-obstructive PVT, the preferred treatment is usually medical therapy, unless the thrombus is large or highly mobile. Finally, TEE has a much greater sensitivity than TTE for the diagnosis of bioprosthesis thrombosis, which is rare when compared to mechanical prostheses.

Another very important issue is the prosthesis - patient mismatch (PPM) phenomenon. Implantation of a prosthetic valve that is too small for the patient's BSA can be prevented by measurement of the LVOT diameter in a 120° long-axis view. If this diameter is less than 23 mm, a stentless valve has to be used. Valve prosthesis – patient mismatch is more commonly seen in aortic than in mitral prostheses.

Finally, prosthetic valve endocarditis (PVE) represents approximately 25% of total endocarditis cases. Mechanical valves are affected equally with bioprosthetic valves. Echocardiographic signs of PVE are vegetation, abscess, valve dehiscence and ruptured abscess or fistula. Transesophageal echocardiography plays an important role in the timing of surgical intervention in PVE, on the basis of the following findings: a) abscess or perivalvular extension, b) large vegetations (≥ 1 cm) with high embolic risk (mobile, anterior mitral leaflet) or (recurrent) embolization during antibiotic treatment, c) paravalvular regurgitation and d) prosthetic valve dehiscence with rocking motion of the prosthetic valve.

References

1. Douglas P, Khandheria B, Stainback R, Weissman N. ACCF/ASE/ACEP/ASNC/SCAI/SCCT/SCMR 2007 appropriateness criteria for transthoracic and transesophageal echocardiography. J Am Coll Cardiol 2007;50(2):187-204.
2. Bach D. Echocardiographic assessment of stentless aortic bioprosthetic valves. J Am Soc Echocardiogr 2000;13:941-8
3. Van den Brink R. Evaluation of prosthetic heart valves by transesophageal echocardiography: problems, pitfalls, and timing of echocardiography. Semin Cardiothorac Vasc Anesth 2006;10:89-100
4. Pibarot P, Dumesnil JG. Prosthesis-patient mismatch: definition, clinical impact, and prevention. Heart 2006;92:1022-9
5. Hill E, Herregods M, Vanderschueren S, Claus P, Peetermans W, Herijgers P. Management of prosthetic valve infective endocarditis. Am J Cardiol 2008;101:1174-8
6. Roudaut R, Serri K, Lafitte S. Thrombosis of prosthetic heart valves: diagnosis and therapeutic considerations. Heart 2007;93:137–42
7. Rahko PS. Assessing prosthetic mitral valve regurgitation by transoesophageal echo/Doppler. Heart 2004;90;476-478
8. Vitarelli A, Conde Y, Cimino E, Leone T, D'Angeli I, D'Orazio S, Stellato S. Assessment of severity of mechanical prosthetic mitral regurgitation by transoesophageal echocardiography. Heart 2004;90:539–544.

Intraoperative Assessment of the Aortic Pathology

F.L. Lorini
Ospedali Riuniti di Bergamo, Bergamo -Italy

I. Introduction

This talk will focus on the pathologic conditions that affect the aorta. Acute dissection of the ascending aorta is a true medical emergency, often necessitating immediate surgical repair. The key to instituting appropriate aorta therapy in acute dissection of ascending aorta, therefore, is an accurate and rapid diagnosis and anatomical assessment of the aorta. Neurologic dysfunction after cardiopulmonary bypass remains a major source of the morbidity and mortality associated with cardiac surgical procedures. Since athereombolic phenomena are a predominant cause of cerebral ischemic events in this patient population, atheromatous disease of the thoracic aorta is a significant risk factor for neurologic injury after cardiopulmonary bypass. Echocardiography may help to identify the patients at risk, and may provide risk reduction strategies.

II. Aortic Dissection

Pathophysiology

Blood accumulation in the medial layer is the characteristic feature of aortic dissection.The dissection of the medial layer may either be localized or split longitudinally. The plane of dissection usually courses along the greater curvature of the ascending and arch of the aorta, while in the descending aorta it is mainly located lateral to the true lumen but may also spiral along its longitudinal axis. Most often, the dissection starts at a tear (rent) in the intimal layer that allows blood to flow between the intimal and medial/adventitial layers (Fig 1). Also, a dissection can occur without any evidence of an intimal tear. It is proposed that this type of aortic dissection is due to medial layer weakness and hemorrhage of vessels in the vasa vasorum.

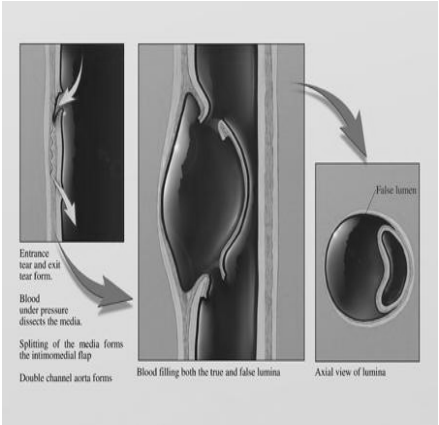


Fig 1.

B. Classification

Aortic dissections may be described by either the DeBakey or Stanford (Daily) classification systems (Fig 2). The simpler Stanford system divides dissections into two groups: type A or type B. Type A describes those dissections that involve the ascending aorta, regardless of the origin of the tear or the extent of dissection (DeBakey types I and II are both included). Type B dissections involve only the descending aorta. Many clinicians have adopted the use of the simplified Stanford classification because it delineates two distinct risk groups and therapeutic approaches. Stanford type A accounts for 50-85% of cases of aortic dissection and is associated with a mortality of 90-95% without surgical intervention. The acute mortality rate of a Stanford type B dissection is about 40% and, accordingly, the therapeutic approach is more conservative.

C . Diagnostic Modalities

A computerized tomography (CT) scan, especially with contrast image enhancement, can aid in determining the extent of the dissection and in detecting the true and false lumina. It can also detect aortic wall thickness and calcium deposits within the aortic wall. The CT scan can also visualize the pericardial and pleural spaces to reveal if there are collections, suggestive of leakage of blood, compression of major structures. The test is relatively rapid and noninvasive. However, there are the associated risks of contrast dye reactions and dye-iduced renal insufficiency. Another significant problem of this diagnostic modality is that it lacks the temporal resolution to identify the site of intimal tear or delineate branch involvement in a reliable fashion. It is also impractical to perform CT scanning on critically ill patients who are hemodynamically unstable.

Magnetic resonance imaging (MRI) produces unrestricted high resolution views of the aorta in the transverse, sagittal and coronal planes. Because of its higher quality images, MRI provides better delineations of the origin and extent of the aortic dissection. Like the CT scan, MRI can obtain images of structures surrounding the aorta that may be acutely affected by the dissection process.

Also, cine MRI has the capability to detect aortic insufficiency. MRI has overcome some of the drawbacks of CT scanning in that it is minimally invasive and does not require contrast dye. The major limitations are that it is time consuming and the facilities are not always available or on-site. For patients who are hemodynamically unstable and/or in respiratory failure, MRI may prove very difficult. These patients need to be transported with appropriate monitoring. During the study, only limited access is afforded to the patient for examination because they are required to lie within a small housing and for an extended period of time. TEE has overcome some of the major disadvantages of the CT and MRI. It is a minimally invasive procedure that has a proven safety record.[1] An examination can be performed within about fifteen to twenty minutes and a diagnosis can usually be obtained at the same time. The test is easily performed at the bedside in critically ill patients. The close anatomic relationship of the esophagus to the aorta and the heart allows TEE to provide excellent high quality images without significant inteference from the overlyng structures (lungs and chest wall). With the introduction of biplane and multiplane TEE probes, a more complete definition of the distal ascending aorta and aortic arch are possible. TEE is performed in real time allowing for its unique ability to give functional and hemodynamic information.

D. Secondary Diagnoses

In addition to revealing the presence and extent of an aortic dissection, special features of the TEE can be used to diagnose and define several important aspects of the dissection. TEE is one of the best methods for accurately identifying the structural and functional status (using DCFI and CW Doppler) of the aortic valve, which has important surgical implications. TEE is also valuable for assessing the degree of involvement and integrity of the coronary arteries in aortic dissection. TEE visualized approximately 70-88% and 25-50% of the left and right coronary artery ostia, respectively. Of the seven patients with proven involvement of the coronary ostium in the study by Ballal et al, six were correctly diagnosed by TEE.9 There are situations, however, where coronary angiography is required to define the need for CABG or coronary angioplasty (e.g., ascending aortic dissection with an acute MI). Aortography should obviously be performed if cardiac catheterization is planned. Flow patterns of the true and false lumina and the location of intimal tears can be further assessed using DCFI and PW Doppler. This can lead to identification of patients at risk for malperfusion. The diagnosis of a left pleural or pericardial effusion or even blood clots in the pericardium can be obtained more rapidly with TEE compared to CT or MRI. TEE can also give real time analysis of cardiac function, which is very critical for medical, surgical and anesthetic management. Rare complications of aortic dissections have also been reported that were diagnosed by TEE and was missed by other modalities. An example is aortic intussusception, where the intimal flap partially or totally separates from the aorta and migrates distally causing obstruction of blood flow to extremities or major organs.

III. Aortic Atherosclerosis

A. Clinical Significance

Prior investigations that focused on cerebral hypoperfusion, nonpulsatility, and air embolization as the major etiologic factors for stroke have been supplanted by studies of atheroemboli, which are now believed to be responsible for a majority of cerebral embolic events during open or closed cardiac procedures.[2] Valvular surgery and open cardiac procedures were once thought to be higher risk procedures for micro and macro-embolization because of the entrainment of air into cardiac chambers and the potential for embolization of calcific and thrombotic debris.[3] Atherosclerosis of the thoracic aorta is now recognized as one, if not the major predictor of postoperative stroke after cardiac surgery.4 As the median age of our population increases, so too does the prevalence of aortic atherosclerosis.

Despite a progressive decline in perioperative mortality, as a result of improved myocardial preservation, surgical, and perfusion techniques, the overall incidence of perioperative stroke remains unchanged at 1-2% and increases with each decile of age. The incidence of early neuropsychological dysfunction may be as high as 80% immediately after surgery and progressively declines to approximately 40% by the time of hospital discharge.5 As the median age of our cardiac surgical population continues to increase, the incidence of age-related adverse events will also increase, unless appropriate preventive or therapeutic measures can be instituted.

When aortic instrumentation is planned, the presence of ascending, transverse, and descending aortic atheromatous disease is a harbinger of potential cerebral embolization. Using echocardiographic techniques aortic atherosclerosis can and should be diagnosed prior to anticipated instrumentation. Ascending aortic plaque is often soft, friable and not able to be palpated by the cardiac surgeon. A number of investigations have shown that palpation underestimates the incidence of aortic plaque when compared with diagnosis via echocardiographic techniques.6 Its presence my only be recognized after it is seen oozing from an aortotomy site. After aortotomy, the embolization of atherosclerotic debris my have already occurred with resultant adverse sequelae. Thus, the preoperative identification of aortic plaque, made possible by improved echocardiographic technologies, has become an area of intense interest and research.

TEE evidence of thoracic aortic atherosclerosis has been shown to be a good predictor of the presence of ascending aortic atherosclerosis as seen by epicardial echocardiography,7 and my thus be a useful screen for patients at risk for cerebral embolization. Further, a negative TEE exam for atherosclerosis is helpful to exclude the possibility of ascending atherosclerosis by epicardial examination.

References

1. Daniel WG, Erbel R, Kasper W, et al : Safety of transesophageal echocardiography: A multicenter survey of 10, 419 examinations. Circulation 83: 817-821, 1991.
2. Mills SA: Risk factors for cerebral injury and cardiac surgery. Ann Thorac Surg 59: 1296-1299, 1995.
3. Nussmeier NA: Adverse neurologic events: risks of intracardiac versus extracardiac surgery. J Cardiothorac Vasc Anesth 10: 31-37, 1996.
4. Bull DA, Neumayer LA, Hunter GC, et al: Risk factors for stroke in patients undergoing coronary artery bypass grafting. Cardiovasc Surg 1: 182-185, 1993.
5. Mora CT, Henson MB, Weintraub WS, et al: The effect of temperature management during cardiopulmonary bypass on neurological and neuropsychological outcomes in coronary revascularization patients. J Thorac Cardiovasc Surg 112: 514-522, 1996.
6. Marschall K, Kanchuger M, Kessler K, et al: Superiority of transesophageal echocardiography in detecting aortic arch atheromatous disease: identification of patients at increased risk of stroke during cardiac surgery. J Cardiothorac Vasc Anesth 8: 5-13, 1994.
7. Konstadt SN, Reich DL, Kahn R, Viggiani RF: Transesophageal echocardiography can be used to screen for ascending aortic atherosclerosis. Anesth Analg 81: 225-8, 1995.

Cardiac masses, tumours and pericardial disease

Bogdan A. Popescu PhD, FESC

"Carol Davila" University of Medicine and Pharmacy
Bucharest, Romania

EACTA / EAE TOE Accreditation Course, Rome, Sep 2008

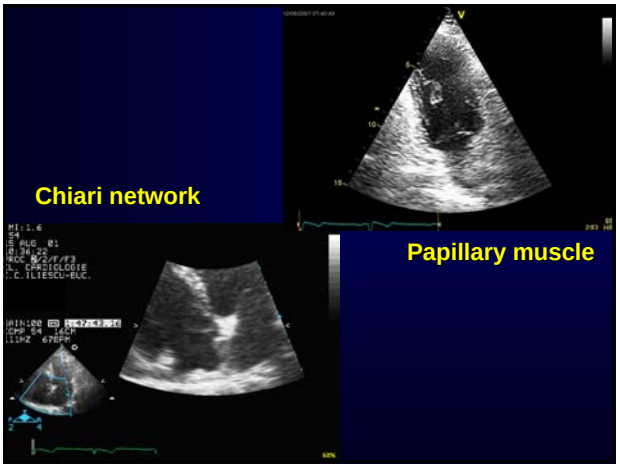
CARDIAC MASSES

NORMAL

- Large Eustachian valve
- Crista terminalis
- Chiari network
- Moderator band (RV)
- False tendons (LV)
- Papilary muscles

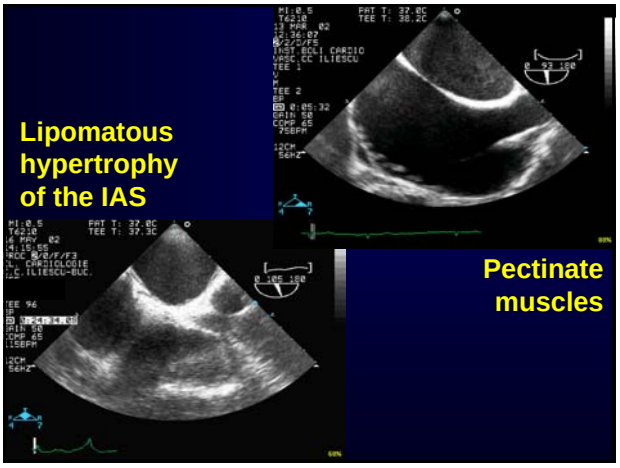
ABNORMAL

- Primary tumor
- Metastatic tumor
- Thrombus
- Vegetations
- Iatrogenic material



Other structures misinterpreted as cardiac masses

- Lipomatous atrial septal hypertrophy
- Pectinate muscles (eg in the LAA, RA)
- Mitral valve annulus calcification
- Hiatal hernia
- Transverse sinus with fibrin material in the presence of pericardial effusion (TOE)



Abnormal masses

(1) Tumors

- Primary: rare (0.01-3/1000 autopsies)
- Metastatic: 30 - 40 x more frequent

Primary cardiac tumors in adults

BENIGN	
Myxoma	27%
Lipoma	10%
Papillary fibroelastoma	10%
Hemangioma	3%
Mesothelioma of the AV node	1%
MALIGNANT	
Angiosarcoma	9%
Rhabdomyosarcoma	5%
Mesothelioma	4%
Fibrosarcoma	3%
Malignant lymphoma	2%
Extraskeletal osteosarcoma	1%
CYSTS	
Pericardial	18%
Bronchogenic	2%

Benign cardiac tumors

Myxoma

- The most frequent intracavitary tumor
- Most often localized in the LA (75%)
- Typically: inserted on the interatrial septum
- Diagnosis usually made by TTE
- TOE is mandatory
- Assessment: localization, number, insertion, extension, hemodynamic consequences

Myxomas – remember:

- Insertion on the interatrial septum
- Use multiple views
- TOE (exclusion of other localizations)
- Assess hemodynamic consequences

Fibroma

- benign
- hyperechogenic
- well delineated (not encapsulated)
- small ones – usually: observation
- large ones - sometimes need surgery

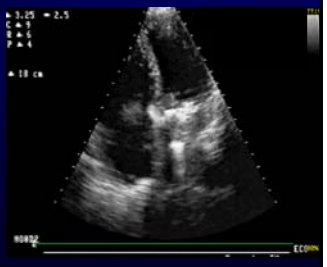
Malignant cardiac tumors

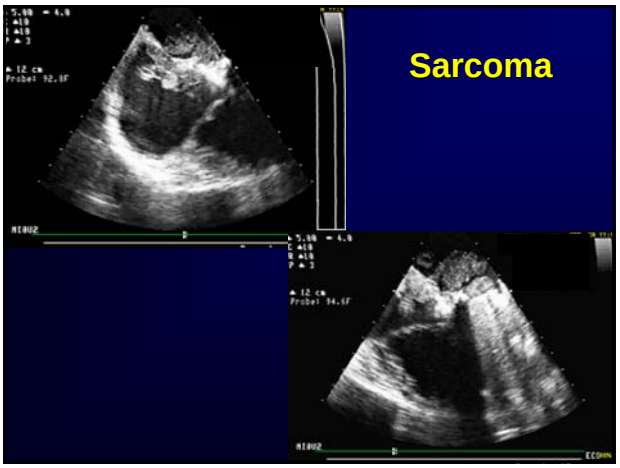
- Sarcomas – the vast majority
  - angiosarcomas
  - rhabdomyosarcoma (most common in children)
  - fibrosarcomas
- Histiocytomas
- Lymphomas
- Extracardiac tumors - by direct invasion

Sarcoma

Angiosarcoma – the most frequent

- tamponade
- atrial wall and pericardial involvement
- extremely invasive





Sarcoma

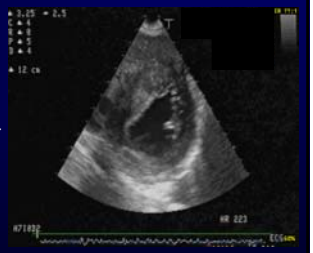
Secondary cardiac tumors

- metastatic
- extension from IVC, SVC or pulmonary veins
- adjacent to the heart: mediastinal or pericardial

Metastases

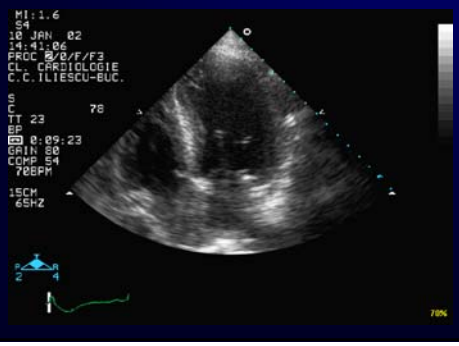
The most common malignant tumors of the heart:

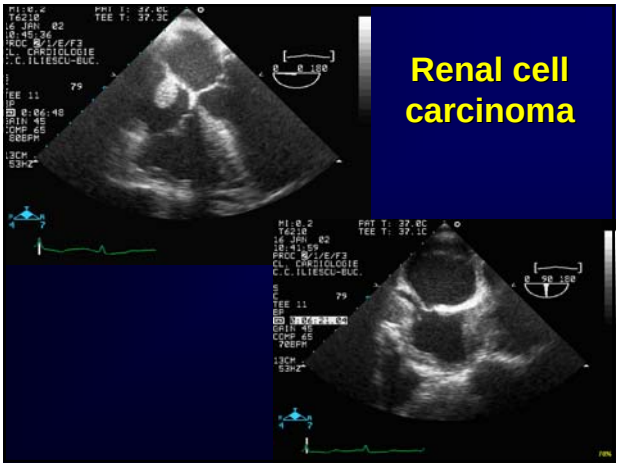
- lung
- breast
- leukaemia and lymphoma
- malignant melanoma
- renal cell carcinoma



Secondary cardiac tumors

Extension via caval veins





Renal cell carcinoma

Abnormal masses

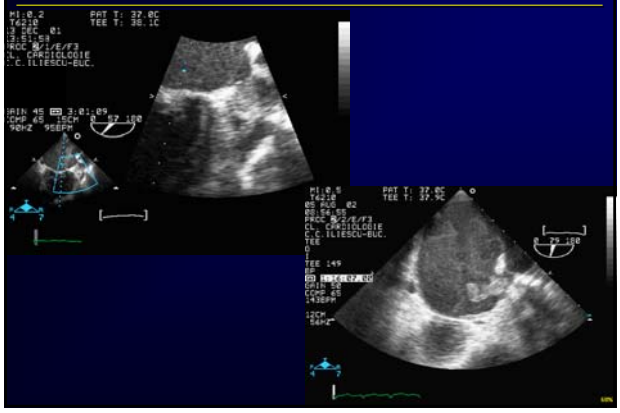
(2) Thrombi

- dislodged from peripheral veins
- formed ‘in situ’ if predisposing local conditions coexist

Thrombus appearance

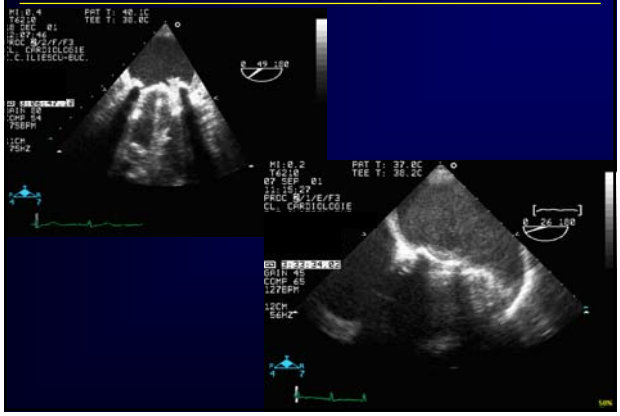
- echogenicity similar to that of the myocardium
- echogenicity less than the myocardium ("jelly-like")
- bright and polypoid in shape
- layered along the ventricular myocardium with impaired contractility (postMI, CMP) or atrial wall (AFib, MS or TS)
- long and thin (RA thrombus dislodged from veins in the extremities)

LAA and LA thrombi



Case presentations

Thrombus on MV prosthesis

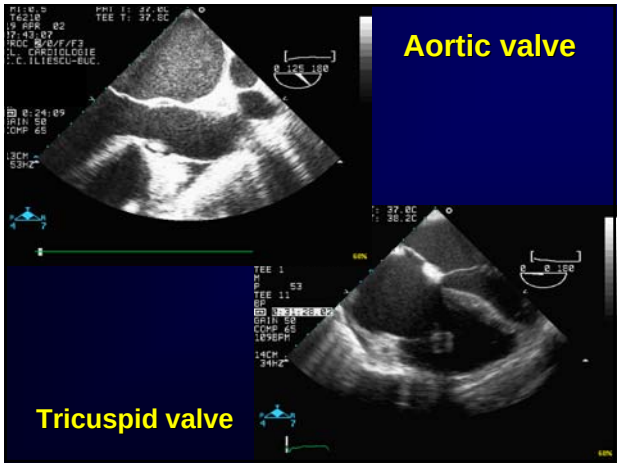


Abnormal masses

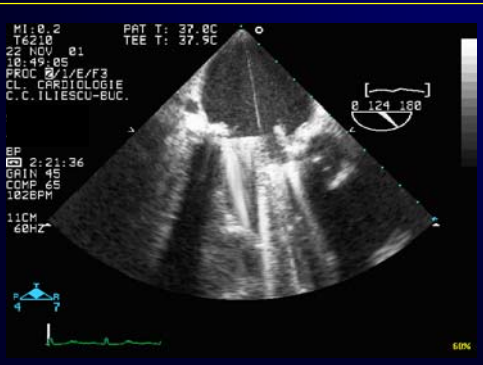
(3) Vegetations

TOE - much higher sensitivity than TTE, especially for:

- small vegetations (< 5 mm)
- MV vegetations
- prosthetic valve vegetations (MV)

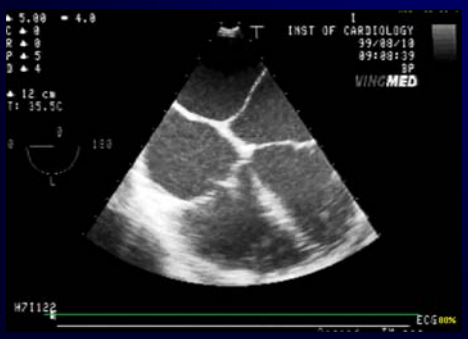


Vegetations on MV prosthesis

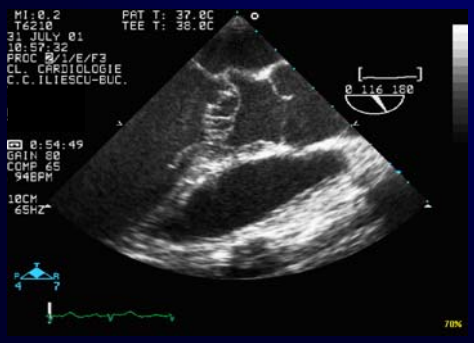


Other cardiac “masses”

Cor triatriatum sinister



Accessory mitral valve



Popescu BA, et al. Echocardiography 2005.

Pericardial diseases

TOE has limited role over TTE

- Localized pericardial effusion
- Postop / postinterventional effusion
- Pericardial cyst
- Selected cases

Echocardiography in infective endocarditis

Gilbert HABIB , MD, FESC, FACC
Hôpital Timone, Department of Cardiology, La Timone Hospital Marseille, France .

Infective endocarditis (IE) is still a severe and life-threatening disease, with high incidence of complications and persistent high mortality. However, IE is also a changing disease, including changes in incidence, underlying disease, microorganisms involved, as well as in therapeutic (medical / surgical) management. Echocardiography plays a crucial role both in the diagnosis, the prognostic assessment, and the therapeutic approach of patients with acute IE.

I – DIAGNOSIS OF ACUTE INFECTIVE ENDOCARDITIS:

Diagnosis of IE is based on a careful clinical evaluation, blood cultures and echocardiography. Echocardiography plays a key-role in the management of IE both for the diagnostic and prognostic assessment of these patients. The advent of the transesophageal approach has improved its diagnostic accuracy. In 1994, Durack proposed a new classification of criteria for IE called Duke criteria. This classification was a big step in the diagnosis of IE because it included echocardiography as a major criterion for IE. The major echographic criteria for IE are vegetation, abscess and new dehiscence of a prosthetic valve. In a series of 93 patients with proved IE, we found that Duke criteria had a 76 % sensitivity for the diagnosis of IE, as compared with only 56 % sensitivity using old von Reyn criteria. However, false negative results of this classification occurred in 24 % of patients with proved IE, emphasizing that diagnosis of IE is still difficult in some situations.
For example, echocardiographic assessment is frequently more difficult in subgroups of patients including prosthetic valve and pacemaker IE. The sensitivity of Duke criteria has been shown to be lower in these patients.

II – PROGNOSTIC ASSESSMENT

Mortality is still high in IE, although it has declined in the recent years. Reported mortality in IE is 10 to 17% in the most recent series. Thus, the identification of factors associated with increased mortality is a crucial challenge, as it will allow the identification of high-risk patients in whom an aggressive strategy will be potentially useful. Several markers have previously been identified including age, occurrence of complications, staphylococcal infection, and prosthetic valve IE. However, in a multicenter European study including 384 patients with definite IE, the size of the vegetation was also found to be the independent predictors of death.
Embolic events are a frequent and life-threatening complication of IE. They are related to the migration of cardiac valvular vegetations into the major arterial beds, including brain, lungs, spleen, and coronary arteries. In addition, cerebral embolism, the most frequent embolic complication, is associated with an increased morbidity and mortality. Thus, an accurate prediction of the embolic risk is a desirable goal. The rate of systemic embolization is estimated to be between 10% and 50%. Echocardiography plays a key role in the prediction of embolic risk, although past studies gave conflicting results. The risk of embolism has been shown to be higher in patients with large and mobile vegetations, especially when vegetations were localized on the mitral valve. Other factors probably influence the risk of embolism including location of vegetation, evolution of the vegetation size under therapy, and biological and microbiological factors.

III – THERAPEUTIC APPROACH

About 50% patients with acute IE are treated with combined medical and surgical therapy. The decision to operate early in IE is always difficult and remains specific for the individual patient. Accepted indications for surgery during acute IE include acute aortic and mitral regurgitation with congestive heart failure, evidence of perivalvular extension, persistent infection after 7-10 days of adequate antibiotic therapy and infection due to microorganisms with a poor response to antibiotic therapy. Decision to operate because of recent embolism or large vegetation is more debated.

Both TTE and TOE are very useful for decision-making in IE. Moreover, intraoperative TOE is particularly helpful in patients in whom conservative surgery of homograft surgery are used. Finally, echocardiography is a useful tool for the follow-up of patients treated either medically or by surgery

Artefacts and Illusions of Echocardiography

Dr. med. Joachim M. Erb, DEAA,
Universitätsklinik für Anästhesiologie mit Schwerpunkt op. Intensivmedizin, Campus Charité Mitte, Berlin, Germany

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.

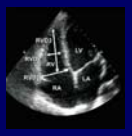
Advice:	*Use multiple imaging planes to evaluate ominous structures. Follow them from side to side, sort out their anatomical relation to other structures and their moving pattern.*

Standard transthoracic exam

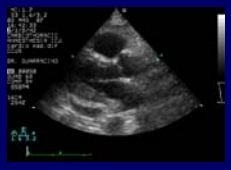
Fabio Guarracino

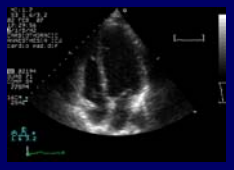
Cardiothoracic Anaesthesia and ICU
Cardiothoracic Department
University Hospital of Pisa, Italy

Ultrasonographic anatomy



Functional anatomy

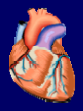




Morphology & Function

Morphology & function

- Pericardium
- Chambers: atria and ventricles
- Valve apparatus



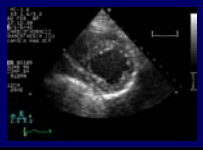
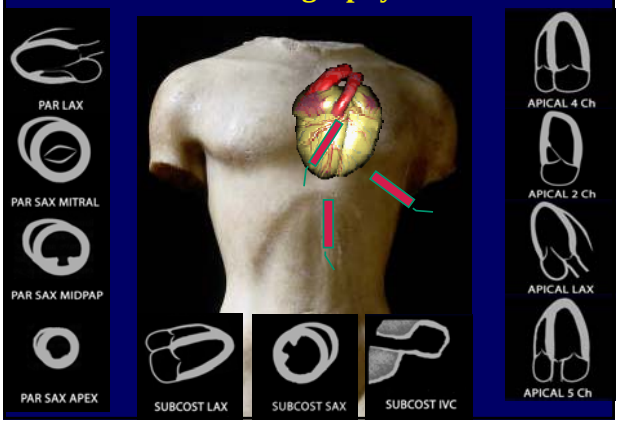


Image incorporation into clinical scenario

- Hemodynamic instability
- Dyspnoea
- Infective endocarditis
- Aortic pathology
- Source of embolization
- Unexplained hypoxemia

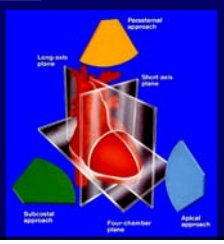
TT echocardiography in ICU

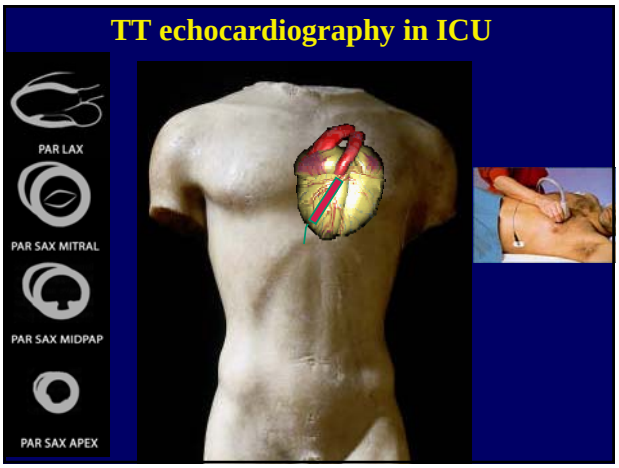


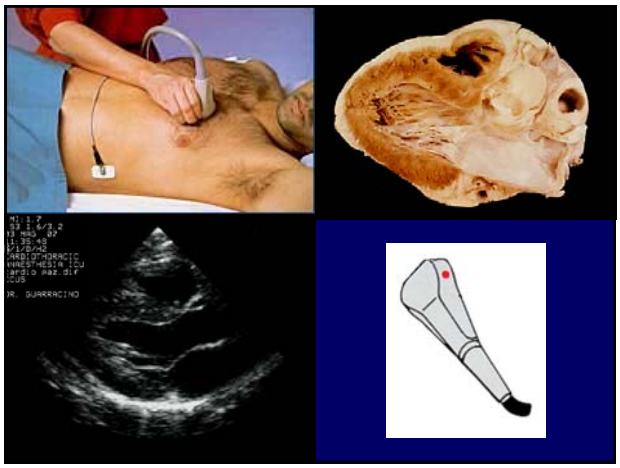
The transducer: positions and views

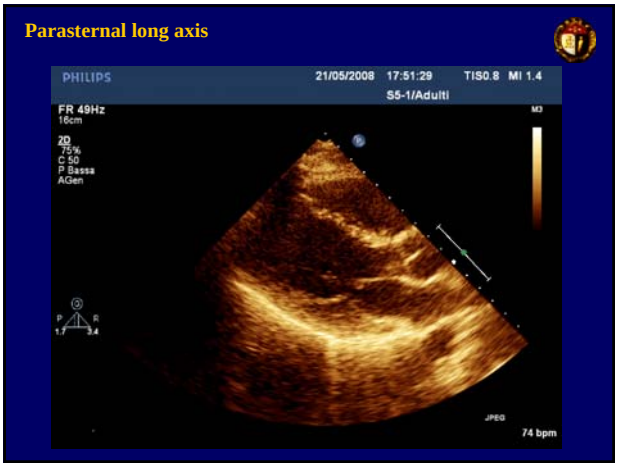
- Apical
- Parasternal
- Subcostal

- Longitudinal view
- Short axis view

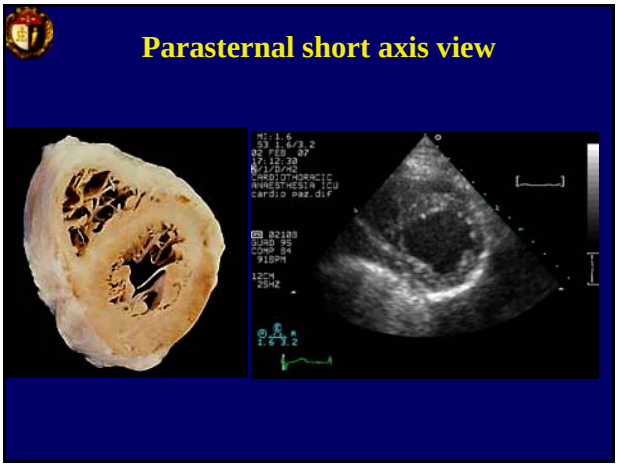


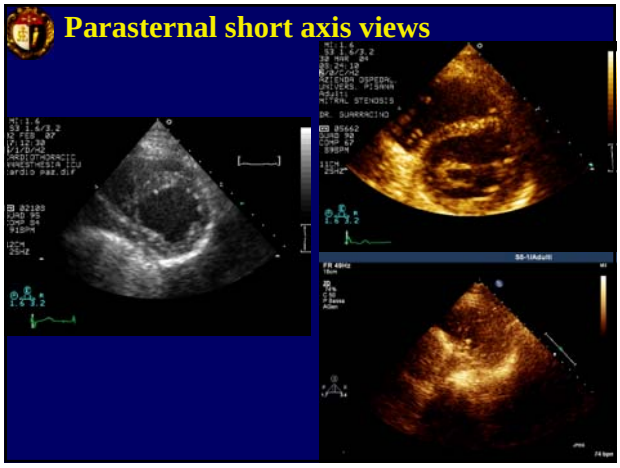

TT echocardiography in ICU
PAR LAX
PAR SAX MITRAL
PAR SAX MIDPAP
PAR SAX APEX



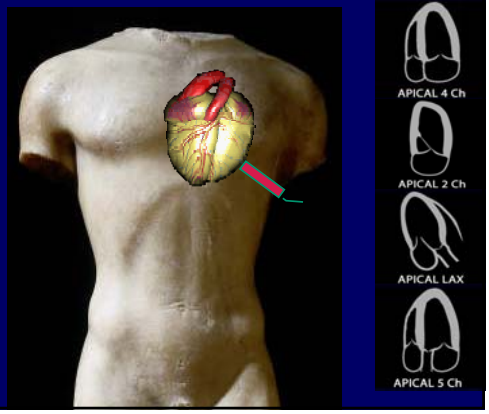

Parasternal long axis
PHILIPS
21/05/2008 17:51:29
S5-1/Adulti
FR 49Hz
74 bpm


PAR SAX MIDPAP
RV
LV


Parasternal short axis view

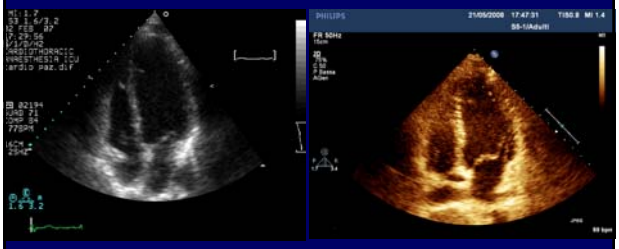

Parasternal short axis views

TT echocardiography in ICU

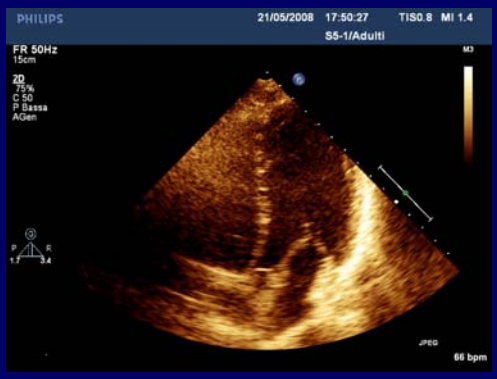


Apical four chamber view

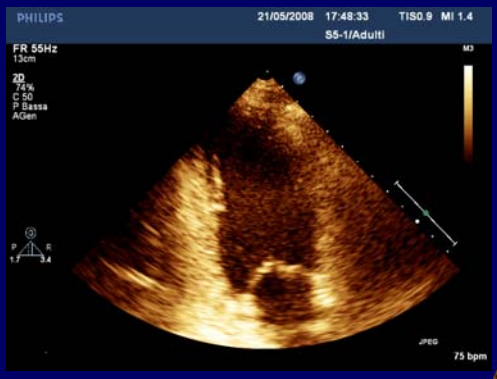




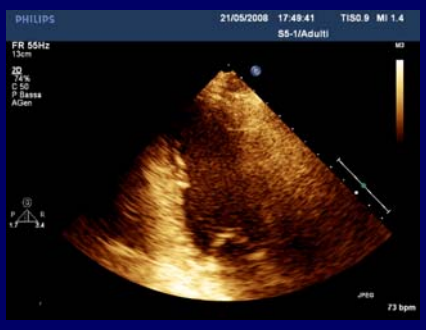
Apical five chamber view



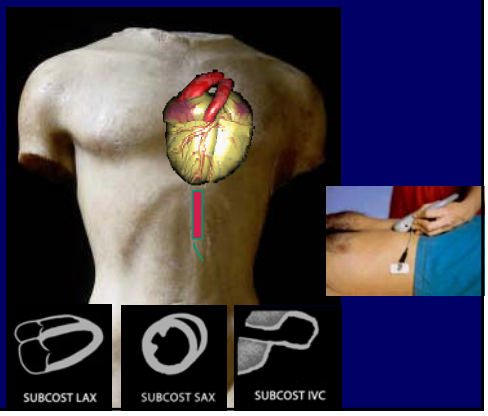
Apical two chamber view



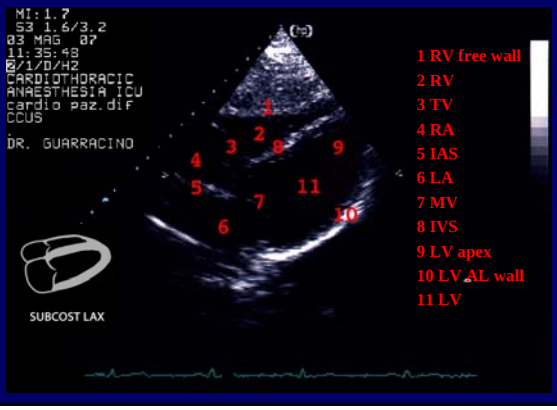
Apical three chamber view



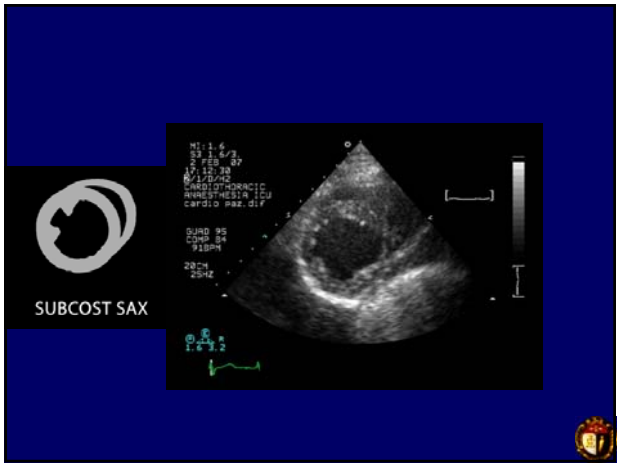
TT echocardiography in ICU



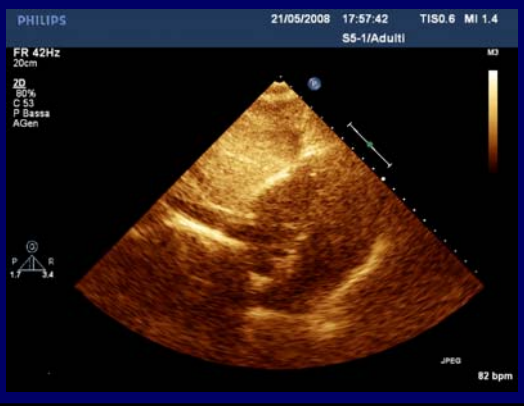
Subcostal view long axis view

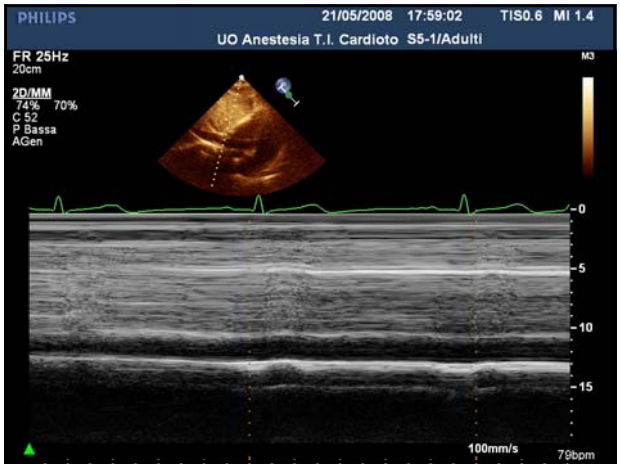


1 RV free wall
2 RV
3 TV
4 RA
5 IAS
6 LA
7 MV
8 IVS
9 LV apex
10 LV AL wall
11 LV

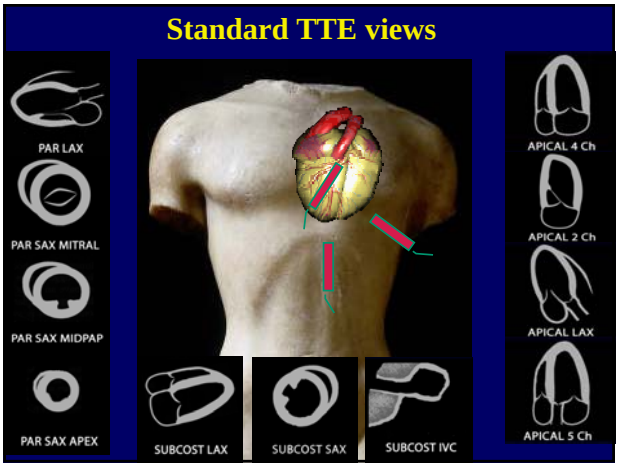


Subcostal view for IVC





Standard TTE views



TTE in the ICU – the FATE protocol

Erik Sloth
Aarhus University Hospital, Skejby, Denmark.

Transoesophageal echocardiography (TOE) is well established as a diagnostic and monitoring tool in the operating room but just like transthoracic echocardiography (TTE) it has hitherto only been sporadically used for hemodynamic optimization in the ICU. 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 (Figure 1), have put more focus on the benefit of abbreviated echo protocols (1-3). Several review articles, which are highly recommended reading, have also been published recently underlining the growing interest and emphasising the need for echocardiography in the evaluation of the critical care patient (4,5).
Echocardiography is, at present, the only method which can provide bedside real-time and dynamic imaging of the heart, pericardium, great vessels, lung and pleura. The combination of 2D imaging, M-mode and Doppler modalities give unique structural and functional information essential for choosing or adjustment of the correct therapy (4,5). There are studies indicating 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 in the critical care environment (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. “True” pocket echo machines have already been advertised and it is only a question of time until an abbreviated echo examination will be mandatory in the evaluation of the ICU patient. A paradigm shift is in process and the echo machine will turn out to be the “Doctors best friend”.

Figure1. 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 Pericardial effusion Pleural effusion/pneumothorax Valvular dysfunction Aorta dissection 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, sub costal drainage and an unfavourable 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). In a recent study carried out in patients who underwent aortic valve replacement it was proved that even sub costal drains did not affect the image quality in the post operative period (10). 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.

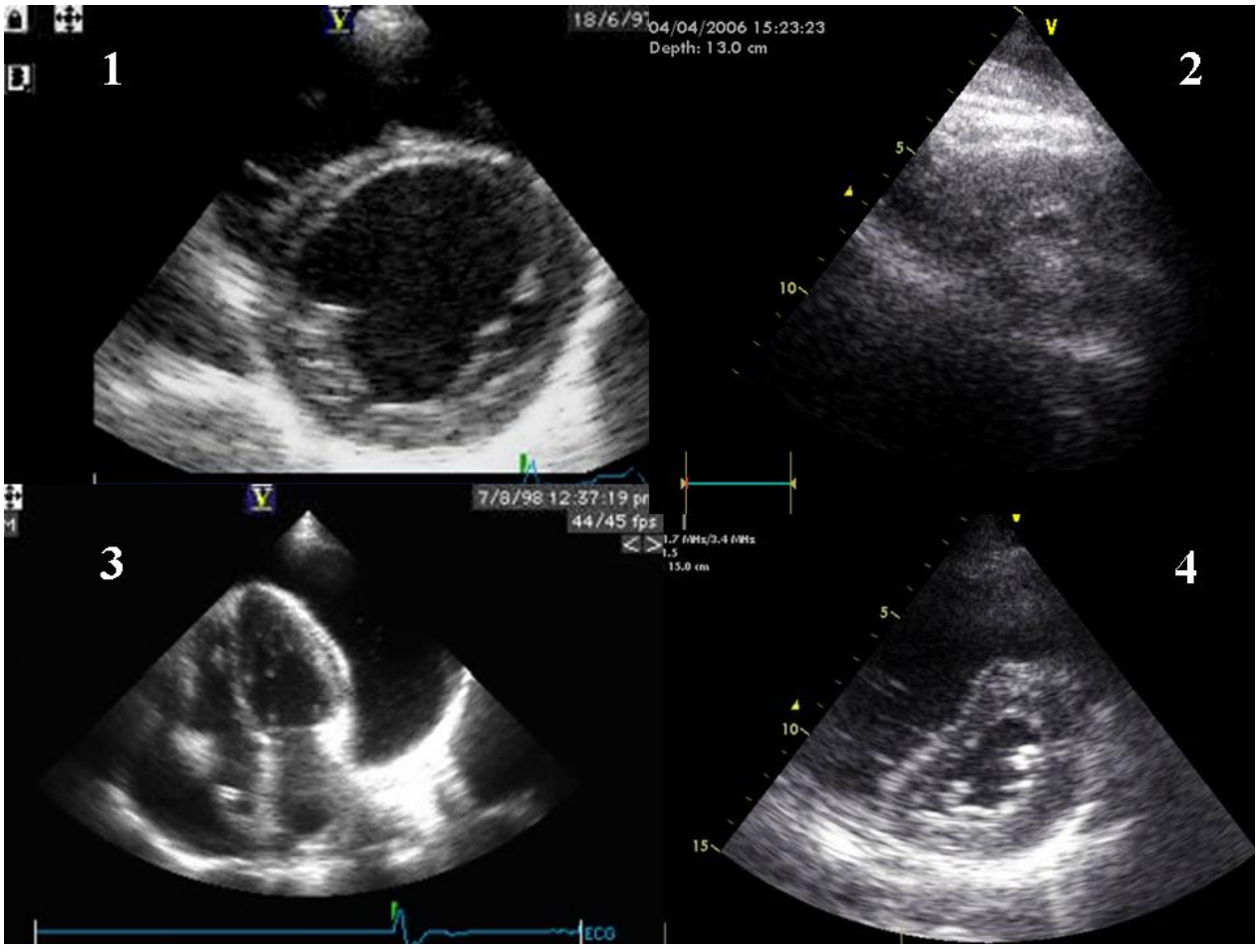


Figure 2, showing the four important causes of shock. 1) dilated and dysfunctioning left ventricle, 2) severe left ventricular hypovolemia (poor but sufficiently image quality in an emergency case), 3) pericardial effusion and 4) right ventricle pressure overload with "D"-shaped left ventricle (pulmonary embolus). These cases are extremely difficult to differentiate *clinically* but very easy to recognise by means of echocardiography.

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 and correct ventilation of the lungs after tracheal intubation can be controlled (11).

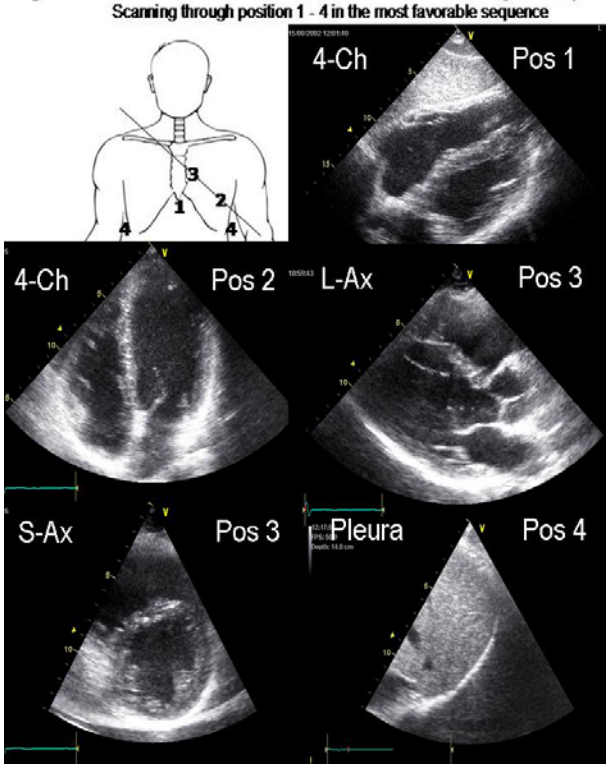
Focus assessed transthoracic echocardiography (FATE)

It is not likely that all emergency team members will learn echocardiography on "top level" - and it is not at all necessary. After almost 20 years experience with echocardiography on high level, it is my opinion, that abbreviated echo protocols which can easily be learned, will solve more than 99% of emergency cases. Therefore, 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 (1). Thus, *FATE should be considered as a supplement to the clinical evaluation- no more no less.*

FATE is a focus assessed ultrasound examination through position 1 – 4 (fig. 3) 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. 3 **Focus Assessed Transthoracic Echo (FATE)**
Scanning through position 1 - 4 in the most favorable sequence



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. Depending on the clinical situation and the quality of the images obtained during the FATE examination a full cardiologic TTE evaluation can be applied together with a diagnostic TEE. It should be emphasised that FATE, TTE and TOE are not in competition with each but

highly complementary.

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. 4).

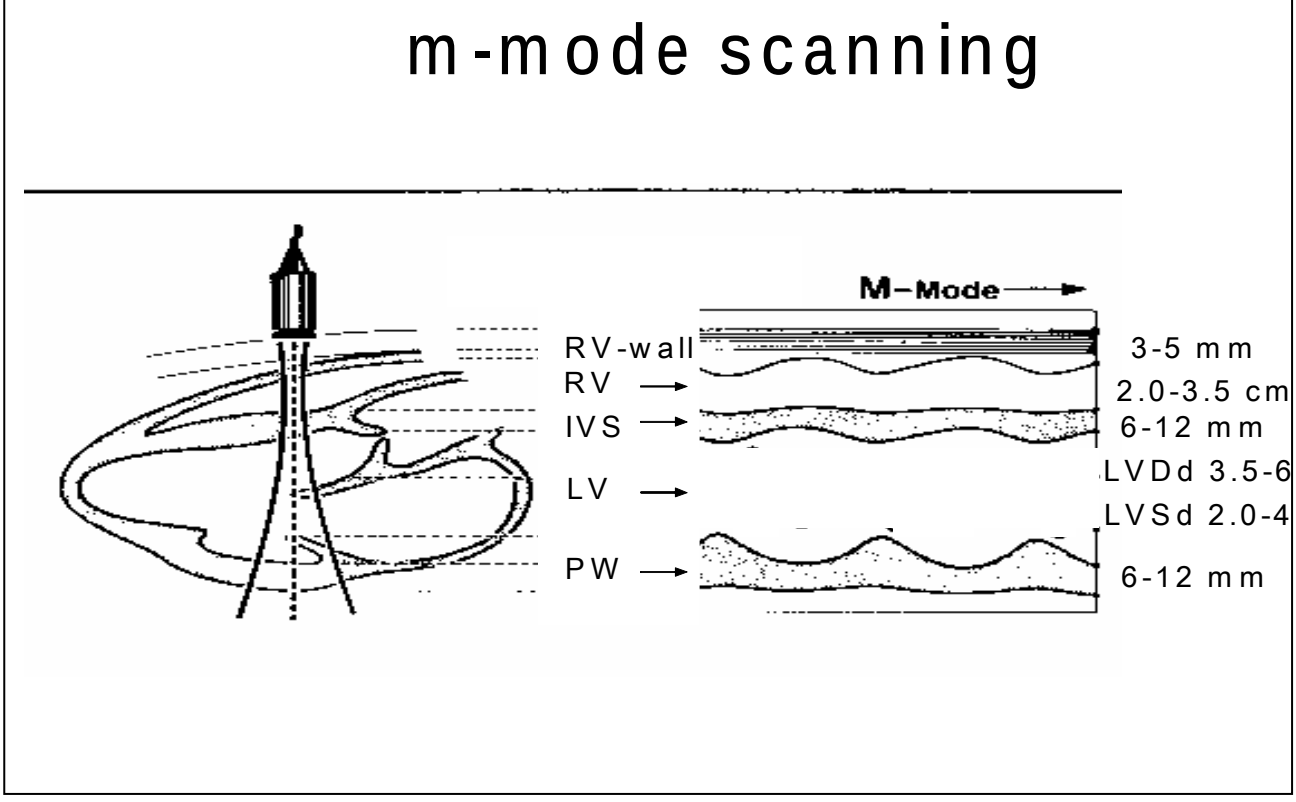
In the parasternal long axis view the RV diameter measures approximately 2.0-3.5 cm in diastole. The normal left ventricular end-diastolic diameter (LVDd) is within 3.5-6.0 cm and left ventricular end-systolic diameter (LVSd) between 2.0-4.0 cm (fig. 2). From these measures *fractional shortening* (FS) 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. 4). 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 4



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. All together this will 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 in the critical care setting instead of the being the fading patients last chance.

References

1. Jensen MB, Sloth E, Larsen K and Schmidt MB. Transthoracic echocardiography for cardiopulmonary monitoring in intensive care. EJA 2004: 21: 700-707.
2. Breitkreutz, Walcher F and Seeger FH. Focused echocardiographic evaluation in resuscitation management: Concept of an advanced life support-conformed algorithm.
3. Kimura B, Shaw DJ, Agan DL, Amundson SA, Ping AC, and DeMaria AN. Value of a cardiovascular limited ultrasound examination using a hand-carried ultrasound device on clinical management in an outpatient medical clinic. Am J Cardiol 2007; 100: 321-325.
4. Price S, Nicol E, Gibson DG, Evans TW. Echocardiography in the critically ill: current and potential roles. Intensive Care Med 2006: 32:48–59.
5. Beaulieu Y. Bedside echocardiography in assessment of the critically ill. Crit Care Med 2007; 35S: S235-S249.
6. Barbier C, Loubières and Schmit C et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responciveness in ventilated septic patients. Intensive Care Med 2004: 30: 1740-46.
7. Lyon M, Blaivas M and Brannam L. Sonographic measurement of the inferior vena cava as a marker of blood loss. American Journal of Emergemcy Medicine 2005: 23:45-50.
8. Cook CH, Praba AC, Beery PR and Martin LC. Transthoracic echocardiography is not cost-effective in critically ill surgical patients. J Trauma 2002; 52: 280-284.
9. Joseph MX and Disney PJS. Transthoracic echocardiography to identify or exclude cardiac cases of shock. Chest 2004; 126: 1992-1597.
10. Jakobsen CJ, Torp P, Sloth E. Perioperative feasibility of imaging the heart and pleura in patients with aortic stenosis undergoing aortic valve replacement. Eur J Anaesthesiol. 2007; 24(7): 589-95.
11. Lichtenstein D. Lung ultrasound in the critically ill. Clinical Intensive Care, 2005; 16 (2): 79-87.

What 3 D TOE adds ?

Dr. Andreas Ziegler
Herzzentrum Bad Krozingen Anesthesia

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. Newest technology as the intraoperative assessment of the right ventricle in 3 D technology will be shown. 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.

The clinical examples presented were acquired using an Acuson CV70 echocardiography system (Siemens, Erlangen) and analyzed 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 artifacts 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 anesthetized 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 and the right ventricle. 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".

Speckle tracking - the next generation for global and regional ventricular function assessment?

Heinz D. Tschernich

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.

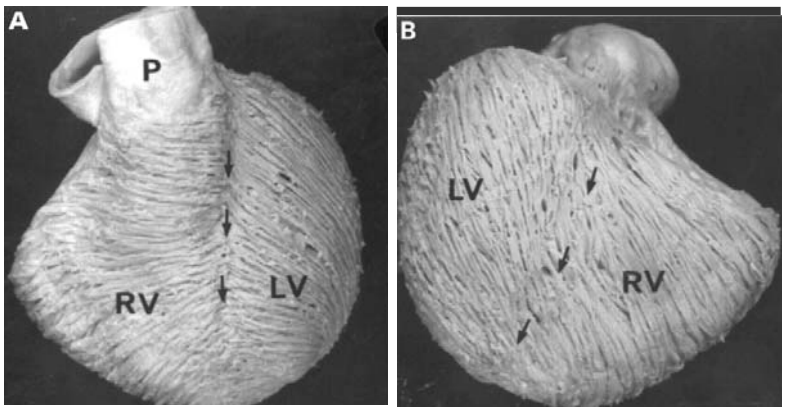
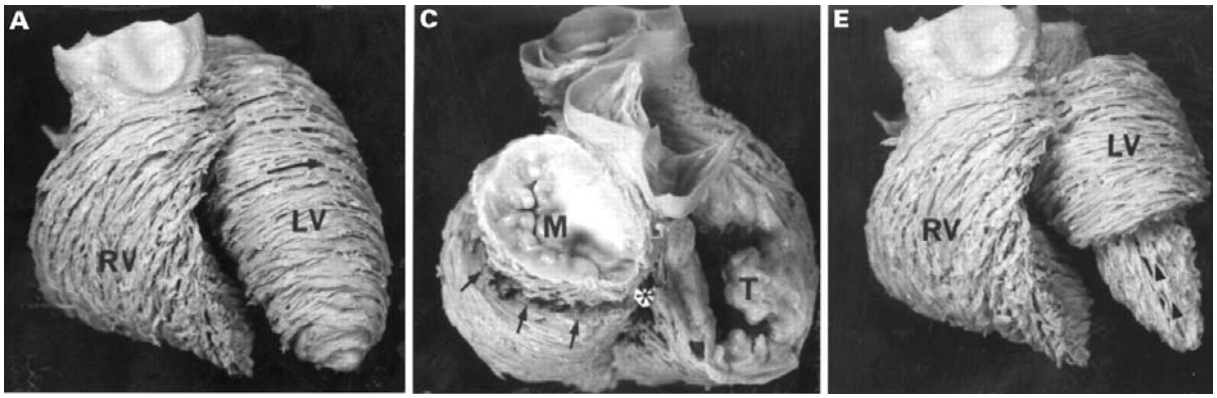


Fig. 1: From „Sanchez-Quintana – Myocardial architecture[2]“
A,B: Subepicardial layer

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



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).

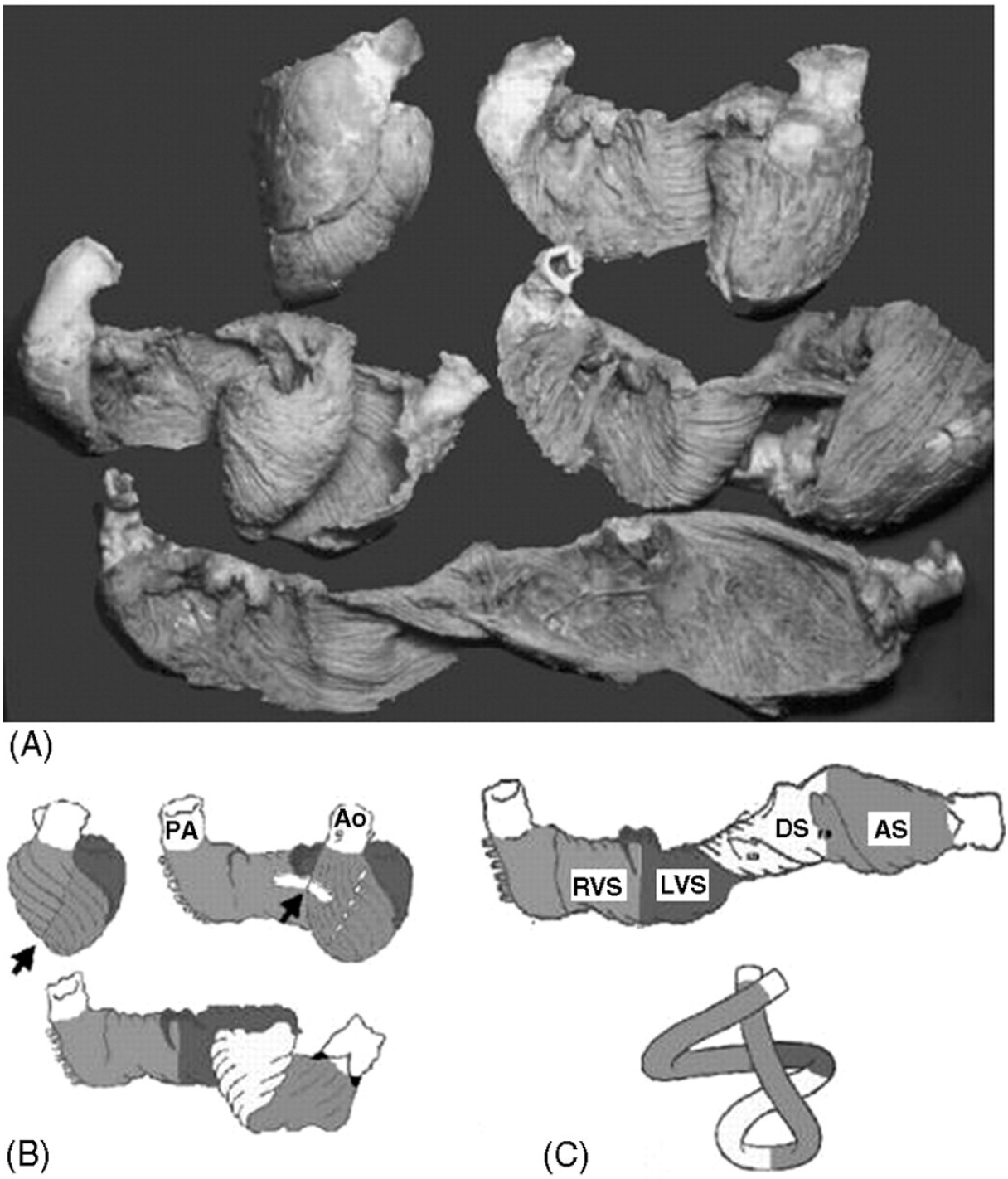


Fig. 3. Unwrapping the heart to a single muscle band from the beginning at the Pulmonary Artery (PA) to the end at the Aorta (AO)[3]

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].

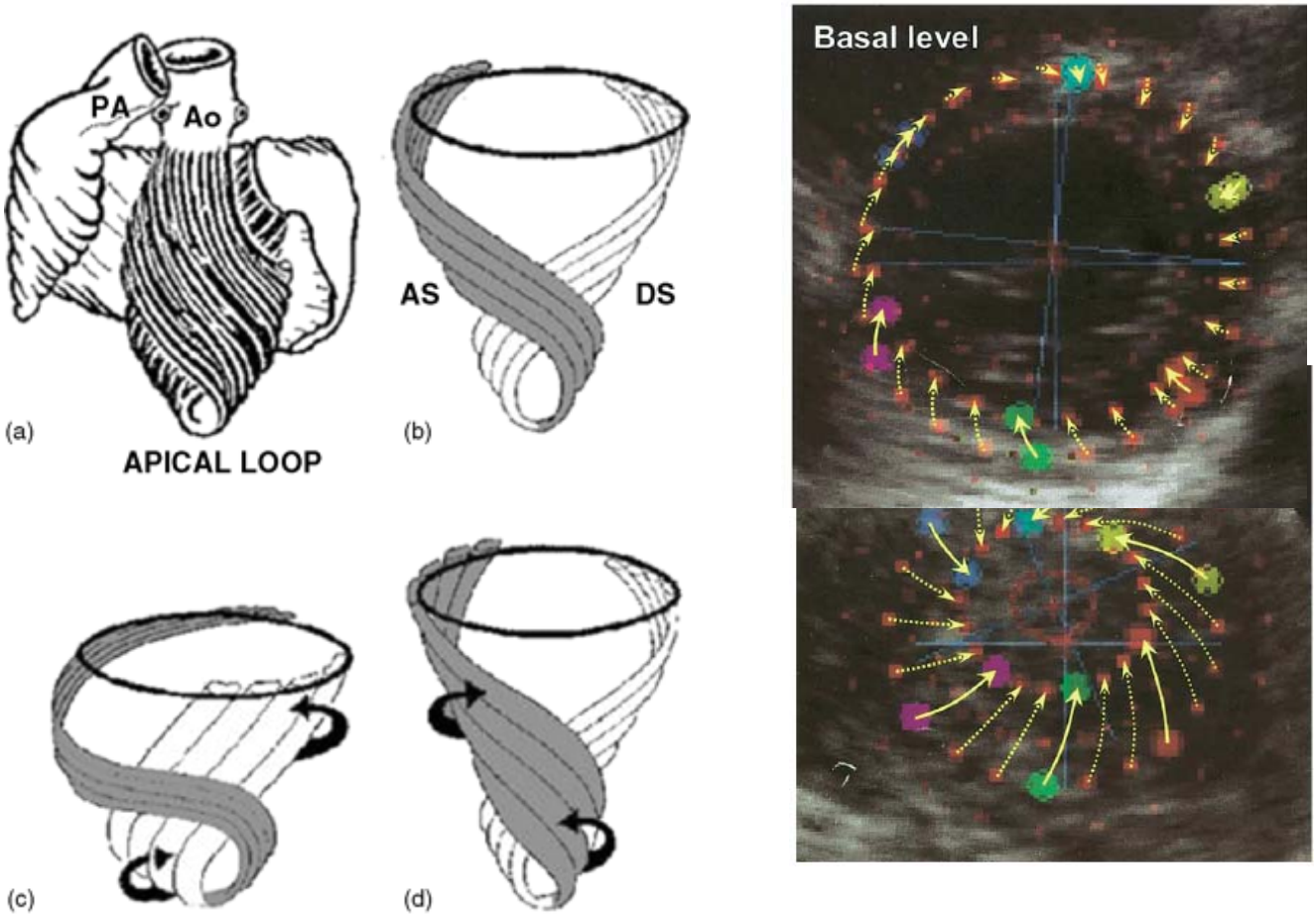
Influences of the Myocardial Architecture on Contraction Pattern

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)

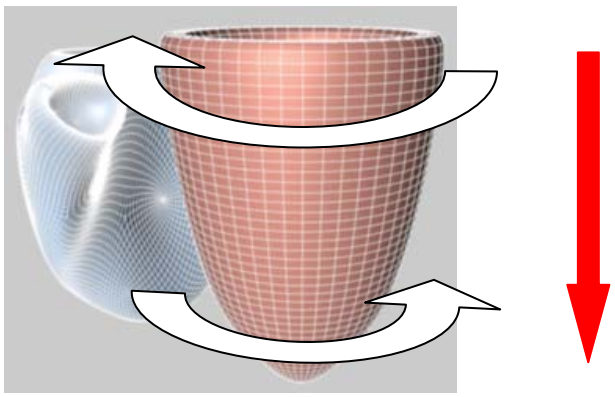
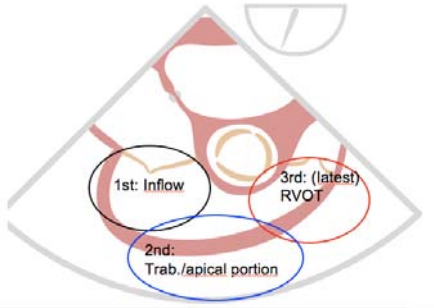
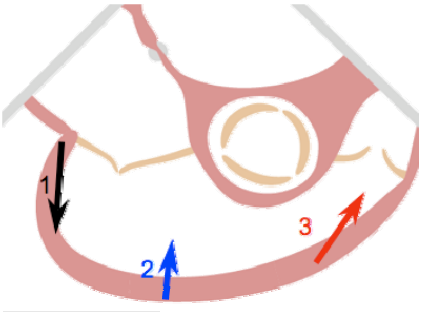


Fig. 5. Clockwise and counter-clockwise rotation and overall shortening in longitudinal direction

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).





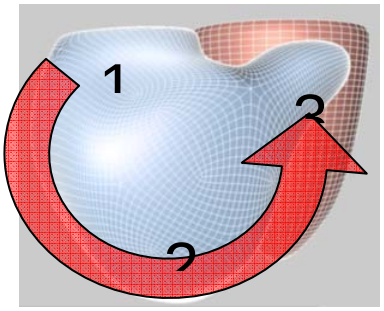


Fig. 6 Fig 7 Fig. 8

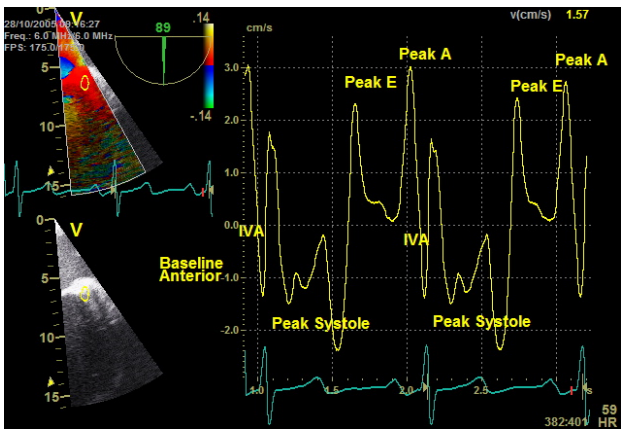
Fig. 7, 8: Sequence of RV-contraction. Wringing motion from the base to RVOT

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



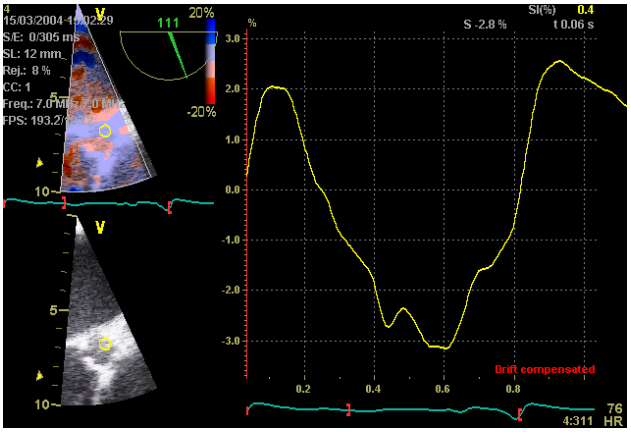


Fig. 10: strain curve – Peak systolic strain

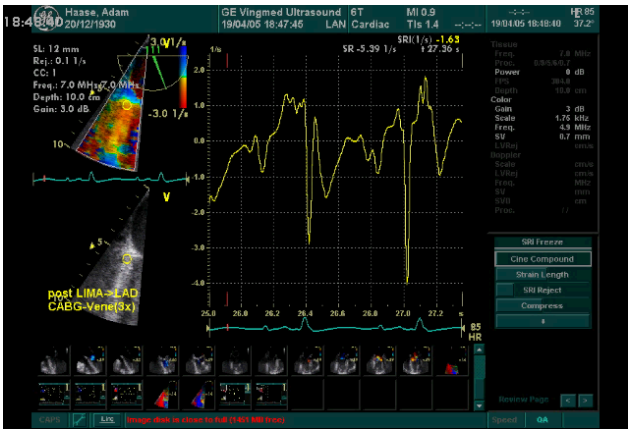


Fig. 11: Strain rate curve – Peak systolic strain rate

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).

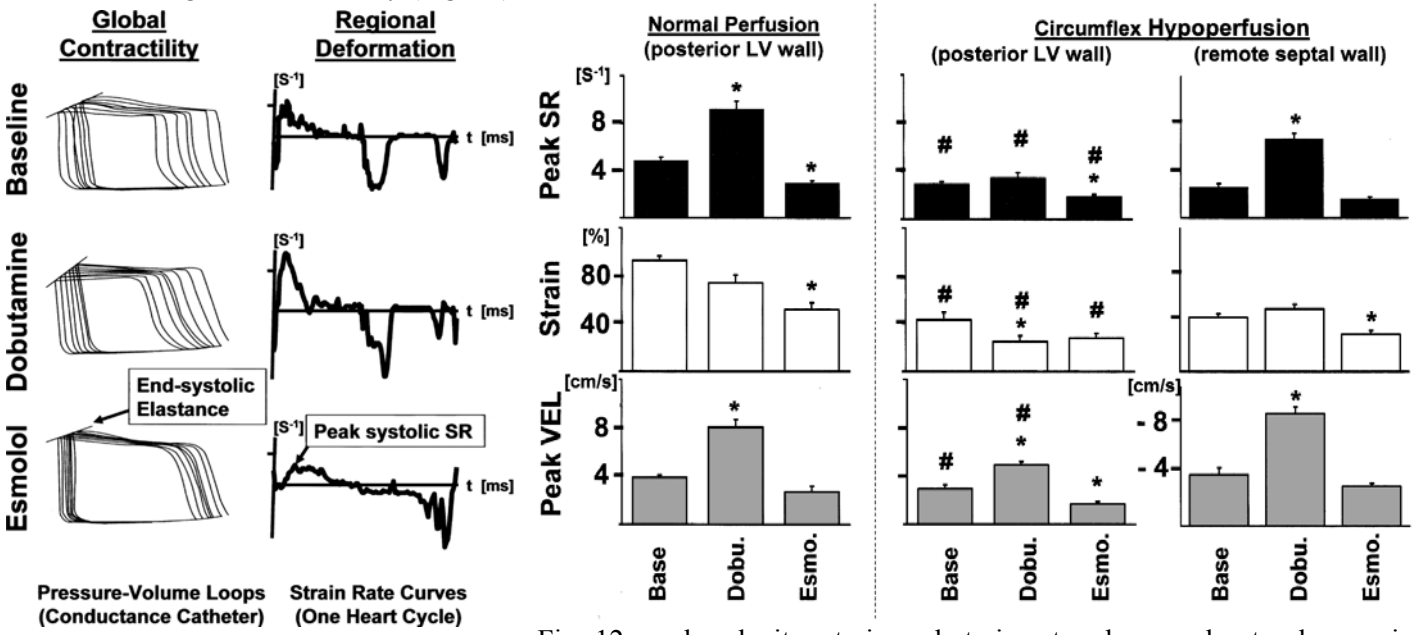


Fig. 12: peak velocity, strain and strain rate: changes due to changes in contractility (dobutamine, esmolol) [17].

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.

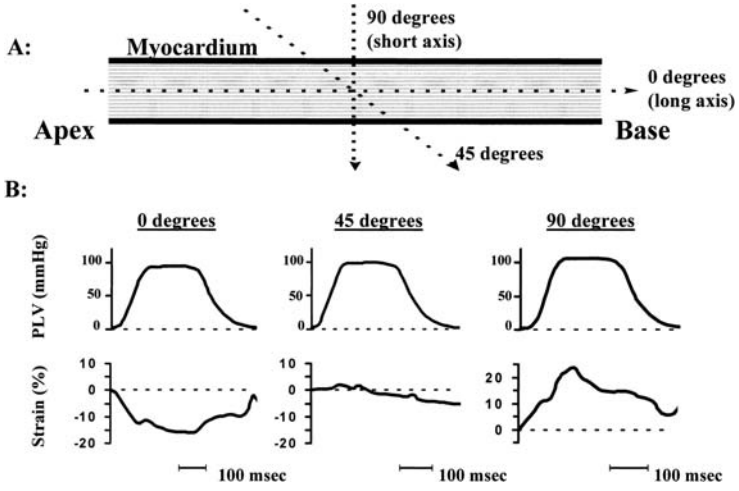


Fig. 13: Angle dependency of strain rate – changing from negative to positive values with increasing angles.

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.

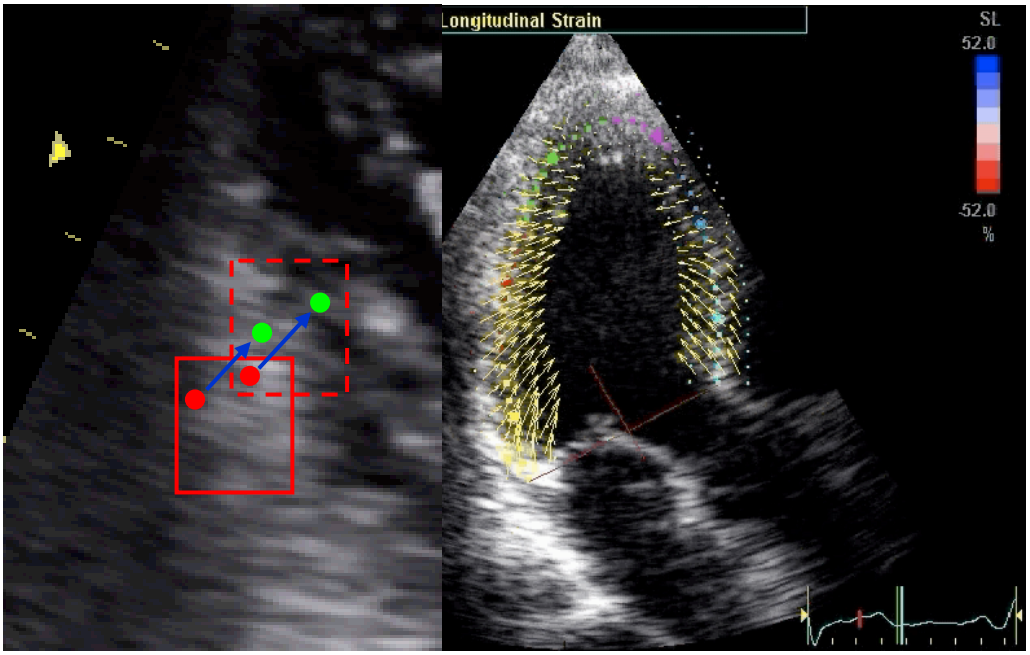


Fig. 14: Speckle tracking and vectorized imaging
By analyzing direction, shift over time (velocity) and spatial relationship between neighboured regions/speckles myocardial deformation can be quantified. (Fig. 14)

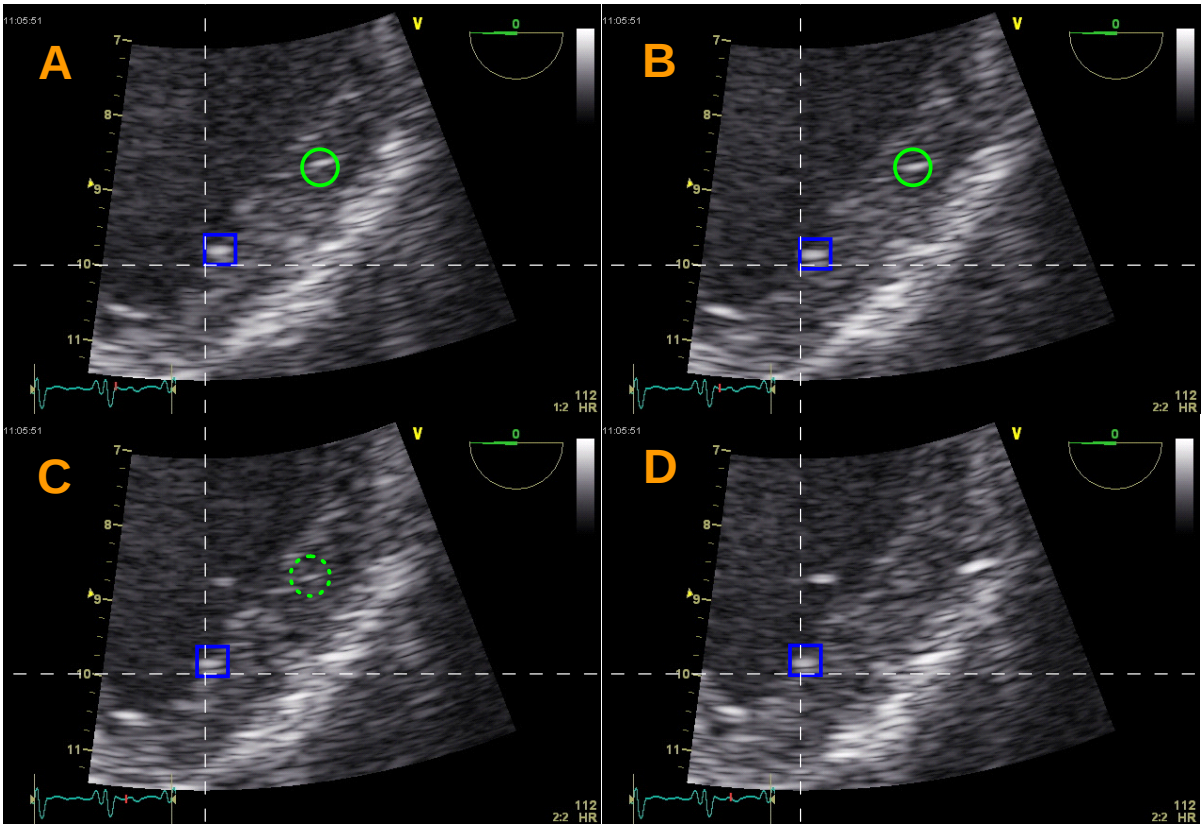


Fig.15: Speckle analysis: speckle tracking throughout the heart cycle

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.

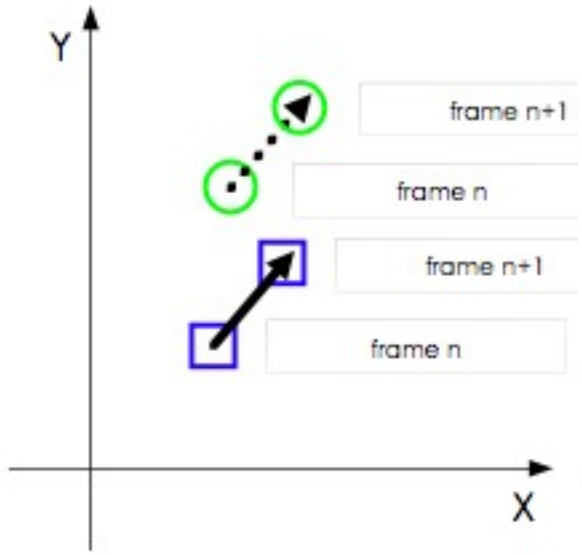


Fig. 16 Speckle tracking: identical velocities

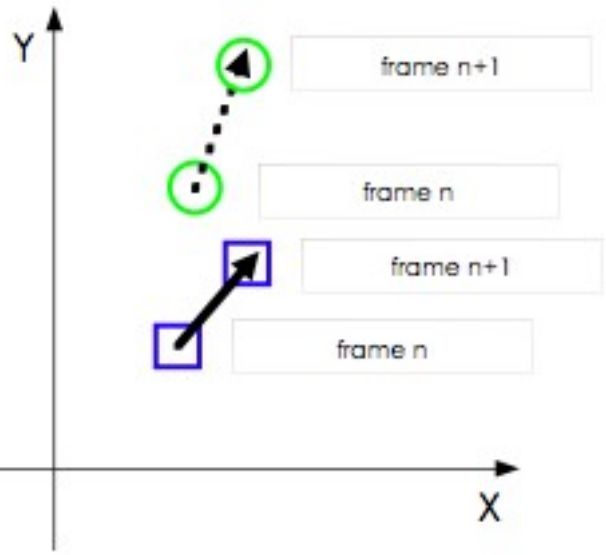


Fig. 17 Speckle tracking: different velocities

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.

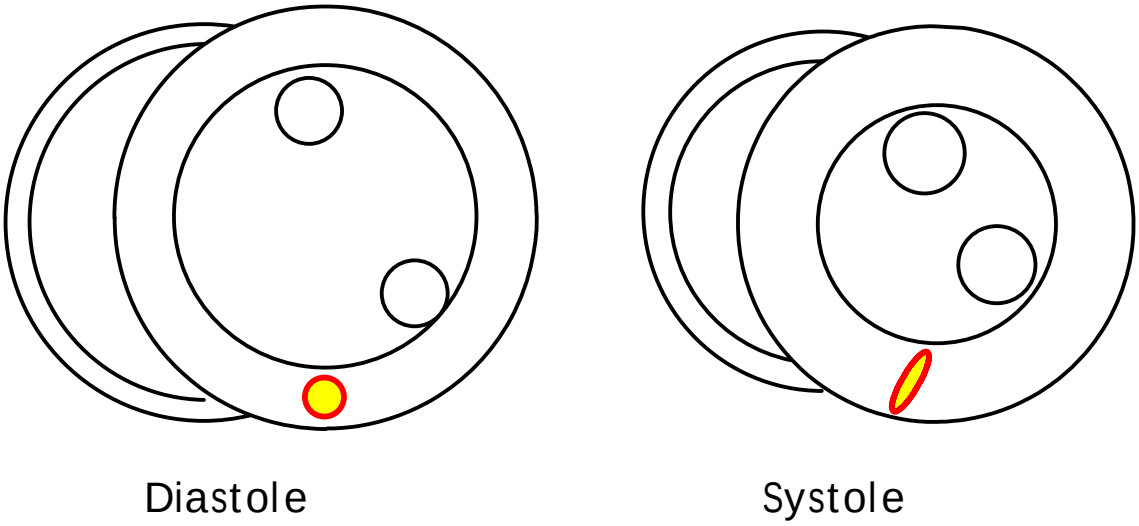


Fig. 18: Tissue deformation: a round tissue area during diastole becomes an ellipse during systole.

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)

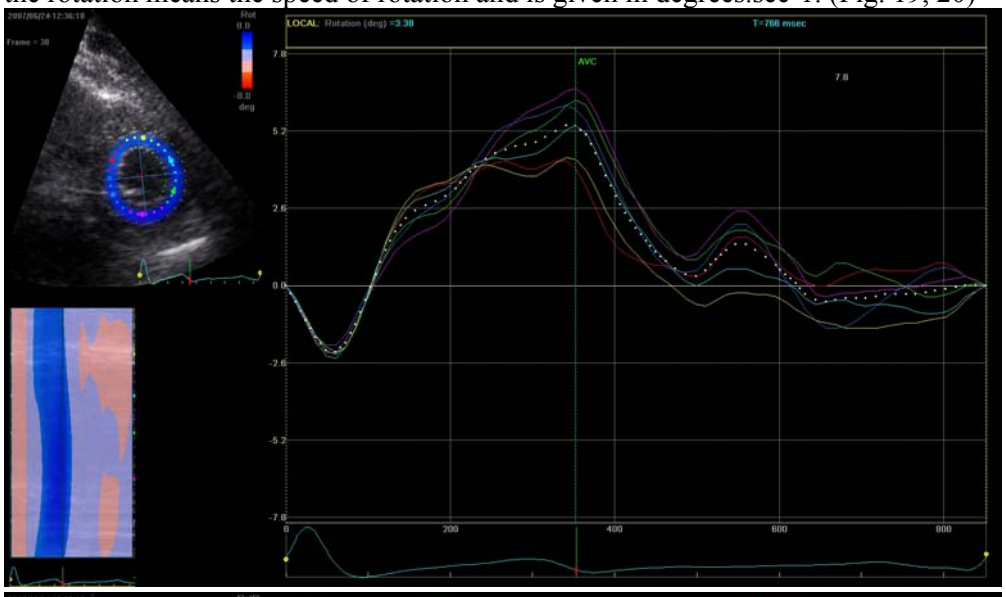


Fig. 19

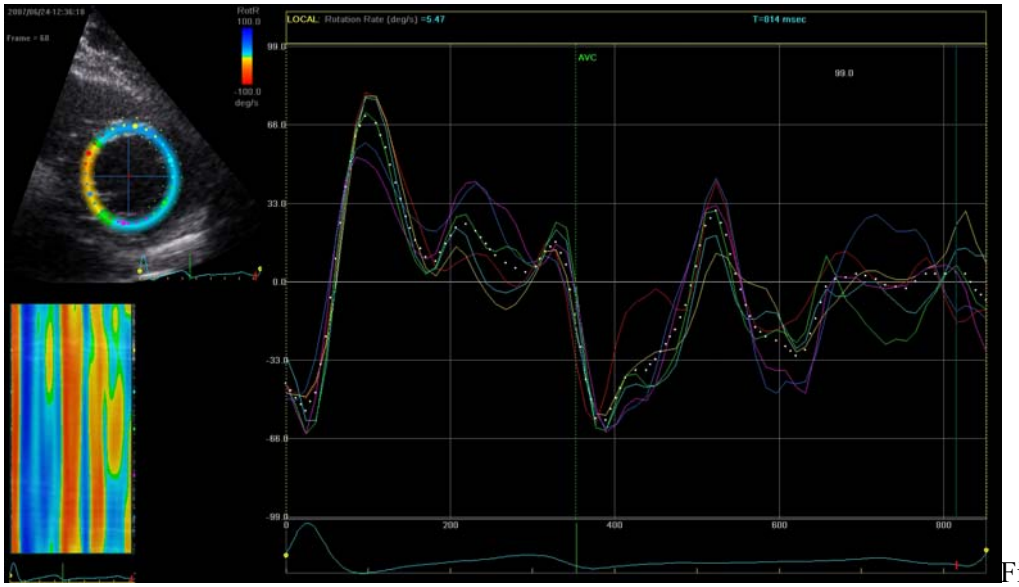


Fig. 20

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)

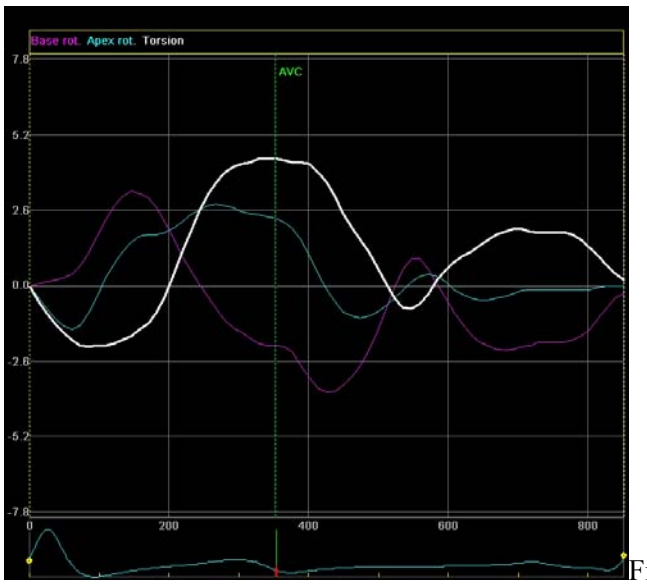


Fig. 21

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)
- 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)

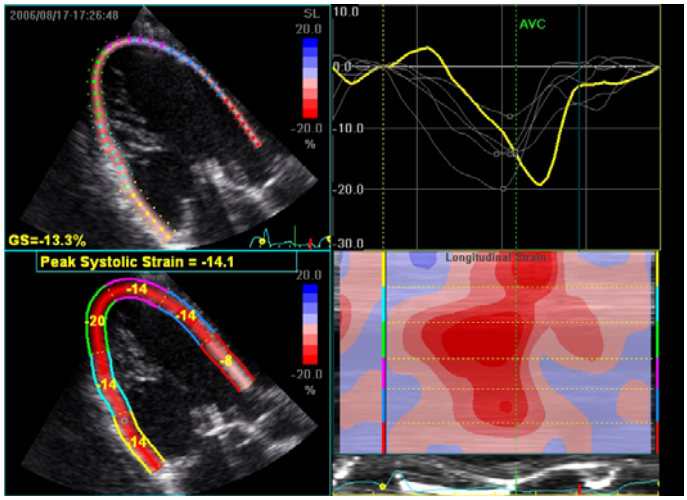


Fig. 19: 2D-strain analysis regardless of the angle between direction of deformation and the scanning plane.

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].

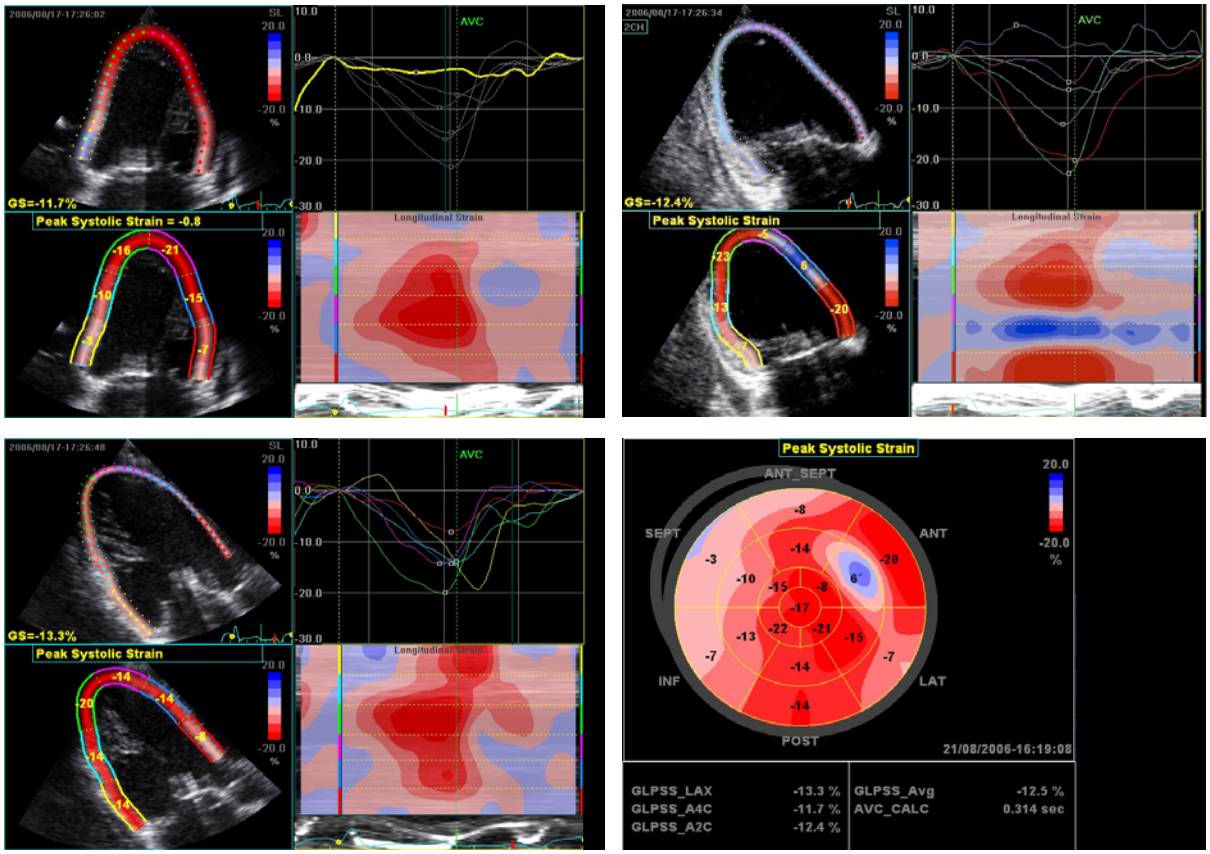


Fig. 20: Global peak systolic strain measurements. ME 4Ch, ME 2Ch and ME LAX are analyzed. GPSS values are obtained from averaging values of all 6 walls of the 3 planes.

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)

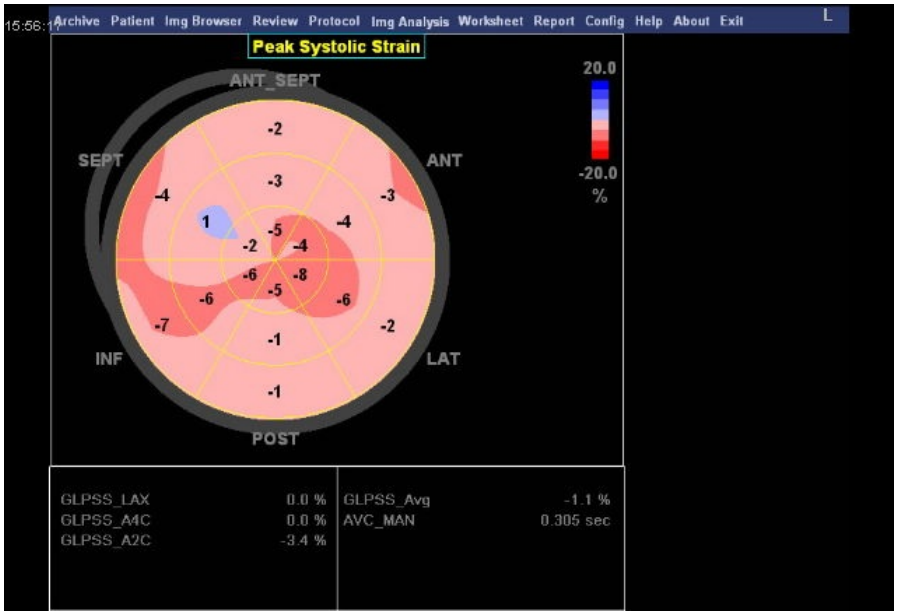


Fig 21: Bull´s eye-view example from a patient with dilative CMP

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)

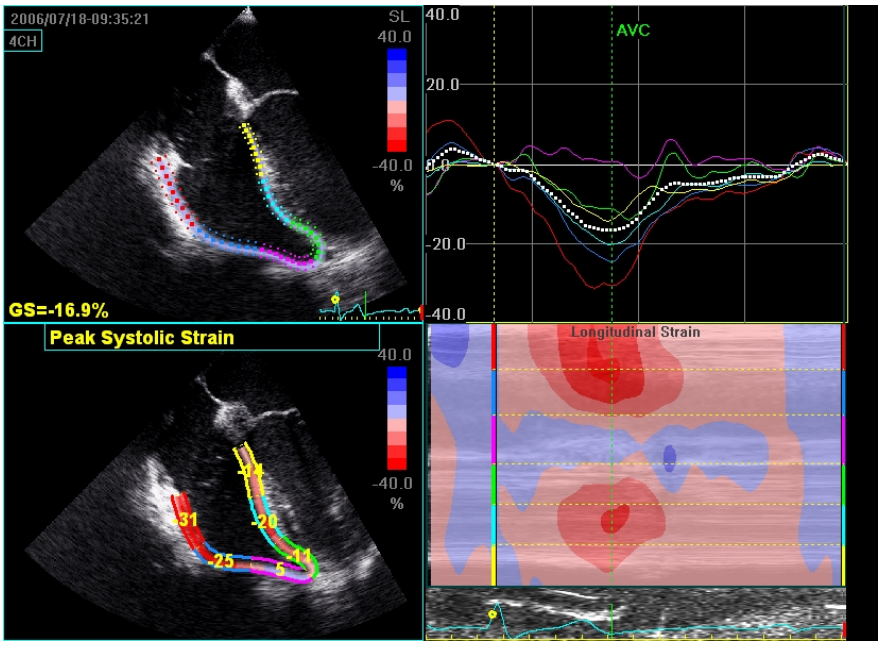


Fig. 22: Longitudinal peak global systolic strain of the right ventricle.

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:

1. 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.
2. 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)

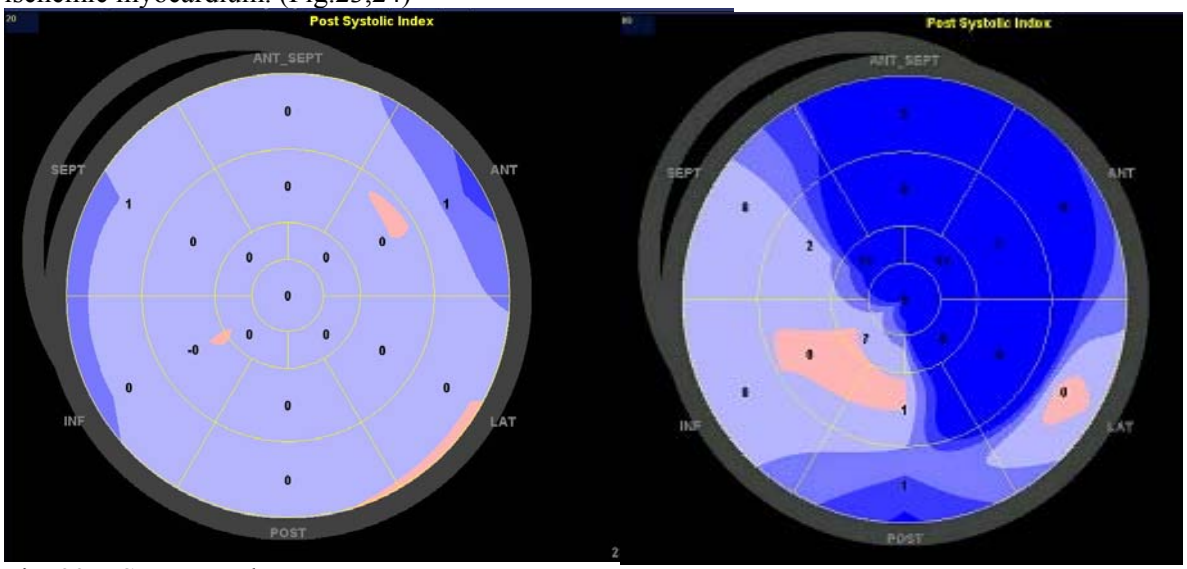


Fig. 23: PSI – normal BE

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)

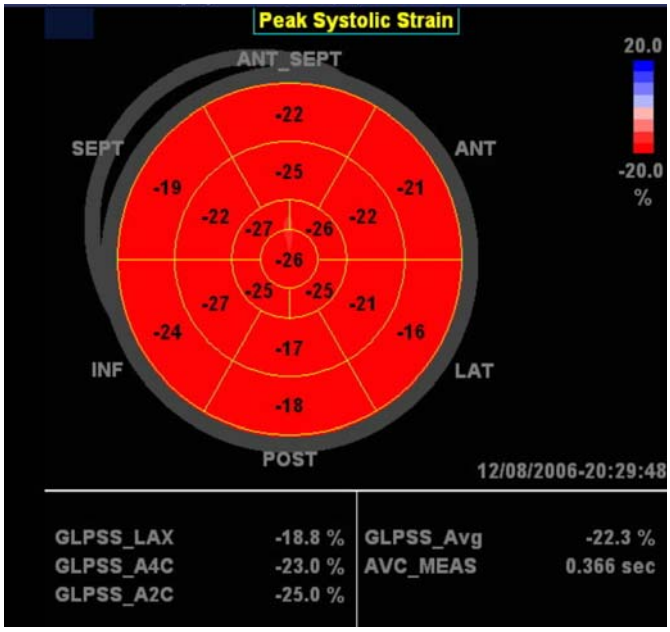


Fig. 26: Ischemia of the LAD

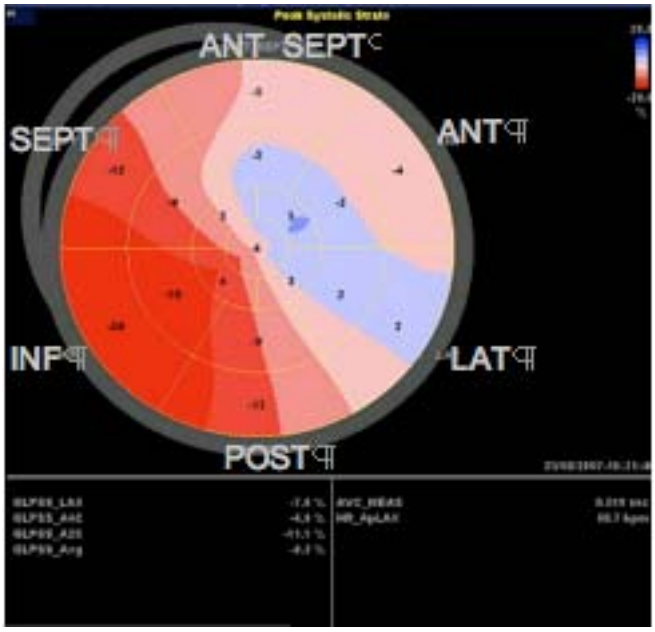


Fig. 25: Normal bull´s eye

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.

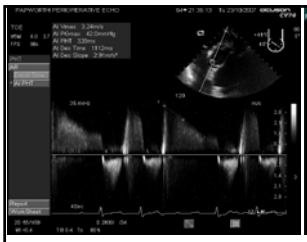
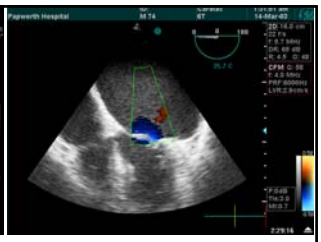
Future aspects

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.

Notes:

References:

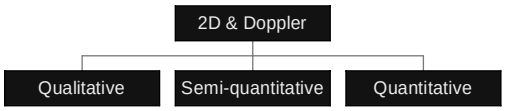
1. McGowan JH and Cleland JGF. Reliability of reporting left ventricular systolic function by echocardiography: A systematic review of 3 methods. Am Heart J 2003;146:388-97
2. Sanchez-Quintana D, Climent V, Ho SY, Anderson RH, et al. Myoarchitecture and connective tissue in hearts with tricuspid atresia. Heart 1999;81:182-91
3. Torrent-Guasp, Francisco, Ballester, Manel, Buckberg, et al. Spatial orientation of the ventricular muscle band: Physiologic contribution and surgical implications. J Thorac Cardiovasc Surg 2001;122:389-392
4. Port S, Cobb FR, Coleman E et al. Effect of age on the response of the left ventricular ejection fraction to exercise. N Engl J Med 1980;303:1133-37
5. Wandt B, Bojö L, Hatle L, et al. Left ventricular contraction pattern changes with age in normal adults. J Am Soc Echocardiogr 1998;11:857-63
6. Notomi Y, Setser RF, Thomas JD, et al. Assessment of Left Ventricular Torsional Deformation by Doppler Tissue Imaging. Validation Study With Tagged Magnetic Resonance Imaging. Circulation. 2005;111:1141-1147
7. Notomi Y, Martin-Miklovic MG, Oryszak SJ, Thomas JD et al. Enhanced ventricular untwisting during exercise: a mechanistic manifestation of elastic recoil described by Doppler tissue imaging. Circulation. 2006;30;113(21): 2524-33.
8. Ballester-Rodes M, Flotats A, Torrent-Guasp F, et al. The sequence of regional ventricular motion. Eur J Cardiothorac Surg 2006; 29: S139-144
9. Weidemann F, Broscheit J, Strotmann J et al. Dobutamine induces ineffective work in regional ischaemic myocardium: an experimental strain rate imaging study. Clinical Science 2004; 106: 173–181.
10. Urheim S, Edvardsen T, Torp H et al. Myocardial Strain by Doppler Echocardiography : Validation of a New Method to Quantify Regional Myocardial Function. Circulation, Sep 2000;102:1158 - 1164.
11. Reisner SA, Lysansky P, Friedman Z, et al. Global longitudinal strain: a novel index of left ventricular systolic function. J Am Soc Echocardiogr 2004;17: 630-3.
12. Thomas Helle-Valle, Jonas Crosby, Thor Edvardsen, et al.New Noninvasive Method for Assessment of Left Ventricular Rotation: Speckle Tracking Echocardiography. Circulation, Nov 2005; 112: 3149 - 3156.
13. Kukulski T, Jamal F, Herbots L, et al. Identification of acutely ischemic myocardium using ultrasonic strain measurements. A clinical study in patients undergoing coronary angioplasty. J Am Coll Cardiol 2003;41:810-9.

Grading valvular dysfunction

John Kneeshaw
Papworth UK

Principles



- There is never a single best test.
- Evaluation is based on multiple modalities
- Loading conditions matter
- Note the extra valvular effects of valvular lesions

Stenotic lesions

Qualitative and semi-quantitative

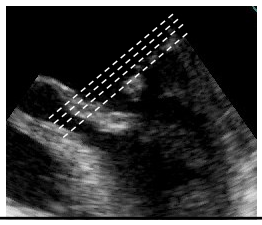
- 2D valve appearances
  - leaflet morphology and motion
  - planimetry
- Extra valvular effects
  - chamber size
  - wall thickness

Qualitative Mitral Stenosis
Mass General 2D echo morphology score

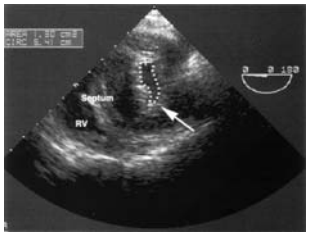
Morphology	Score
Mobility	
Highly mobile valve with only leaflet tips restricted	1
Leaflet middle and base portions have normal mobility	2
Valve continues to move forward in diastole, mainly from the base	3
No or minimal forward movement of the leaflets in diastole	4
Leaflet Thickening	
Leaflets near normal in thickness (4–5 mm)	1
Midleaflets normal, marked thickening of margins (5–8 mm)	2
Thickening extending through the entire leaflet (5–8 mm)	3
Marked thickening of all leaflet tissue (>8–10 mm)	4
Subvalvular Thickening	
Minimal thickening just below the mitral leaflets	1
Thickening of chordal structures extending up to one-third of the chordal length	2
Thickening extending to the distal third of the chordal length	3
Extensive thickening and shortening of all chordal structures	4
Calcification	
A single area of increased echo brightness	1
Scattered areas of brightness confined to leaflet margins	2
Brightness extending into the mid portion of the leaflets	3
Extensive brightness throughout much of the leaflet tissue	4
Total score is obtained by adding the scores for each of the four features	

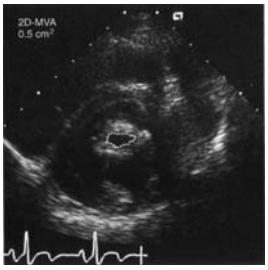
MV Area by Planimetry

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

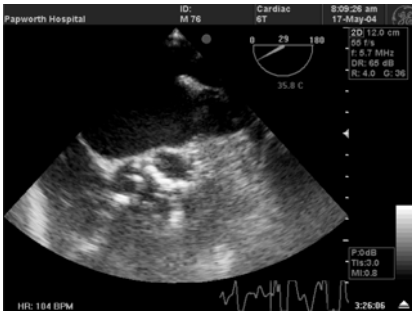


Examples

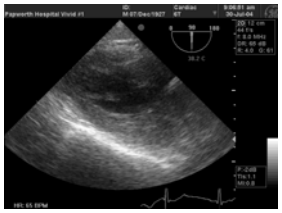


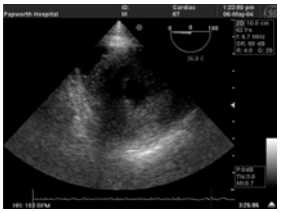


Planimetry?

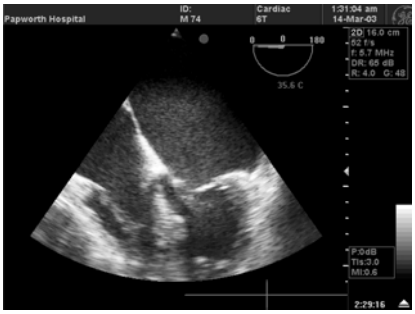


Effect of valve on chamber





Chamber size



Stenotic lesions

Quantitative principles

- Conservation of flow
- Orifice flow
- Flow acceleration
- Pressure gradient
- Pressure half time

Conservation of flow

- Continuity
- Flow in two continuous conduits is equal
- Flow = cross sectional area x velocity

  $cm^3/sec = cm^2 \times cm/sec$
- If you know two the third can be calculated

Two point continuity

Volume 1 = Volume 2

Volume = area x VTI

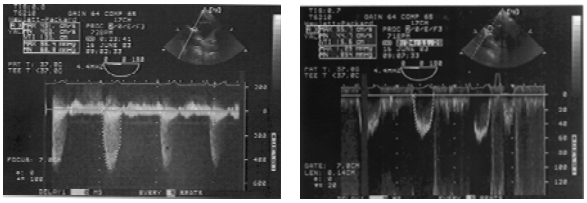
$cm^2 \times cm = cm^3$

Area 1 x VTI 1 = Area 2 x VTI 2

$$\text{Area 1} = \frac{\text{Area 2} \times \text{VTI 2}}{\text{VTI 1}}$$

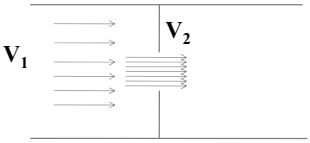
Aortic valve area continuity

Aortic valve VTI **LVOT VTI**



Needs LVOT area from ME SAX view.
NB error of squares

Orifice flow



$\Delta P = 4(v_2^2 - v_1^2)$ + total acceleration + viscous losses

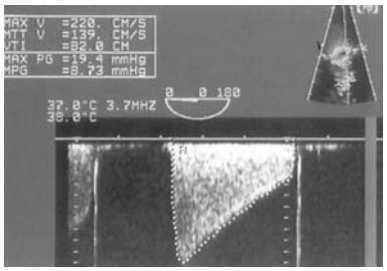
$\Delta P = 4(v_2^2 - v_1^2)$

$\Delta P = 4(v_{max}^2)$

Mitral Stenosis

- Mean pressure gradient
- Pressure half time
- Continuity
- PISA

Mean transmitral pressure gradient



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

Problems

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

Generally

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

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

PHT and spectral Doppler

Time taken for the pressure gradient to decrease by ½

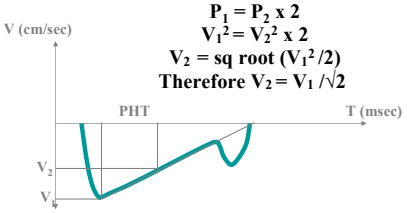
$P_1 = P_2 \times 2$ and Press gradient = $4V^2$

$P_1 = P_2 \times 2$

$V_1^2 = V_2^2 \times 2$

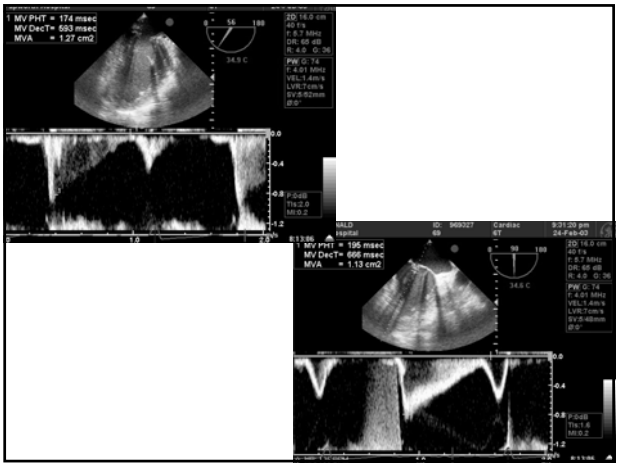
V_2 = sq root (V_1^2 /2)

Therefore $V_2 = V_1 / \sqrt{2}$



Comparison with invasively derived Gorlin formula data led to

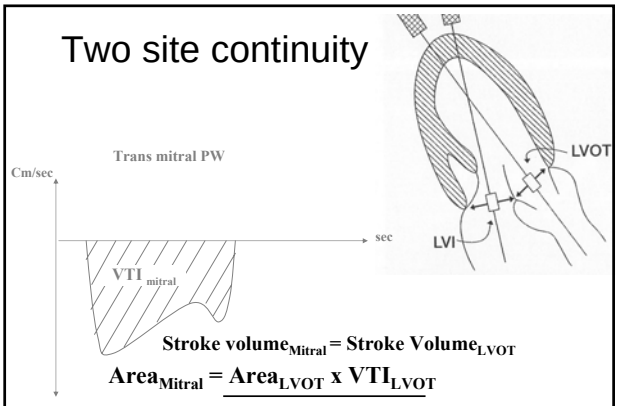
MVA = 220/ $T_{1/2}$



Limitations

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

Two site continuity

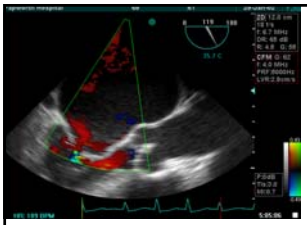


$Stroke\ volume_{Mitral} = Stroke\ Volume_{LVOT}$

$$Area_{Mitral} = \frac{Area_{LVOT} \times VTI_{LVOT}}{VTI_{Mitral}}$$

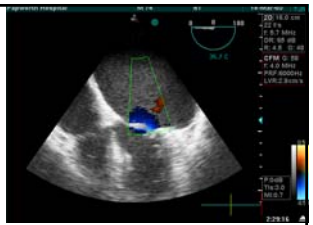
Continuity

- Stroke volume can be VTI x Area at any site (LVOT if no AR, or Pulmonary artery)
- Only applicable if there is minimal Mitral Regurgitation



MS with central MR.

Flow convergence and acceleration with PISA



MS with trivial MR.

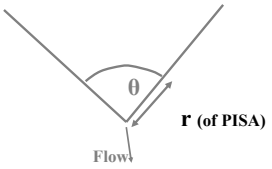
PISA

Proximal Isovelocity Surface Area

Proximal Isovelocity Surface Area

- Theoretically able to calculate MVA even in the presence of MR.
- Adjust colour flow variables to produce a PISA
- Measure PISA radius
- Note aliasing velocity
- Note angle of interception of mitral leaflets
- Measure peak transmitral velocity

Maths



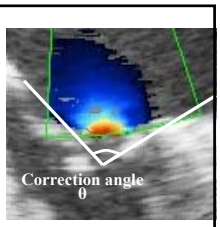
Flow through valve = Flow at PISA

$$MVA \times V_{max} = Velocity_{alias} \times PISA$$

$$MVA \times V_{max} = V_{alias} \times 2\pi r^2 \times \frac{Valve\ angle(\theta)}{180}$$

Example

r = 1 cm
Alias Velocity = 0.58 m/sec
Angle of leaflets = 100^0
V_{max} = 1.6 m/sec



$$MVA \times V_{max} = V_{alias} \times 2\pi r^2 \times \frac{Valve\ angle(\theta)}{180}$$

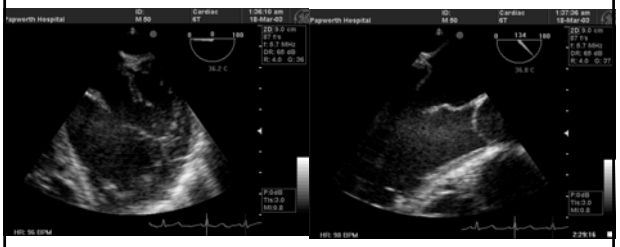
MVA x 1.6 = 0.58 x 2 x 3.14 x $(1)^2$ x 100/180
MVA = 2.024/1.6
MVA = 1.26cm^2

Regurgitant lesions

Qualitative and semi-quantitative

- 2D valve appearances
  - leaflet morphology and motion
- Doppler
  - jet density
  - jet direction
- Extra valvular effects
  - back flow effects
  - chamber size
  - wall thickness

2D views



What's big IS big

CW Regurgitant signal strength in MR

How densely filled is the jet spectral display

- Dependent on jet direction
- Very densely filled is a good correlate of severe MR

CW Regurgitant signal strength in AR

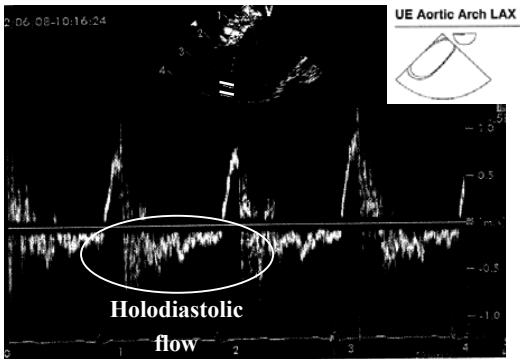
At constant setting gain

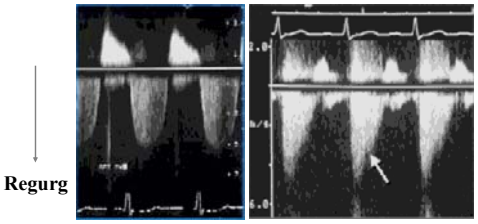
More intense Doppler signals → more severe regurgitant lesions

Aortic Diastolic Flow Reversal

- Retrograde flow in ascending, descending aorta or aortic arch
- UE aortic arch LAX view
- With tortuous aorta
  - UE aortic arch SAX view

- Normally
  - Minor retrograde flow in the ascending and proximal descending aorta
- AR
  - Holodiastolic retrograde flow
  - More severe
    - The retrograde flow relative to antegrade flow increase
    - Further distally of holodiastolic retrograde

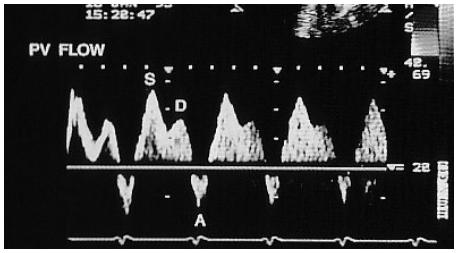




Mild – Mod **Severe**

TTE images

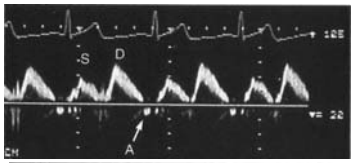
Pulmonary venous PW spectral display



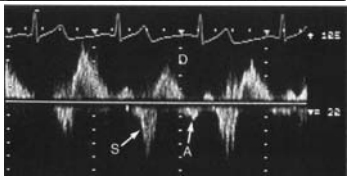
Normal velocity pattern S/D > 1

Left Upper Pulmonary vein velocities

Systolic blunting S/D < 1

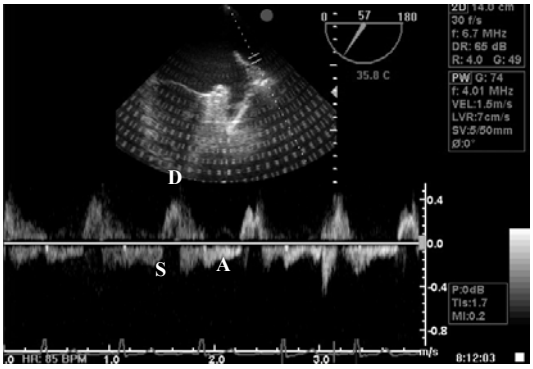


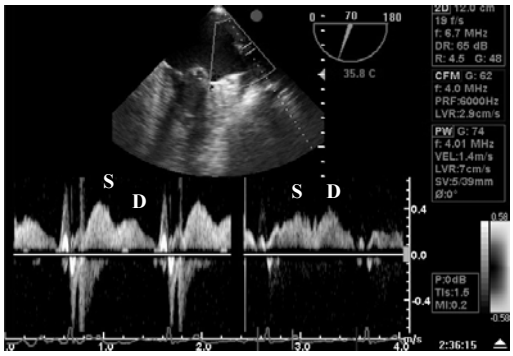
Systolic reversal S/D < 0



Limitations

- Flow reversal highly predictive of severe MR
- Dependent on LA and LV compliance
- Affected by MR jet direction
- Blunted flow pattern not a reliable predictor of mod MR often seen in LV dysfunction with raised LA pressure





Reliable?
Normal & blunted

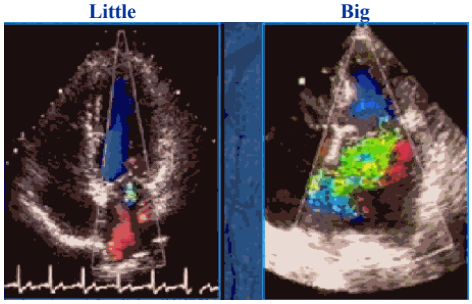
Regurgitant lesions
Quantitative principles

- Jet mapping
- Vena contracta
- Conservation of flow
- Pressure half time
- Flow acceleration (PISA)

Spatial area mapping

TTE images

Little **Big**

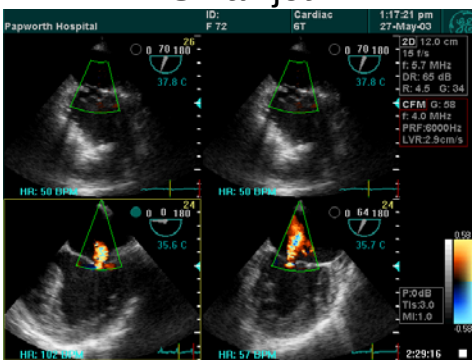


Area Mapping

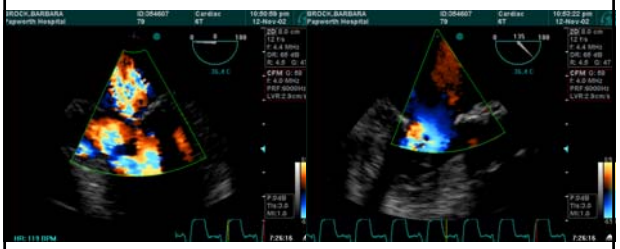
Mild	Small Jet	<20% LA	<4cm^2
Mod	Bigger Jet	20-40% LA	4-8cm^2
Severe	Biggest Jet	>50% LA	>8cm^2

Beware % area - TOE cannot see the whole LA
Multiple planes
Note effects of instrument settings
Beware eccentric jet – Choanda effect
LA compliance

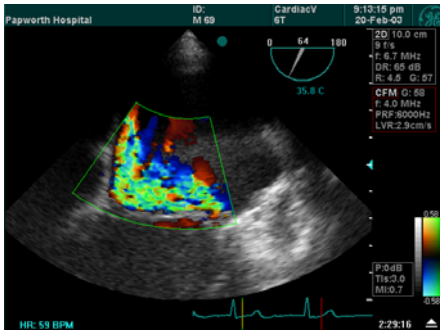
Small jet



Bigger jet



Eccentric Jet



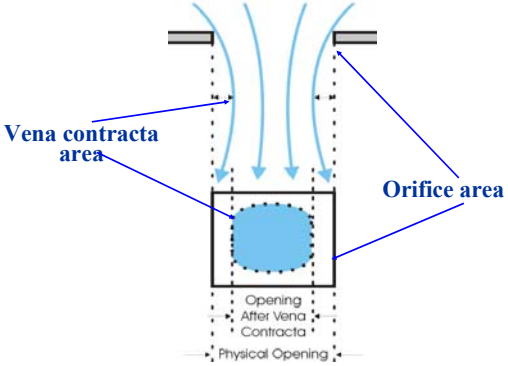
Vena Contracta Measurement

Well known in the field of fluid dynamics.

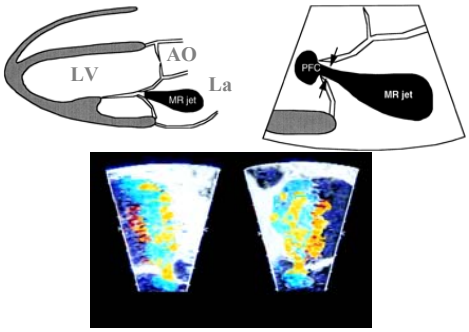
The Vena Contracta effect occurs when fluid flows through any opening.

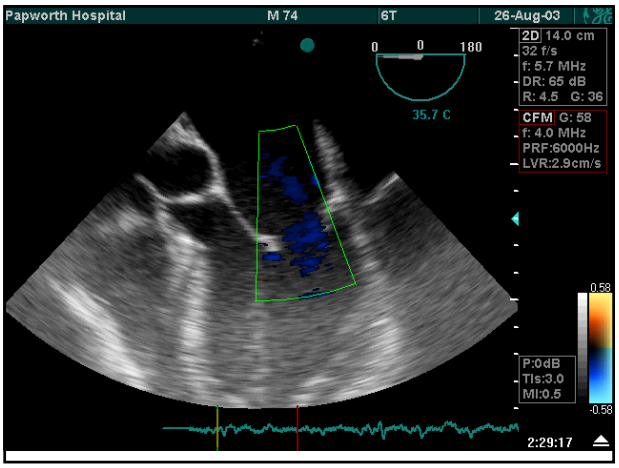
The fluid "sticks" to the edges of the opening, thus effectively reducing the size of the opening.

Fluid dynamics diag



MR Vena Contracta





Vena Contracta Measurement

Measure the VC diameter in at least 2 orthogonal planes

VC > **6.5mm** has a 90% sensitivity and 96% specificity for angiographic 4+ MR

VC is not independent of flow or loading

Aortic

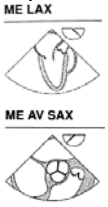
New Index for Grading the Severity of Aortic Regurgitation Based on the Cross-Sectional Area of Vena Contracta Measured by Color Doppler Flow Mapping.

NOZAKI S(Kagawa Medical Univ., Jpn) MIZUSHIGE K(Kagawa Medical Univ., Jpn) TAMINATO T(Kagawa Medical Univ., Jpn) OBAYASHI N(Takamatsu Obayashi Hospital, Kagawa, Jpn) MATSUO H(Kagawa Medical Univ., Jpn)

Circulation. VOL.67;NO.3;PAGE.243-247(2003)

Mapping the Vena Contracta

- Vena contracta
  - The narrowest point of the jet crossing the valve plane
- Severe AR
  - ME long-axis view
    - Width > 6 mm
  - ME AV short-axis view
    - Area > 7.5 mm²



AR Jet mapping

Jet height / LVOT diameter

- ME AV long-axis view (120º)
- MV five-chamber view
- Color M-mode

Jet height/ LVOT diameter

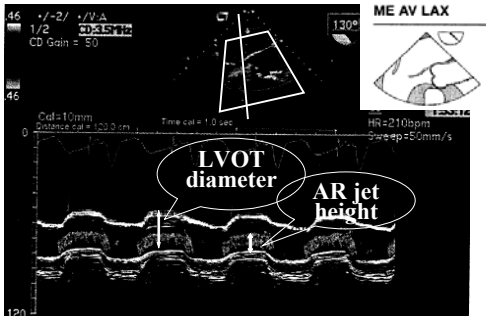


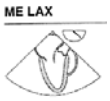
TABLE 11.1. SCORING OF SEVERITY OF AORTIC REGURGITATION

Method of evaluation (view)	Trivial ($0–1^+$)	Mild ($1^+–2^+$)	Moderate ($2^+–3^+$)	Severe ($3^+–4^+$)
AI jet height/LVOT diameter (ME AV LAX)	1%–24%	25%–46%	47%–64%	>65%
AI areal/LVOT area (ME AV SAX)	<4%	4%–24%	25%–59%	>60%

- Most used intraoperative method
- Most easily applied method
- Affected by changing in preload conditions

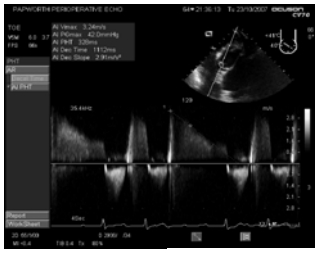
Color Flow Mapping of the Depth of the Regurgitation jet

- Recorded in relation to MV structures
  - >90% toward ant. mitral leaflet



Method	Trivial (0~1+)	Mild (1+~2+)	Moderate (2+~3+)	Severe (3+~4+)
Jet depth mapping	LVOT	Mid ant. Mitral leaflet	Tip ant. Mitral leaflet	Papillary m. head

Pressure Half-Time Measurement



Factors affect the estimation
- LV and aortic compliance
- High LVEDP
  - Heart failure, restrictive physiology and diastolic dysfunction

→ Artifactually worsen the severity of the lesion

Calculation of Regurgitation Volume

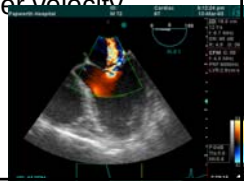
AR volume = LVSV - RVSV

$= A_{LVOT} \times TVI_{LVOT} - A_{RVOT} \times TVI_{RVOT}$

Time-consuming and inaccurate (squares errors)

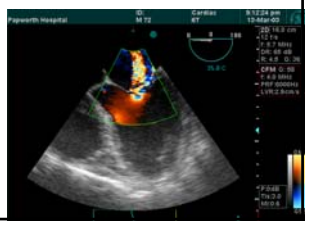
PISA Mitral regurgitation

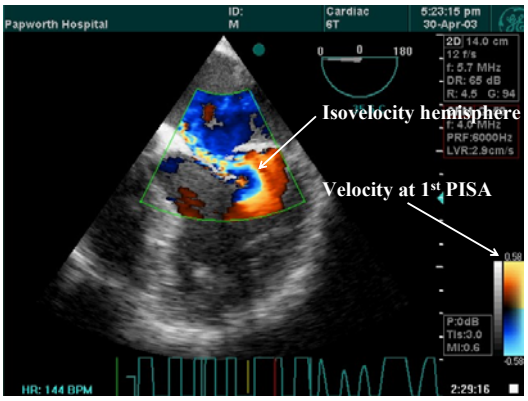
- Present in moderate to severe MR
- Area of Doppler flow convergence proximal to the regurgitant orifice (Flow acceleration)
- Represented by series of concentric hemispheres of blood at same velocity
- Closer hemispheres = higher velocity



Continuity theory

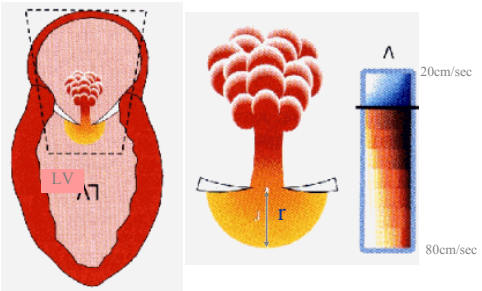
- Blood flow through the PISA = blood flow through the regurgitant orifice
- Flow = Velocity x Area
- $cm^3.sec^{-1}$ = $cm.sec^{-1}$ x cm^2





Flow at PISA = $2\pi r^2$ x Aliasing velocity

cm^3/sec = cm^2 x cm/sec



Regurgitant orifice area (ROA)

ROA = $\frac{\text{MR flow rate } (cm^3.sec^{-1})}{\text{MR peak velocity } (Vmax_{MR})}$

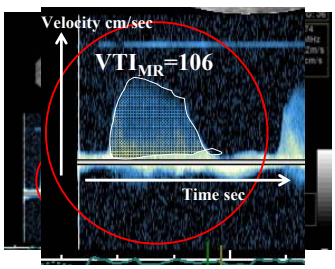
ROA = $\frac{136.7\ cm^3.sec^{-1}}{430cm.sec^{-1}}$

ROA = 0.32 cm^2

Regurgitant volume

Volume (cm^3) = Area (cm^2) x VTI (cm)

Regurgitant volume (cm^3) = ROA (cm^2) x VTI_{MR} (cm)



Regurgitant volume

Volume (cm^3) = Area (cm^2) x VTI (cm)

Regurgitant volume (cm^3) = ROA (cm^2) x VTI_{MR} (cm)

= 0.32 cm^2 x 106cm

= 34 cm^3

Normal aortic valve

	Mean	Range
Valve area cm^2	3	2 - 4
Ascending aorta velocity (m/sec)	1.35	1.0 – 1.7
LVOT velocity (m/sec)	0.9	.07 – 1.1
LVOT diam (cm)	2.0	1.8 – 2.2

Aortic stenosis

	Mild	Moderate	Severe
Peak aortic velocity (m/sec)	2.5 - 3.0	3.0 – 4.0	> 4.0
Peak instantaneous pressure gradient (mm Hg)	25 – 35	35 – 60	>60
Mean pressure gradient (mm Hg)	15 – 20	20 – 35	>35
Aortic valve area (cm^2)	1.2 – 1.5	0.7 – 1.2	< 0.7
Velocity ratio $_{LVOT/AV}$	0.35 – 0.4	0.25 – 0.35	<0.25

Aortic regurgitation

	Mild	Moderate	Severe
Structural factors LV size Aortic leaflets	 Normal Normal or abnormal	 Normal or dilated Normal or abnormal	 Usually dilated Abnormal: Flail or wide coaptation defect
Doppler Jet width in LVOT colour flow Doppler Jet density CWD Jet deceleration PHT (msec) Flow reversal in desc aorta	 Small in central jet Incomplete or thin >500 Slow	 Intermediate Dense 500 -250	 Large if central; variable if eccentric Dense <200 very steep
Quantitative VC width (cm) Jet width/LVOT) width (%) Jet CSA/ LVOT CSA (%) Regurg vol (ml/beat)	 <0.3 <25 <5 <30	 0.3 – 0.6 25 – 45 / 46 – 64 5 – 20 / 21 – 59 30 – 44 / 45 – 59	 >0.6 <65 >60 >60

Mitral stenosis

	Mild	Moderate	Severe
Mean pressure gradient (mm Hg)	<6	6 – 12	> 12
Pressure half time (msec)	90 – 150	150 – 219	>220
Mitral valve area (cm^2)	1.5 – 2.5	1.0 – 1.5	<1.0

Mitral regurgitation

	Mild	Moderate	Severe
Structural factors LA size LV size Leaflets and subvalvar structures	 Normal Normal Normal or abnormal	 Normal or dilated Normal or dilated Normal or abnormal	 Usually dilated Usually dilated Abnormal or flail leaflet or pap muscle
Doppler Colour flow jet area Mitral inflow PWD Jet density CWD Jet contour CWD Pulmonary vein flow	 Small central jet (Usually <4cm^2 or <20% of LA area) A-wave dominant Incomplete / faint Parabolic Systolic dominant	 Variable Variable Dense Mainly parabolic Systolic blunting	 Large jet (>10cm or > 40% LA area) any direction or variable wall hugging, to back of LA E-wave dominant (>1.2m/sec) Dense Early peak triangle Systolic reversal
Quantitative VC width (cm)	 <0.3	 0.3 – 0.65	 >0.65

Tricuspid regurgitation

	Mild	Moderate	Severe
Valve	Usually normal	Normal or abnormal	Abnormal or flail leaflet or poor coaptation
RA / RV/ IVC size	Normal	Normal or dilated	Usually dilated
Jet area (central jets) (cm^2)	<5	5 - 10	>10
VC width (cm)	-	Usually <0.7	>0.7
PISA radius (cm)	<0.5	0.6 – 0.9	>0.9
Jet density & contour (CWD)	Soft & parabolic	Dense, variable contour	Dense, early peak triangle
Hepatic vein flow	Systolic dominant	Systolic blunting	Systolic reversal

TOE support in cardiac assist devices

Emanuele Catena, MD
Division of Cardiothoracic Anesthesia and Intensive Care, Cardiologic Department "A. De Gasperis" Niguarda Cà Granda Hospital, Milan, Italy.

Introduction

Mechanical circulatory support is nowadays a well established treatment modality for end stage heart failure (HF). Patients candidates for cardiac transplantation who decompensate while awaiting graft can be supported by long-term devices. These devices are intra-corporeal pumps, expensive and complicated to install, but offer the advantages of a high cardiac output and a good patient mobility.
Short-term devices are utilized as acute support after the initial resuscitation of the patient. These devices are bedside extra-corporeal pumps, less expensive and less complicated to install. They provide a brief but sufficient time to wait patient recovery ("bridge to recovery") or to evaluate further therapies ("bridge to long term device" or "bridge to transplantation").
Both long-term and short-term devices require a careful echocardiographic management before implant, during surgical procedure, and during postoperative course.

Long-term devices

Cardiac transplantation is the most effective treatment for patients with HF refractory to medical or surgical therapy. However, its epidemiologic impact remains limited because < 3000 donor organs are available annually worldwide. Left ventricular assist device (LVAD) insertion is usually performed either because of a deteriorating circulatory state in patients taking maximal medical therapy, impending organ damage in absence of donor, or in patients who have temporary contraindications. LVAD implantation allows the hemodynamic normalization, improves end-organ dysfunction, and provides a reasonable quality of life.
Successful experience with the use of a mechanical assist device as a bridge to transplantation, particularly among those with prolonged duration of implantation, justifies evaluating the last generation of these devices as long-term or destination therapy for chronic HF in patients who are not candidates for heart transplantation for a variety of reasons, including cancer, age, severe comorbidity, and debilitation health. To be eligible for destination therapy, these patients must be at high risk for mortality (>75% in 1 year).

Long-Term devices : the characteristics of the pumps

The implantable devices utilized for long term support can be pulsatile pumps or continuous flow pumps. Continuous flow axial pumps are the new-generation of LVADs. Axial flow pumps currently implanted as a bridge to heart transplantation or destination therapy are the MicroMed DeBakey VAD (MicroMed Technology, Inc., Houston, TX), the Incor (Berlin Heart AG) VAD, and the Jarvik 2000 FlowMaker (Jarvik Heart Inc., New York, NY). They have several potential advantages over current pulsatile devices: the pump is significantly smaller and therefore can be used in smaller patients; it's silent and relatively simple mechanics, without compliance chamber or valves, requires lower energy. These small pumps, placed into a small abdominal pocket, are designed to achieve from 5 to 7 L/min of blood continuous flow. The implantation requires median sternotomy and cardiopulonary bypass. The pump is connected to the left ventricular apex by an inflow cannula and to the aorta by an outflow graft. The pump housing unit contains the impeller (only moving part) and the motor. A motor cable is exteriorized above the right iliac crest and connected to the external control system. The control system consists of a small controller where pump flow, pump speed, and current signal values are displayed, and two batteries. Figure 1 shows MicroMed DeBakey long-term LVAD and its components.

Long-term devices: the role of echocardiography

Echocardiography is usually applied to any kind of LVAD to accomplish the following:

a) Preoperative evaluation

Echocardiography has to be used for: (1) search of masses or trombotic material into left ventricular (LV) apex where pump inflow cannula has to be inserted; (2) measurement of apex wall thickness to choice inflow cannula size; (3) search for atrial septal defects or patent forame ovale because they may create right-to left shunt, systemic desaturation, or paradoxical embolus; (4) search of aortic regurgitation. The presence of aortic regurgitation reduces the forward flow produced by the LVAD causing regurgitation into LV cavity; (5) diagnosis of mitral stenosis that would limit LV filling; (6) evaluation of ascending aorta to exclude aneurismal dilatations and damages of the aortic wall where outflow cannula has to be applied; (7) to evaluate right ventricular function because adequate LVAD circulatory support must be warranted by adequate transpulmonary blood flow.

The complete preoperative evaluation of right ventricular function is pivotal because the right and left hearts are connected in series and therefore the output of one ventricle become the input of the other. Due to the in-series nature of the right and left hearts, any increase in flow to the systemic circulation from the LVAD will result in an increase in venous return to the right ventricle. The right ventricle must be capable of increasing its cardiac output to at least the amount being pumped by the LVAD, an approximate doubling of flow from preoperative values. The in-series concept explains how an LVAD can unmask pre-existing right ventricular dysfunction by attempting to increase venous return beyond the capability of the right ventricle.

Right ventricular failure can manifest early in the post-LVAD setting, often immediately after placement and activation of the device in operating room. In the setting of preoperative assessment of right ventricular (RV) function, echo can help to identify two group of patients: (1) patients needing for left ventricular support alone and medical treatment and (2) patients at risk for severe RV failure requiring right mechanical support

The following preoperative echocardiographic predictors can be used to grade RV dysfunction:

Group 1. Before LVAD implant, most of these patients show a significant increase in right ventricular afterload due to passive pulmonary hypertension secondary to elevated left atrial and pulmonary venous pressures. Echocardiography usually shows moderate tricuspid regurgitation, RV-RA pressure drop from 30 to 50 mmHg, RV-FAC from 30% to 35%, right ventricular outflow tract fractional shortening (RVOT fs %) from 20% to 40%, TAPSE near 15 mm. LVAD support alone has the ability to produce dramatic improvements in right ventricular afterload due to reversal of passive pulmonary hypertension: it improves right heart function by reducing left atrial pressure and enhancing transpulmonary blood flow.

Patients showing severe RV walls hypokinesia and RV-FAC $\leq$ 25%, TAPSE $\leq$ 10 mm, RVOT fs $\leq$20% before LVAD implant, suffer RV failure when device is started. Pulmonary arterial pressures may be misleadingly low, a phenomenon more likely reflective of poor output state of the right ventricle than a true decrease in right-sided pressures; tricuspid regurgitation may be light or absent. These patients usually require maximal pharmacologic support to obtain a gradual improvement of right ventricular function.

***Group 2*.** Echocardiographic predictors of severe RV failure requiring RVAD after LVAD insertion are considered the following: anterior and/or inferior right walls akinesis, severe right ventricle enlargement (RV end diastolic diameter >> LV end diastolic diameter). To predict RV dysfunction after LVAD preoperative clinical and haemodynamic variables must be also considered: right atrial pressure (RAP) > 20 mmHg, transpulmonary gradient > 16 mmHg with fixed pulmonary resistances, low pulmonary artery pressure (PAP) and right ventricular stroke work index (RV-SWI), PAP-RAP < 4 mmHg.

b) Intraoperative evaluation when circulatory support starts

Intraoperative TEE allows the documentation of LVAD function immediately after implantation. Echographic signs of good LVAD function are the following: (1) neutral septum position indicating adequate LV filling and RV function; (2) inflow cannula correctly oriented toward the mitral valve without abutting any wall; (3) closed aortic valve indicating adequate LV unloading. Especially

during continuous flow pumps circulatory support it's important to ensure a good LV unloading and an adequate LV filling.

LV unloading. Detection of blood flow through the inlet cannula by means of color Doppler shows unidirectional continuous low-velocity flow and pulsed wave Doppler a continuous flow with slightly pulsatility. In our experience peak filling velocity patterns are variable: when pump achieves 5 L/min of blood flow with a proper blood drainage, they range from 60 to 120 cm/sec according to preload and to the remaining pumping action of the patient's heart. Transesophageal four-chambers view or transgastric long-axis view allow to assess the correct positioning of inflow conduit. Cannula obstruction can be assessed by means of color Doppler as high-velocity aliased flow at the cannula orfice with manifest convergence area. Cannula kinking causes the loss of Doppler signal in any echo views. Inflow obstructions are also described during circulatory support with pulsatile flow pumps. Similarly, echo shows aliased flow at the cannula orifice with significantly high velocities (>2.5 m/sec).

Mid-esophageal longitudinal view at 100-120 degrees shows outlet cannula perpendicularly stitched on the anterior wall of the ascending aorta. Normal blood outflow appears as a low-velocity flow at color Doppler and pulsed wave Doppler documents an unidirectional slightly pulsatile flow with peak velocity usually ranging from 1.0 to 2.0 L/min. Cannula obstruction can be assessed by means of color Doppler as high-velocity aliased flow at the cannula orfice with manifest convergence area. Cannula kinking causes the loss of Doppler signal in any echo views.

The primary variable that determine blood continuous flow is the vascular resistances. Intense vasoconstriction can result in an critical LV unloading, poor peripheral perfusion until to pump failure. Intraoperative transesophageal echocardiography (TEE) shows rightward septum shift, increased mitral regurgitation degree, aortic valve opening, sometimes spontaneous contrast echo into left chambers and pulsed wave Doppler interrogation documents continuous flow with an abnormal increased pulsatility (peak velocity >2.0 m/sec). Hypertensive crisis may be treated in order to maintain mean arterial blood pressure less than 85-90 mmHg.

LV filling. Left ventricular filling can be compromised by hypovolemia or by right ventricular failure. Severe hypovolemia or very poor transpulmonary flow can be dangerous with pulsatile devices: this condition lead to falling left–sided filling pressures with consequent collapse of left chambers and, because a pulsatile LVAD keeps pumping and tries to fill against an empty and collapsed left ventricle, a subatmospheric pressure of up to 5 mmHg can be generated. This may allow entry of air through sites (venting needle holes, the interstices of the graft and suture lines) that are otherwise airtight and generate systemic air embolism.

When severe right ventricular failure occurs, right ventricle is depicted as enlarged in the four-chamber view and left ventricular chamber may collapse around the pump inflow cannula with severe decrease of LVAD flow. In some cases of RV failure you can observe mitral valve in fixed open position. Severe tricuspid regurgitation may develop as a consequence of right ventricular dilation and the severe leftward shift of the septum results in a further decreased septal systolic contribution to RV ejection.

Strict treatment protocols need to be instituted early to optimize volume replacement therapy and titrate pharmacologic support. Our experience suggests as standard therapy high dosage of adrenaline (0.06-0.14 mcg/Kg/min) and/or milrinone to sustain right ventricle contractility, and the use of selective pulmonary vasodilators (inhaled nitric oxide 20-30 ppm) to improve trans-pulmonary blood flow. It may be necessary to optimize dosage of systemic vasodilators and LVAD flow to contain venous return to the insufficient right ventricle.

We consider right ventricular failure "extreme" requiring right ventricular assist device (RVAD) when transpulmonary flow is unable to ensure LVAD filling, despite maximal dosage of inotropic drugs and selective pulmonary vasodilators. If right ventricular support is required after LVAD starting, TEE has to guide cannulas placement. The echocardiography has to ascertain that the tip of inflow cannula is in the center of the right atrium without obstruction by atrial septum or tricuspid valve. The outflow cannula is introduced into the main pulmonary artery through the right ventricle

outflow tract and the pulmonary valve with off-pump technique. The tip of the outflow cannula must lie 1.5-2.0 cm beyond the pulmonary valve plane.
RV mechanical support improves organ function by increasing both cardiac output and return to the left atrium and ventricle, as well as by relieving venous congestion. RV assistance has to be continued until the patient displays signs of recovery: stable decrease in central venous pressure and pulmonary artery diastolic pressure, increase of urine output and recovery of organ function.

c) postoperative evaluation during ICU course

The main determinant of haemodynamic instability in the early phase of intensive care management is low pump flow rates. TEE is used to rule out mechanical and circulatory complications. The differential diagnoses include hypovolemia, tamponade, RV failure, and cannulae obstruction.
Because LVAD flow is volume dependent, appropriate preload must be maintained to ensure its adequacy. Extreme *hypovolemia* may generate occlusion of inflow cannula by left ventricular collapse around the inlet orifice and pump failure, as above described.
It can be very difficult to diagnose pericardial *tamponade* during LVAD support because the unusual physiology of supported LV make impossible standard Doppler assessment for tamponade and rarely cause classical clinical or echocardiographic features of tamponade. Moreover, collections may be loculated and cardiac tamponade can be determined by very small collections or a limited thrombus (for example substernal or posterior).
During postoperative phases it's frequent to observe *RV dysfunction*. Echocardiographic quantitative and qualitative daily assessment of RV function is crucial in optimizing volume replacement and titrating pharmacologic support. The gold standards during circulatory support are to warrant a good LV unloading with adequate pump flow but also to maintain RAP <10 mmHg. In fact, RAP is the only consistent haemodynamic value reported to be associated with end-organ failure and adverse outcome.

d) controls for suspected LVAD malfunction

Continuous flow LVADs use is becoming widespread so that durations of support are becoming extended and pump dysfunction will be increasingly prevalent. After the first 15 days of support the main causes of pump malfunction are consequent to thrombosis and thromboembolism. Investigators have described during LVAD support the induction of activated coagulation and fibrinolytic cascades despite normal coagulation values and platelet counts when a stress situation is imposed (such as infection, sepsis, ongoing bleeding, or an operation), resulting in thromboembolic events. This complication, described in patients with axial flow pump support, requires in some cases device replacement.
Premonitory signs of pump malfunction are increases in current and power as well as sudden and short episodes of decreases in flow rate. In our experience, three patients had pump failure due to thromboembolic material and they showed infection some days before pump failure.

In the event that the LVAD stops operating, it is imperative that the cause of pump malfunction must be identified and corrected as soon as possible and all attempts to restart the pump must be made immediately. Echocardiography allows to document the pump arrest as a retrograde flow from ascending aorta, through the ouflow cannula – pump – inflow cannula, to left ventricle. TEE is an invaluable tool to assess urgently cardiac function and LVAD components: it is mandatory to research inflow and outflow cannulae obstruction and any source of thromboembolic materials. Left atrial appendage and left ventricular apex must be examined carefully. It's possible to detect thrombotic material into the small "pocket" next to the LVAD inflow cannula orfice and interventricular septum-inferior wall. This area can be considered a "risk zone" for blood stagnation: in all patients pulsed wave Doppler interrogation with simple volume in this area shows very low blood velocity.
After TEE exclusion of thrombi in the left ventricle, in inflow and outflow cannulae, in the vascular graft or at the aortic anastomosis, and of other causes of pump malfunction, it is mandatory to suspect impeller thrombosis, to start heparin administration (resolution in 3 cases in our experience)

and eventually low-dose thrombolysis into left ventricle. This last approach eliminates the risks associated to high-dose systemic thrombolysis and no cases of peripheral neurologic deficit are reported.

Pump failure may be a consequence of an abnormal increase of systemic vascular resistances. In one case of our experience we observed a sudden pump failure on a 105th day of support during severe hypertensive crisis. Mean arterial pressure was >130 mmHg. In this case, TEE showed poor left ventricle empting by LVAD as indicated by rightward septum shift, mitral valve regurgitation (degree ++/IV or more) and minimal aortic valve opening; continuous wave Doppler interrogation documented continuous flow with an abnormal increased pulsatility (peak velocity >2.0 m/sec). We treated this hypertensive crisis with a short-acting vasodilator (nitroprusside) in order to maintain mean arterial blood pressure less than 80-85 mmHg until complete hemodynamic and pump recovery.

Short-term device

Indications for using short-term mechanical circulatory support devices generally occur in 3 broad categories: (1) postcardiotomy shock, in which patients having undergone a cardiac operation either cannot be weaned from cardiopulmonary bypass or develop low cardiac output in the postoperative period; (2) acute cardiogenic shock resulting from myocardial infarction; (3) various forms of cardiogenic shock or low cardiac output state caused by acute myocarditis.

Medical therapy with inotropic drugs and vasodilators when possible, and use of intra aortic balloon pump (IABP) is the first line treatment to provide cardiac output adequate to preserve end-organ function awaiting myocardial recovery. Mechanical support is the treatment of choice for acute low cardiac output state refractory to maximal medical treatment and IABP.

If recovery within several days or weeks is expected, intracorporeal LVAD designed primarily for long-term support (as bridge or destination therapy) should be avoided and it is better to use "short term" systems as first line mechanical support: extracorporeal membrane oxygenation (ECMO), intraventricular axial pump Impella recover (IR), or paracorporeal devices (Medos, Thoratec).

Short-Term devices: the characteristics of the pumps and the role of echocardiography

ECMO. The ECMO circuit consisted of a centrifugal pump which propelles blood through a hollow-fiber membrane oxygenator, an integrated heat exchanger and two cannulas, venous and arterial. The blood enters the pump and is accelerated through the actions of a rotating impeller. The flow rate is dependent on the available amount of blood to be pumped, pump revolutions per minute, and arterial pressure. Figure 2 shows ECMO circuit for a patient with fulminant myocarditis. ECMO is an ideal means of rescue as temporary support for patients with both right and left failure. It can work as "first line" short-term device: if recovery does not occur, it is always possible to switch toward a long-term mechanical support as bridge to recovery or transplantation ("short bridge to long bridge"). Cannulation can be central (right atrium to ascending aorta) or peripheral (femoral vein to femoral artery). ECMO has the advantage of easy and rapid setup without requiring sternotomy at bedside under local anesthesia and its cost is much lower than other devices.

TEE has an important role during peripheral ECMO implantation. Venous blood is drained by gravity from a large-bore cannula inserted in either the right internal jugular or the femoral vein and advanced so that its tip is near the right atrium. The echocardiography has to ascertain that the tip of the cannula is in the center of the right atrium and is not obstructed by the atrial septum or the tricuspid valve, and to exclude tip malposition in the superior vena cava or in the subclavian vein. During postimplant course echocardiographic monitoring has to prevent insufficient left ventricular unloading, left ventricular distension, echo contrast enhancement with blood stagnation and intracardiac clot formation. Inotropic drugs has to be reduced to decrease myocardial oxygen demand, but adequate dosage is necessary to maintain a slightly pulsatility. Left ventricular

distension can lead to severe mitral regurgitation, lung edema and compromise the recovery of the myocardium.

IMPELLA RECOVER: Impella Recover (IR) is a miniaturized axial endovascular pump powered by external batteries and driven by a software placed in an external console. The pump, placed across the aortic valve, aspirates blood from the LV cavity to the ascending aorta with a non-pulsatile flow up to 4.5-5.0 l/min. The pump can be surgically inserted directly into the femoral artery by means of a J guidewire or through a small vascular presthesis stitched on the ascending aorta al least 7 cm above the valve plane with off-pump technique. A percutaneous version of device is available from 2004. IR presents the following advantages: no cardio-pulmonary-bypass for implantation; a reduced blood-contact surface, thus minimizing the systemic inflammatory response; no systemic anti-coagulation; low cost. Disadvantages include limited blood outflow and short-term application.

The first goal of IR is "bridge-to-immediate survival". Despite sub-maximal flow support, in patients with border-line indication to traditional LVAD support (septic shock, fever, advanced hepatic or renal damage), minimizing the biological cost of procedure can result in hemodynamic improvement and organ function stabilization. Patients may subsequently be candidates for recovery, for emergent heart transplantation or for the application of more sophisticated devices.

Echocardiography has a role for preoperative anatomic and functional evaluation of the heart and for correct intraventricular positioning and function of device.

Specific items unique to IR 100 has to be applied: (1) morphologic study of aortic valve to detect stenosis that does not allow IR 100 positioning; (2) morphologic study of mitral valve to evaluate opening and closing motions with particular reference to the anterior mitral leaflet; (3) morphologic study of intraventricular septum to exclude obstructive hypertrophic cardiomyopathy; (4) evaluation of ascending aorta to exclude aneurysmal dilatations and atherosclerotic damages of the aortic wall; (5) complete evaluation of abdominal and thoracic aorta when the device is inserted into femoral artery.

When left ventricle is dilated (>120 ml), IR can work at the highest levels of performance without risk of malfunction due to contact with LV wall. During IR insertion, TEE with off-axis 100-120° views allows: (1) to visualize device insertion through aortic valve to avoid positioning into Valsalva Sinuses; (2) to verify that the tip of the device reaches anterior mitral valve leaflet; (3) to perform measurements for definitive positioning after checking adequacy of VAD's position. The tip of endoventricular IR must be located 3.0-3.5 cm far from the aortic valve plane; (4) to detect blood flow through the device by means of color Doppler. The turbulence caused by blood flow output from the pump into the ascending aorta must remain far (1.5-2.0 cm) from Valsalva sinuses.

During postoperative period echocardiography allows to detect the most common reasons for hemodynamic instability in ICU: hypovolemia, RV dysfunction, and cardiac tamponade. All the above mentioned events can be well documented by transesophageal and transthoracic examination. During circulatory support echocardiography has an adjunctive role to control its correct endoventricular position as displacement is a possible cause of LVAD malfunction.

PARACORPOREAL DEVICES. Medos VAD (Medos, Stolberg, Germany), **Thoratec VAD**, **Abiomed VAD** are paracorporeal systems configured for univentricular or biventricular support. In paracorporeal device the pump (one for each ventricle) is positioned on the external abdominal wall with cannulae tunnelled subcostally into the mediastinum. For left ventricular support, the inflow cannula can be placed in the left ventricular apex or the left atrium and the outflow cannula is anastomosed to the ascending aorta. For right ventricular support, the inflow cannula is placed in the right atrium or ventricle and the outflow cannula in the main pulmonary artery. Mechanical bileaflet valves within the inflow and outflow conduits ensure unidirectional pulsatile blood flow. An external drive console sends pressurized air to the pump, which compresses the blood sac and causes blood flow ejection. The role of echocardiography in the positioning and management of paracorporeal devices shows the same aspects of intracorporeal devices with or without RVAD.

Short-term devices: echocardiographic parameters to evaluate myocardial recovery

All patients undergoing short mechanical circulatory support should be systematically evaluated for evidence of myocardial recovery.
The assessment of myocardial recovery and the decision of LVAD explantation has to be based on a combination of clinical, hemodynamic and echocardiographic parameters: the criteria for short-term support weaning included mixed venous oxygen saturation greater than or equal to 70%, good peripheral perfusion, stable hemodynamics with low dose inotropic support, and echocardiographic determination of the absence of tamponade, right and left ventricular distension, or presence of intracardiac clot.
Weaning off short term support is usually not attempted in the first 3 days. The mechanical blood flow has to be gradually reduced with gradual reloading of the ventricles under TEE monitoring and removed within 24-36 hours if hemodynamics remained stable, left ventricular ejection fraction of greater than or equal to 40-45%, RVFAC greater than or equal to 40%. During weaning pharmacologic support is titrated as necessary.

SUGGESTED READINGS

1. Vitali E, et al. Different clinical scenarios for circulatory mechanical support in acute and chronic heart failure. Am J Cardiol 2005;96:34L-41L.

2. Goldstein DJ. Worldwide experience with the Micromed DeBakey ventricular assist device as a bridge to transplantation. Circulation 2003;108:272-277.

3. Catena E, et al. Echocardiography and cardiac assist devices. Minerva Cardioangiol 2007;55:247-65.

4. Chumnanvej S, et al. Perioperative echocardiographic examination for ventricular assist device implantation. Anesth Analg 2007;105:583-601.

5. Scalia GM, et al. Clinical utility of echocardiography in the management of implantable ventricular assist device. J Am Soc Echocardiogr 2000;13:754-63.

6. Catena E, et al. Left ventricular support by axial flow pump: the echocardiographic approach to device malfunction. J Am Soc Echocardiogr 2005;18:1422e7-1422e13.

7. Garatti A, et al. Impella recover 100 microaxial left ventricular assist device: the Niguarda experience. Transplant Proc 2004;36:623-626.

8. Catena E, et al. Echocardiographic approach in a new left ventricular assist device: Impella recover 100. J Am Soc Echocardiogr 2004;17:470-473.

9. Acker M. mechanical circulatory support for patients with acute fulminant myocarditis. Ann Thorac Surg 2001;71:73-6.

10. Catena E, et al. Mechanical circulatory support for patients with fulminant myocarditis: role of echocardiography to address diagnosis, choice of device, management, and recovery. J Cardiothorac Vasc Anesth 2008 “in press”.

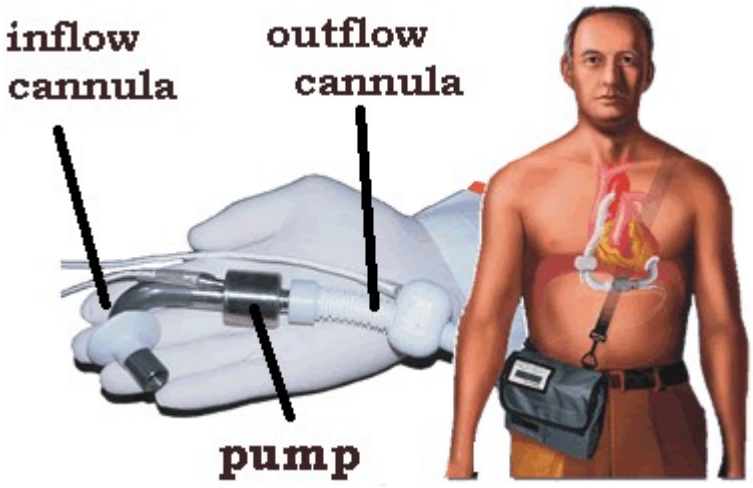

inflow
cannula
outflow
cannula
pump

Fig 1

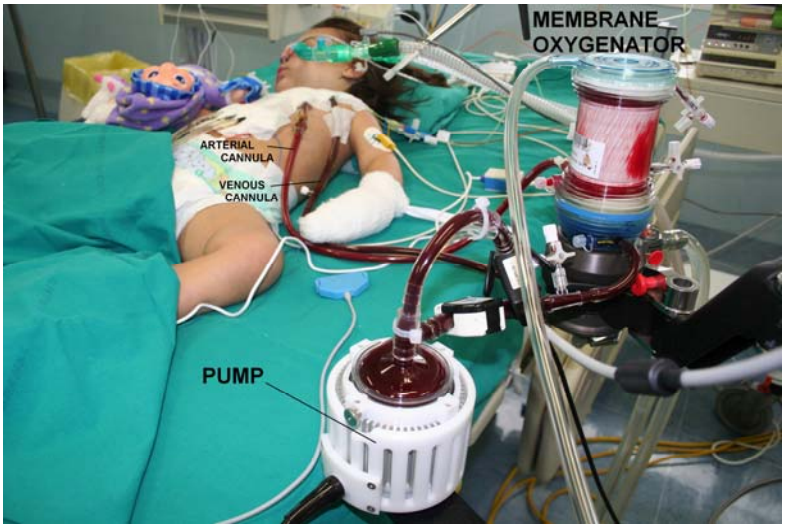

MEMBRANE
OXYGENATOR
ARTERIAL
CANNULA
VENOUS
CANNULA
PUMP

Fig 2

How to create an Echo report

Heinz D. Tschernich
Clinical Department of Cardiothoracic and Vascular Anaesthesia & Intensive Medicine, Medical University of 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

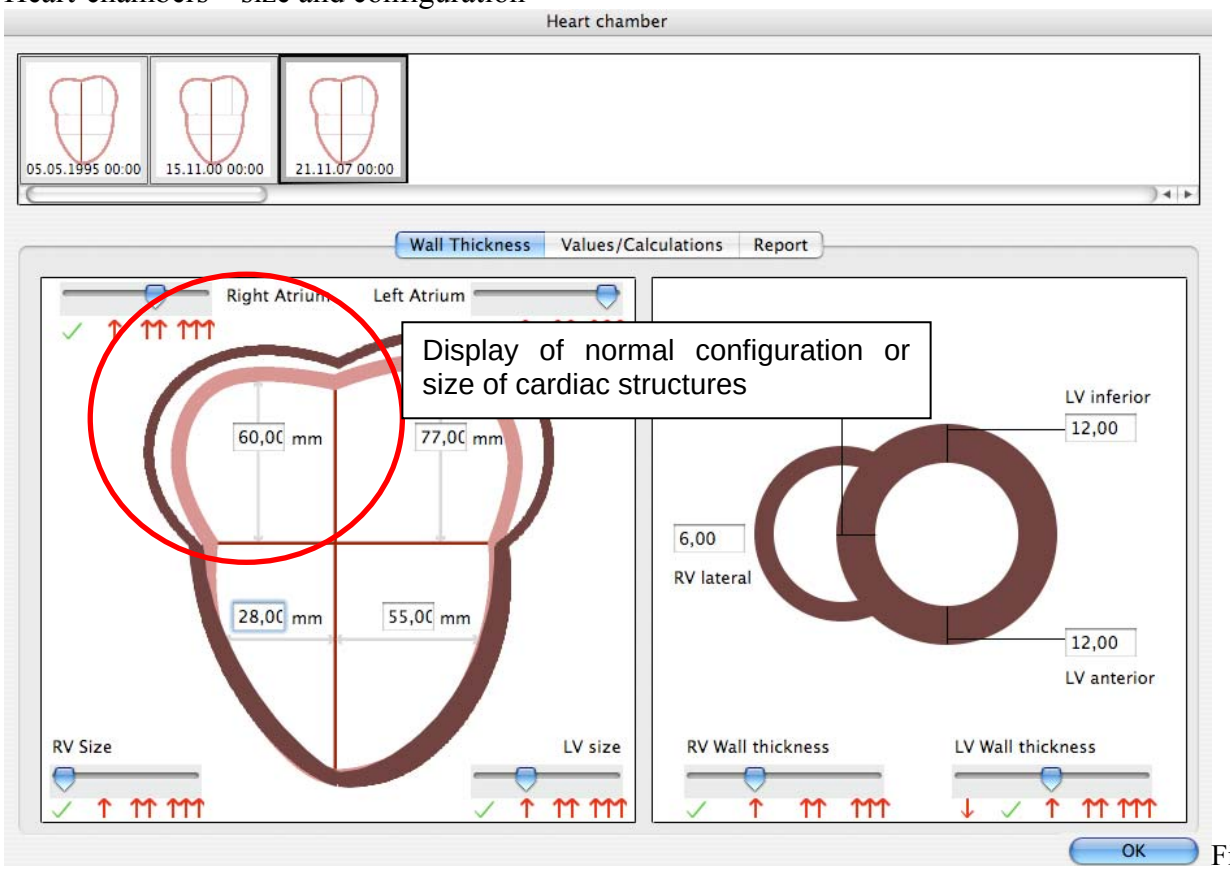


Fig. 1

Reporting by TEE-planes – ME 4CH

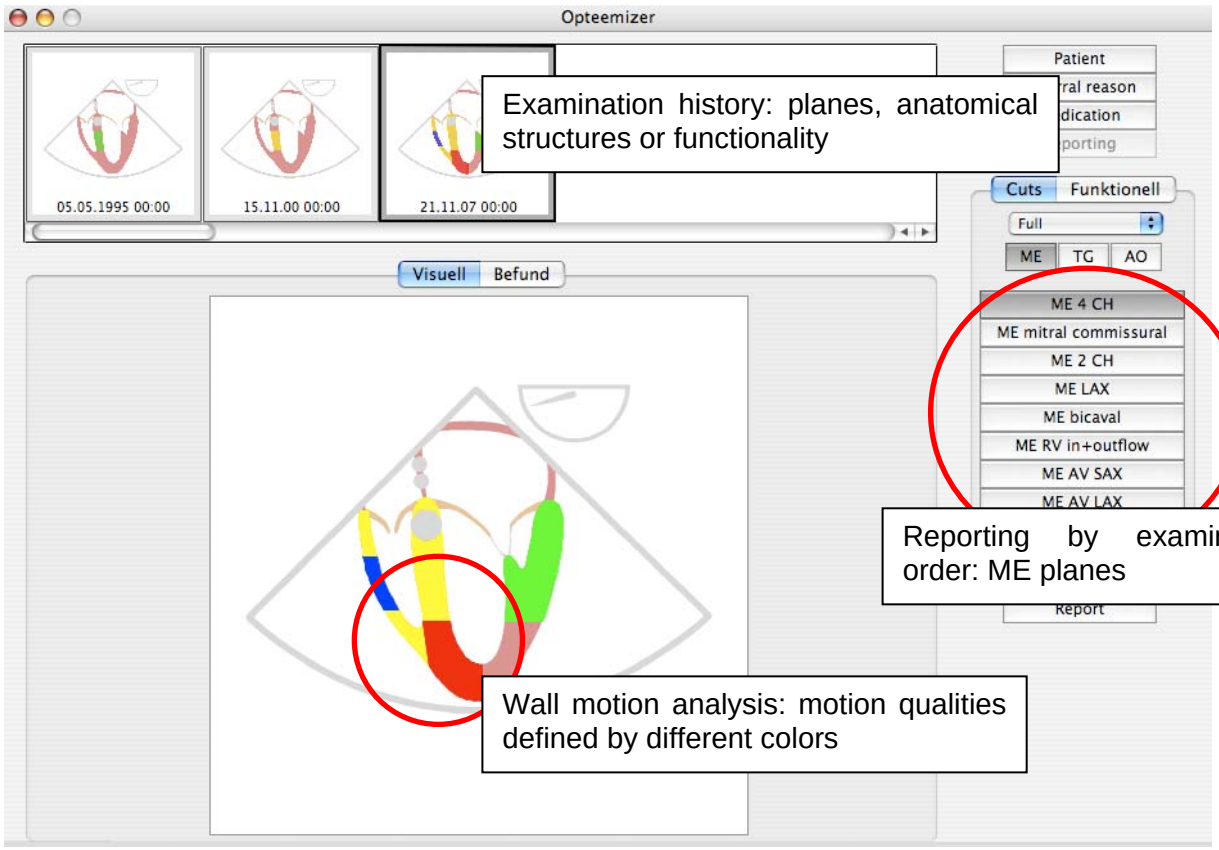


Fig. 2

Mitral valve

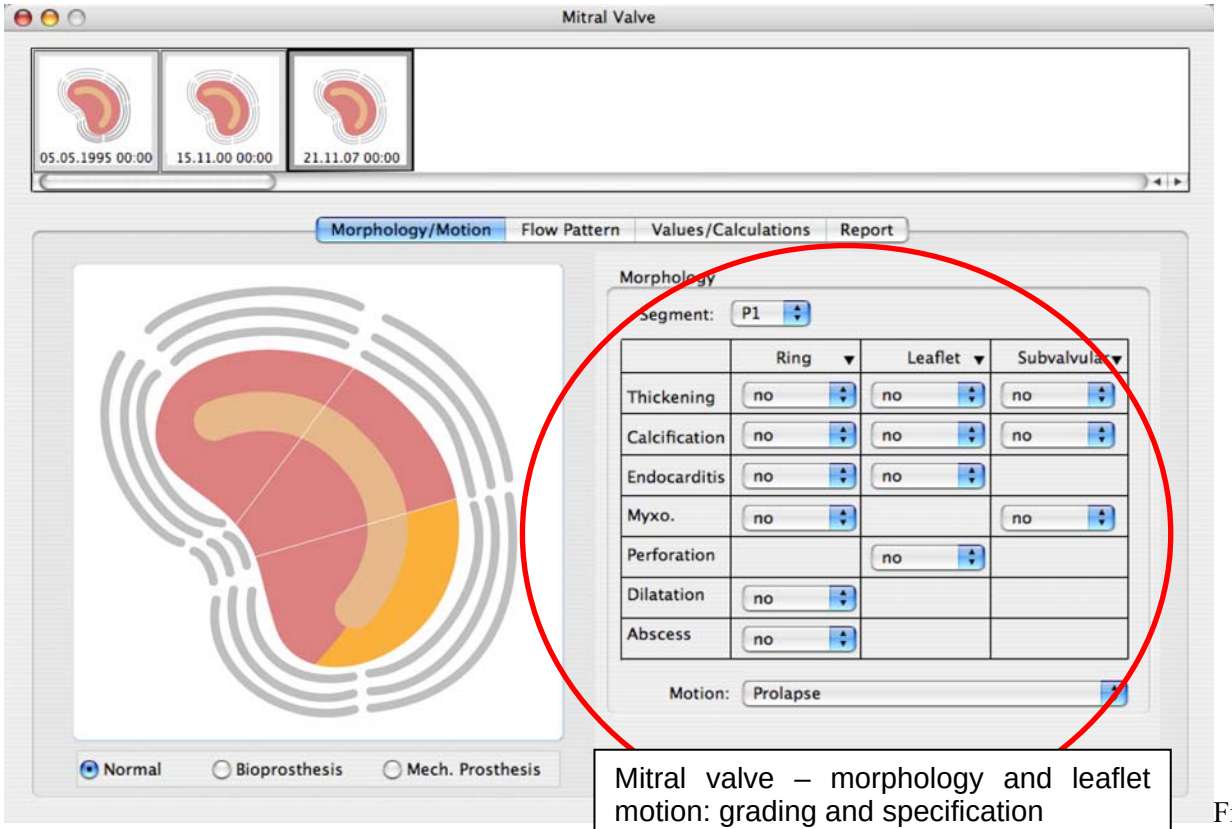


Fig. 3

Tricuspid Valve

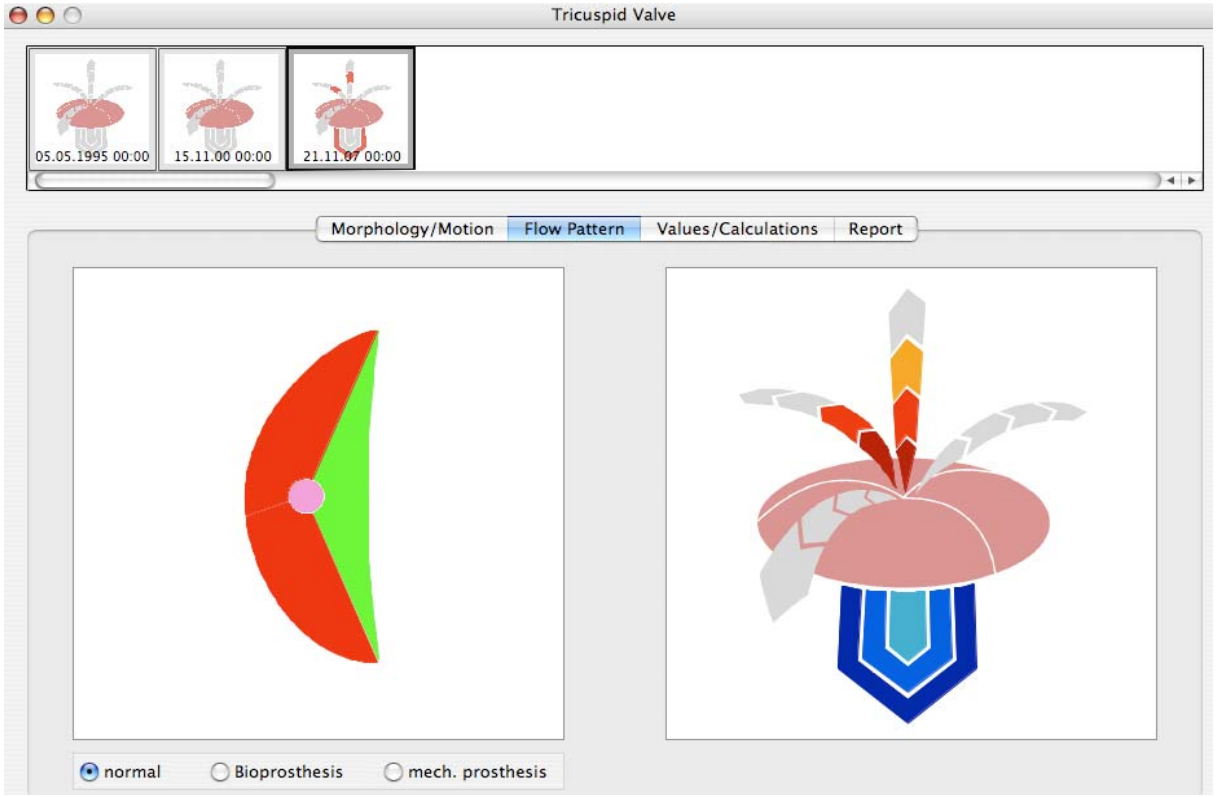


Fig. 4
Aorta

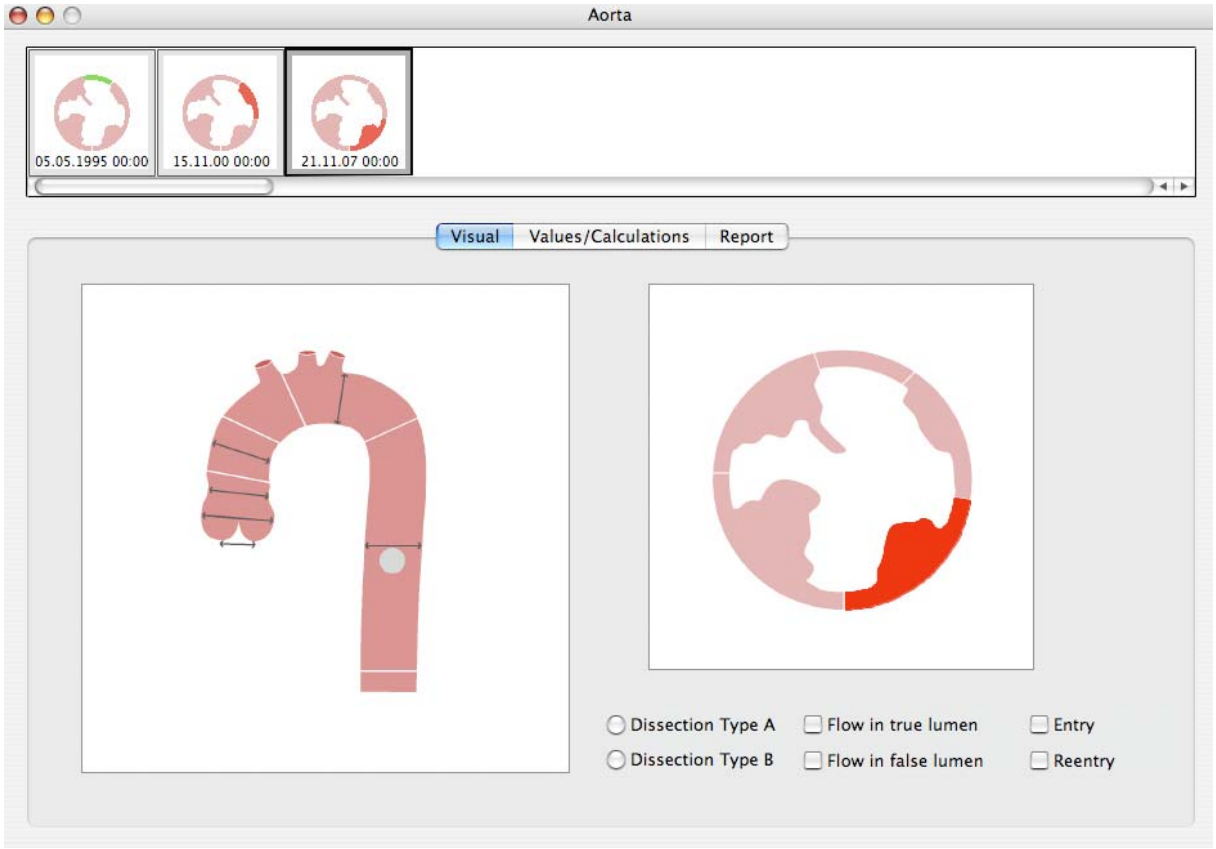


Fig. 5

Notes: