Tutorial 1 - Basic physics of ultrasound and the Doppler phenomenon

BASIC PHYSICS OF ULTRASOUND AND THE DOPPLER PHENOMENON

Medical ultrasound imaging consists of using high pitched sound bouncing off tissues to generate images of internal body structures.

Frequency

Frequency refers to the number of cycles of compressions and rarefactions in a sound wave per second, with one cycle per second being 1 hertz. While the term ultrasound generally refers to sound waves with frequencies above 20,000 Hz (the frequency range of audible sound is 20 to 20,000 Hz), diagnostic ultrasound uses frequencies in the range of 1-10 million (mega) hertz.

Wavelength

The wavelength is the distance traveled by sound in one cycle, or the distance between two identical points in the wave cycle i.e. the distance from a point of peak compression to the next point of peak compression. It is inversely proportional to the frequency. Wavelength is one of the main factors affecting axial resolution of an ultrasound image. The smaller the wavelength (and therefore higher the frequency), the higher the resolution, but lesser penetration. Therefore, higher frequency probes (5 to 10 MHz) provide better resolution but can be applied only for superficial structures and in children. Lower frequency probes (2 to 5MHz) provide better penetration albeit lower resolution and can be used to image deeper structures.

Propagation velocity

The propagation velocity is the velocity at which sound travels through a particular medium and is dependant on the compressibility and density of the medium. Usually, the harder the tissue, the faster the propagation velocity. The average velocity of sound in soft tissues such as the chest wall and heart is 1540 metres/second.

ULTRASOUND TISSUE INTERACTIONS

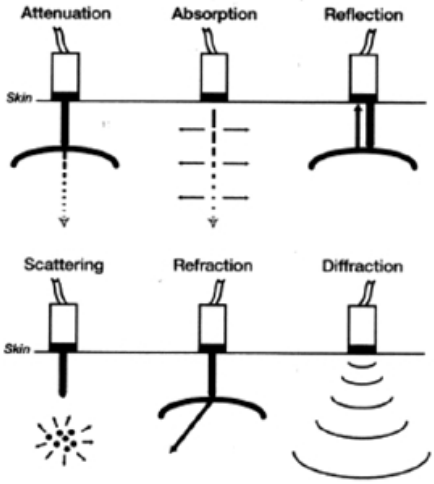

Figure 1: Interactions of Ultrasound with tissue: Echocardiography, Bonita Anderson, Dutoit, Wiley-Blackwell

The interaction of ultrasound waves with organs and tissues encountered along the ultrasound beam can be described in terms of attenuation, absorption, reflection, scattering, refraction and diffraction.

Attenuation

Sound energy is attenuated or weakened as it passes through tissue because parts of it are reflected, scattered, absorbed, refracted or diffracted.

Reflection

A reflection of the beam is called an echo and the production and detection of echoes forms the basis of ultrasound. A reflection occurs at the boundary between two materials provided that a certain property of the materials is different. This property is known as the acoustic impedance and is the product of the density and propagation speed. If two materials have the same acoustic impedance, their boundary will not produce an echo. If the difference in acoustic impedance is small, a weak echo will be produced, and most of the ultrasound will carry on through the second medium. If the difference in acoustic impedance is large, a strong echo will be produced. If the difference in acoustic impedance is very large, all the ultrasound will be totally reflected. Typically in soft tissues, the amplitude of an echo produced at a boundary is only a small percentage of the incident amplitudes, whereas areas containing bone or air can produce such large echoes that not enough ultrasound remains to image beyond the tissue interface.

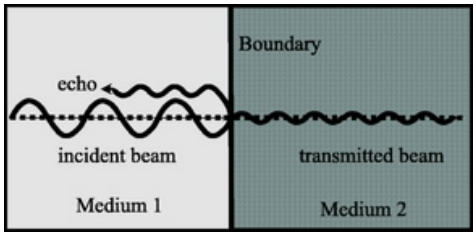

Figure 2: Production of an echo depending on relative acoustic impedances of the two media: From: Aldrich: Crit Care Med, Volume 35(5) Suppl.May 2007.S131-S137

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Boundary	% Reflected
Fat/ muscle	1.08
Fat/kidney	0.6
Soft tissue/water	0.2
Bone/fat	49
Soft tissue/air	99

Table 1: Percentage reflection of ultrasound at boundaries: From: Aldrich: Crit Care Med, Volume 35(5) Suppl.May 2007.S131-S137

At a tissue–air interface, 99% of the beam is reflected, so none is available for further imaging. Transducers, therefore, must be directly coupled to the patient's skin without an air gap. Coupling is accomplished by use of gel between the transducer and the patient.

Scattering

Not all echoes are reflected back to the probe. Some of it is scattered in all directions in a non-uniform manner. This is especially true for very small objects or rough surfaces. The part of the scattering that goes back to reach the transducer and generate images is called backscatter.

Absorption

Tissue absorption of sound energy contributes most to the attenuation of an ultrasound wave in tissues.

Refraction

The change in the direction of a sound wave on being incident upon a tissue interface at an oblique angle is refraction and is determined by Snell's law. Follow this link for an explanation of this law: **http://www.ndt-ed.org /EducationResources/CommunityCollege/Ultrasonics/Ph...**

TRANSDUCERS

Inside the core of the transducer are a number of peizo-electric crystals that have the ability to vibrate and produce sound of a particular frequency when electricity is passed through them. This is how ultrasound waves are formed. These transducers also act as receivers for the reflected echoes as they generate a small electric signal when a sound wave is incident upon it.

Duty factor

In most modes of ultrasound operation, only 1% of the time is spent in generating a pulse of ultrasound waves and 99% of the time is then spent listening for the echoes. This is called the duty factor…1% in such a case.

Pulse repetition frequency (PRF)

The PRF is the number of pulses (send and listen cycles) of ultrasound sent out by the transducer per second. It is dependent on the velocity of sound and on the depth of tissue being interrogated. The deeper the tissue being examined, the longer the transducer has to wait for echoes to come back, hence a lower PRF.

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Beam

The ultrasound beam is focused by the transducer so as to be as close to a flat plane as possible. The beam is made up of tens to hundreds of scan lines.

Orientation

There is usually a dot, groove or light on one ends of the transducer to assist orientation. A corresponding marking is also displayed on the screen to help give an orientation to the images.

Resolution

Axial resolution:The ability to resolve objects in the line of the ultrasound beam. Factors affecting axial resolution include Spatial Pulse Length (SPL) and frequency.

Lateral resolution: Resolution at 90° to the direction of the beam. Factors affecting lateral resolution are width of the beam, distance from the transducer, frequency, side and grating lobe levels.

Temporal resolution: Refers to the ability to detect moving objects in the field of view in their true sequence. The number of frames generated per second (frame rate) determines temporal resolution.

DOPPLER EFFECT

Doppler shift is given by the following formula:

$F_D = (V \times F_O) / C$

where

F_D is the Doppler shift

F_O is the original frequency

V is the velocity of blood

C is the speed of sound in tissues

Therefore $V = (F_D \times C) / F_O$

A further refinement of this formula is: $V = (F_D \times C) / (2F_O \times \cos\theta)$

The original frequency is multiplied by 2 because the Doppler shift occurs twice – when the original wave is incident on the moving RBC and when the moving RBC reflects it back. Cosine theta (Cosθ) is applied as a correction for the angle between the ultrasound beam and the direction of blood flow. Cosθ = 1 if the beam is parallel to the direction of blood flow and maximum velocity is measured. Cosθ = 0 if the beam is perpendicular to the direction of blood flow and zero velocity is measured

It is noteworthy that for Doppler, maximum velocity information is obtained with the ultrasound beam aligned parallel to the direction of the blood flow being studied. Otherwise the peak velocity and consequently the pressure gradient (see below) will be underestimated. This is in sharp contrast to conventional echo where the best image quality is obtained with the ultrasound beam aligned perpendicular to the structure being studied.

Since the original frequency value ($2F_O$) is in the denominator in the equation, it is important to remember that maximum velocity information is obtained using low frequencies (usually 2 MHz). This is in contrast to conventional 2-D echo where higher frequencies deliver higher resolutions.

There is a direct relationship between the peak flow velocity through a narrow valve and the pressure gradient across it. Understandably, when the valve orifice is small, blood flow has to accelerate in order to eject the same stroke volume. The smaller the orifice, the higher the acceleration and velocity. This

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increase in velocity can be measured using Doppler echo. The pressure gradient across the valve can be calculated using the simplified Bernoulli equation:

Pressure gradient = $4\ v^2$

Where 'v' is the peak flow velocity of the jet through the orifice.

This equation is frequently used during Doppler evaluation of stenotic valves, regurgitant lesions and intra-cardiac shunts. The velocity information provided by Doppler complements the anatomical information provided by M-mode and 2-D echocardiography.

Analysis of the returning Doppler signal not only provides velocity information but also information about flow direction. By convention, velocities towards the transducer are displayed above the baseline and velocities away from the transducer are displayed below the baseline.

The returning Doppler signal is a spectral trace of velocity against a time axis. The area under the curve (AUC) of this spectral trace is known as the Velocity Time integral (VTI)….also known variably as TVI or FVI (flow velocity integral). The value of the VTI is determined by peak flow velocity and ejection time and can be calculated by the processor of most echo machines.

ARTIFACTS

The following section on artifacts has been taken in toto from: Basic physics of ultrasound imaging; Critical Care Medicine - Volume 35, Issue 5 Suppl (May 2007)

Artifacts are errors in images. They are normally caused by physical processes that affect the ultrasound beam and that in some way alter the basic assumptions the operator makes about the beam. To understand artifacts, one needs to consider the basic assumptions made in producing an ultrasound image:

- Sound waves travel in straight lines.
- Reflections occur from structures along the central axis of the beam.
- Intensity of reflection corresponds to the reflector scattering strength
- Sound travels at exactly 1540 m/sec.
- Sound travels directly to the reflector and back.

These assumptions do not always hold true. There are numerous cases of exceptions to these assumptions. Although some of these artifacts may actually provide useful information or allow for novel interpretations, the majority are potential pitfalls that may confuse the examiner if not considered. The major artifacts encountered in critical care ultrasound are outlined below.

Reverberation

Reverberation artifacts appear as multiple equally spaced lines along a ray line. Reverberation is caused by the sound bouncing back and forth between tissue boundaries and then returning to the receiver.

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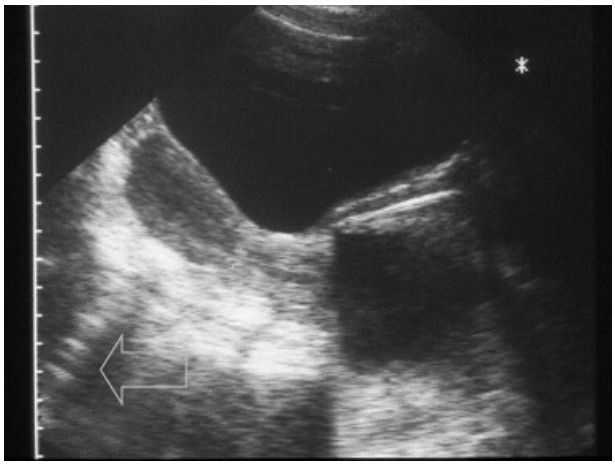

Figure 3: Example of a reverberation artifact.

Ring Down

Ring-down artifacts are produced when small crystals such as cholesterol or air bubbles resonate at the ultrasound frequency and emit sound. Because the sound is emitted after the transducer receives the initial reflection, the system thinks the emitted sound is coming from structures deeper in the body. Air bubbles in the abdomen are shown.

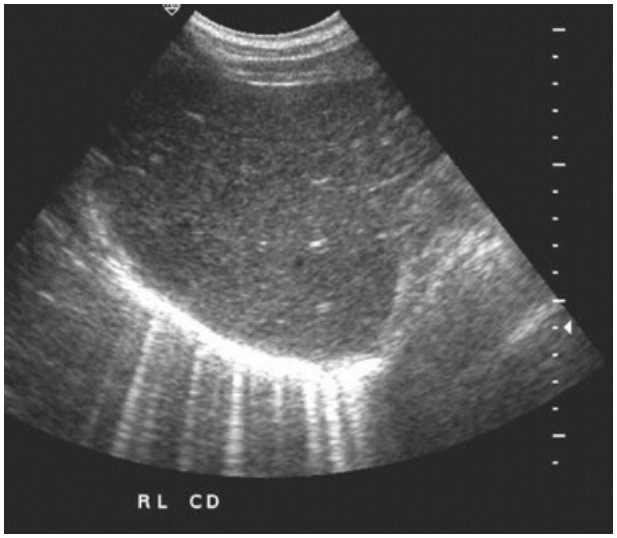

Figure 4: Example of a ring-down artifact.

Mirror Images

Sound can bounce off a strong, smooth reflector such as the diaphragm. The surface acts as mirror and reflects the pulse to another tissue interface. The ultrasound system believes the second interface is beyond the first surface, and this is where it appears on the scan. In Figure 5, the arrow shows the real object, which appears as if reflected in a mirror.

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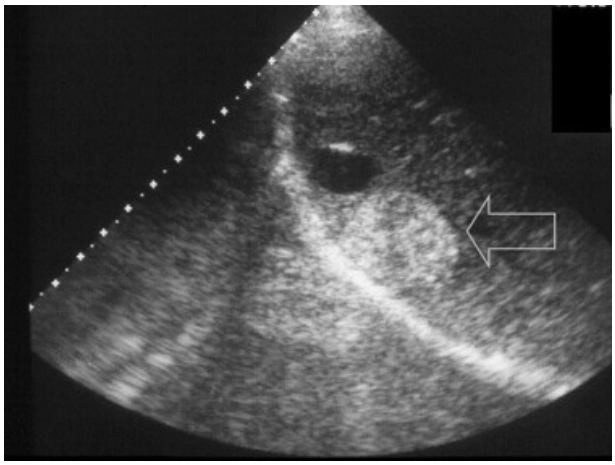

Figure 5: Example of a mirror-image artifact.

Reflections

Reflection is somewhat similar to the mirror image described above but has a very different appearance and is caused by multiple reflections. Sound can bounce off a strong, smooth reflector, such as the posterior bladder wall, and be reflected back to the transducer, giving the appearance of the structure deep to the bladder wall as would be seen with fluid collection.

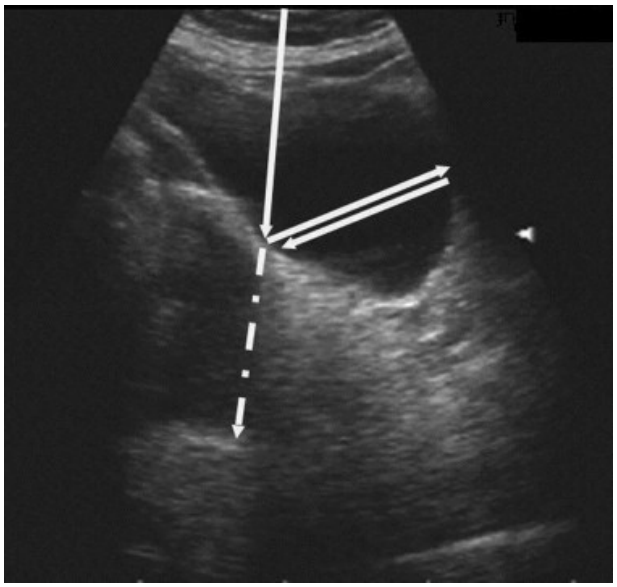

Figure 6: Example of a reflection artifact.

Enhancement

Enhancement is seen as an abnormally high brightness. This occurs when sound travels through a medium with an attenuation rate lower than surrounding tissue. Reflectors at depths greater than the weak attenuation are abnormally bright in comparison with neighboring tissues. Enhancement of tissues (See arrow in Fig. 7 below) deeper than cysts or ducts is common. The attenuation of the sound through the fluid in these tissues is less than that of the surrounding tissues and results in this abnormal brightness. The tissues deeper than the gallbladder show abnormal brightness.

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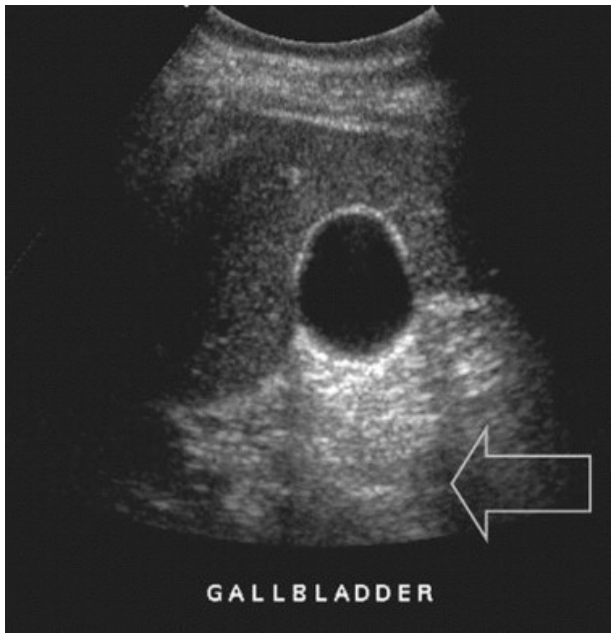

Figure 7: Example of an enhancement artifact.

Attenuation

Tissues deeper than strongly attenuating objects, such as calcification, appear darker because the intensity of the transmitted beam is lower. In the scan of the gallbladder in Figure 8 below, the left side shows enhancement as described above; the right side shows decreased beam intensity because of attenuation in calcified gallstones (arrow).

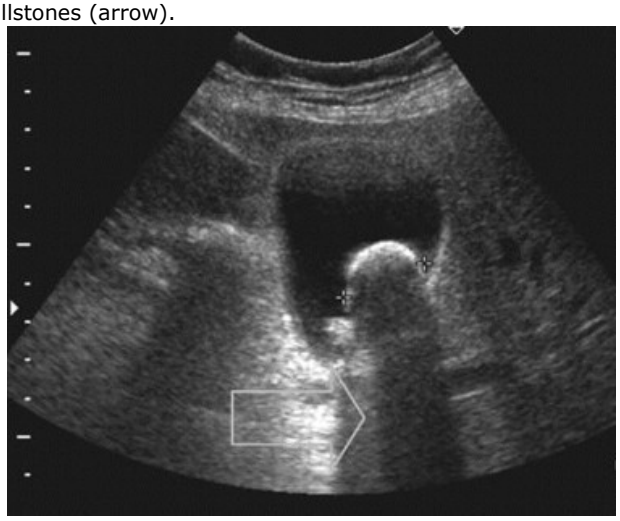

Figure 8: In this scan of the gallbladder, the left side shows enhancement described above; the right side shows a hypoechoic shadow distal to a brightly echogenic stone

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Tutorial 2 - Modes of Ultrasound

MODES OF ULTRASOUND

The principal modes of ultrasound in echocardiography are

1.2-D or 2 dimensional mode
2.M-mode or motion mode
3.Colour flow doppler imaging
4.Pulse wave doppler
5.Continuous wave doppler
6.Tissue doppler

2D

This is the default mode that comes on when any ultrasound / echo machine is turned on. It is a 2 dimensional cross sectional view of the underlying structures and is made up of numerous B-mode (brightness mode) scan lines. This is the most intuitive of all modes to understand. The field of view is the portion of the organs or tissues that are intersected by the scanning plane. Depending on the probe used, the shape of this field could be a sector - commonly seen with Echo and abdominal ultrasound probes or rectangular or trapezoid - seen with superficial or vascular probes.

Multiple images of the field or frames are generated every second on the screen, giving an illusion of movement. A frame rate of at least 20 frames per second is needed to give a realistic illusion of motion.

On a grey scale, high reflectivity (bone) is white; low reflectivity (muscle) is grey and no reflection (water) is black. Deeper structures are displayed on the lower part of the screen and superficial structures on the upper part.

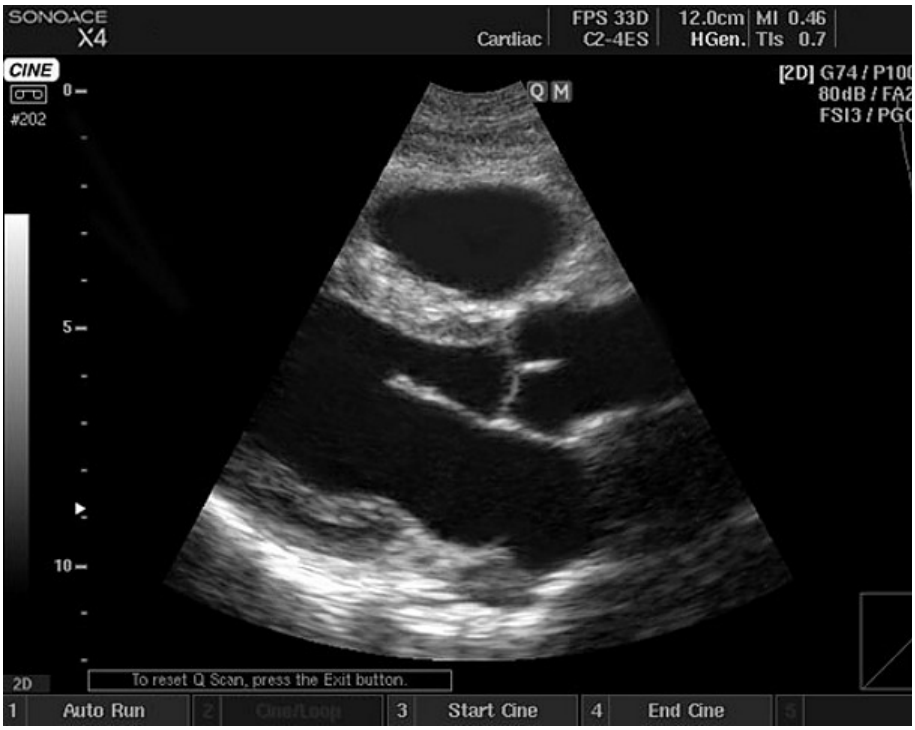

Figure 1: An example of 2D imaging: From www.medison.ru/uzi/eho408.htm

The main uses for 2-D mode are to measure cardiac chamber dimensions, assess valvular structure & function, estimate global & segmental ventricular systolic function, and improve accuracy of interpretation of Doppler modalities.

While this mode is useful to accurately represent the 2- dimensional structure of the underlying tissues, it does not resolve rapid movements well and may misrepresent 3-dimensional nature of structures.

If you would like some more information about 2D, the section on 2-Dimensional imaging at **http://folk.ntnu.no/stoylen/strainrate/Ultrasound/** is very informative with excellent graphical explanations.

M-mode

This represents movement of structures over time. Initially a 2-D image is acquired and a single scan line is placed along the area of interest. The M-mode will then show how the structures intersected by that line move toward or away from the probe over time.

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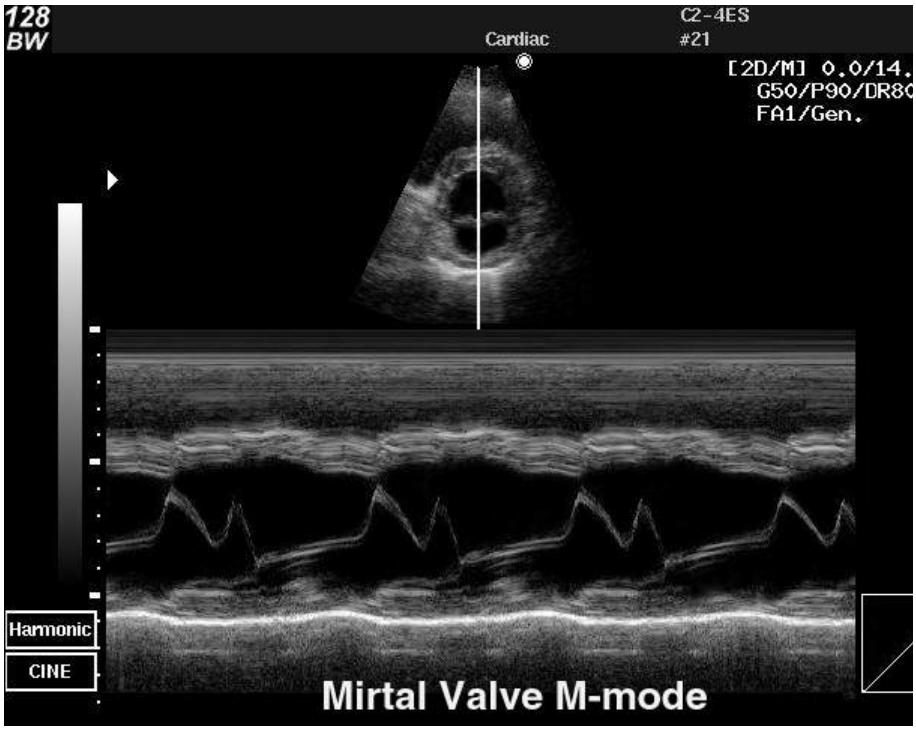

Figure 2: An example of M Mode showing movements of the mitral leaflets over time: http://www.medison.ru/uzi/eho36.htm

The M-mode has good temporal resolution, so it is useful in detecting and recording rapid movements. We can also correlate and time events with ECG or respiratory pressure waveforms traced alongside the M-mode tracings. The M-mode is commonly used for measuring chamber dimensions and calculating fractional shortening and ejection fraction.

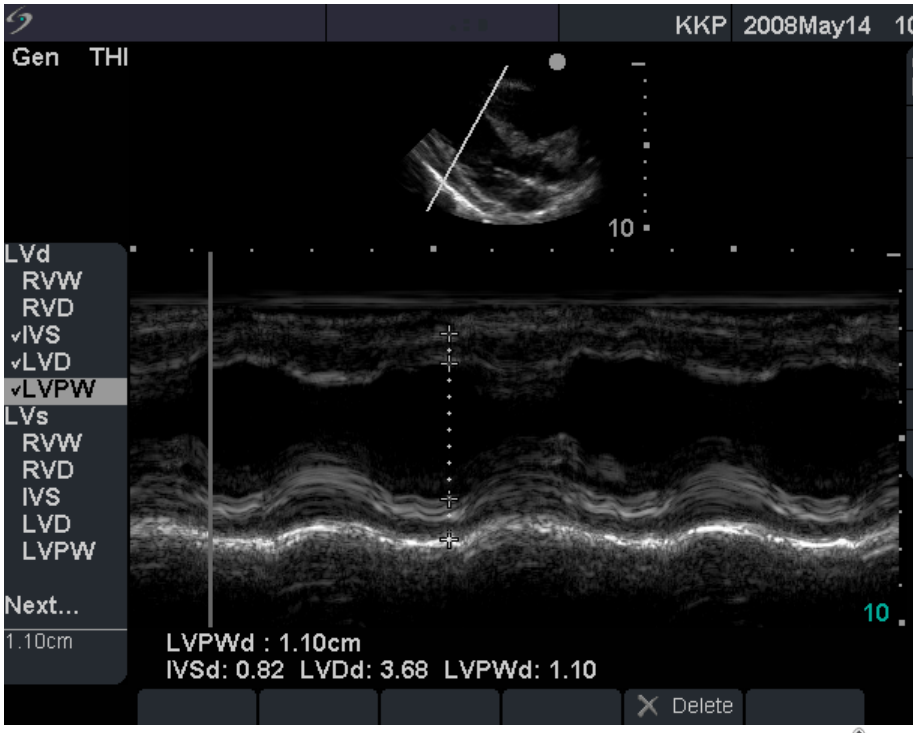

Figure 3: Another example of M mode through the left ventricle showing movement of the walls over time

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Colour flow Doppler imaging (CFI)

In this mode, the velocity and direction of blood flows are depicted in a color map superimposed on the 2-D image.

It uses pulse wave Doppler signals to derive this image. This is usually done with lower frequency ultrasound waves and hence the resolution of the 2-D image deteriorates in this mode. As it takes many pulses in each scan line to derive the color image, the frame rate is reduced compared to 2-D mode. Reducing the depth and size of the color box and reducing the scanning sector width can compensate for this.

Although it can be changed, by convention, blood flowing away from the probe is depicted in blue and that flowing toward the probe in red. (BART: blue away, red toward). Blood flowing perpendicular to the scanning plane will appear black. Areas of turbulent flow may be depicted in green or white.

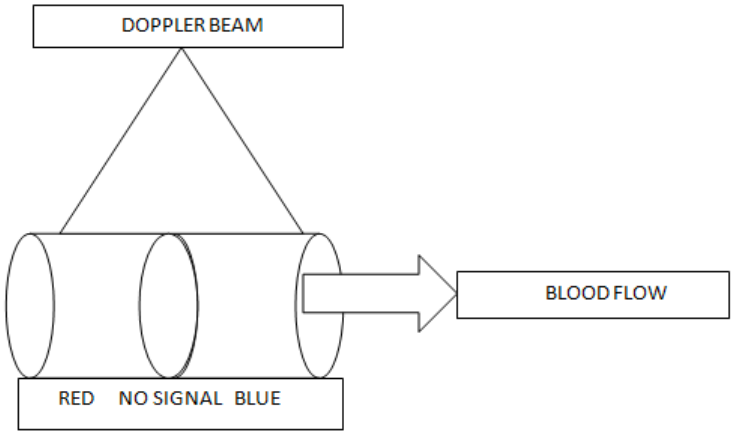

Fig 4: Schematic diagram of Colour Doppler: The mid-zone has no signal because the beam is perpendicular to flow. The conventional colour codes are – BART: Blue Away, Red Towards, but can be changed.

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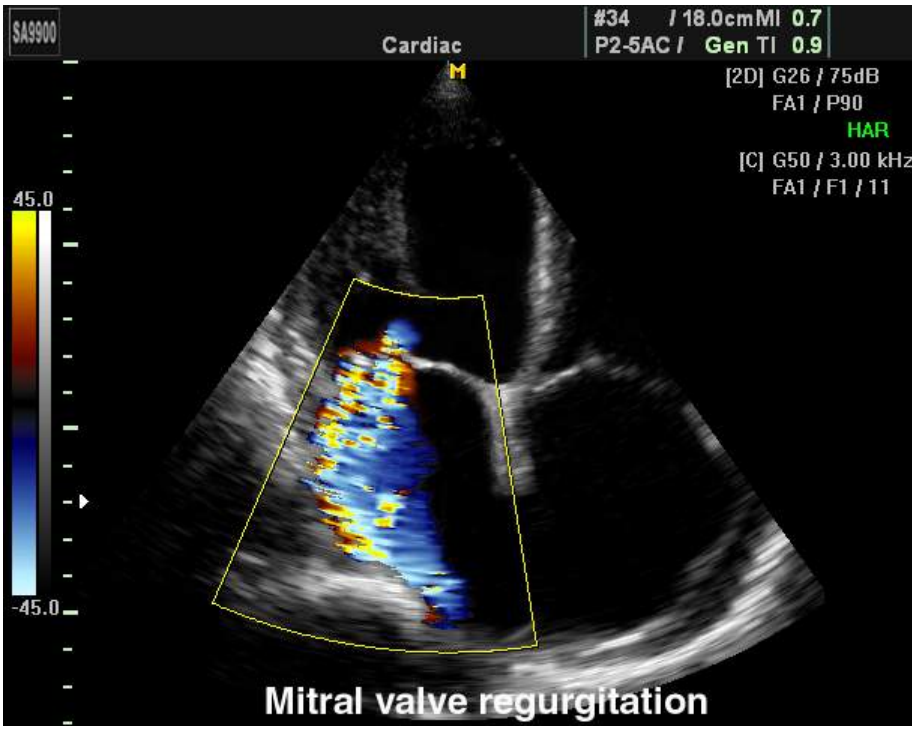

Figure 5: An example of Color Flow Imaging of a mitral regurgitation jet:
http://www.medison.ru/uzi/eho187.htm

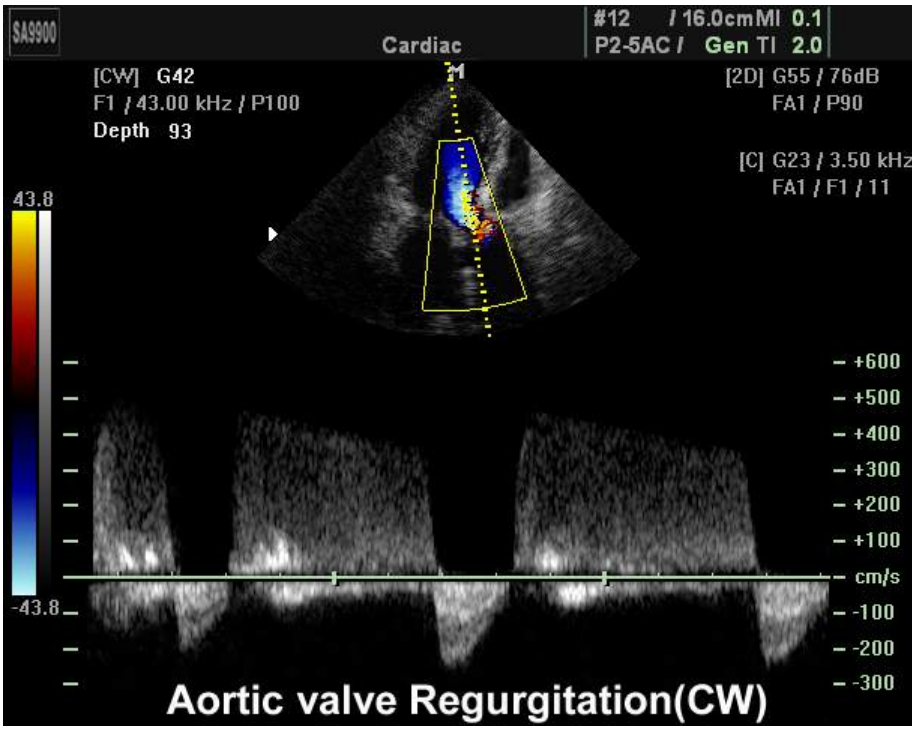

Figure 6: Using Colour flow to guide CWD in aortic regurgitation:
http://www.medison.ru/uzi/eho189.htm

Color flow imaging tells us about intra-cardiac blood flows in relation to the anatomy. Hence it is useful in visualizing and semi-quantitatively assessing regurgitant jets and other abnormal flows. It can also be used to guide the accurate placement of the cursor

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for pulse and continuous wave Dopplers.

In addition to the poor 2D resolution, the reduced frame rate also reduces temporal resolution. Estimates of velocity and direction of blood flow are not as accurate as in Continuous wave or pulse wave Dopplers.

Pulse wave Doppler (PWD)

This is a pulsed Doppler technique in which the Doppler signal arising from a specific position in the scanned tissue is analyzed to depict velocity and direction of flow.

The transducer crystal transmits the ultrasound and receives it after a preset delay. This allows it to precisely localize the site of origin of a velocity signal. For this, a cursor or 'sample volume' is placed over the 2-D image at the region of interest.

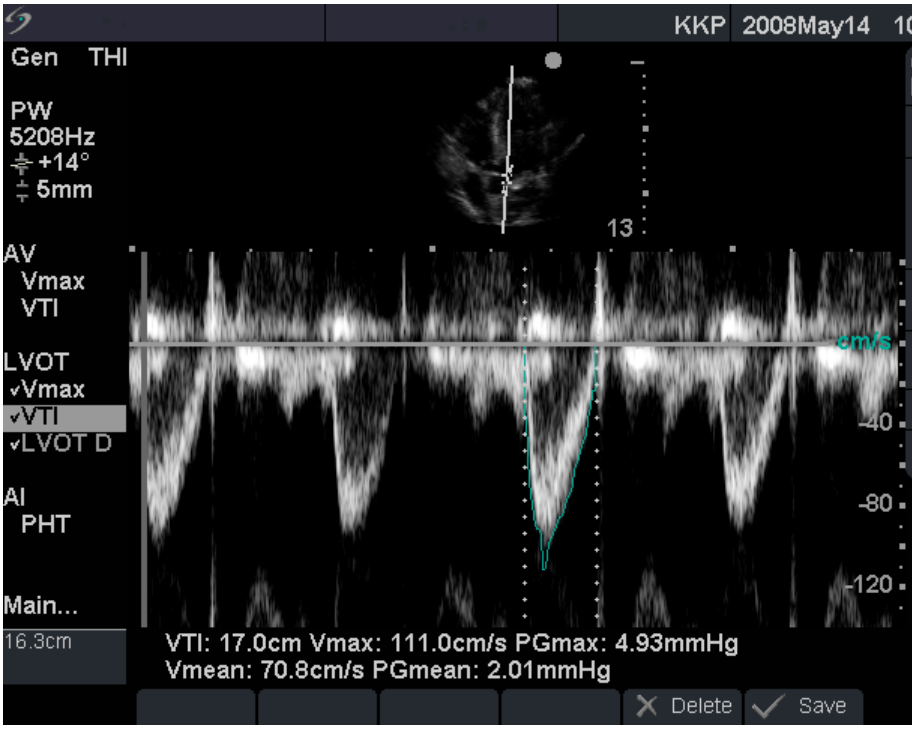

Figure 7: An example of Pulsed Wave Doppler of normal flow through the left ventricular outflow tract

The PWD also gives us information about the nature of flow-laminar or turbulent. In laminar flows, since most of the RBCs are traveling at the same velocity, the Doppler waveform has a thick white edge but is black within. In turbulent flow - e.g. across a stenotic valve, there is a wide distribution of RBC velocities and the Doppler signal appears filled-in. This is known as spectral broadening.

A key disadvantage with PWD is the inability to measure high velocities accurately. High velocities result in a phenomenon called 'aliasing'. This causes the velocity waveform to wrap around both sides of the baseline. Direction and velocity information cannot be interpreted for an aliased waveform.

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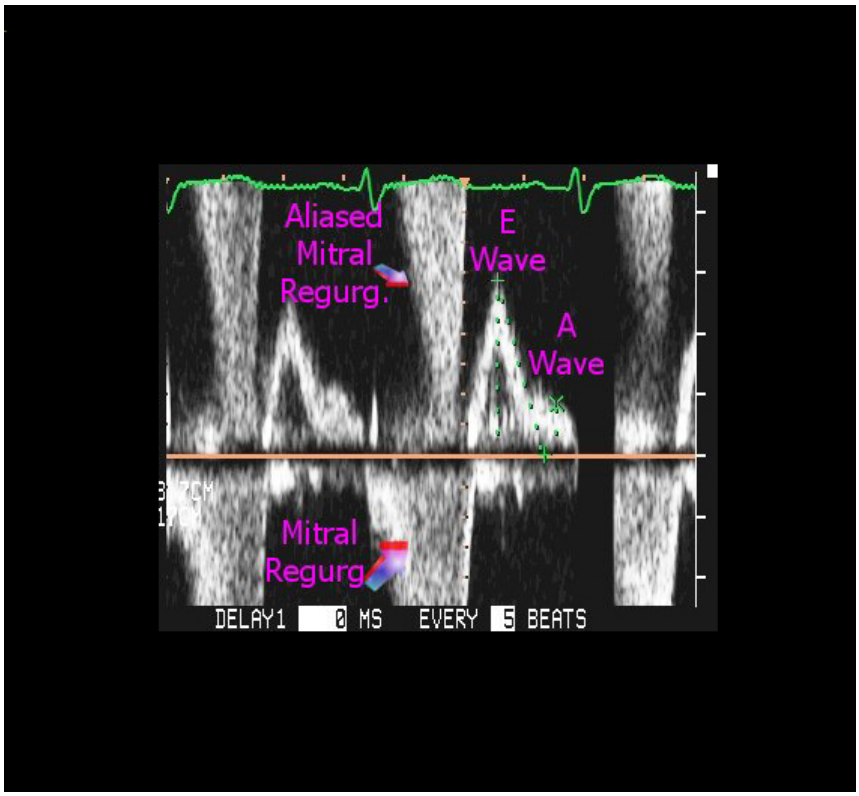

Figure 8: This Pulsed Wave Doppler of mitral inflow shows an aliased mitral regurgitation waveform: http://www.heartweb.com.au/Videos/Echobk/CH3/Aliasing_PW.jpg

Aliasing usually sets in when the Doppler shift being measured exceeds one-half of the pulse repetition frequency (PRF). At usual settings, this is seen to happen above a velocity of 2m/sec.

To minimize the possibility of aliasing, the following can be done:

1.Shift the baseline and the scale to accommodate the maximum velocity possible

2.Reduce the depth of the sample volume, if that is possible

3.Use a lower frequency

4.Use a high PRF mode.

Steps 3 and 4 may not be possible on all machines. If the velocities are still too high, use continuous wave Doppler.

PWD is used to analyze Mitral and Tricuspid inflow patterns, measure velocities of flow at the left ventricular outflow tract (LVOT) and pulmonary and hepatic venous flow patterns.

Continuous Wave Doppler (CWD)

In this mode, a part of the transducer is continuously transmitting and a part of the transducer is continuously receiving the Doppler signal along a single line that is placed on the 2-D image. This method gives very good resolution of high velocities, but it does not give any information about the location of the signal, which may originate anywhere along the preset line of the ultrasound beam. As it measures velocities along the entir

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line, there will be a range of RBC velocities and the Doppler waveform is normally filled-in in contrast to the PWD.

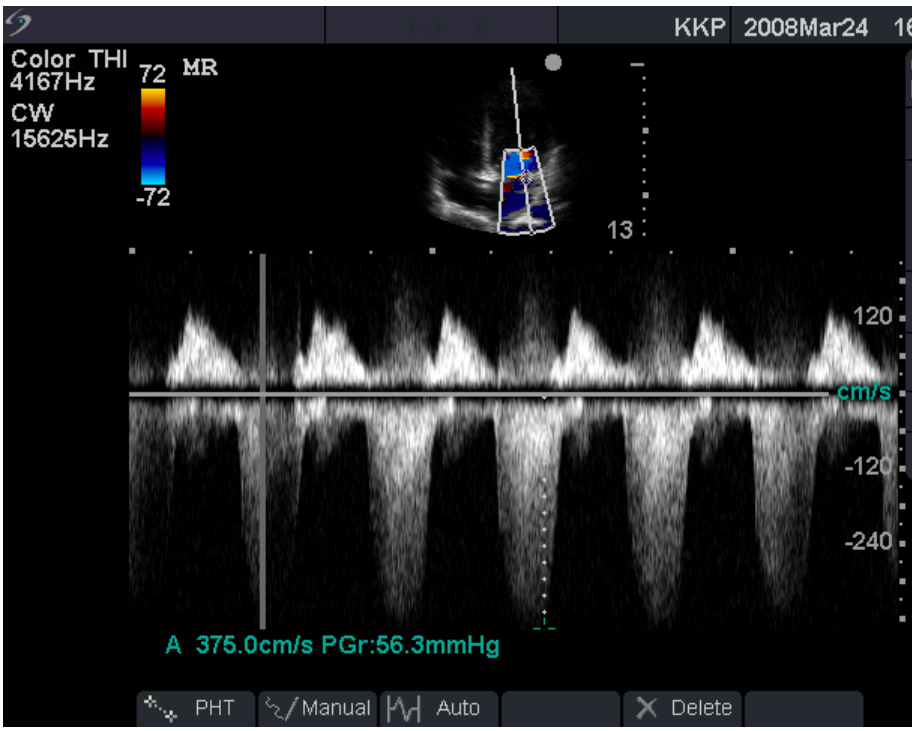

Figure 9: An example of Continuous Wave Doppler of a mitral regurgitation jet.

CWD is used to measure velocities of Tricuspid, Pulmonary, Mitral and Aortic regurgitation, and velocity of systolic flow through the aortic valve.

Tissue Doppler

This mode is similar to the Pulsed Wave Doppler except that it is used to measure velocities of tissue movement, which are much lower than blood velocities. The cursor or sample volume is placed on the 2-D image over the tissue of interest and the Doppler waveforms are acquired. The machine filters out the high velocities and displays a waveform that is very similar in appearance to the PWD waveform.

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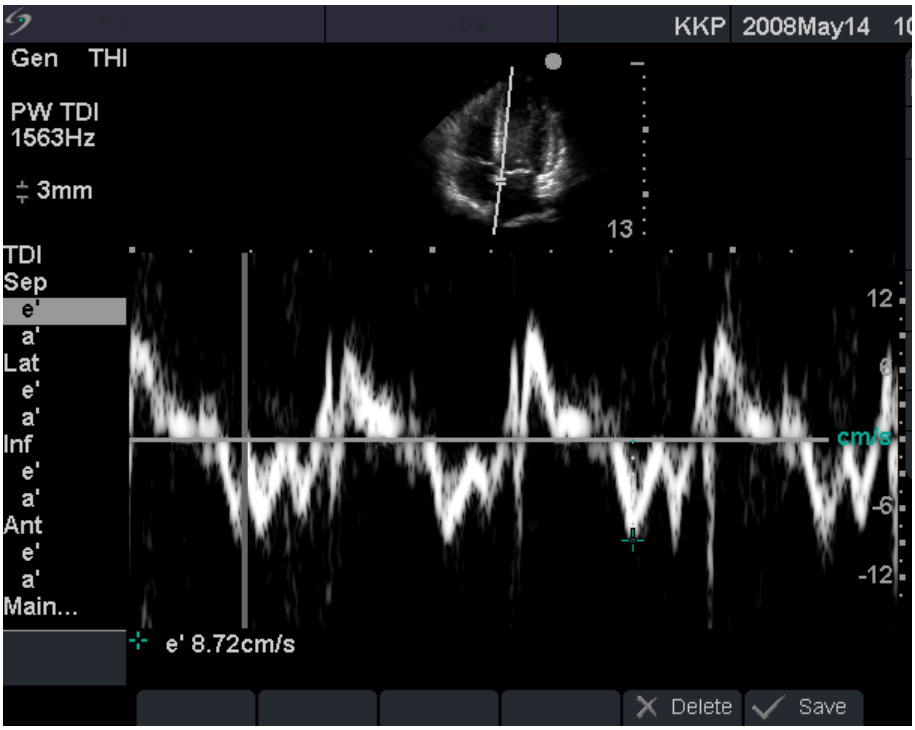

Figure 10: An example of Tissue Doppler through the medial mitral annulus

Tissue Doppler is used to measure tricuspid and mitral annulus velocities to assess RV systolic function and estimate LA pressure.

Tissue Harmonic Imaging

In this modality, the transducer looks for reflected echoes at twice the frequency of that which was emitted. This results in darker cavities and brighter walls leading to better endocardial definition, better resolution even at greater depths and reduced near field clutter.

It is better to leave this mode on at all times throughout the echocardiographic examination. Other modes can be used concomitantly with this.

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Video 1. A demonstrative lecture on ultrasound physics and basic modes from the Indira Gandhi National Open University's Youtube channel

Note: This is a large video file and may take time to download.
Pause play back for a couple of minutes at the start of the video and then play it. This will help prevent interrupted playback on slow connections

Video 2. A demonstrative lecture on Doppler principles and its applications from the Indira Gandhi National Open University's Youtube channel

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Tutorial 3 - Setting up and acquiring images

SETTING UP AND ACQUIRING IMAGES

Optimal positioning for most echo examinations is the left lateral position. A small subset of patients may have good visualization of the apical 4 and 5 chamber views in supine position. The subcostal view requires the patient to be in supine position.

The examination can be done from the right or left side of the patient. People in different centers have different preferences. However, from the point of view of infection control in the ICU, it is better for the examiner to stand on the left of the patient as this reduces direct contact with the patient. This would mean handling the probe with the left hand and operating the knobs on the machine with the right.

- Adjust the height of the patient bed for your comfort. Keep the machine close so your other non-probe hand can operate controls.
- Connect the ECG cables of the Echo machine.
- Dim the lights.
- Use adequate ultrasound jelly on the probe. It may be better to use separate sachets of jelly for each patient.
- 2-D:
  Adjust the overall gain knob till the picture is neither too dark nor too bright.
- Adjust the time-gain control (a series of sliders arranged in a row) so that the picture has uniform brightness from top to bottom.
- Use depth knob to set the minimum depth required. The main structure of interest should be in the center of the screen.
- Doppler:
  CFI- Turn up colour gain till speckling occurs in tissues (speckling indicates too much gain), then turn it down until the speckling disappears (correct gain).
- PWD and CWD:
  Adjust baseline and scale to so that the entire waveform is seen.

- Measurement:
  All machines have caliper and trace functions either with a trackball or a touch pad, which allow measurement of distance, time and areas. These form the basis of most of the hemodynamics calculations such as velocities, gradients, areas and size. More sophisticated machines have automated measurement devices and a bewildering array of calculation options.

VIEWS

It is useful to visualize the heart as being transected by three main axes of imaging.

Three Orthogonal Planes:

- Long Axis - transects the heart from aortic root to the left ventricular apex

- Short Axis - runs from left mid clavicle to the right hip
- Four Chamber Axis - runs from apex to the base; perpendicular to other 2 axes

Parasternal long axis view (PLAX)

Transducer position: left sternal edge; 2nd to 4th intercostal space.

Marker dot direction: points towards right shoulder

Structures seen are illustrated below.

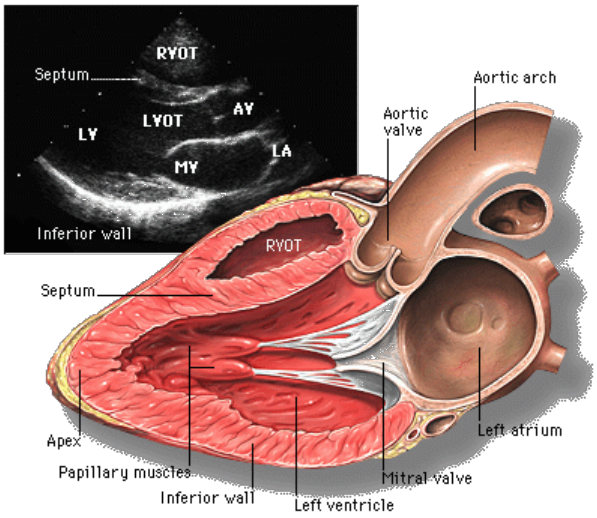

Figure 1: Parasternal long axis view: http://www.med.yale.edu/intmed/cardio/echo_atlas/contents/index.html

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Video 1. This video shows a normal parasternal long axis view.

The parasternal long axis view is commonly the first view obtained in an echo examination and is useful for assessing contractility visually, calculating ejection fraction in M-mode, detecting regional wall motion abnormalities and measuring LV outflow tract diameter for cardiac output studies.

Parasternal short axis view (PSAX)

Transducer position: left sternal edge; 2nd to 4th intercostal spaces

Marker dot direction: points towards left shoulder (90 degrees clockwise from PLAX view)

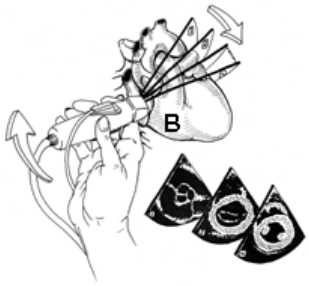

Figure 2: Changing the angle of the ultrasound probe results in being able to view short axis images from the aortic root down to the apex

By tilting the transducer on an axis between the left hip and the right shoulder, short-axis views are obtained at different levels, from the aorta to the LV apex. This angulation of the transducer from the base to the apex of the heart for short axis views is known as "breadloafing". Short axis sections are viewed at Aortic root level, mitral valve level and papillary muscle level.

The structures seen are illustrated below.

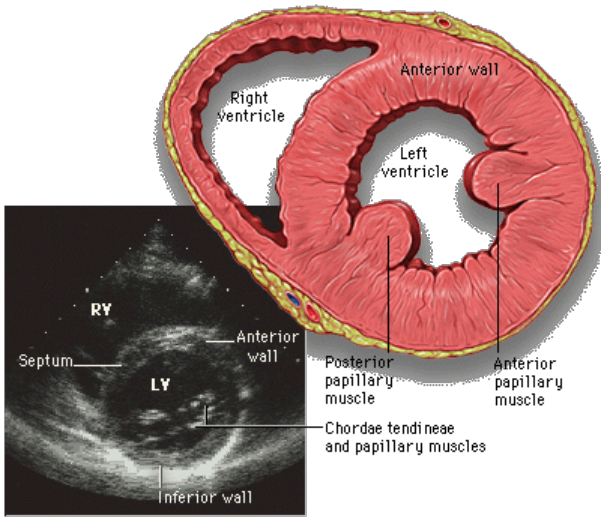

Figure 3: Short axis view at the level of the papillary muscles: http://www.med.yale.edu/intmed/cardio/echo_atlas/contents/index.html

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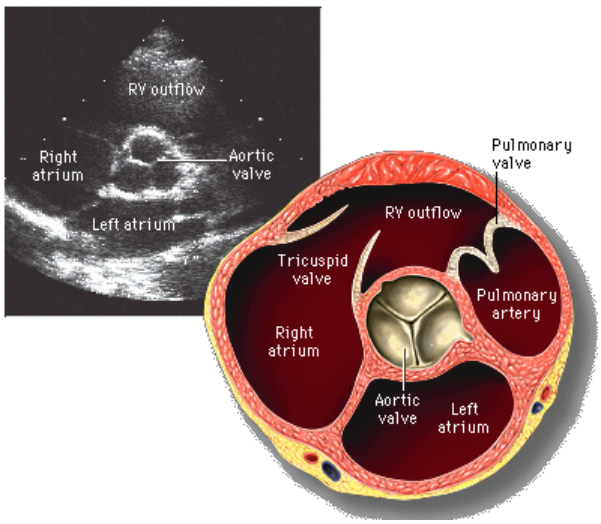

Figure 4: Short axis view at the level of the aortic valve: http://www.med.yale.edu/intmed/cardio/echo_atlas/contents/index.html

Video 2. Short axis view at the level of the AV valve

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Video 3. Short axis view at the level of the mitral valve

Video 4. Short axis view at the level of the papillary muscles

The parasternal short axis view at the level of the aortic valve is used to look for vegetations, and other abnormalities of aortic valve structure.

The parasternal short axis view at the level of the mitral valve is used to measure mitral valve area.

The parasternal short axis view at the papillary muscle level is useful for assessing the LV end diastolic area (an index of volume status), visual gestalt assessment of contractility, detecting regional wall motion abnormalities, detecting right ventricular enlargement and assessing interventricular septum motion abnormalities in acute cor pulmonale.

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Apical 4 chamber view (A4C)

Transducer position: apex of the heart

Marker dot direction: points towards left shoulder

Structures seen are illustrated below.

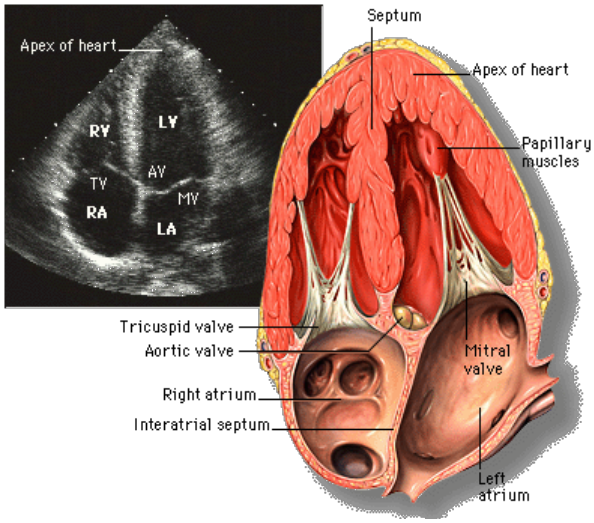

Figure 5: Apical 4 chamber view: http://www.med.yale.edu/intmed/cardio/echo_atlas/contents/index.html

Video 5. Apical 4 chambered view. note the pulmonary veins entering the left atrium

The apical 4 chambered view is used to detect and quantify mitral and tricuspid regurgitant and stenotic lesions using color flow imaging and Doppler. It is also used to detect right ventricular enlargement, visually assess RV and LV contractility, calculate ejection fraction using the 2D method, assess mitral and tricuspid inflow patterns and

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measure pulmonary vein inflow for diastolic function .

Apical 5 chamber view (A5C)

Transducer position and marker dot direction are same as the A4C view. The A5C view is obtained from the A4C by slight anterior angulation of the transducer towards the chest wall. The 5th chamber added is the LVOT.

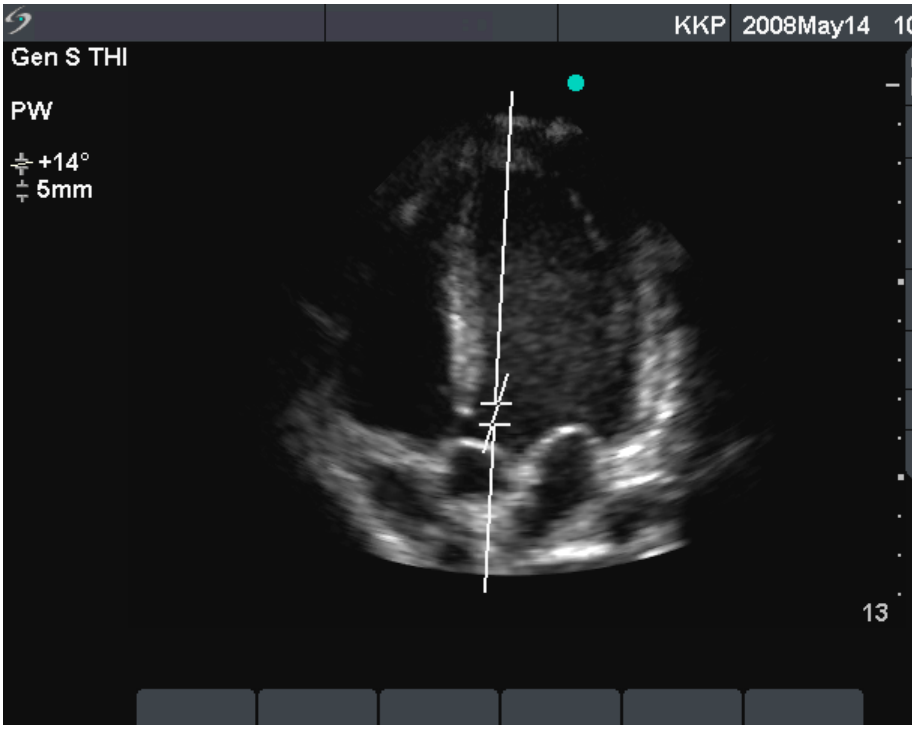

Figure 6: Figure 6: Apical 5 chamber view - the LVOT, aortic valve and root are visualized

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Video 6. The LVOT has been opened up to show the apical 5 chambered view

The apical 5 chambered view is used to measure LV ouflow tract velocities to measure cardiac output. It is also useful to detect and quantify aortic regurgitation and stenosis using color flow imaging and Doppler.

Apical 2 chamber view (A2C)

Transducer position: apex of the heart

Marker dot direction: points towards left side of the neck (45 degrees anti-clockwise from A4C view)

Structures seen are illustrated below

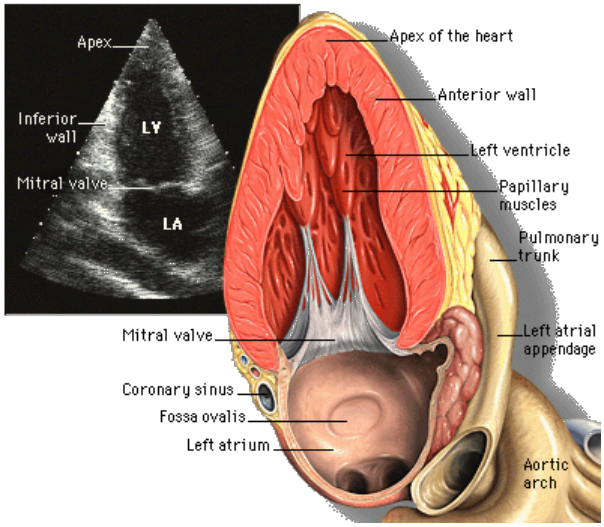

Figure 7: Apical 2 chamber view: http://www.med.yale.edu/intmed/cardio/echo_atlas/contents/index.html

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Video 7. Apical 2 chambered view. Note the left atrial appendage appearing as a cul-de-sac towards the right, and the coronary sinus, seen in cross section to the left of the atrioventricular junction

The apical 2 chambered view is used to measure ejection fraction using the Simpson's method. It is also useful to visualize the left atrial appendage to look for a thrombus. Mitral regurgitation jets may sometimes be seen better in this view than in the A4C.

Apical long axis or 3 chamber view (A3C)

Transducer position: apex of the heart

The transducer is placed at the same position as for a A4C view and then turned clockwise by 60°.

Structures visualized: It is similar to a parasternal long axis view seen from the apex and characterized by the presence of the mitral and aortic valves in the same plane.

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Video 8. Apical 3 chambered view

The apical 3 chambered view is used to measure LV outflow tract velocities when the A5C is suboptimal.

Subcostal view

The patient should ideally be placed in a supine position for a subcostal view examination. The abdomen has to be relaxed to allow indentation of the probe into the epigastrium.

Transducer position: under the xiphisternum

Marker dot direction: points towards left shoulder

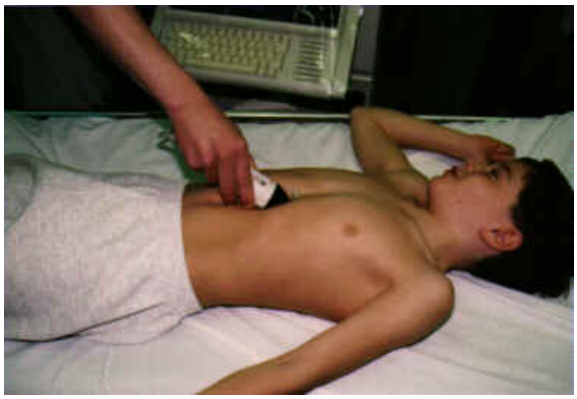

Figure 8: Probe position for the subcostal view: http://www.hcmcem.com/frmain.htm

The structures seen are illustrated below.

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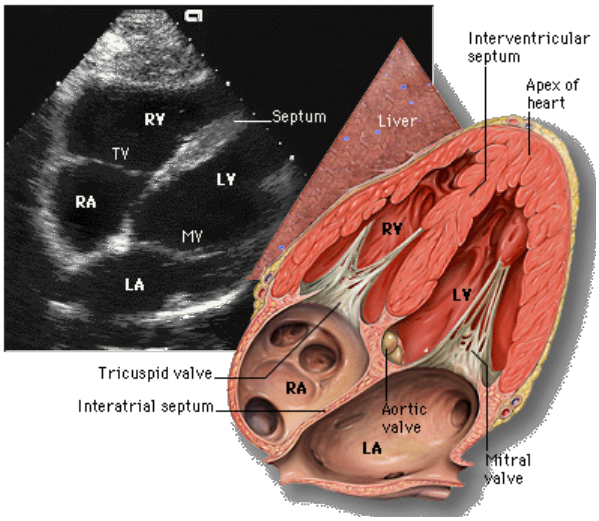

Figure 9: Subcostal 4 chamber view: http://www.med.yale.edu/intmed/cardio/echo_atlas/contents/index.html

The inferior venacava (IVC), descending aorta, interatrial septum and pericardial effusions are best seen in this view. This view is also particularly useful when obesity, emphysema or chest wall deformity prevents satisfactory transthoracic views from being obtained.

It is used to measure IVC variability to assess volume status, diagnose small pericardial effusions, measure RV free wall thickness to diagnose chronic cor pulmonale.

Video 9. A demonstrative lecture on probe positions for various views from the Indira Gandhi National Open University's Youtube channel

Note: This is a large video file and may take time to download. Pause play back for a couple of minutes at the start of the video and then play it. This will help prevent interrupted playback on slow connections

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Video 10. A demonstrative lecture on structures to be evaluated for various views from the Indira Gandhi National Open University's Youtube channel

Note: This is a large video file and may take time to download. Pause play back for a couple of minutes at the start of the video and then play it. This will help prevent interrupted playback on slow connections

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Tutorial 4 - Volume status and preload responsiveness assessment

Volume status and preload responsiveness assessment

A reliable assessment of volume status in the haemodynamically unstable patient is valuable in guiding management. Many a times, a more crucial question to answer is whether, the patient will respond to a bolus of fluids by raising his cardiac output, blood pressure or both. The echocardiogram can be used to answer both these questions.

The following measurements and indices are used for this purpose:
1.IVC diameter
2.IVC collapsibility index
3.LV end diastolic area
4.LVOT VTI variation with respiration
5.Peripheral artery Vmax variation
6.LVOT VTI variation with passive leg raise

IVC diameter and variability

Echocardiography of the IVC can easily be done by a transthoracic, subcostal approach. The transducer position is just below the xiphisternum 1-2cms to the right of the midline, with the marker dot pointing towards the sternal notch.

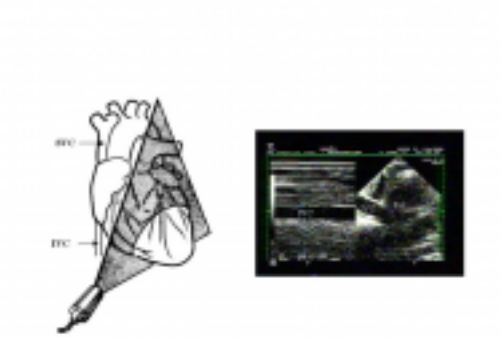

Figure 1: Longitudinal view of the inferior vena cava (IVC): RA: right atrium. From http://www.pifo.uvsq.fr/hebergement/webrea/index.php?option=com_content&task=view&id=36&Itemid=93

Recorded using two-dimensional and M-mode echocardiography. The transthoracic probe sensor must be positioned subcostally and slowly turned 90° counter clockwise till the optimal view is obtained.

be positioned subcostally and slowly turned 90° counter clockwise till the optimal view is obtained.

After obtaining a 2-D image of the IVC entering the right atrium and verifying that the IVC visualization is not lost during movements of respiration, place a M-mode line through the IVC 1-2cms from its junction with the right atrium, and obtain a M-mode tracing.

If the patient is spontaneously breathing, ask him to take a short quick inspiratory effort ("a sniff") during the M-mode recording. If the patient is mechanically ventilated, record the M-mode through 3 or 4 respiratory cycles.

Freeze the M-mode image and using calipers, measure the maximum and minimum diameter of the IVC tracing.

IVC diameter

Low CVP is increasingly is likely as IVC diameter (IVCD) gets smaller than 1 cm and abnormally high CVP increasingly likely as IVCD increases above 2cm. However, there is wide variation and the absolute measurements are not applicable with positive pressure ventilation. The IVC size is an indicator of volume status and not volume responsiveness - these two are not the same.

Sometimes the IVC is completely collapsed and may be difficult to visualize (virtual IVC). Such a situation in a mechanically ventilated or spontaneously breathing patient always indicates severe hypovolemia in the absence of raised intra-abdominal pressure.

IVC collapsibility index

Measurement of IVC diameter in different phases of respiration differentiates normal subjects from patients with elevated right atrial pressure. In a spontaneously breathing, healthy subject, cyclic variations in pleural pressure, which are transmitted to the right atrium, produce cyclic variations in venous return, which is increased by inspiration, leading to an inspiratory reduction of about 50% in IVC diameter.

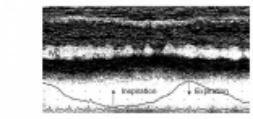

Figure 2: Physiological

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respiratory variations in IVC diameter in a healthy volunteer breathing quietly.: From: http://www.pifo.uvsq.fr/hebergement/webrea/index.php?option=com_content&task=view&id=36&Itemid=93

IVC diameter decreases on each inspiration.

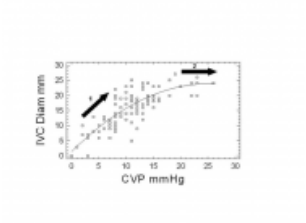

Figure 3: Simultaneous measurements of the central venous pressure (CVP) and IVC diameter at the end of expiration in 108 mechanically ventilated patients.: From: http://www.pifo.uvsq.fr/hebergement/webrea/index.php?option=com_content&task=view&id=36&Itemid=93

The relation pressure/IVC diameter is characterized by an initial ascending curve (arrow 1) where the compliance index (slope) does not vary, and an almost horizontal end part where the compliance index progressively decreases, because of the distension.

When the IVC is dilated, the relation between the diameter and pressure is located on the horizontal part of the curve: respiratory variations in diameter, produced by a low inspiratory pressure, are therefore abolished. This is what is seen in cardiac tamponade, and in severe right ventricular failure.

The **IVC collapsibility index** is expressed as the difference between the value of the maximum diameter and the minimum diameter, divided by the max of the two values. It should be noted that the denominator here is the **maximum** diameter. This index is used only for **spontaneously breathing non ventilated** patients. This is an index of volume status (hypovolemia, hypervolemia) and right atrial pressure, but has never been studied as an indicator of volume responsiveness. Its most studied uses include estimating CVP non-invasively and monitoring fluid removal during hemodialysis and ultrafiltration.

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Mechanically ventilated patients:

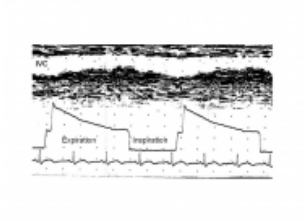

Fig. 4: Respiratory variations in IVC diameter in a patient on controlled ventilation: IVC diameter increases on each inspiration. From:http://www.pifo.uvsq.fr/hebergement/webrea/index.php?option=com_content&task=view&id=36&Itemid=93

In a patient requiring ventilatory support, the inspiratory phase induces an increase in pleural pressure, which is transmitted to the right atrium, thus reducing venous return. The result is an inversion of the cyclic changes in IVC diameter, leading to increases in the inspiratory phase and decreases in the expiratory phase. The respiratory variations in IVC diameter in a mechanically ventilated patient are therefore only observed when right atrial pressure is normal, that is low. In a patient presenting with signs of circulatory insufficiency, this finding may indicate hypovolemia. Measurement of IVC diameter in a patient receiving mechanical ventilation does not accurately predict right atrial pressure. Absence of respiratory variations in IVC diameter in a mechanically ventilated patient presenting with signs of circulatory insufficiency suggests that volume expansion will be ineffective in 90% of cases.

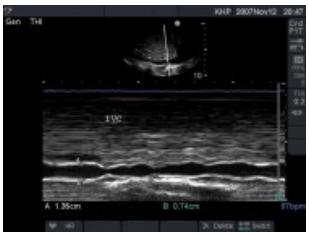

Figure 5: Measuring the maximum and minimum diameters in a M-mode tracing of the IVC showing marked IVC variability

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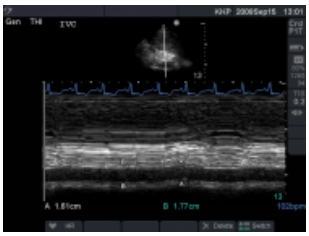

Fig.6 Measuring the maximum and minimum diameters in a M-mode tracing of the IVC showing insignificant IVC variability

In mechanically ventilated patients, a 12% or more variation identified patients likely to respond to vascular filling, in terms of increased cardiac output, from those who would not respond, with a positive predictive value of 93% and a negative predictive value of 92%. It must be remembered that the measurements should be taken during mandatory ventilator breaths and the tidal volume should be at least 8 ml/kg.
In spontaneously breathing patients, the normal IVC variation is approximately 50%. This index is not reliable in spontaneously breathing patients. Other markers of volume responsiveness in such patients are discussed below.
The great merit of this technique is that it is a dynamic, noninvasive parameter to evaluate the potential benefit of volume expansion. Moreover, the examination of the IVC is particularly easy and can be done by someone with limited experience in echocardiography.

Left Ventricular end diastolic area (LVEDA)

First, obtain a 2-D parasternal short axis view at the level of the papillary muscles. Freeze it and scroll back and forth to identify a frame showing the left ventricle in end diastole. You can use the ECG to time this. Using a caliper, trace along the endocardium to measure the area of the left ventricle at end diastole. You do not have to trace around the papillary muscles and they can be included inside the circle.

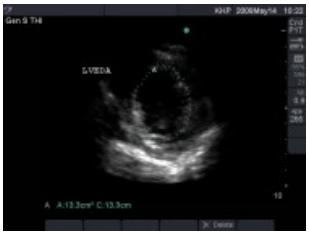

Fig. 7: Tracing the endocardial margin of the LV at end diastole

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An LVEDA of less than $10cm^2$ or a LVEDA index (LVEDA / BSA) of less than $5.5cm^2/m^2$ indicates significant hypovolaemia.

Another sign that suggests severe hypovolaemia is the "kissing papillar muscle sign" where opposing papillary muscles come in contact with each other at end systole.
One thing to remember is that severe concentric hypertrophy can reduce LVEDA even without any hypovolaemia.

Left ventricular outflow tract (LVOT) Velocity Time Integral (VTI) variation with respiration

In the apical 5-chamber view, place a PWD sample volume in the middle of the LVOT just adjacent to the aortic valve. The sample cursor should not overlie the valve. Obtain a PWD tracing. Make sure there is no valve opening artifact in front of the systolic flow waveform. That means that the cursor is placed over the aortic valve and needs to be moved into the LVOT by a few millimeters.

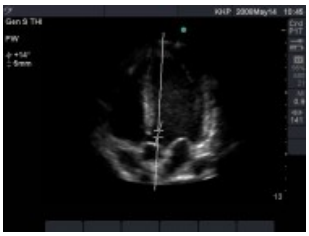

Figure 8: Correct placement of the PWD cursor in the LVOT proximal to the aortic valve

Once you have obtained the waveform over 3 or 4 respiratory cycles, freeze the image. Reducing the horizontal sweep speed enables capture of a larger number of LVOT ejections. Scroll back and forth till you can identify the largest (usually at end inspiration, if mechanically ventilated) and the smallest waveforms over a single respiratory cycle. Go to 'calculations'...'aortic continuity equation'....'LVOT VTI' and trace the edge of the waveforms using the trackball.

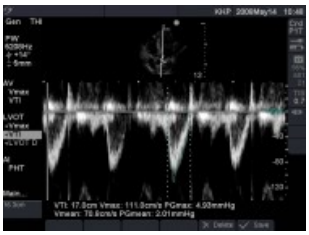

Figure 9: Tracing the VTI of the PWD at the LVOT

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The machine will calculate the VTIs for these two waveforms. The VTI variation is then calculated as the difference between the maximum and the minimum VTI divided by the mean of the two values.

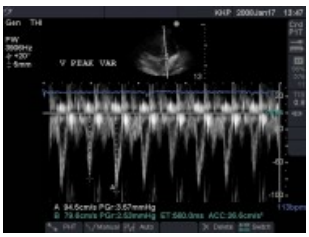

Figure 10: Measuring the maximum and minimum Vmax at the LVOT

A VTI variation of more than 12% predicts fluid responsiveness (defined as an increase in cardiac output by at least 15% in response to a standard fluid bolus) with a sensitivity of 100% and a specificity of 89%.

An alternative to tracing the VTIs is to measure the maximum and minimum peak velocities (Vmax) instead. This is quicker and easier, and again, more than 12% variation suggests fluid responsiveness.

Peripheral artery Vmax variation

Some have validated variation of peak velocities (Vmax) measured with a Doppler probe in peripheral arteries such as the brachial artery, the cutoff being the same. This is a quick and simple way of assessing fluid responsiveness.

Left ventricular outflow tract (LVOT) Velocity Time Integral (VTI) variation with Passive Leg Raise (PLR)

The LVOT VTI is measured with a PWD in the A5C view as described above. 2 assistants then lift both lower limbs of the patient to a 45° angle. A repeat LVOT VTI is measured after 1 minute.
Passive leg elevation (PLR to 45°) induced increase in VTI by > 12.5% predicts an increase in stroke volume by > 15% after saline infusion (500ml over 15minutes). The sensitivity of PLR induced response is 77% and the specificity is 100%.

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Tutorial 5 - Assessment of LV systolic function

Assessment of LV systolic function

A knowledge of the LV systolic function is crucial in the undertanding of and management of unstable hemodynamics or a failing heart in the ICU. As with fluid status assessment, a composite of different measures should be used rather than any one. The different methods commonly used in the echocardiographic assessment of LV systolic function are:

1.Ejection fraction - M-mode LV dimensional method
Simpsons method
Visual gestalt
2.dP/dT of the mitral regurgitant jet
3.Doppler measurement of stroke volume...and therefore cardiac output

Ejection fraction

This refers to the percentage of the end diastolic LV blood volume that is ejected out of the LV during systole. The normal ejection fraction is above 50%. It is a widely used measure of LV contractility. Simplicity and familiarity are advantages of the EF as a measure of LV systolic function.
For a certain degree of contractility (the intrinsic contractile strength of the myocardium or the amount of work that the heart can perform at a given load), the stroke volume (SV) of the ventricle is determined by the preload (the end diastolic ventricular volume, pressure or stretch) and afterload (the force opposing ejection).
The ideal indicator of myocardial contractility should not be affected by preload or afterload.
Ejection fraction (an indicator of contractility) is less dependent of loading conditions as compared to SV. However, the EF is afterload dependent and is depressed in situations with a high afterload. EF is measured in the ICU in three ways.

M-mode LV dimensional method:

First obtain a parasternal long axis view and place a M-mode cursor is placed through the septal and posterior LV walls just beyond the tip of the mitral leaflets.
In the resultant M-mode image take measurements of the RV internal dimension, interventricular septum thickness, LV internal dimension and LV posterior wall thickness at end-diastole (timed on ECG or point of largest LV internal dimension) and at end-systole (ECG timed or point of smallest LV internal dimension).

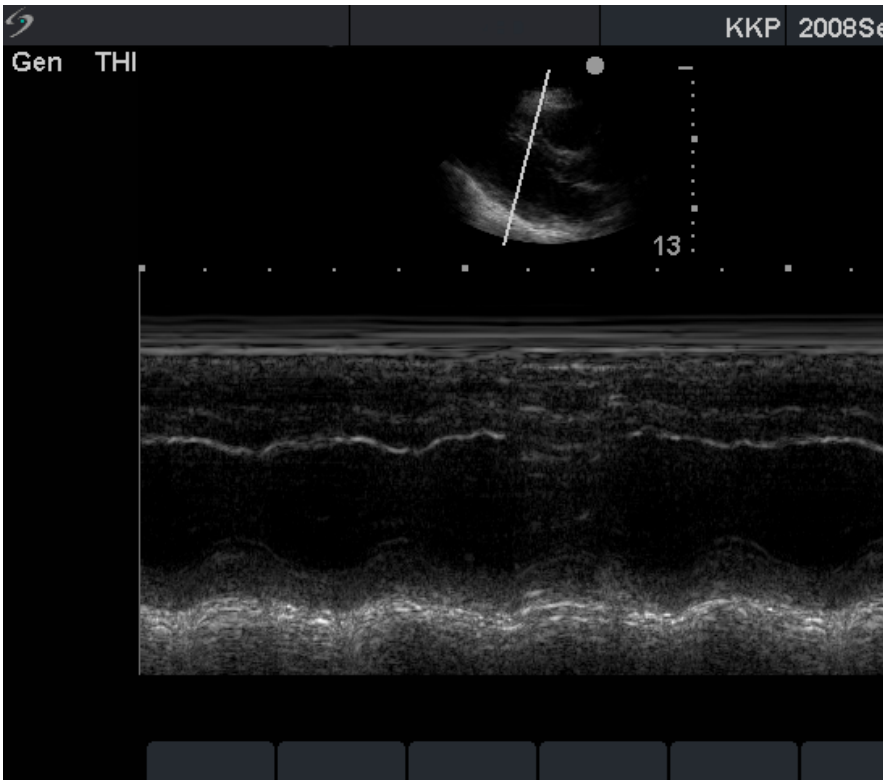

Fig.1 M-mode of the LV in PLAX view

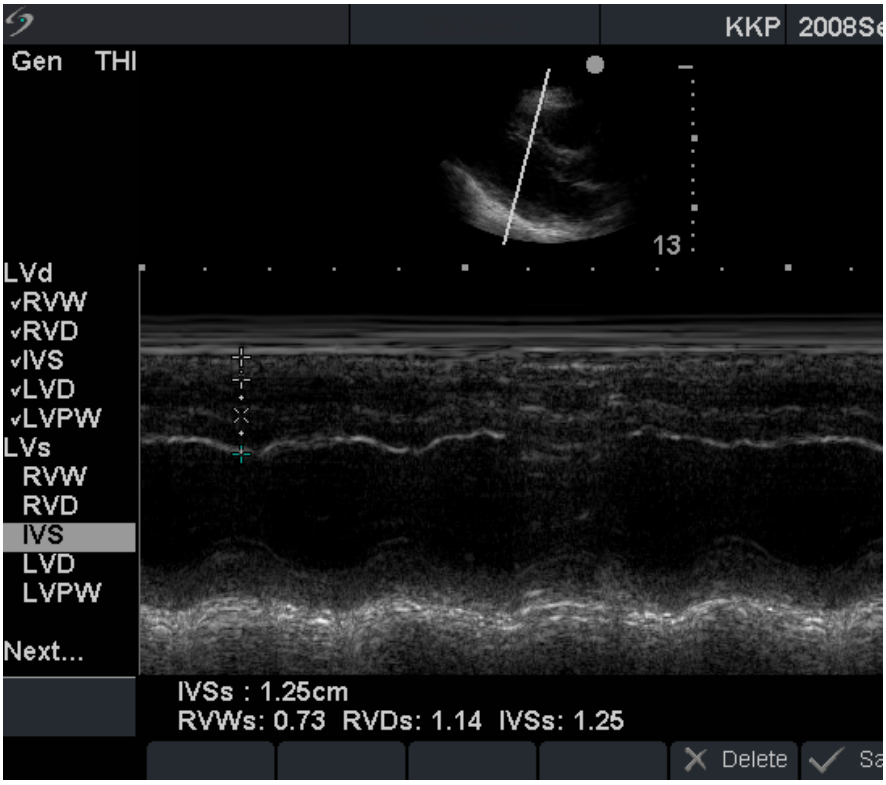

Fig.2 Systolic measurements with a caliper in progress

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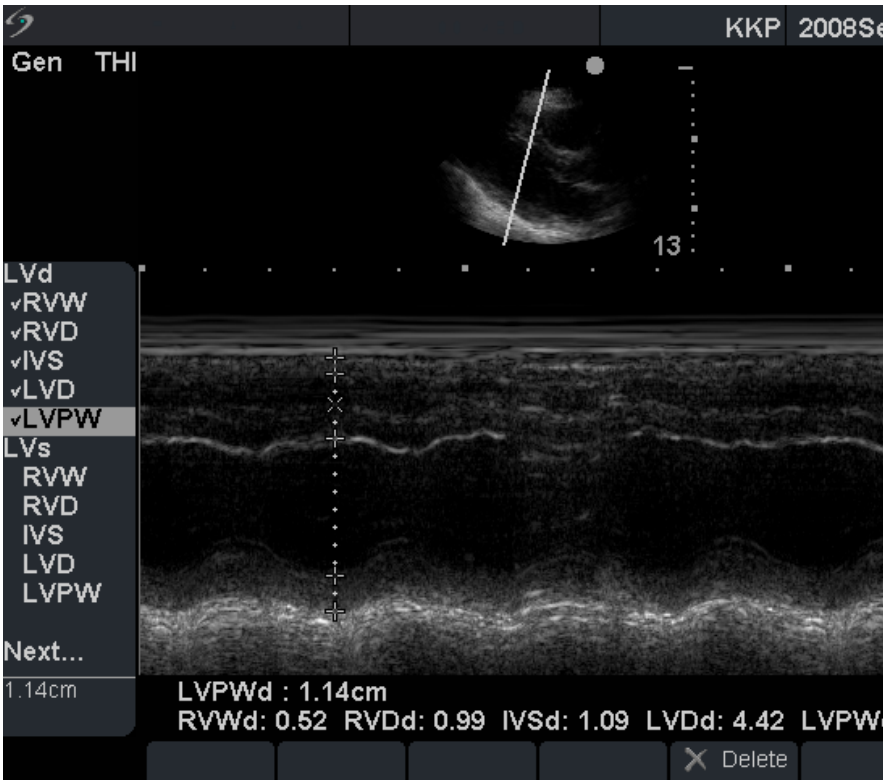

Fig.3 Diastolic measurements done with the calipers

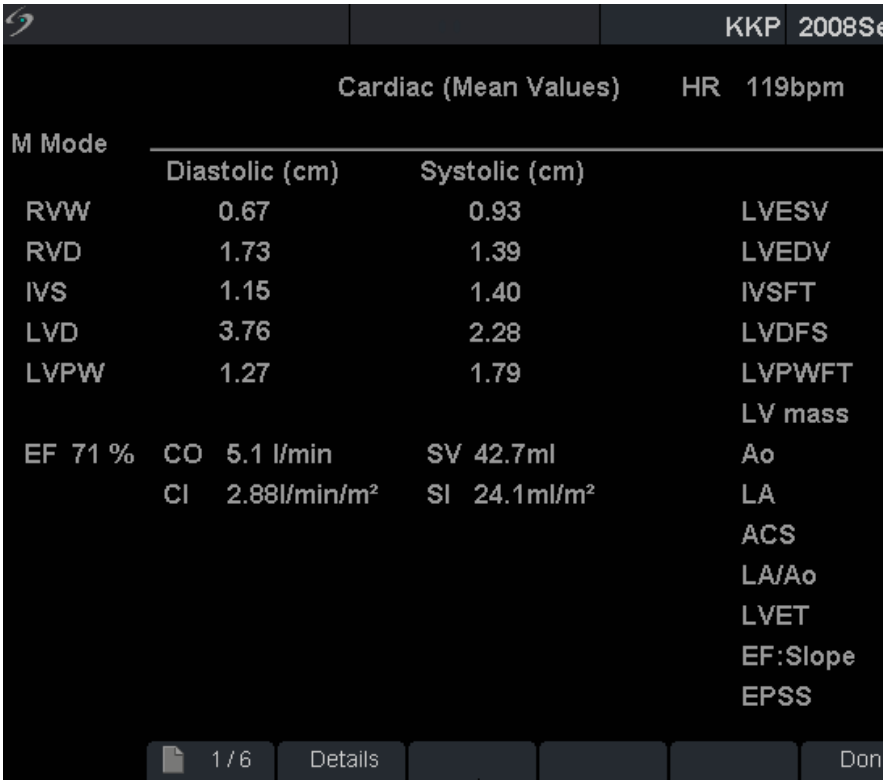

Fig.4 Report of EF and FS generated

With this information, most machines will be able to generate two numbers, the fractional shortening and the ejection fraction.
Fractional shortening is (LVEDd-LVESd) / LVEDd expressed as a percentage. The normal value is 30% to 45%.
Ejection fraction is calculated from derived volumes, which are computed based on the "cubed" or "Teichholtz" equations. The geometric assumptions made with

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the cubed method limits its usefulness in the abnormal ventricle. In dilated and spherical ventricles, the ratio of long to short axis in the ventricle increases and LV volumes will be overestimated. Using a derived formula by Teichholz instead of the cubed equation compensates for ventricles of abnormal size but only in the absence of asynergy (no RWMA's):
The normal ejection fraction is 50% to 75%.

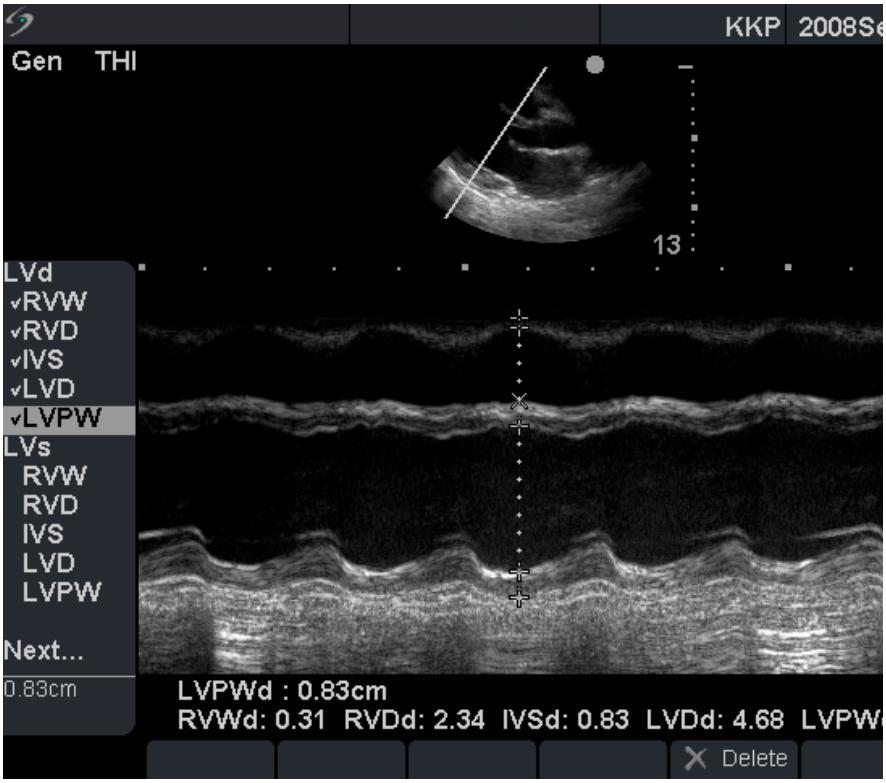

Fig.5 M-mode of LV showing moderate LV dysfunction

While the M-mode method of calculation of EF is easy to learn and perform, it has some drawbacks. The M-mode assessment provides information about contractility along a single line. In a patient with coronary artery disease and regional wall motion abnormalities, the severity of the dysfunction may be underestimated if only a normal region is interrogated or overestimated if the M-mode beam transits through the wall motion abnormality exclusively. Another disadvantage of the M-mode asessment is that it does not reflect the true minor axis dimension. This is particularly common in elderly patients and in some patients with emphysema, in whom there is an angulation of the interventricular septum. In such cases, the M-mode beam traverses the ventricle in a tangential manner and often overestimates the true internal dimension

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In this method, acquire A4C or A2C views, making sure that the endocardial borders are visualised well. Freeze the image and scroll backward and forward to identify a frame at end diastole. This can be timed using the appearance of the ventricle - identifying a frame where the ventricle appears to have the largest volume; or with the ECG trace, where the peak of the R wave corresponds to end-diastole.
Open the "calculations" menu and select "LV volumes" and "A4C diastolic" or "A2C diastolic", whichever is appropriate. Place the cursor on the endocardial border where the anterior mitral leaflet meets the interventricular septum and trace the entire endocardial border of the left ventricle. You do not have to trace around the papillary muscles. Once this is done, the LV volume in diastole will be calculated.

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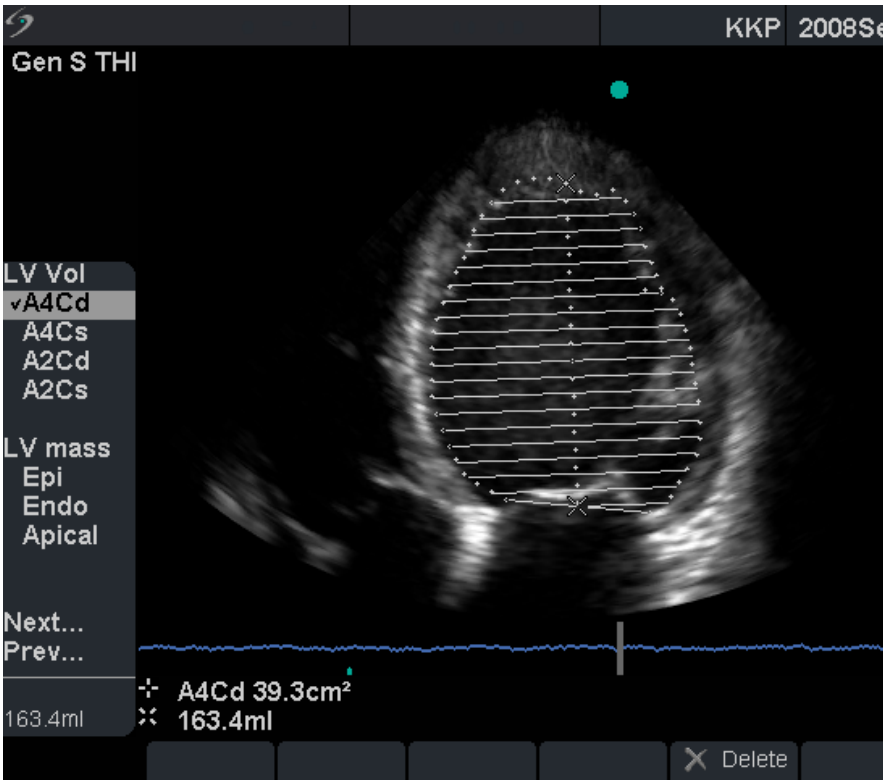

Fig.6 Calculation of LV volume in end-diastole

The frozen image is then scrolled forward or backward to identify a frame at end-systole. Again this can be done by identifying a frame where the ventricle appears to have the smallest volume, or correlating with the ECG trace, where the peak of the T wave corresponds to end-systole. Select "systolic LV volume" on the calculations menu and trace the outline of the endocardial border of the LV. Once this is done, the LV volume in systole will be calculated.

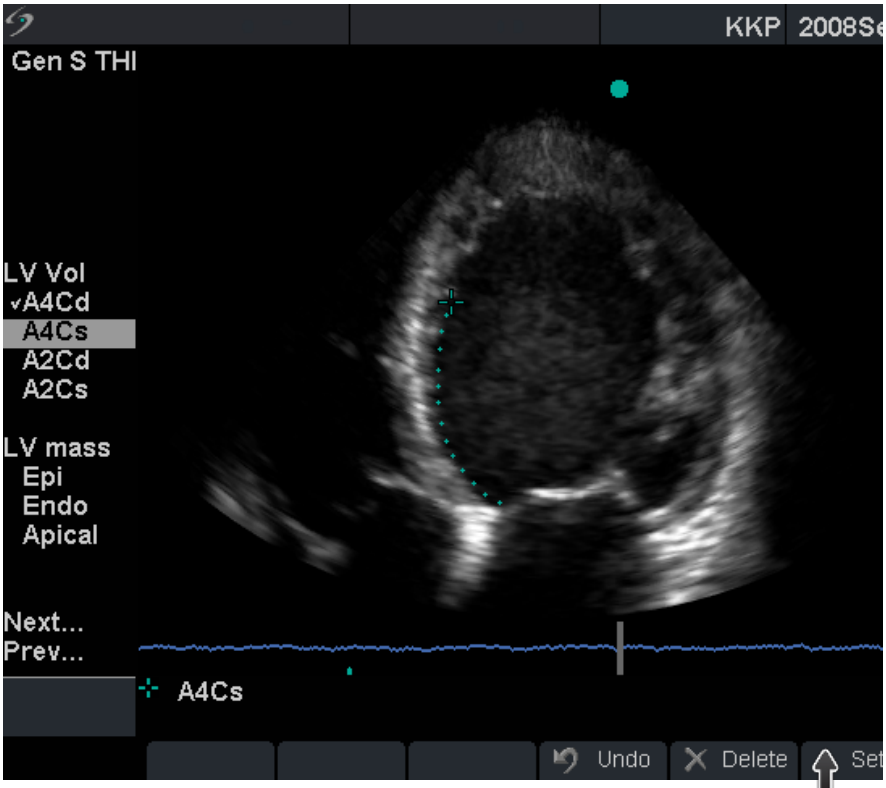

Fig.7 Tracing endocardial border in end-systole

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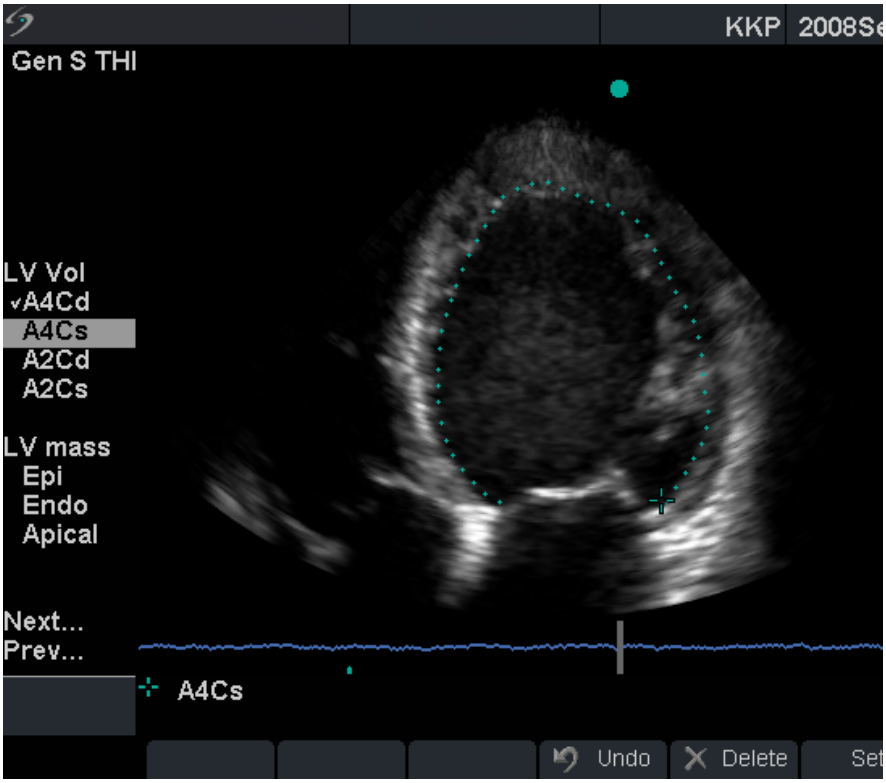

Fig.8 Tracing of endocardium in end-systole completed

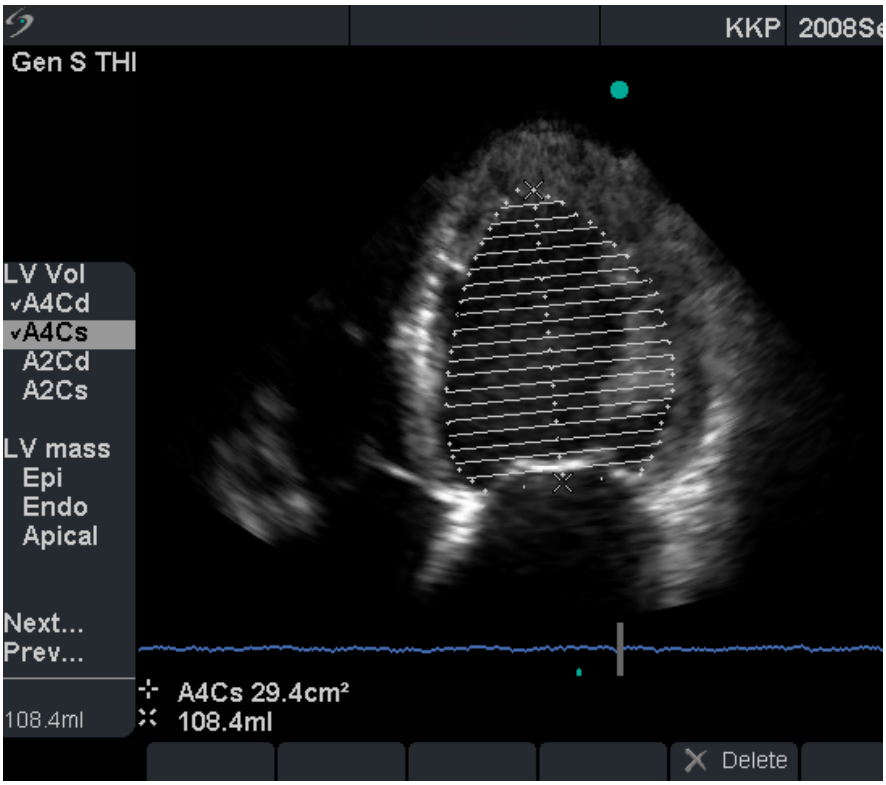

Fig.9 Calculation of volume in end-systole

The machine will then calculate the ejection fraction by using the formula:

Ejection fraction = LVEDV - LVESV
LVEDV

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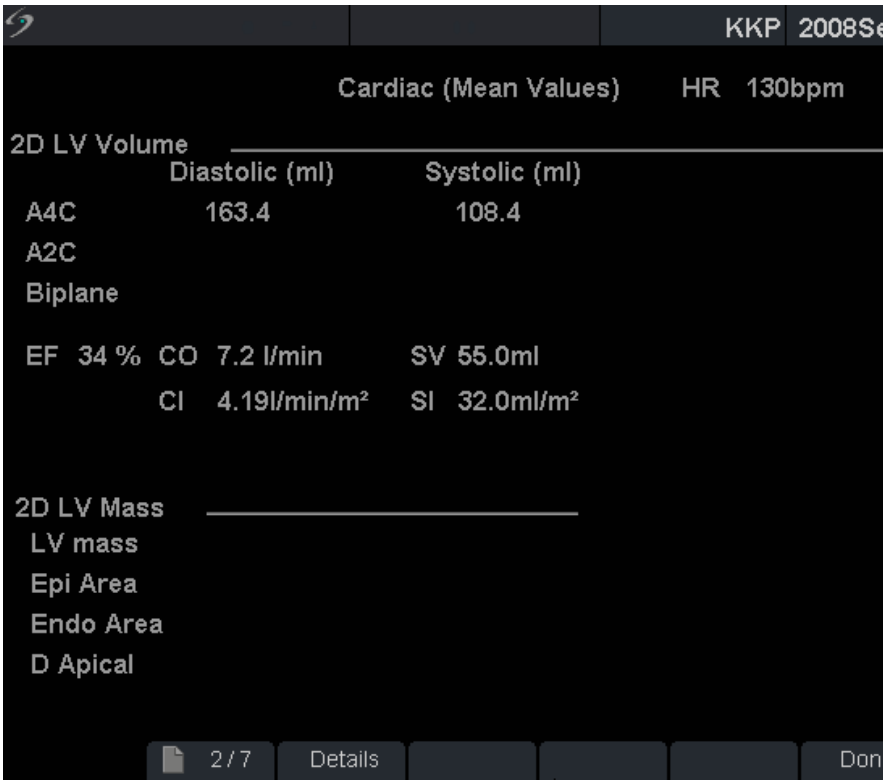

Fig.10 Report of ejection fraction generated

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2.Because they are parallel to the ultrasound beam, some parts of the endocardial border are not well delineated, causing uncertainty in deciding where to trace the outline of the LV cavity. This results in inter and intra-observer variability of LV Volumes and EF.
3.Another cause of such variability is the choice of frame at end diastole and systole.
4.The LV volumes are calculated using some assumptions made about the shape of the LV cavity, which are not always valid, particularly in a heart with regional LV dysfunction.

However, this remains one of the most widely used methods to calculate LVEF.

Visual Gestalt

Experienced echocardiographers frequently estimate EF by looking at the overall size and contractility as well as the inward movement and thickening of the various segments of the LV walls without actually taking measurements. Although it is dependent on the experience of the echocardiographer, it has been shown to correlate fairly well with angiographic assessment of the EF.

Parasternal long axis view:

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Video 1. Normal LV function

Video 2. Moderate LV dysfunction

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Video 3. Severe LV dysfunction

A4C view:

Video 4. Normal LV function

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Video 5. Moderate LV dysfunction

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One method is to distinguish three grades of global LV systolic function based on the subjective radius change of the LV short axis in systole: Normal (³ 30% change in radius), moderate dysfunction (10-30% decrease in radius) and severe dysfunction (< 10% change in radius). A fourth grade, hyperdynamic can be used to describe the very vigorous ventricle that is seen in severe vasodilatation or mitral regurgitation with preserved LV function.

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It is prone however to intra-observer variation. This assessment may also be unreliable for serial evaluation of LV function and when LV volumes critically influence the timing of cardiac surgery.

Mitral regurgitaion dP/dT

This is another, traditionally underutilized indicator of LV function. Whilst, EF is affected by afterload, MR dP/dT is afterload independent but is influenced by the preload. This is because this is a measure of contractility of the LV in the isovolumic contraction phase.
This can only be used in patients who have a measurable mitral regurgitation. An A4C view is obtained and the mitral regurgitant jet is identified using color flow imaging. The continuous wave doppler cursor line is placed over the origin of the MR jet and a doppler trace is obtained.
After selecting MR dP/dT on the "calculations" menu, the cursor is used to mark a point on the slope of the MR jet trace at 1m/sec and another at 3m/sec. The time interval in seconds between these two points is noted (Ti). The dP/dT is given by the formula:
dP/dT = 32/Ti
Most machines will provide reference lines at 1 and 3 m/sec and will calculate and display dP/dT automatically.
The normal dP/dT is>1200mmHg/sec. 800 to 1200mmHg/sec suggests mild LV dysfunction and <800mmHg/sec severe LV contractile dysfunction.

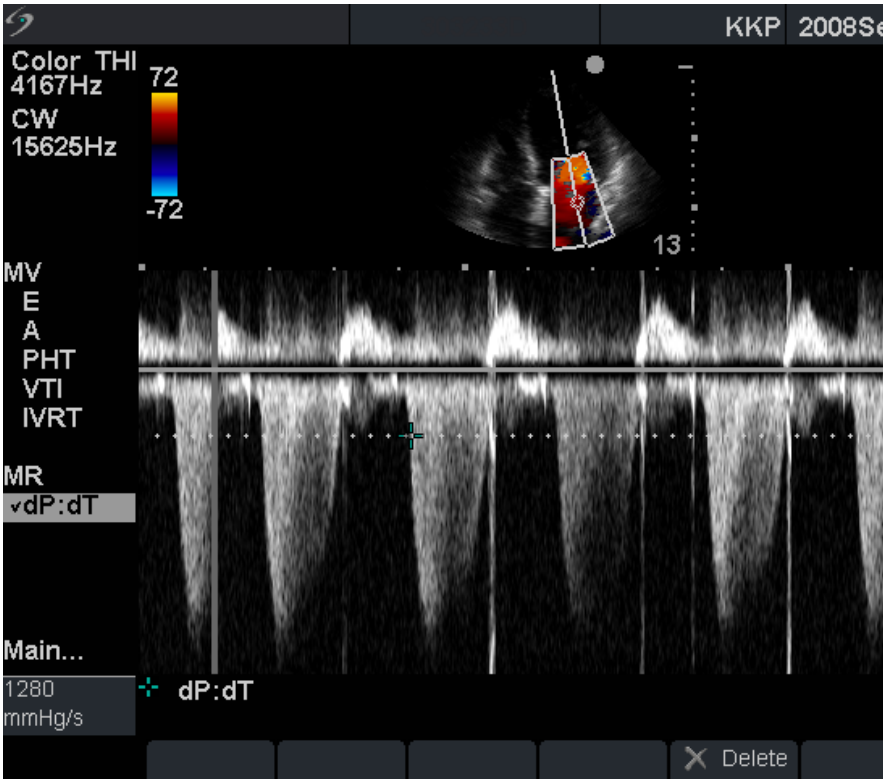

Fig.11 Placement of the first cursor point on the 1m/s line

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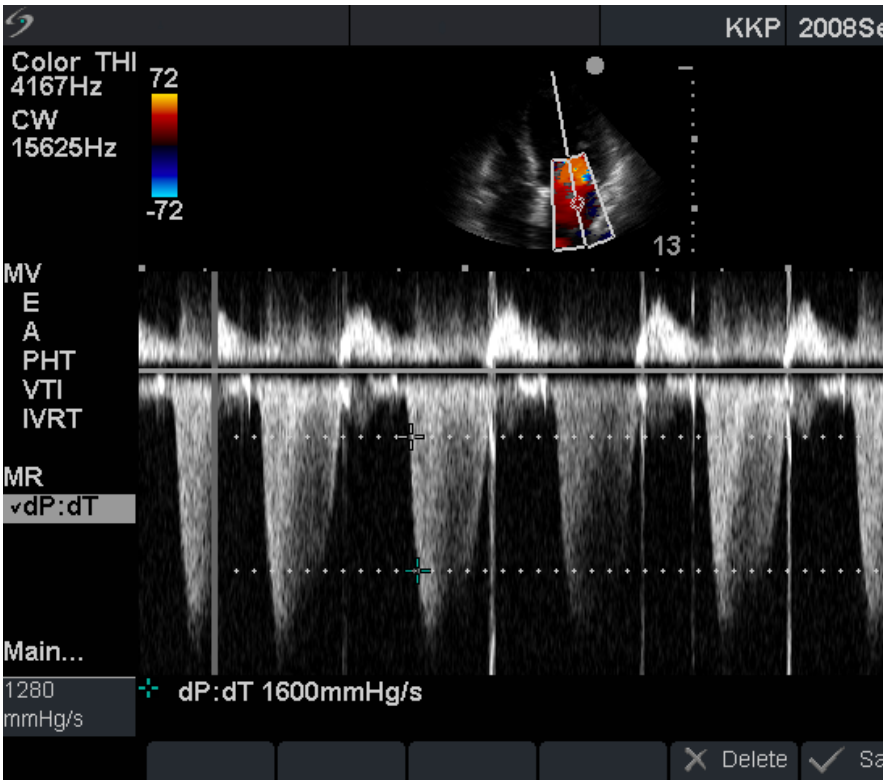

Fig.12 Placement of the second cursor point on the 3m/s line: The dP/dT in this case is normal

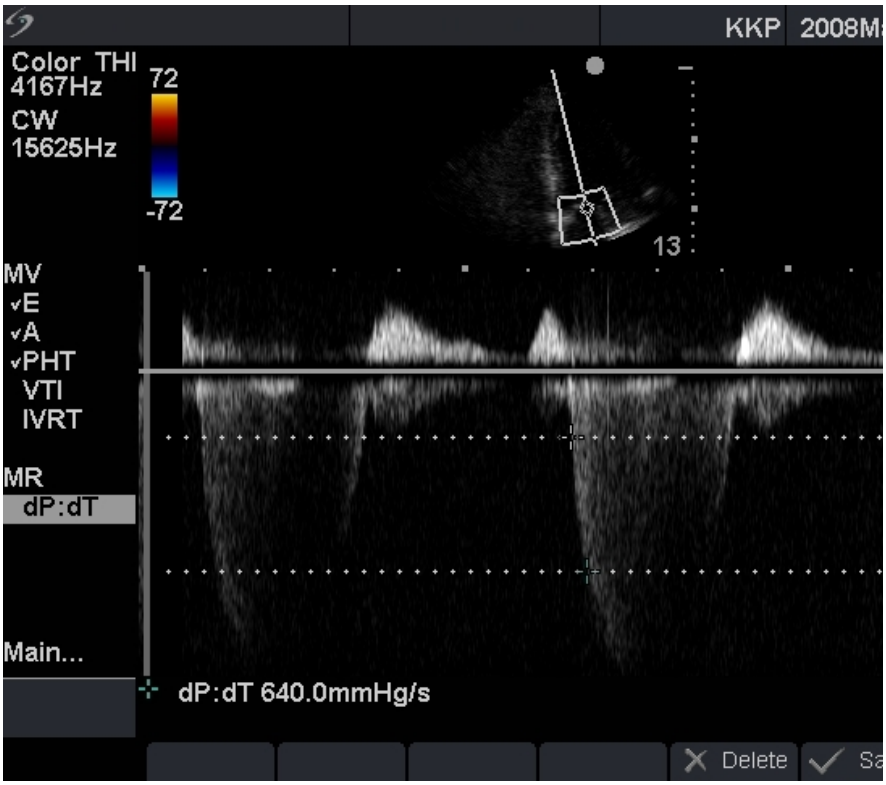

Fig.13 dP/dT in a patient with severe LV dysfunction

Limitations:

This method is only useful in patients with enough MR to obtain a well-defined velocity curve.

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LA should be compliant.
Click artifact (caused by valve closure) can obscure the descending limb of the CWD envelope, which makes measurements difficult.
Eccentric MR jets may not reflect true velocity and will result in underestimation of dp/dt unless careful colour Doppler examination of the jet is made to minimize CWD error.
A normal dp/dt maybe present in hypertension and aortic stenosis even with impaired LV function.

Doppler assessment of cardiac output

Although the above techniques are useful to assess the contractility of the mocardium, what is really of interest to the intensivist is the net result of myocardial stretch and contractility....the stroke volume and cardiac output. Although cardiac output can be calculated using doppler at any of the valve orifices of the heart, the mitral or tricuspid annuli, the RVOT or the LVOT, the measurement is done most commonly at the LVOT.

First measure the LVOT diameter on a Parasternal Long Axis view. This is done by zooming into the LVOT on the PLAX view using the zoom tool and freezing the image. The images are scrolled backward and forward to capture a frame in which the aortic valve leaflets are wide open. The LVOT diameter is measured adjacent to the points of attachment of the leaflets. The machine will then calculate the cross sectional area (CSA) of the LVOT.

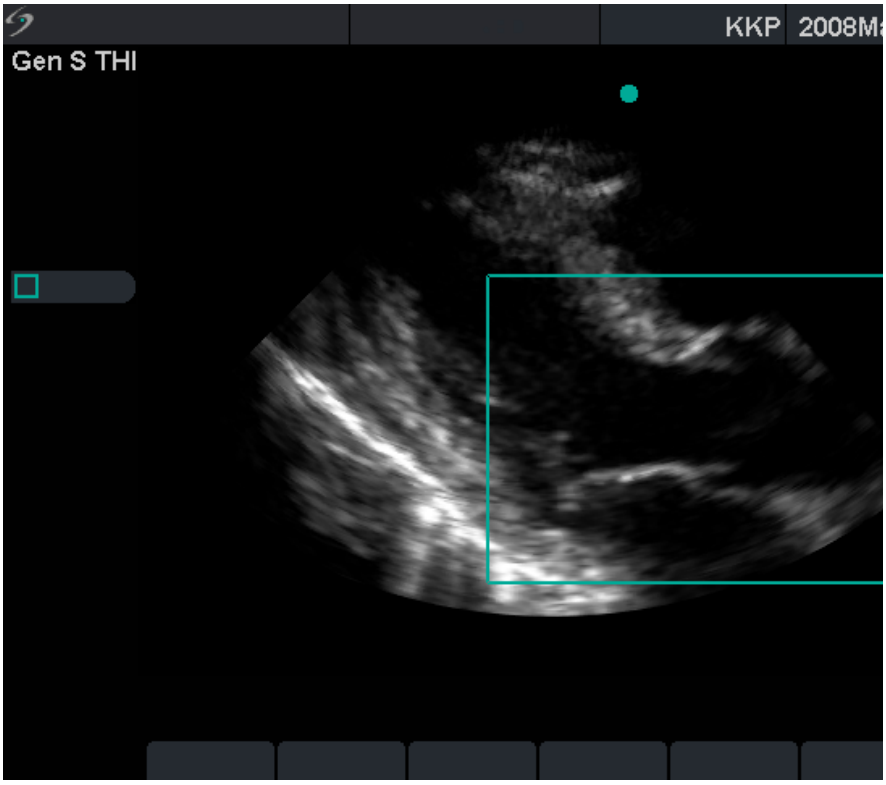

Fig.14 Zooming in on the LVOT in the PLAX view

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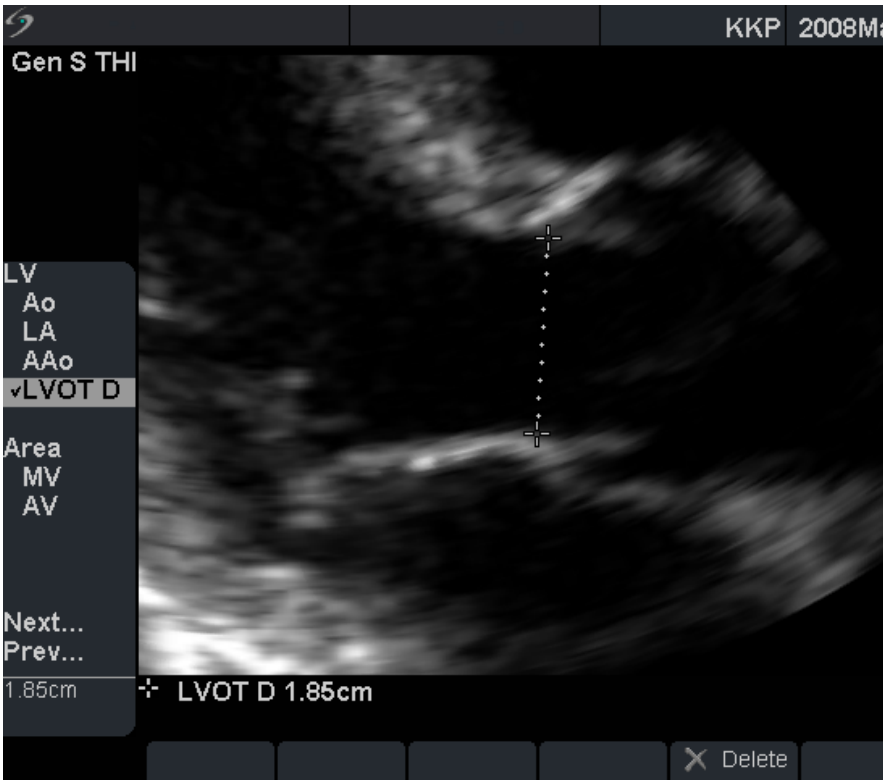

Fig.15 Measuring the LVOT diameter at attachments of the aortic leaflets

Next, obtain an apical 5-chamber view of the heart. As mentioned earlier, the A5C view is obtained from the A4C by slight anterior angulation of the transducer towards the chest wall. The 5th chamber added is the LVOT.
Place the Pulsed Wave Doppler cursor in the LVOT as close to the aortic valve as possible without including it in the sample volume. Acquire the PWD trace. The trace may be considered satisfactory if the closing click of thr aortic valve is visulaised. However, if the opening click is distincly seen before the ejection waveform, it means that the sample volume is too close to the aortic valve and needs to be moved a little away from the valve before another tracing is obtained.

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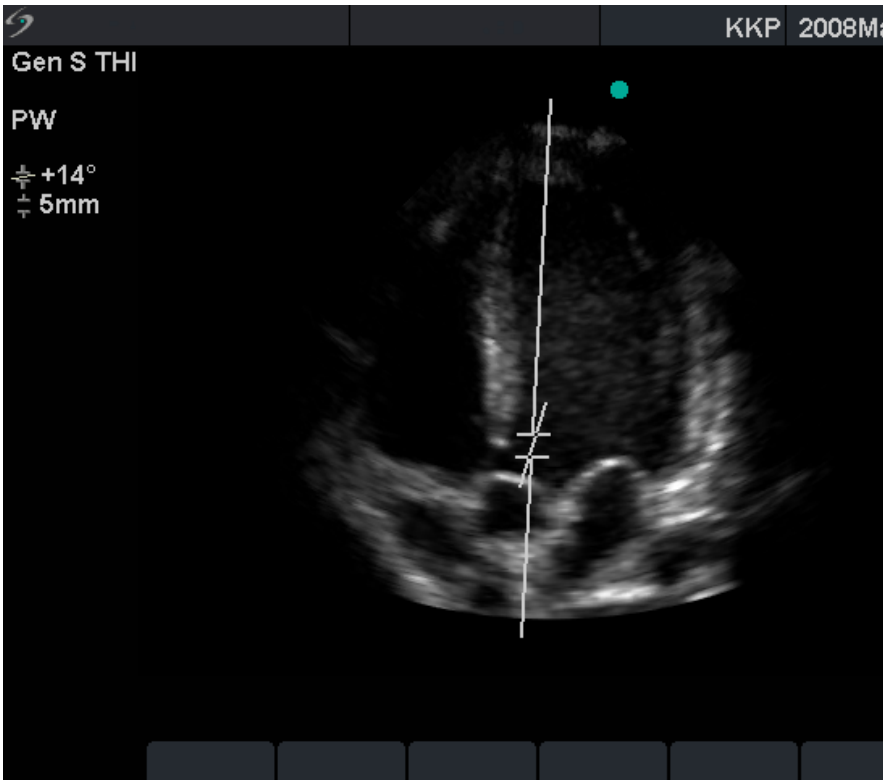

Fig16. Placement of the PWD cusor in the LVOT in A4C view

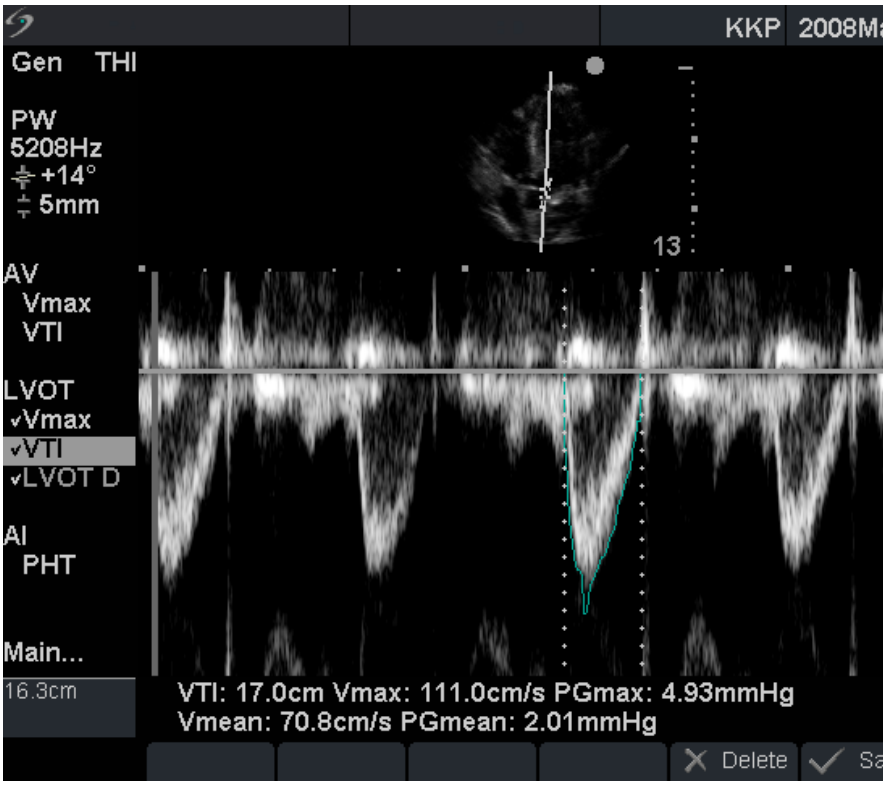

Fig.17 Tracing the PWD waveform obtained in the LVOT

Then choose LVOT VTI from the "calculation" menu and manually trace the PWD waveform. Some machines may be able to do this automatically. The machine

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will calculate the area under the curve and represent it as a Velocity Time Integral (VTI) in cms. Repeat the VTI measurement thrice to reduce sampling bias.
The stroke volume at the LVOT is then obtained by multipying the LVOT VTI with the LVOT CSA.

LVOT VTI X LVOT CSA = Stroke volume

Stroke volume X Heart rate = Cardiac output

Cardiac output = Cardiac index
Body surface area

This is a simple, non-invasive method of measuring cardiac output in ICU patients. It correlates well with measures of cardiac output obtained by thermodilution (r=0.95) with a tendency to underestimate it by about 0.24 l/min.
This measurement can be done repeatedly to see the trend of cardiac output. The LVOT CSA does not need to be calculated for repeat measurements as it does not change.

There are problems however with this technique.
1.Sometimes an adequate A5C view may not be obtainable. In such a case, an Apical 3-chamber view can be tried.
2.The LVOT may not be aligned with the direction of the PWD, leading to underestimation of velocities. In this situation, an apical 3-chamber view may sometimes offer better alignment. The other workaround is to use an angle correction factor. Although this is generally not advocated, it may be acceptable if the angle is kept to less than 20 degrees.
3.When the parasternal long axis view is not obtainable, a LVOT diameter of 2cms for males and 1.75cms for females can be assumed.
4.In patients who are taking deep breaths, the entire cardia may move with respiration making it very difficult to ensure that the PWD sample volume stays at the same place in the LVOT through the respiratory cycle. This can lead to variations in the VTI with respiration, which is not due to hypovolaemia.

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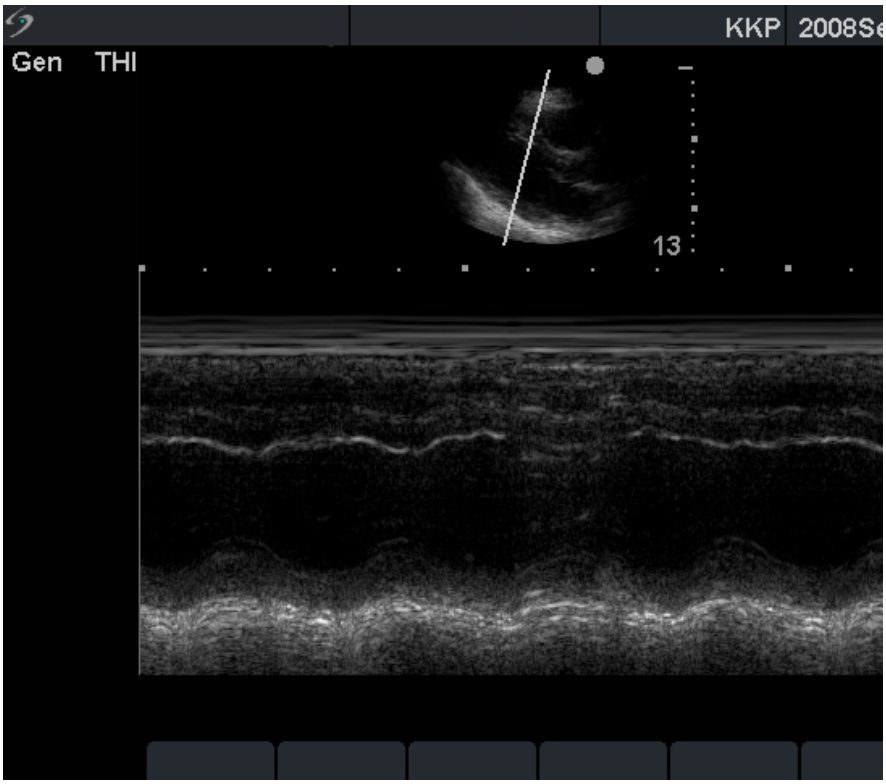

Fig.1 M-mode of the LV in PLAX view

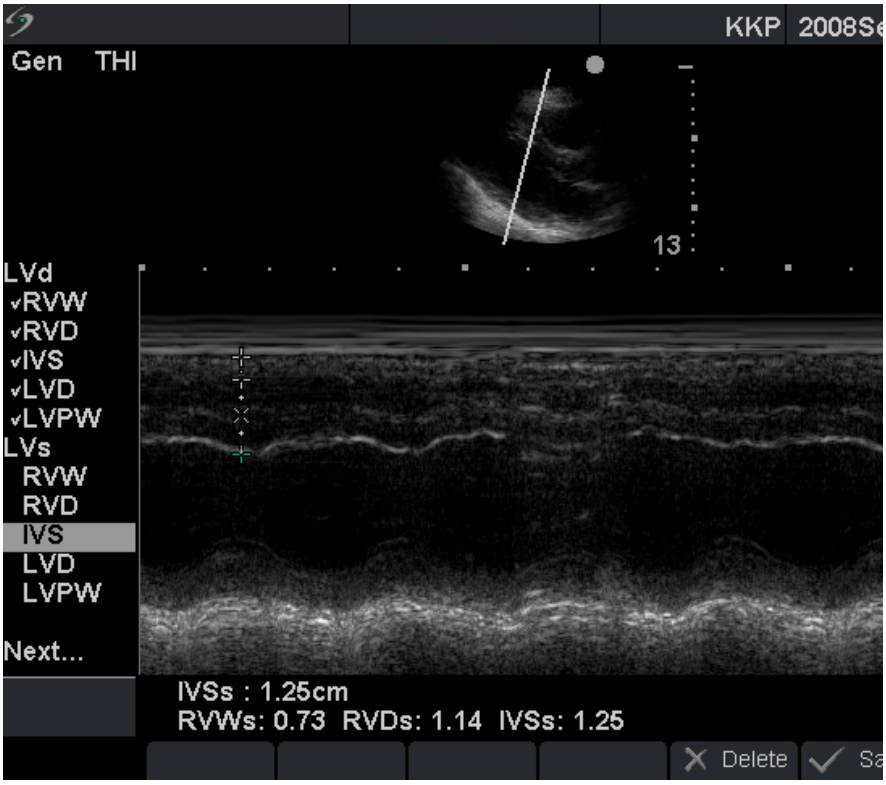

Fig.2 Systolic measurements with a caliper in progress

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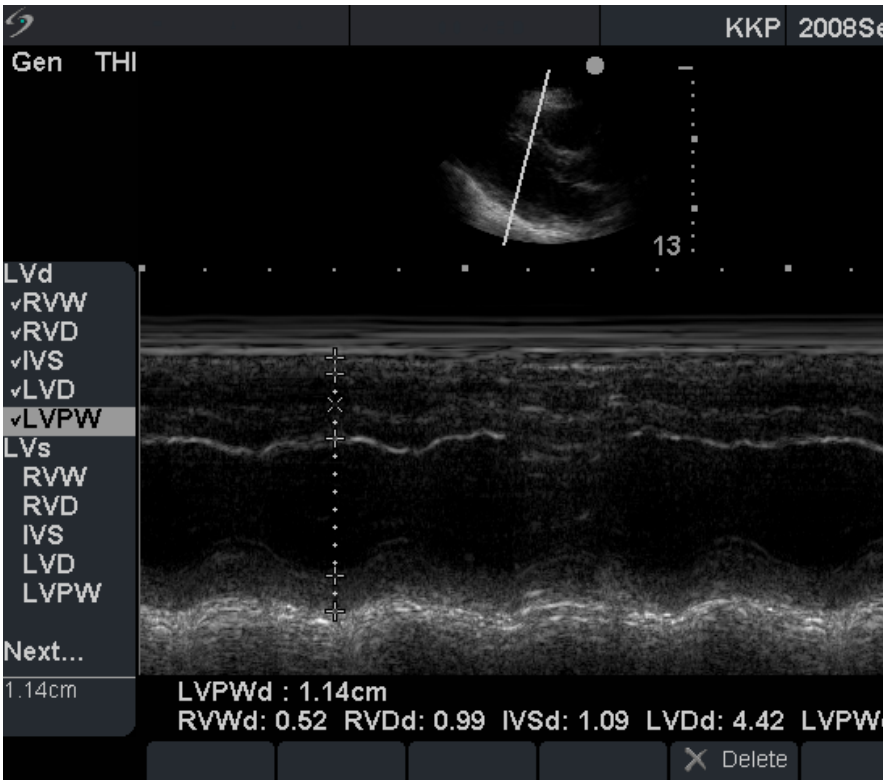

Fig.3 Diastolic measurements done with the calipers

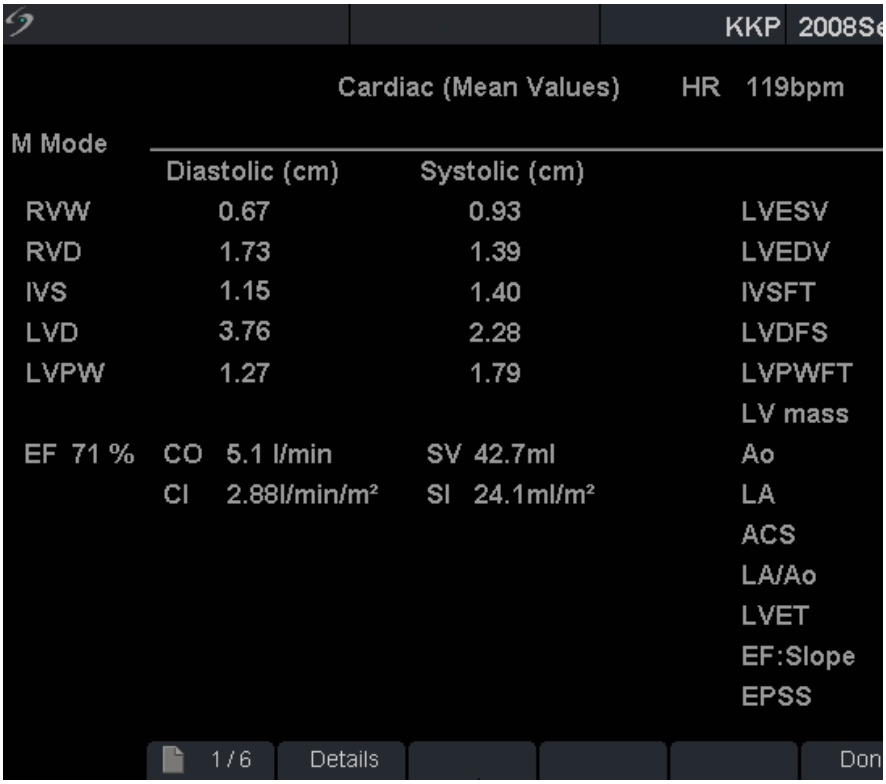

Fig.4 Report of EF and FS generated

With this information, most machines will be able to generate two numbers, the fractional shortening and the ejection fraction.
Fractional shortening is (LVEDd-LVESd) / LVEDd expressed as a percentage. The normal value is 30% to 45%.
Ejection fraction is calculated from derived volumes, which are computed based on the "cubed" or "Teichholtz" equations. The geometric assumptions made with

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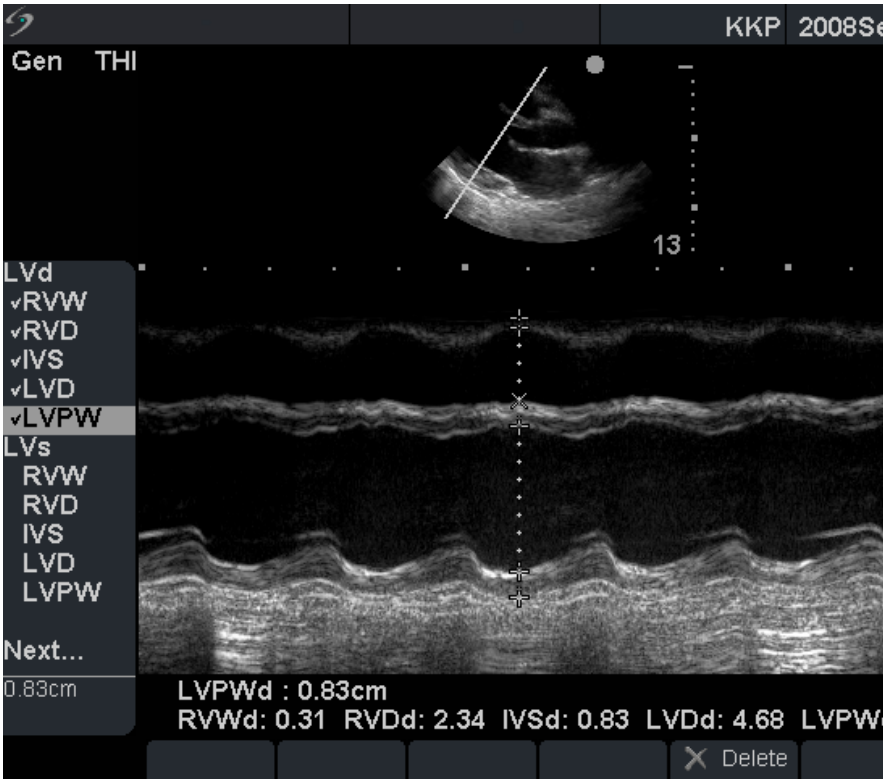
the cubed method limits its usefulness in the abnormal ventricle. In dilated and spherical ventricles, the ratio of long to short axis in the ventricle increases and LV volumes will be overestimated. Using a derived formula by Teichholz instead of the cubed equation compensates for ventricles of abnormal size but only in the absence of asynergy (no RWMA's):
The normal ejection fraction is 50% to 75%.



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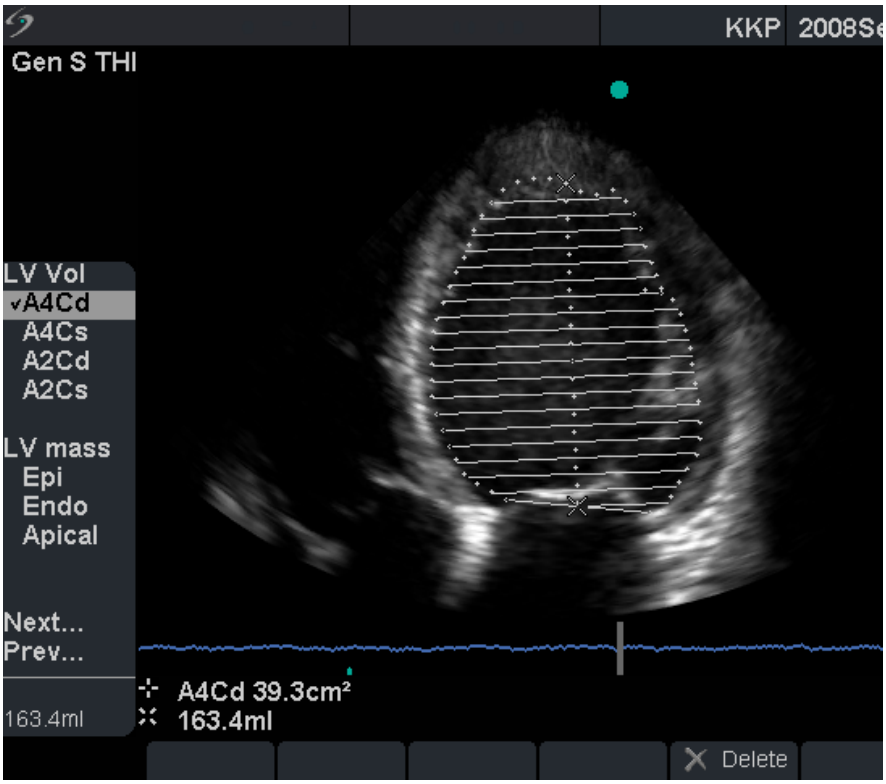

Fig.6 Calculation of LV volume in end-diastole

The frozen image is then scrolled forward or backward to identify a frame at end-systole. Again this can be done by identifying a frame where the ventricle appears to have the smallest volume, or correlating with the ECG trace, where the peak of the T wave corresponds to end-systole. Select "systolic LV volume" on the calculations menu and trace the outline of the endocardial border of the LV. Once this is done, the LV volume in systole will be calculated.

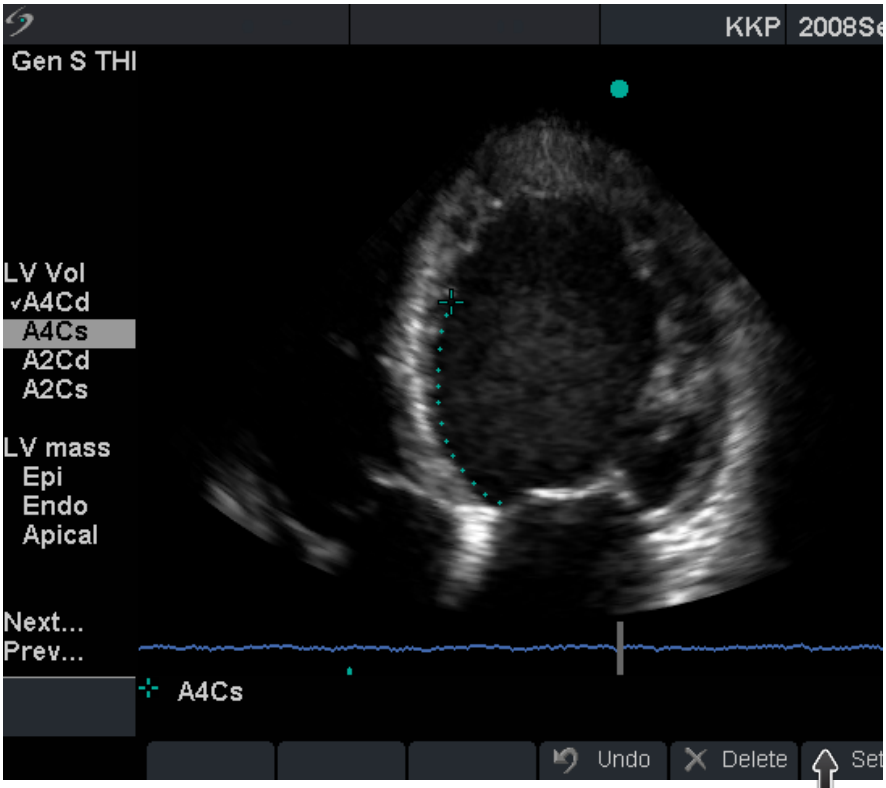

Fig.7 Tracing endocardial border in end-systole

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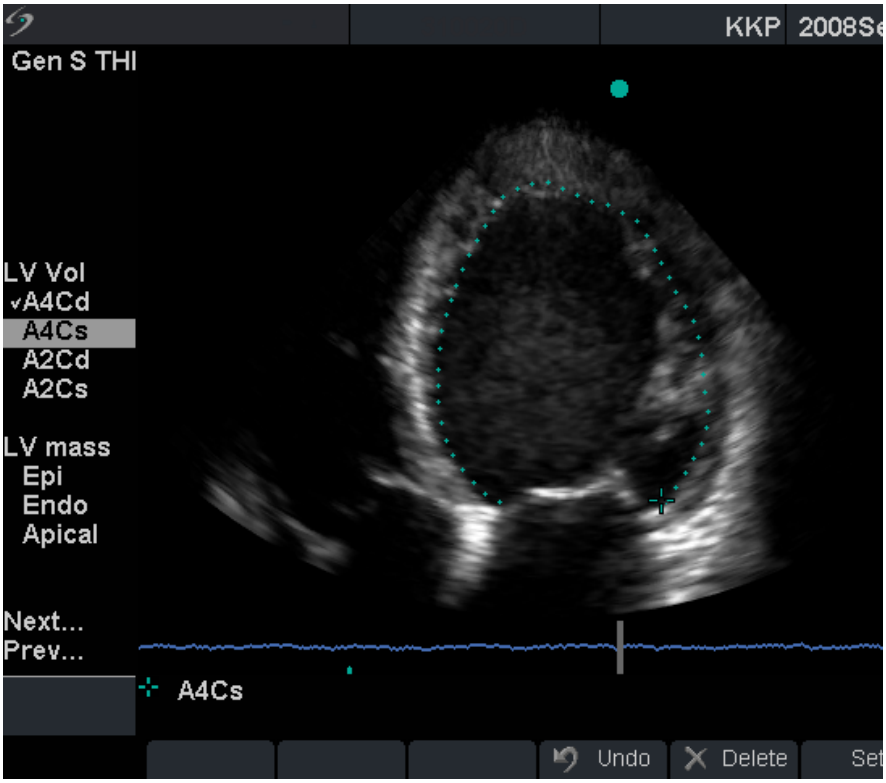
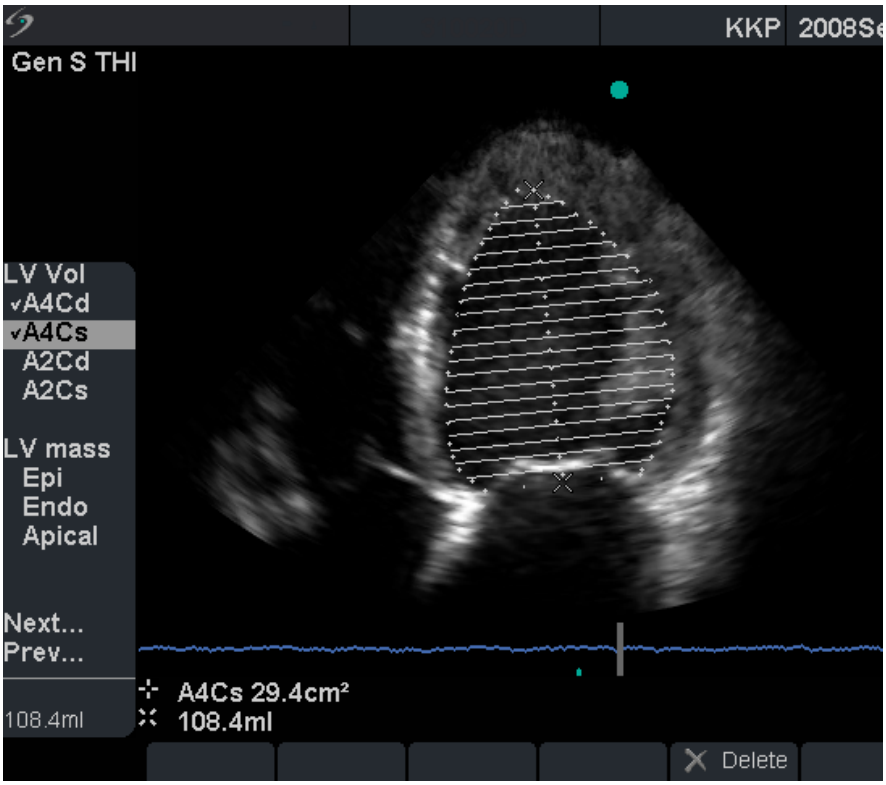


Fig.8 Tracing of endocardium in end-systole completed



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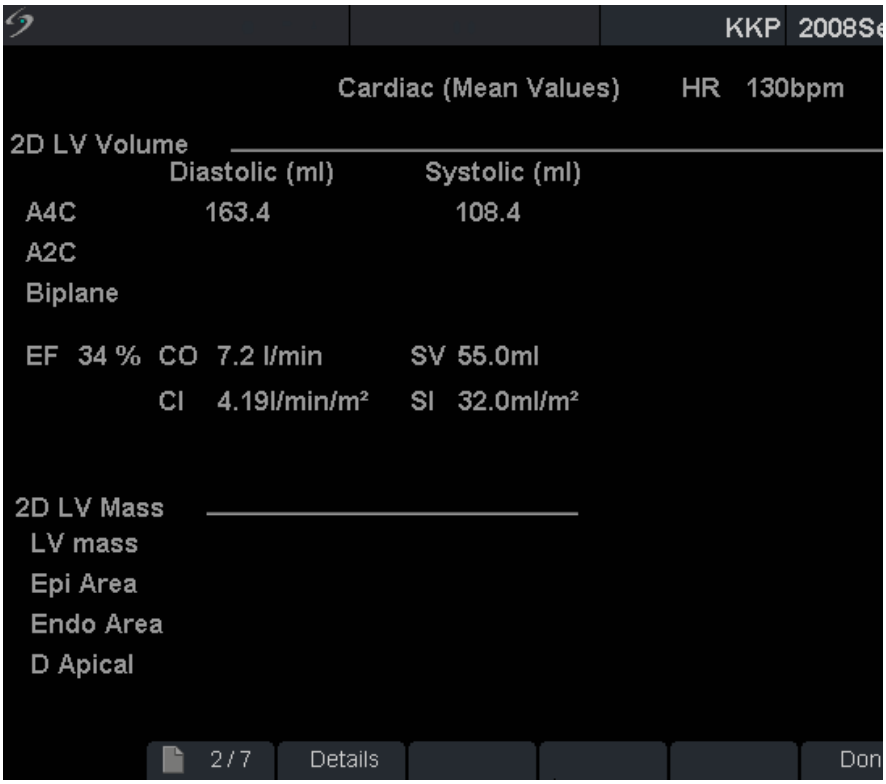

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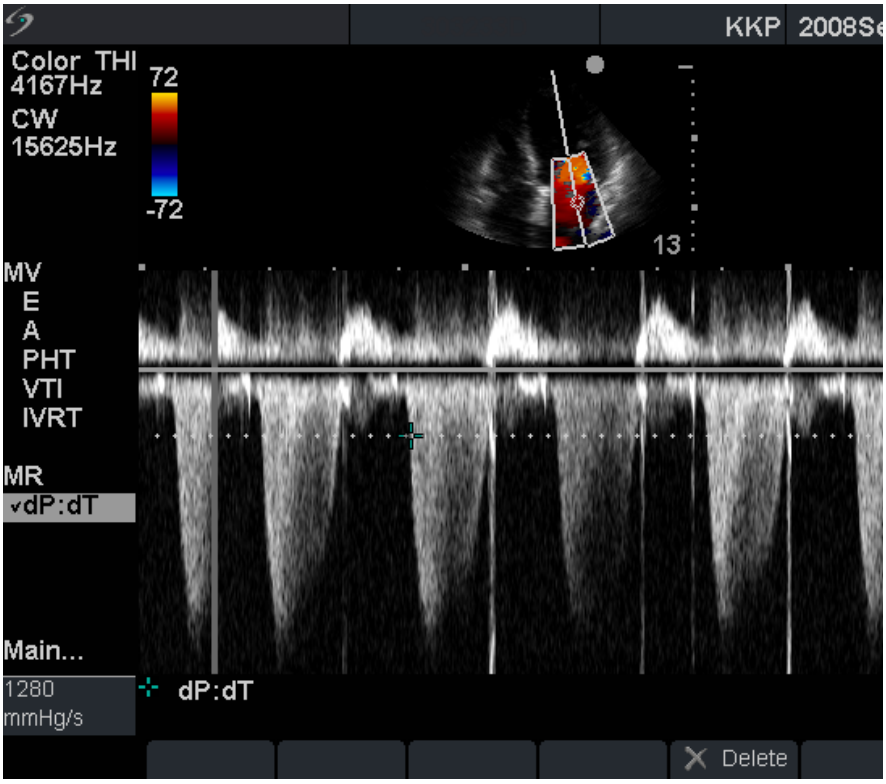

Fig.11 Placement of the first cursor point on the 1m/s line

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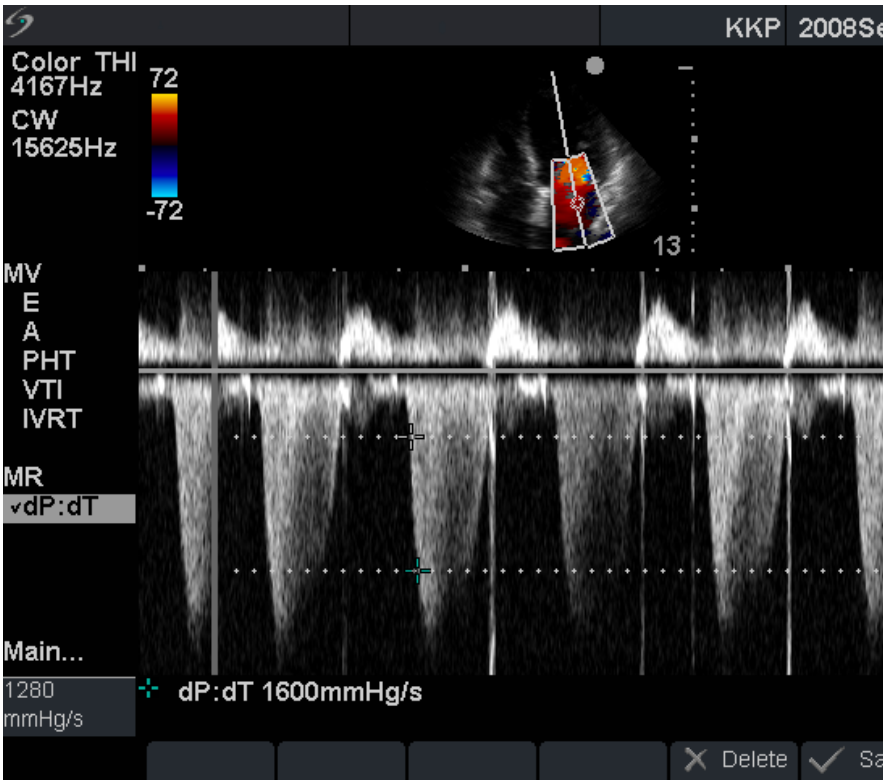
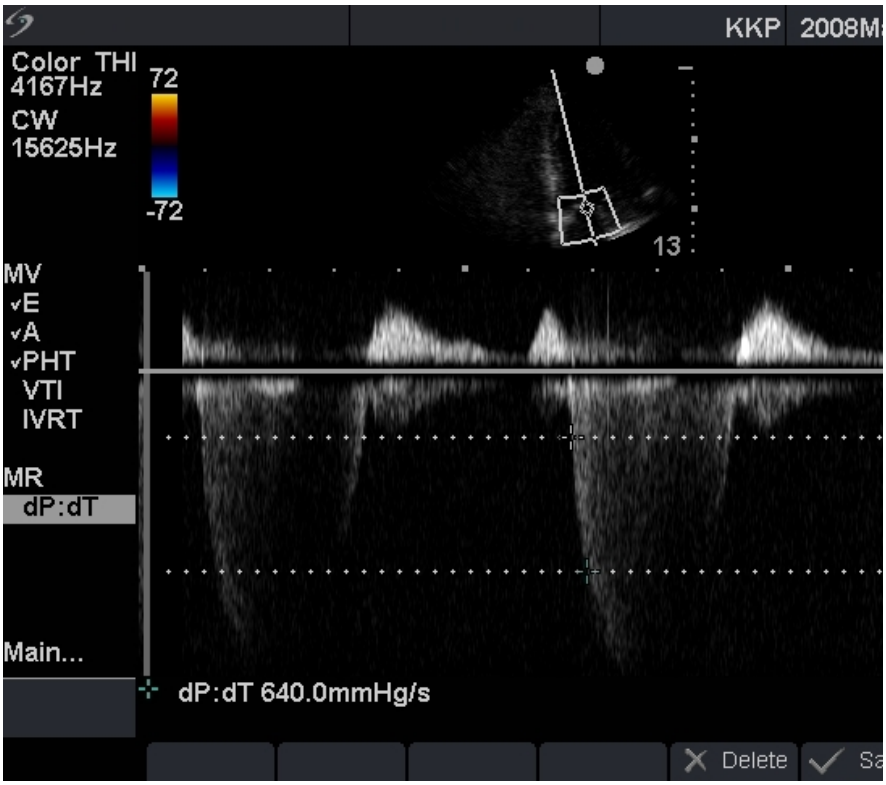


Fig.12 Placement of the second cursor point on the 3m/s line: The dP/dT in this case is normal



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Although the above techniques are useful to assess the contractility of the mocardium, what is really of interest to the intensivist is the net result of myocardial stretch and contractility....the stroke volume and cardiac output. Although cardiac output can be calculated using doppler at any of the valve orifices of the heart, the mitral or tricuspid annuli, the RVOT or the LVOT, the measurement is done most commonly at the LVOT.

First measure the LVOT diameter on a Parasternal Long Axis view. This is done by zooming into the LVOT on the PLAX view using the zoom tool and freezing the image. The images are scrolled backward and forward to capture a frame in which the aortic valve leaflets are wide open. The LVOT diameter is measured adjacent to the points of attachment of the leaflets. The machine will then calculate the cross sectional area (CSA) of the LVOT.

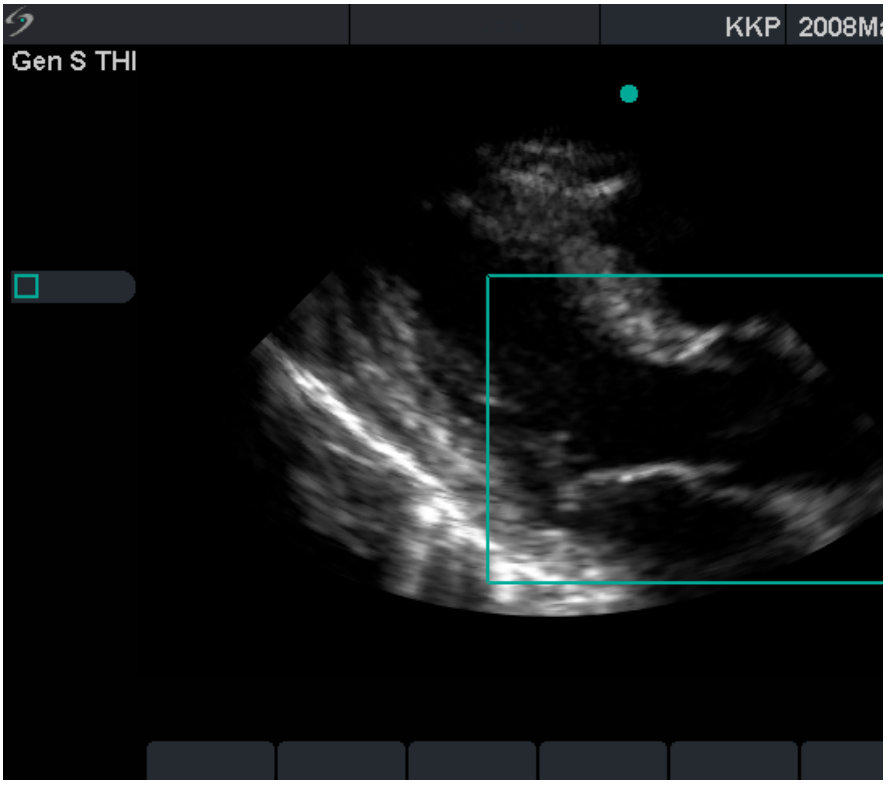

Fig.14 Zooming in on the LVOT in the PLAX view

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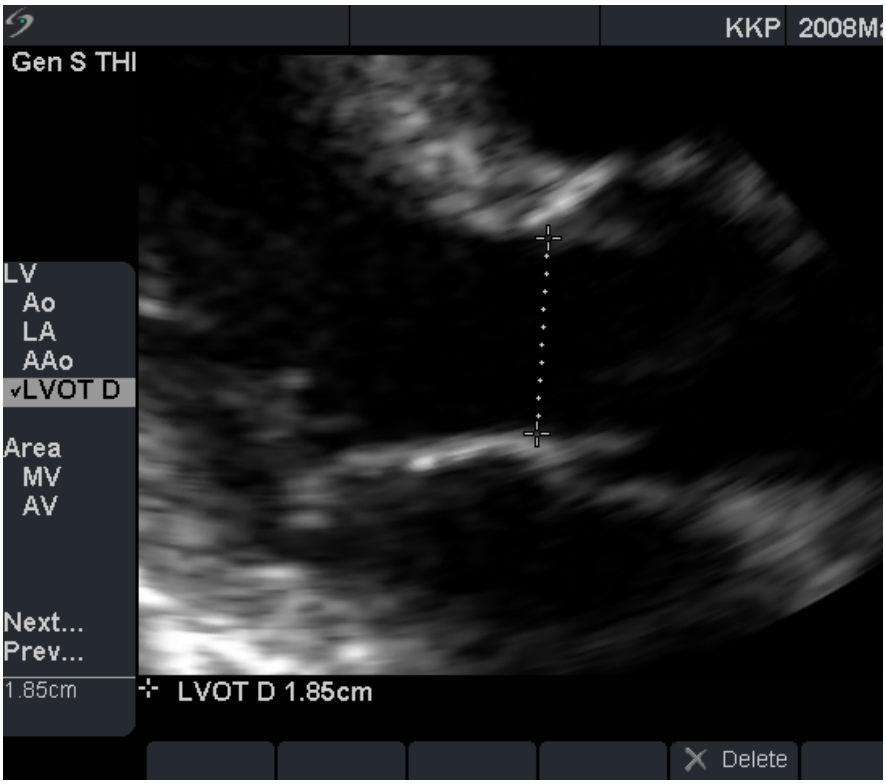

Fig.15 Measuring the LVOT diameter at attachments of the aortic leaflets

Next, obtain an apical 5-chamber view of the heart. As mentioned earlier, the A5C view is obtained from the A4C by slight anterior angulation of the transducer towards the chest wall. The 5th chamber added is the LVOT.
Place the Pulsed Wave Doppler cursor in the LVOT as close to the aortic valve as possible without including it in the sample volume. Acquire the PWD trace. The trace may be considered satisfactory if the closing click of thr aortic valve is visulaised. However, if the opening click is distincly seen before the ejection waveform, it means that the sample volume is too close to the aortic valve and needs to be moved a little away from the valve before another tracing is obtained.

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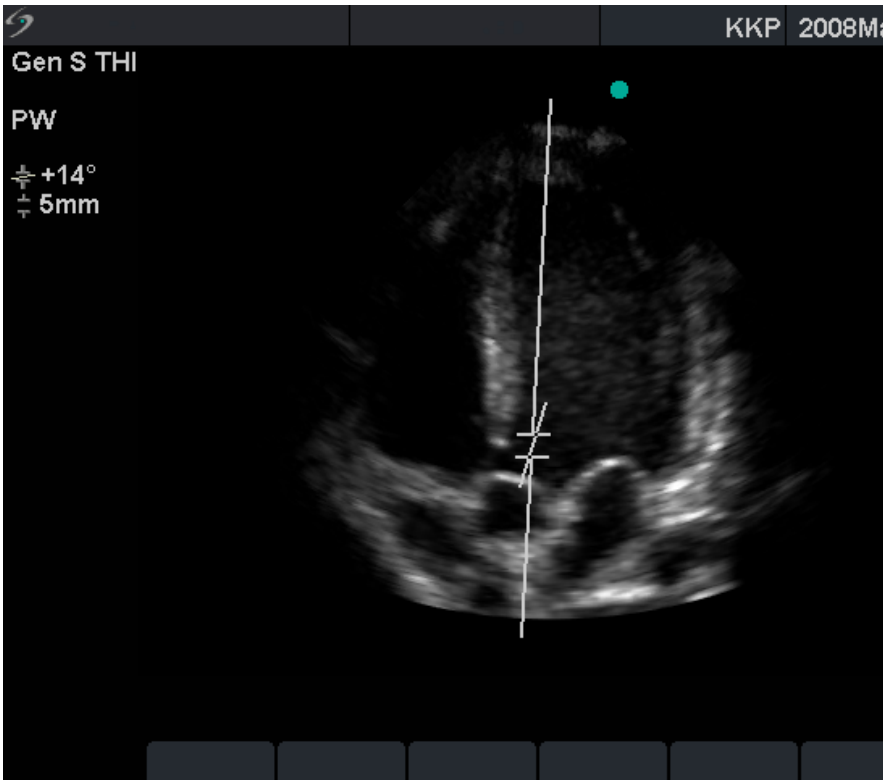

Fig16. Placement of the PWD cusor in the LVOT in A4C view

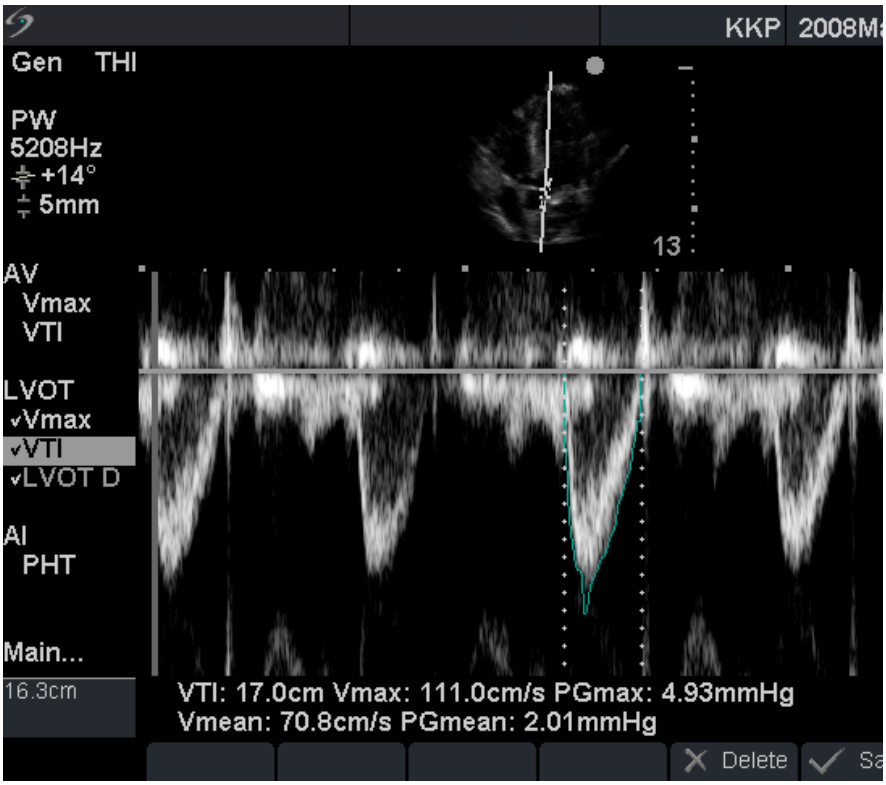

Fig.17 Tracing the PWD waveform obtained in the LVOT

Then choose LVOT VTI from the "calculation" menu and manually trace the PWD waveform. Some machines may be able to do this automatically. The machine

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will calculate the area under the curve and represent it as a Velocity Time Integral (VTI) in cms. Repeat the VTI measurement thrice to reduce sampling bias.
The stroke volume at the LVOT is then obtained by multipying the LVOT VTI with the LVOT CSA.

LVOT VTI X LVOT CSA = Stroke volume

Stroke volume X Heart rate = Cardiac output

Cardiac output = Cardiac index
Body surface area

This is a simple, non-invasive method of measuring cardiac output in ICU patients. It correlates well with measures of cardiac output obtained by thermodilution (r=0.95) with a tendency to underestimate it by about 0.24 l/min.
This measurement can be done repeatedly to see the trend of cardiac output. The LVOT CSA does not need to be calculated for repeat measurements as it does not change.

There are problems however with this technique.
1.Sometimes an adequate A5C view may not be obtainable. In such a case, an Apical 3-chamber view can be tried.
2.The LVOT may not be aligned with the direction of the PWD, leading to underestimation of velocities. In this situation, an apical 3-chamber view may sometimes offer better alignment. The other workaround is to use an angle correction factor. Although this is generally not advocated, it may be acceptable if the angle is kept to less than 20 degrees.
3.When the parasternal long axis view is not obtainable, a LVOT diameter of 2cms for males and 1.75cms for females can be assumed.
4.In patients who are taking deep breaths, the entire cardia may move with respiration making it very difficult to ensure that the PWD sample volume stays at the same place in the LVOT through the respiratory cycle. This can lead to variations in the VTI with respiration, which is not due to hypovolaemia.

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Tutorial 6 - Assessment of LV diastolic function and filling pressures

Assessment of LV diastolic function and filling pressures

Diastolic LV dysfunction is common in the intensive care unit. A knowledge of the prescence of diastolic dysfunction and its severity is useful in the optimising volume status and hemodynamics of critically ill patients. Similar to earlier assessments, a composite of different indices are used to make an assessment of diastolic function. These indices are:

1.Mitral inflow patterns : E/A, decceleration time, IVRT
2.Mitral annulus velocities on tissue doppler: E/e' ratio
3.Pulmonary venous inflow patterns

Mitral inflow patterns

E/A ratio
The flow from the left atrium to the left ventricle occurs in 3 phases: An initial rush of blood as soon as the valve opens causes a peaking of velocity in early diastole, the E wave. This is followed by a period of low or no flow, also known as diastasis. In end-diastole, atrial contraction produces a final rush of blood into the ventricle, the A wave. While these waves can be analyzed by studying the movement of the anterior mitral leaflet in M-mode, it is best done with Pulsed wave Doppler.
The PWD cursor is placed between the tips of the open mitral leaflets in the A2C or A4C views. The typical flow pattern obtained is seen below (Fig1.)

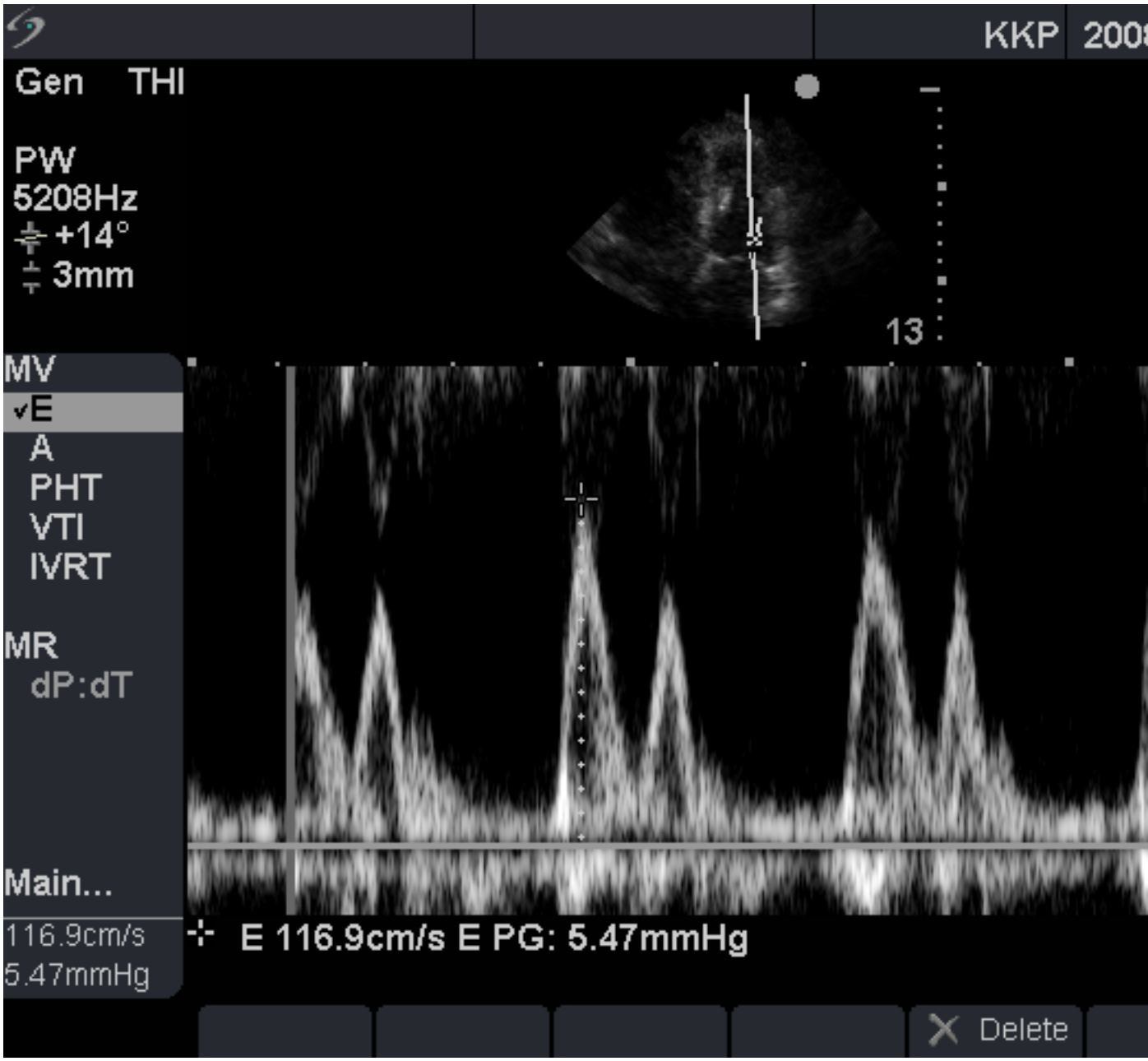

Fig.1 Normal Mitral inflow pattern

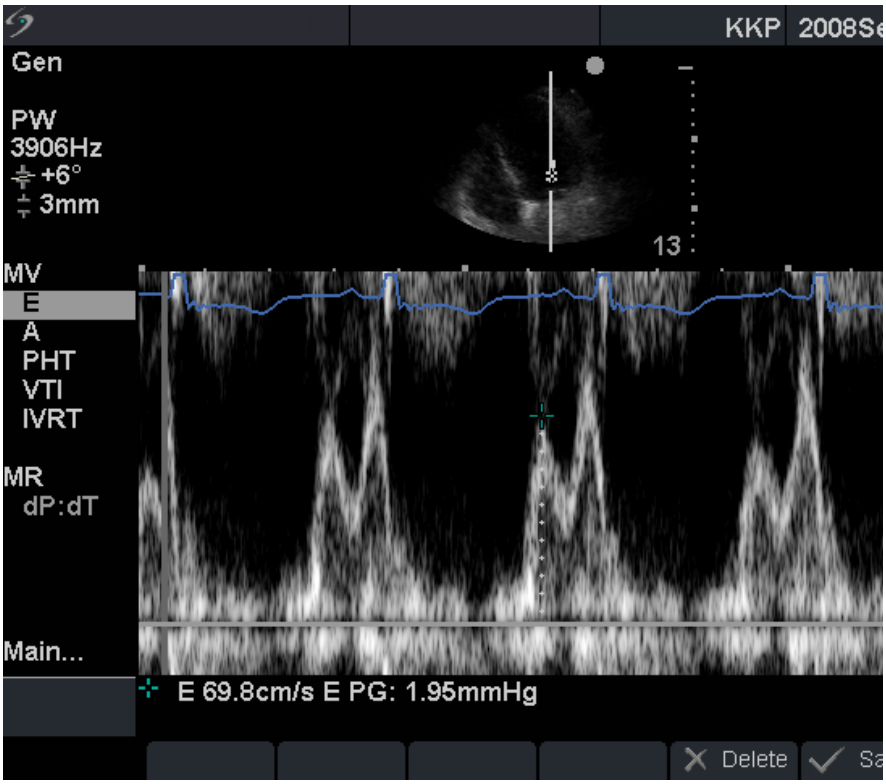

Fig.2 Measuring the E wave velocity

Obtain the mitral inflow wave pattern on PWD and freeze the image. After selecting "E" under "mitral valve" in the calculations menu, the height of the E wave is measured to get the E velocity (see above). Similarly the A wave velocity is also measured. The E/A ratio will be calculated by most echo machines automatically.

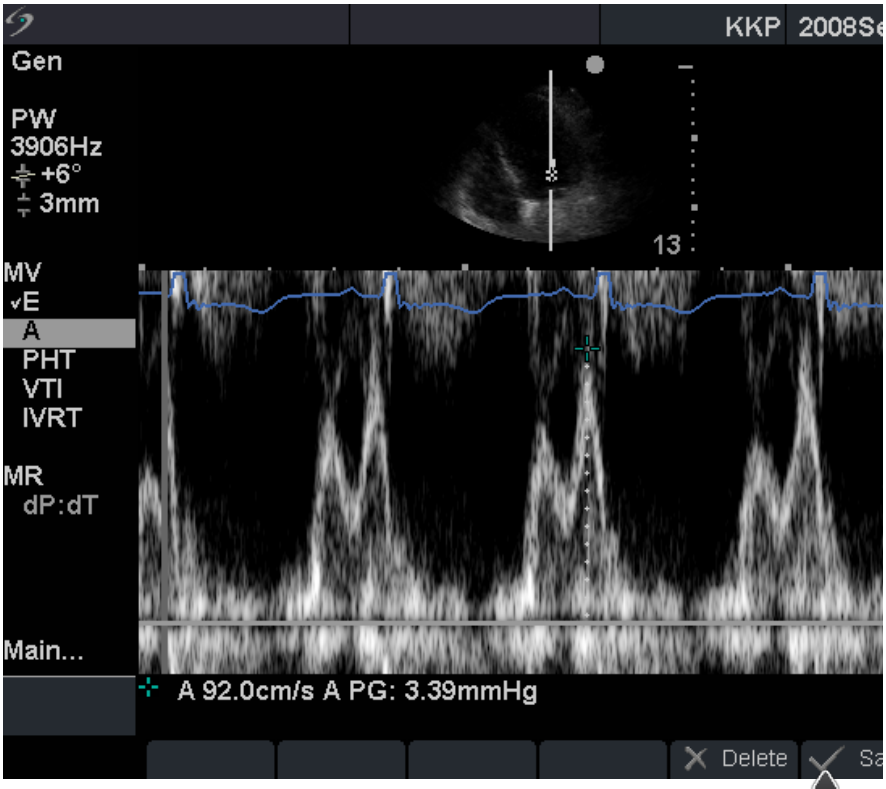

Fig.3 Measuring the A wave velocity

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Decceleration time (Dct)

Obtain the mitral inflow wave pattern on PWD and freeze the image. After selecting Dct under "Mitral Valve" in the calculations menu, the cursor is first placed at the peak of the E wave. On pressing select, another cusor point appears, connected to the first with a line. Pull this cursor point to the baseline and move it around till the line connecting the two points aligns itself along the downslope of the E wave. Sometimes, only the upper part of the line may actually be in contact with the E waveform. This is acceptable as long as the slope of the line faithfully reflects the slope of the E wave. The machine automatically calculates the decceleration time from this.

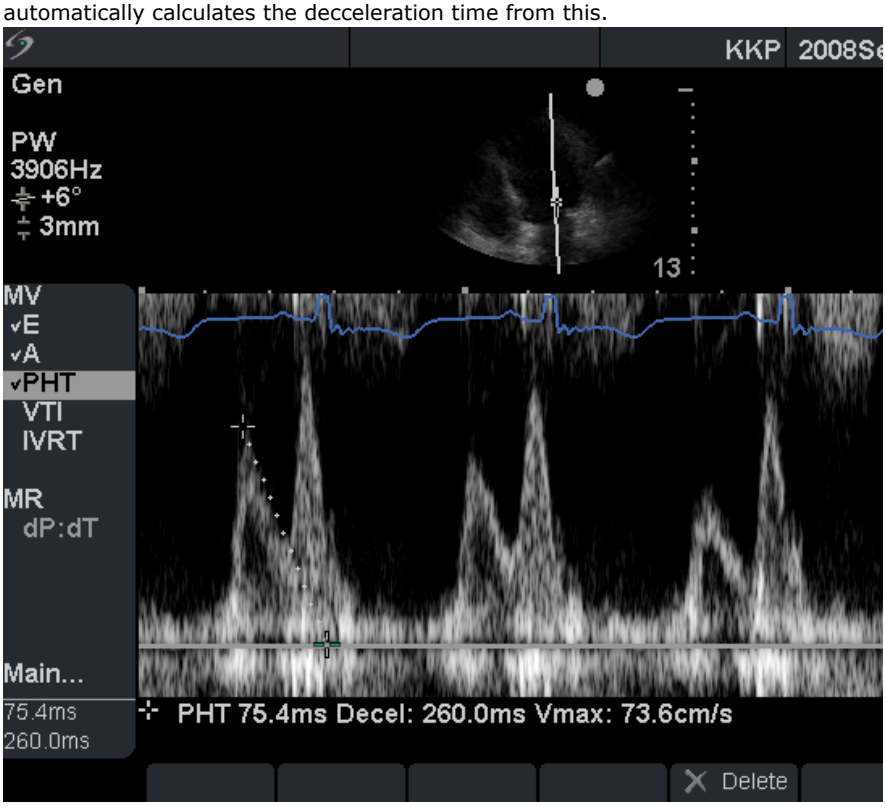

Fig.4 Marking the slope of the E wave to measure Dct

E/e' ratio

In this the diastolic peak velocities of the mitral annulus, are measured both medially and laterally using tissue doppler. These peak velocities are designated e' (medial) and e' (lateral).
This is done by first acquiring an A4C view. PWD is selected and the tissue doppler imaging (TDI) is switched on. A 2mm to 5mm sample volume is placed over the medial mitral annulus, at the base of the mitral leaflet, as shown below (fig.5)

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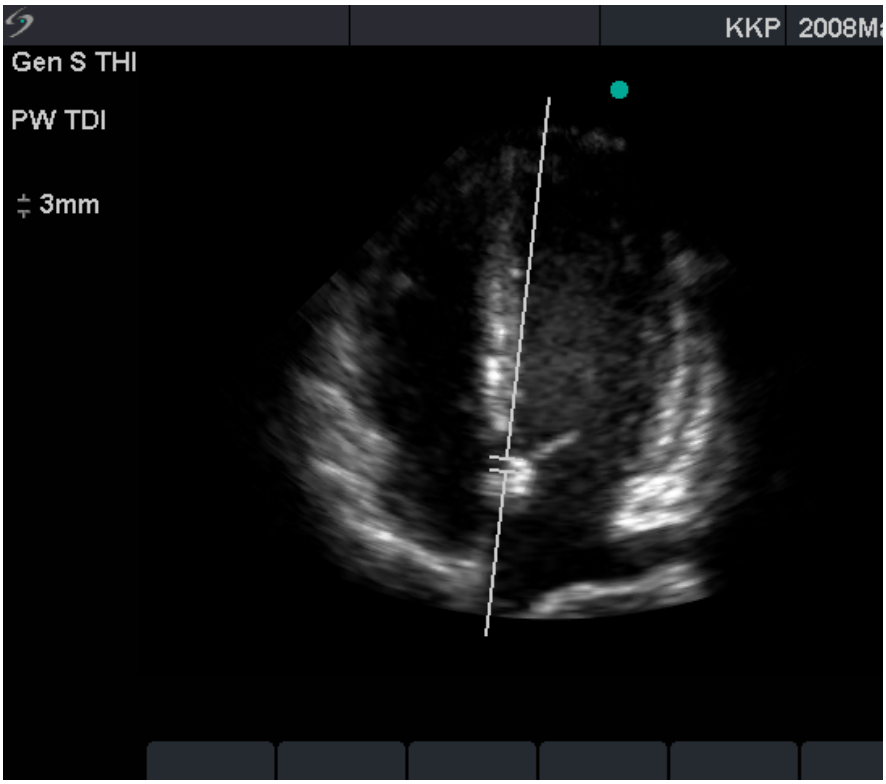

Fig.5 Placement of the TDI cursor on the medial mitral annulus

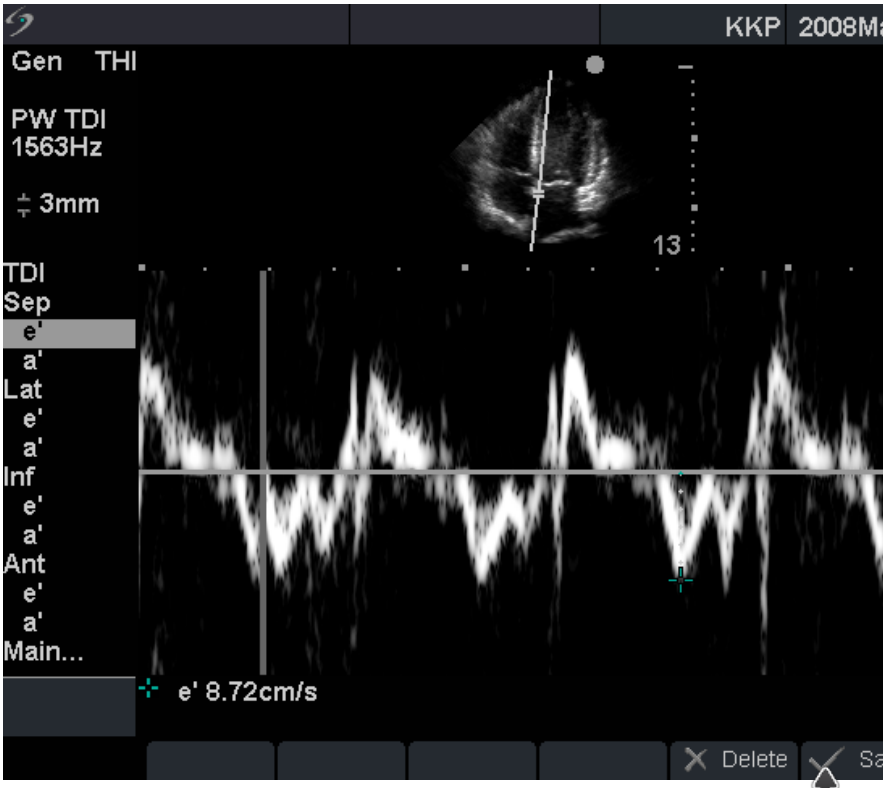

Fig.6 Measuring e' on the medial annulus tissue doppler trace

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Typically, 2 negative waves in diastole and 1 positive wave in systole is seen. The first of the diastolic waves is the result of movement of the annulus towards the left atrium during initial filling of the LV. This wave is referred to as e'. The second diastolic wave is referred to as a'. The systolic wave is labelled s'.

Once the waveform is acquired, freeze it and choose "e' (medial)" under "TDI" in the calculations menu. Measure the peak e' velocity with the cursor. Then, unfreeze and place the TDI cursor over the lateral mitral annulus in the A4C view, at the base of the posterior mitral leaflet.

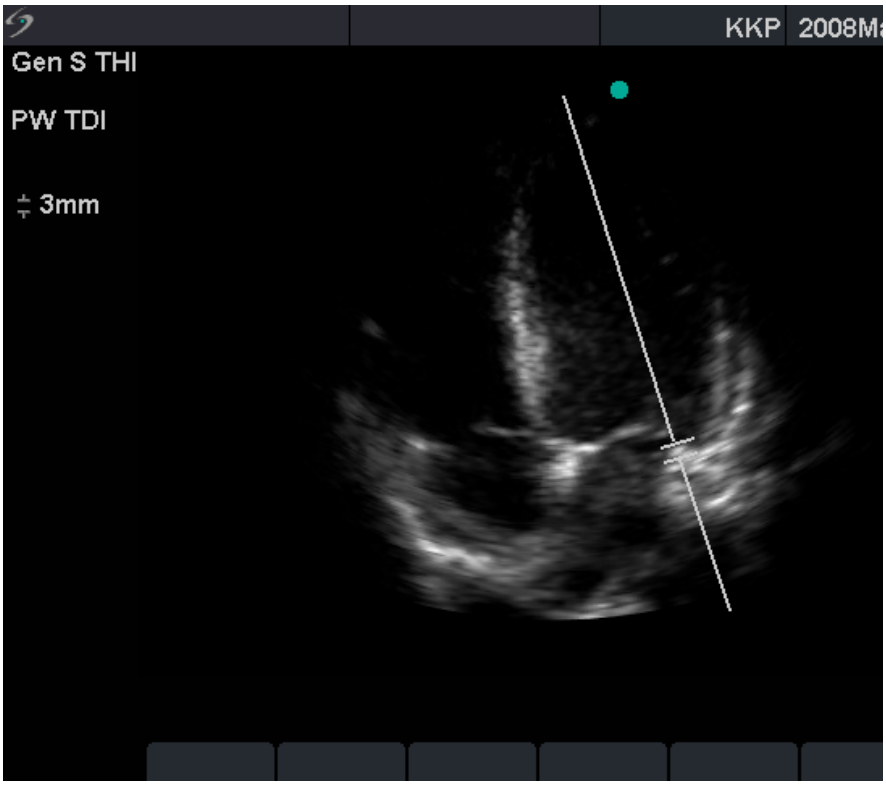

Fig.7 Placement of the TDI cursor on the lateral mitral annulus

Repeat the above procedure for the measurement of e' (lateral). Since E is already known from earlier measuremants of the mitral inflow, E/e' ratio-medial and lateral can be calulated. Many of the newer echo machines offer this calculation automatically.
Normally the e' velocities from the lateral mitral annulus are higher(15cm/sec) than those from the medial annulus.

Pulmonary vein inflow

This is studied by placing a pulsed doppler cursor at the entry of the pulmonary veins into the left atrium identified on an apical 4 chamber view. The use of colour flow imaging may help to locate this as a jet of flow can be seen entering the left atrium from the pulmonary veins. The opening of the pulmonary veins are often difficult to identify clearly on a transthoracic examination. Once identified, a PWD trace is obtained and the trace is frozen.
The normal pulmonary vein flow profile is usually biphasic with a predominant systolic forward flow (S wave) and a less prominent diastolic forward flow wave (D wave). Occasionally, there may be a triphasic flow pattern with two distinct systolic flow waves of which the initial flow into the left atrium results from atrial relaxation followed by a further inflow due to the increase in pulmonary venous pressure. The D-wave occurs when there is an open conduit between the pulmonary vein, LA and LV and reflects the transmitral E wave. A retrograde flow wave into the pulmonary vein (AR wave) occurs during atrial contraction and its amplitude and duration are related to LV diastolic pressure, LA compliance and heart rate.
See fig.8 for the timing correlates of this waveform

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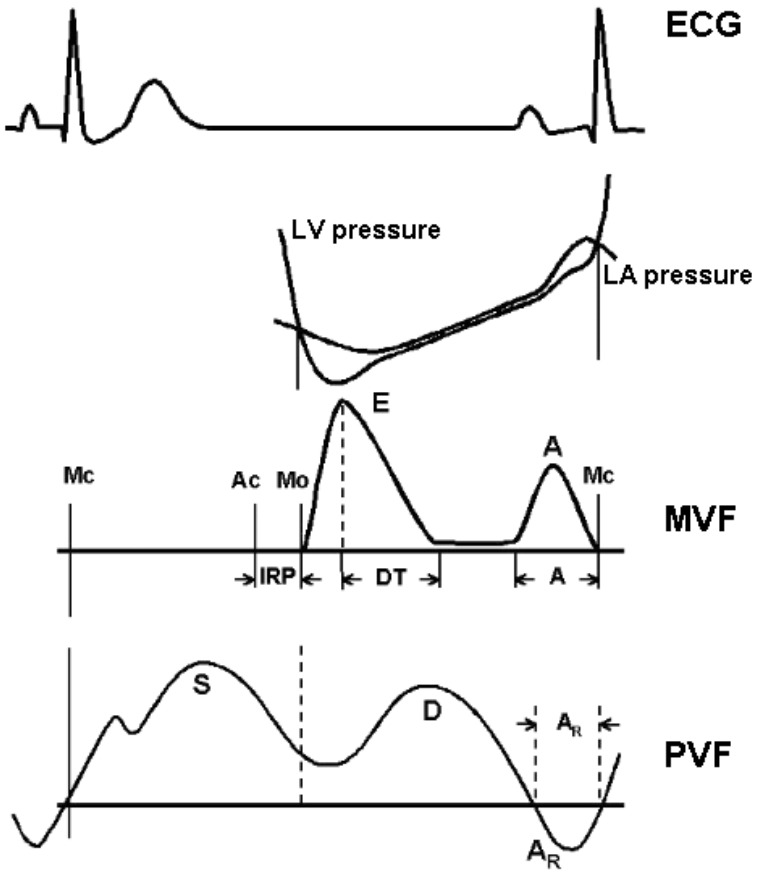

Fig.8 Timing correlates of LA pressure, mitral inflow and pulmonary venous flow: http://www.fac.org.ar/scvc/llave/echo/roeland/roelandi.htm

Interpretation of measured variables

Mitral inflow patterns:

The normal E/A ratio is between 1 and 2. This gradually reduces with age and a E/A ratio >0.75 may be considered normal above 75 years. The normal decceleration time of the E wave is 160 - 240ms.

E/e':

The normal E/e' ratio from the medial annulus is <8 and suggests a normal left atrial pressure. While values between 8 and 12 are indeterminate, a value >12 is indicative of an elevated left atrial pressure or PCWP (>18mmHg). The ranges for E/e' from the lateral mitral annulus are <5, 5 -10 and >10 respectively.

Pulmonary venous flow profile:

The 2 components to note are the diastolic forward flow (D) and the diastolic flow reversal (AR). The D wave is normally equal to or smaller than the S wave. Changes in the D wave with increases in LA pressure parallel changes in the transmitral E wave, initially decreasing and then increasing to become much larger than the S wave. A S/D ratio of < 40% suggests a LA pressure more than 20mmHg. The decceleration time of the D wave also shortens with increasing LA pressures.

The atrial reversal wave increases in amplitude and duration with increasing LA

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pressures. An AR amplitude more than 25cms/sec and a AR duration 30ms more than the transmitral A wave duration suggest a LA pressure more than 20mmHg.
Hepatic venous flow profile is similar to the pulmonary venous flow profile and is an indicator of Right Ventricular filling patterns. (see fig.9)

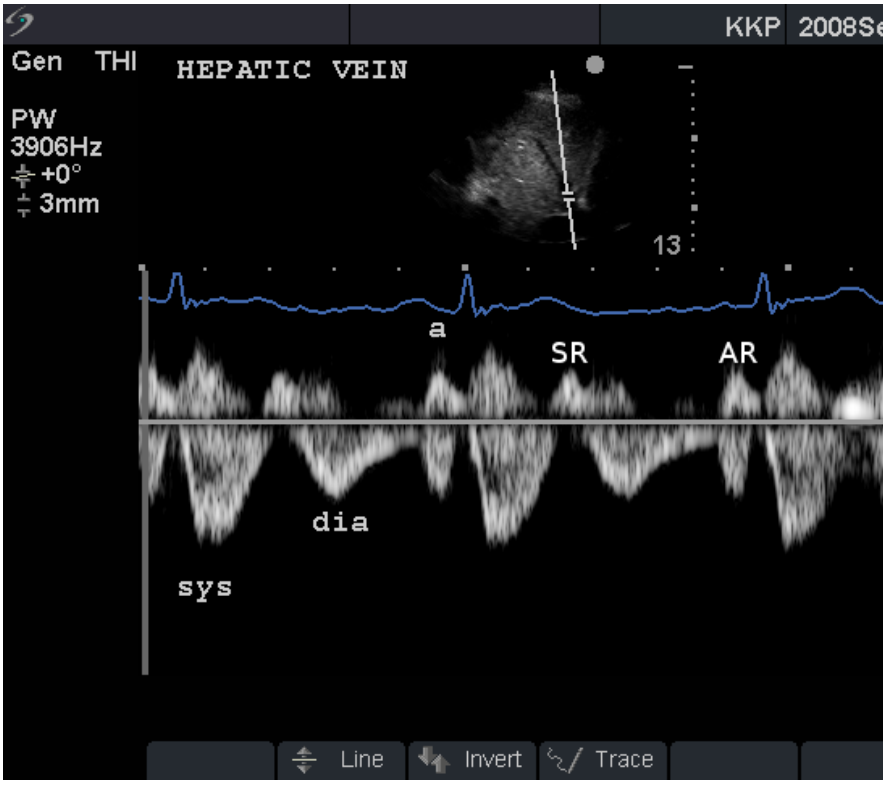

Fig.9 Hepatic venous flow profile

Grading of diastolic dysfunction or diastolic filling pattern

Diastolic filling abnormalities can be graded as follows:

Grade 1: Impaired relaxation pattern with normal filling pressures
Grade 1a: Impaired relaxation pattern with elevated filling pressures
Grade 2: Psuedonormalized pattern
Grade 3: Reversible restrictive pattern
Grade 4: Irreversible restrictive pattern

Grade 1 diastolic dysfunction (Impaired myocardial relaxation)

In diastolic dysfunction from any cause, the initial abnormality is impaired myocardial relaxation. The common causes are increasing age, LV hypertrophy and myocardial ischemia.
The E/A ratio is < 1, with a prolonged Dct (>240ms).
In the tissue doppler assessment, e' is also reduced with a resultant E/e' ratio (medial) <8, suggesting a normal LA pressure.
The D wave of the pulmonary venous inflow is smaller than the S wave and the AR wave is normal.

Grade 1a diastolic dysfunction (Impaired myocardial relaxation with elevated filling pressures)

The pattern is similar to Grade 1, with the exception of the E/e' ratio (medial) which is > 15, suggestive of a high LA pressure. See fig.10 and 11

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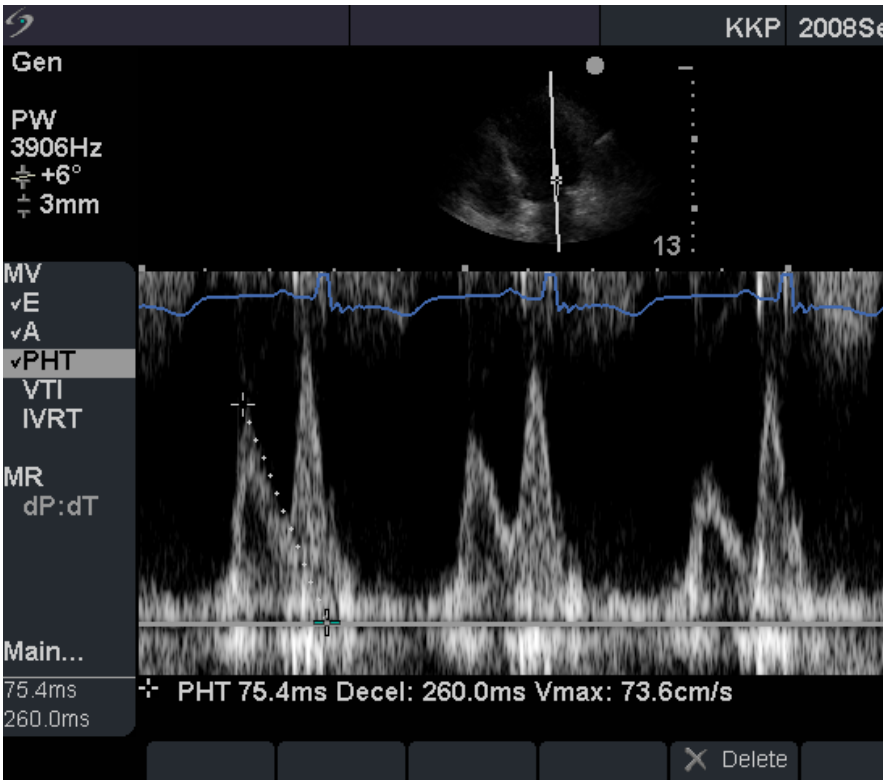

Fig.10 The E/A ratio is < 1, with a prolonged Dct

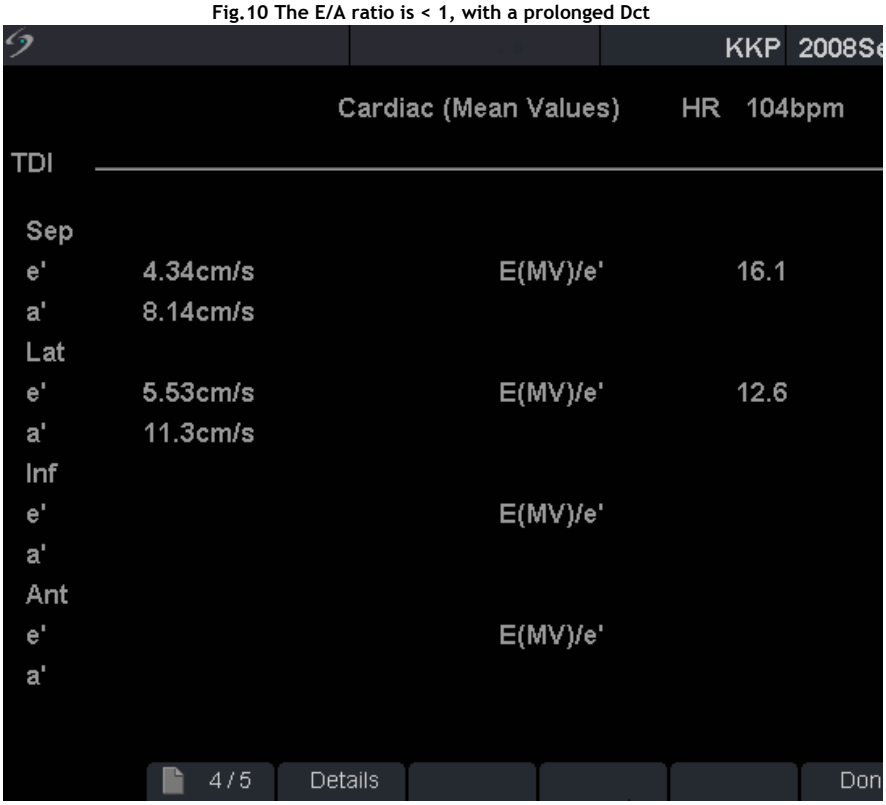

Fig.11 E/e' (medial) >15

Grade 2 diastolic dysfunction (Psuedonormalized pattern)

When diastolic LV function deteriorates, LV compliance progressively decreases and there is an increase of LA pressure and the diastolic filling pressure. The transmitral E wave velocity progressively increases and the Dct decreases. As it does so, it goes through a phase that resembles a normal filling pattern. The

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E/A ratio is between 1 and 2 and the Dct between 160 and 240ms. This pseudo-normal pattern is a transition pattern from impaired relaxation to restrictive filling and is a result of a moderately increased LA pressure superimposed on a relaxation abnormality. The following clues help distinguish this from a normal filling pattern
E/e' ratio (medial) >15
Pulmonary venous flow AR >25cm/sec and longer than transmitral A wave
Presence of LA enlargement or LV hypertrophy

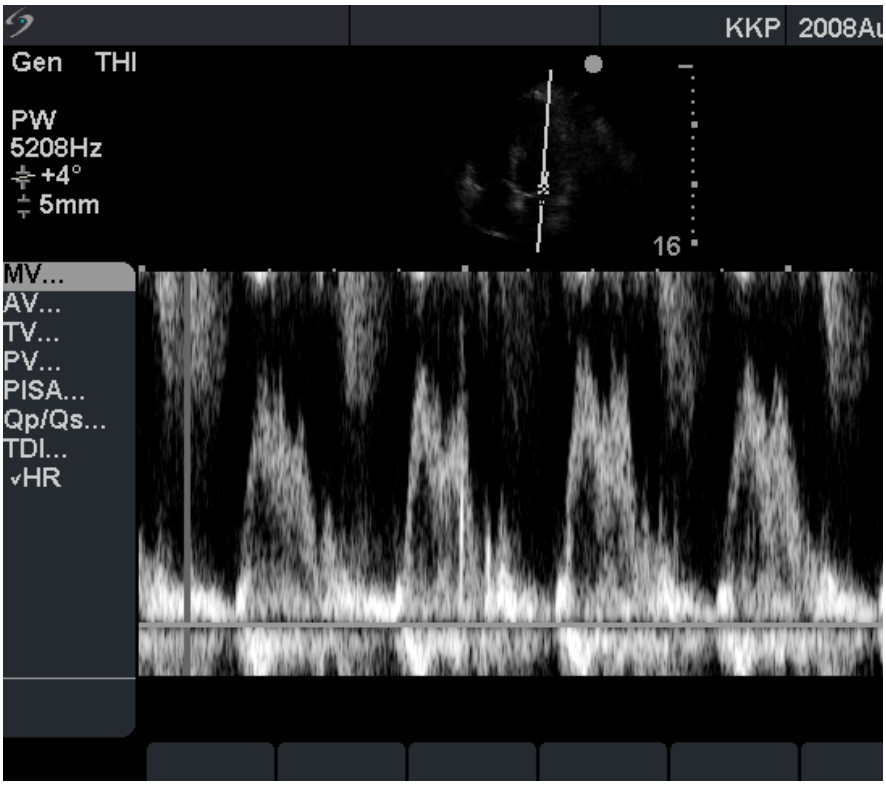

Fig. 12 E/A >1.0

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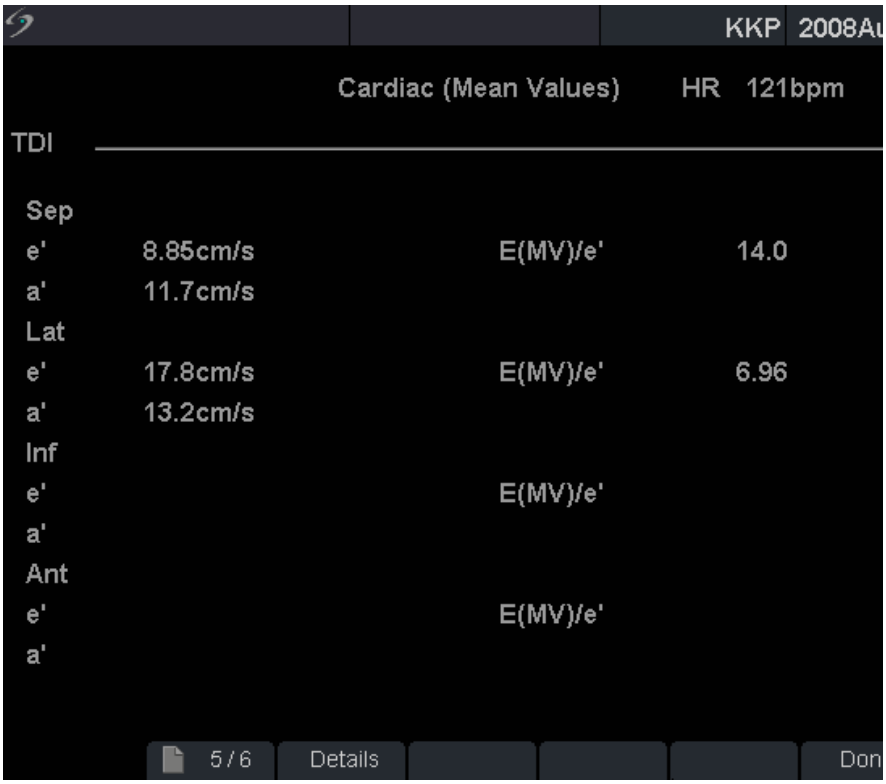

Fig. 13 E/e' is elevated but just short of meeting criteria for grade 2

Grade 3 and 4 diastolic dysfunction (restrictive pattern)

With more severe diastolic dysfunction, LV compliance reduces and LA pressures rise. The low compliance of the LV causes a rapid increase in the early LV pressure and a shortened inflow and DT. The E/A ratio is > 2. Dct is < 160ms. The high LA pressure manifests as a E/e' ratio >15 at the medial annulus. Forward diastolic pulmonary vein flow stops in mid-late diastole and during atrial contraction there is a significant flow reversal resulting in a prolonged AR. A reversal to grade 1 or 2 on reducing the preload by performing Valsalva manouvre or administering nitroglycerine suggests reversibility of the cardiac restriction and is termed grade 3. Diastolic filling should be graded as irreversible (grade 4) in the absence of such a reversal.

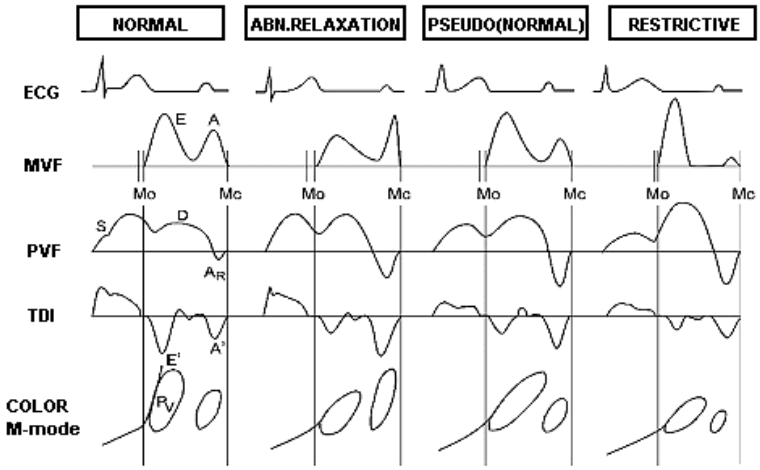

Fig.14 Represents the different diastolic filling patterns: http://www.fac.org.ar/scvc/llave/echo/roeland/roelandi.htm

http://www.fac.org.ar/scvc/llave/echo/roeland/roelandi.htm

Assessment of diastolic function in atrial fibrillation and

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fused EA waves

Atrial fibrillation is common in the critically ill and eliminates the use of the E/A ratio as the A wave is absent. The decceleration time varies as it is dependent on the cardiac cycle length. The only parameter that can be used with confidence is the E/e' ratio.
In sinus tachycardia, the E and A waves frequently fuse, making assessment of diastolic filling pattern based on transmitral flows difficult. In this situation, the E/e' ratio correlates with filling pressures well.

Estimation of LV filling pressures

Other than in the grading of diastolic dysfunction, estimation of LV filling pressures also influence desicions on fluid resuscitation in hemodynamically unstable patients and in titrating diuretics and fluids in patients with diastolic heart failure. While the markers of an elevated LV filling pressure have been mentioned earlier and tabulated below, several formulae have been evaluated to arrive at a numerical value.

Markers of elevated LV filling pressure:

E/A > 2
Dct < 160ms
E/e' (medial) >15
E/e' (lateral) >10
PFV S/D <40%
PFV AR amplitude >25 cm/sec
PFV AR duration > 30ms more than A wave
LA enlargement
LV hypertrophy

Formulae to calculate LA pressure

Sinus rhythm
2+1.2(E/e')

Sinus tachycardia
1.5+1.5(E/e')

Atrial fibrillation
6.5+0.8(E/e')

The E/e' included in the above calculations indicates that obtained from the medial mitral annulus.

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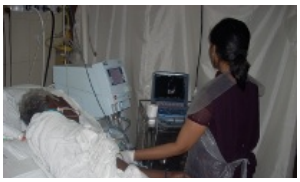

ICU Sonography

Demystifying sonology in intensive care

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Tutorial 7 - Assessment of the right heart

Assessment of the Right heart

The right heart assessment clinically and echocardiographically is not a very important part of mainstream cardiology. In the ICU, however, acute right heart failure is common and assessment of the right heart is sometimes the only way to diagnose pulmonary embolism, a common diagnosis in the critically ill. The presence of RV dysfunction is independently associated with an increased mortality. It is also important to diagnose right heart failure in the ICU as its management is completely different from that of left heart failure.

In ICU echocardiography, the right heart is assessed along three lines:

Detecting enlargement of the RA and the RV
Assessment of RV function
Estimation of pulmonary artery pressures

Detection of RA and RV enlargement

Since the assessment of the right heart does not entail a separate set of views, it is done as part of the usual view sequence. Hence the signs of RA, RV enlargement will be described in the various views.

Parasternal long axis view

If the RV cavity is visualised well on the parasternal long axis view, its largest diameter can be measured with a caliper. If this exceeds 30mm, it indicates a dilated RV. The key however, is getting a plane the right ventricular cavity is well represented. This might be easier to achieve by using a lower intercostal space than the usual PLAX and angulating the beam a little toward the left shoulder. Failure to do this might result in underestimating the RV internal diameter.

Parasternal short axis view

Typically this view shows the round LV with the thin, crescenteric RV cavity anterior and to the right of it hugging the round LV contour. The RV cavity cross section is smaller than the LV cavity cross section.

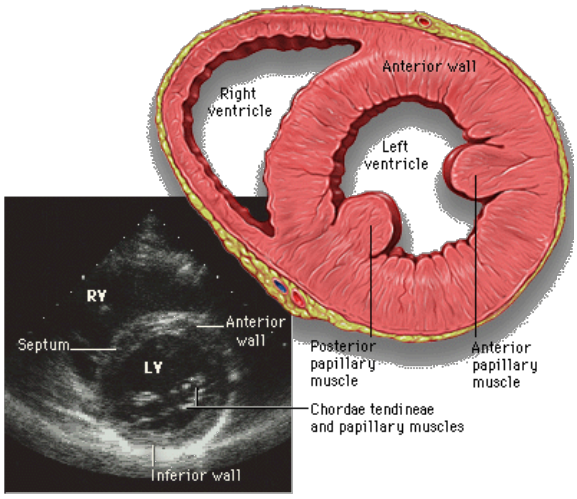

Fig.1 Normal appearance of RV in SAX: http://www.med.yale.edu/intmed/cardio/echo_atlas/contents/index.html

When the RV dilates, 2 things are noticed. First, the RV cross section becomes as large as, or larger than the LV cross section. Secondly, the RV cavity becomes more rounded with the IVS moving into the LV cavity during part (early diastole) or whole of the cardiac cycle. This causes the typical round cross section of the left ventricle to become "D-shaped".

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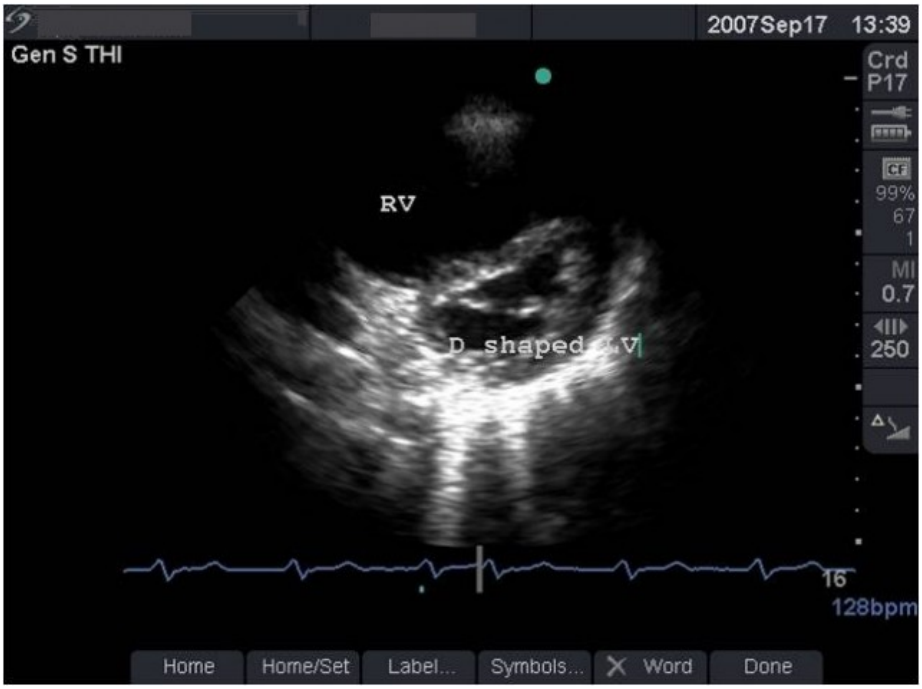

Fig.2 Enlarged, rounded RV with flattening of IVS in diastole

Apical 4 chamber view

Normally, the right ventricular cavity is smaller than the left ventricular cavity in this view. If the view is frozen in end diastole and the cavities traced with a caliper, the areas of the two ventricular cavities can be recorded. A ratio of RV/LV area of 0.6 to 1.0 suggests mild enlargement of the right ventricle, while a ratio of 1 to 2 suggests severe and >2 suggests extreme enlargement of the RV. However, it is tedious to do and if the RV is not opened out enough by turning the probe to achieve an exact 4-chamber view, the RV area may be underestimated. More practically, the RV can be said to be significantly enlarged if the RV cavity appears to be as large as or larger than the LV cavity on this view. In addition, the RV loses its usual triangular shape and becomes more oval. In severe RV enlargement, the RV apex may extend beyond the LV apex.

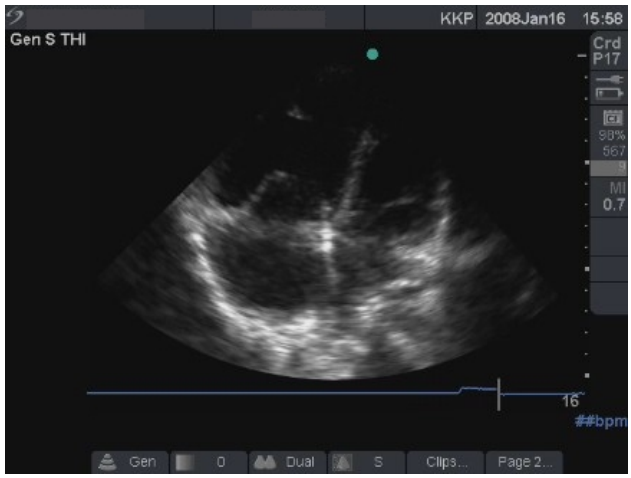

Fig.3 RA and RV enlargement in a patient with pulmonary embolism

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Video 1. The RA and RV are grossly dilated compared to the LA and LV

Subcostal view

In the subcostal 4 chamber view, a comparison of the areas of the RV and LV cavities can be made as described in the A4C view, either by measurement or visual gestalt. In addition, this view affords a good look at the RV free wall. RV free wall thickness more than 0.5cm at end-diastole suggests RV hypertrophy, which is a response to chronic pressure overload.
The subcostal view also affords a good view of the interatrial septum. Normally the IAS is curved, bulging into the RA. However, in patients with right heart overload, the RA is enlarged and the IAS curves towards the LA (see video).

Video 2. Subcostal view showing IAS bulging into the LA, with ballooning of the floor of the fossa ovalis. The round chamber on the left is the right atrium, with the partially visible left atrium to the right

25% of the population have a probe patent foramen ovale. In such patients, an increase in RA pressures can cause a small right to left shunt through it. To look for this, a CFI box is placed over the IAS in the subcostal view. Flow can usually be appreciated moving away from the probe into the LA (see Fig.4).

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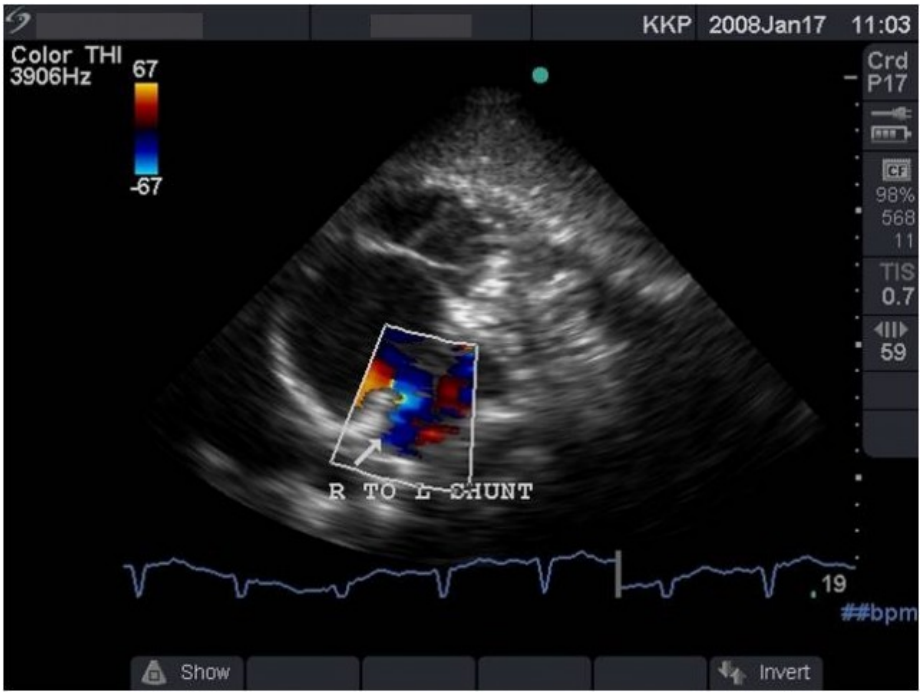

Fig.4 Subcostal view showing right to left shunt through a patent foramen ovale

Sometimes a contrast echo with 10ml of agitated saline may be needed to demonstrate the right to left shunt.

Technique of Contrast Echocardiography

Contrast echoes are most easily done using agitated saline as a contrast medium. For this 2, 10ml syringes are connected to a 3-way connection which is connected to the central or peripheral line.

9.5ml of saline and 0.5ml of air is taken in one of the syringes. This saline and air is rapidly pushed alternatingly from one syringe to the other through the 3-way, so that microbubbles form in the saline. It is better to use luer lock syringes as the non lock syringes tend to disconnect during the agitation process.

An Echo view is then obtained that permits good visualization of the right and left sided chambers, preferably apical 4 chambered view or subcostal 4 chambered view. The agitated saline is then injected rapidly. The Right atrium and ventricle opacify (becomes white) as the microbibbles stream past the echo window. The left heart, however normally remains black as the microbubbles dissipate within the pulmonary circulation and do not enter the left heart. If there is a right to left shunt, the left heart also opacifies along with the right, distal to the level of the shunt. We do not attempt to trace individual bubbles, but only note the pattern of opacification. A patent foramen ovale is diagnosed if the left atrium opacifies within three cardiac cycles or right atrium opacification. An intrapulmonary shunt takes longer, usually only after 5 cardiac cycles.

In the video below, the right atrium and ventricle have completely opacified with the contrast microbubbles while the left heart remains black.

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Video 3. The right sided chambers on the left side of this video are completely white due to microbubbles

A left to right shunt can also be visualized using the same technique provided the machine has a rapid frame rate. The right sided chambers are white due to the microbubbles on the sonogram. If there is a left to right shunt, it will be seen as a bubble free area in the right sided chambers (a dark "space occupying area") as bubble free blood from the left flows into the right side. The level of this space occupying area will depend on the level of the shunt.

Assessment of RV function

A knowledge of RV systolic function may be essential in making hemodynamic decisions, considering thrombolysis in patients with pulmonary embolism and normal blood pressure and in prognostication in RV infarction and chronic cor pulmonale.
A visual gestalt of the contractility of the RV can be made by looking at the 2 major components of RV contraction, excursion of the free wall and the movement of the tricuspid annulus towards the apex. Although many different ways of calculating the ejection fraction of the RV have been described, none is convenient for everyday use.
2 surrogate markers of RV function which have been shown to be reliable are Tricuspid Annular Displacement (TAD) and Tricuspid Annular Peak Systolic Velocity (TAPSV)

TAD

In an Apical 4 chambered view, a M-mode cursor line is paced through the lateral tricuspid annulus and a M-mode tracing is obtained and frozen. A sinusoidal line representing the motion of the annulus is noted. Using calipers, the distance from the peak to the trough of this wave is measured (see figure). This distance is the TAD. In this case it is 1.2cm.
A TAD <1.75 suggests RV dysfunction with 80% sensitivity and specificity, and correlates with an Right ventricular EF of <45% measured by radionucleotide ventriculography.
TAD is also affected by LV systolic function and patients with LV dysfunction but normal RV function have TADs between 1.75 and 2cms, while those with normal biventricular function, demonstrate TADs >2.0cms.

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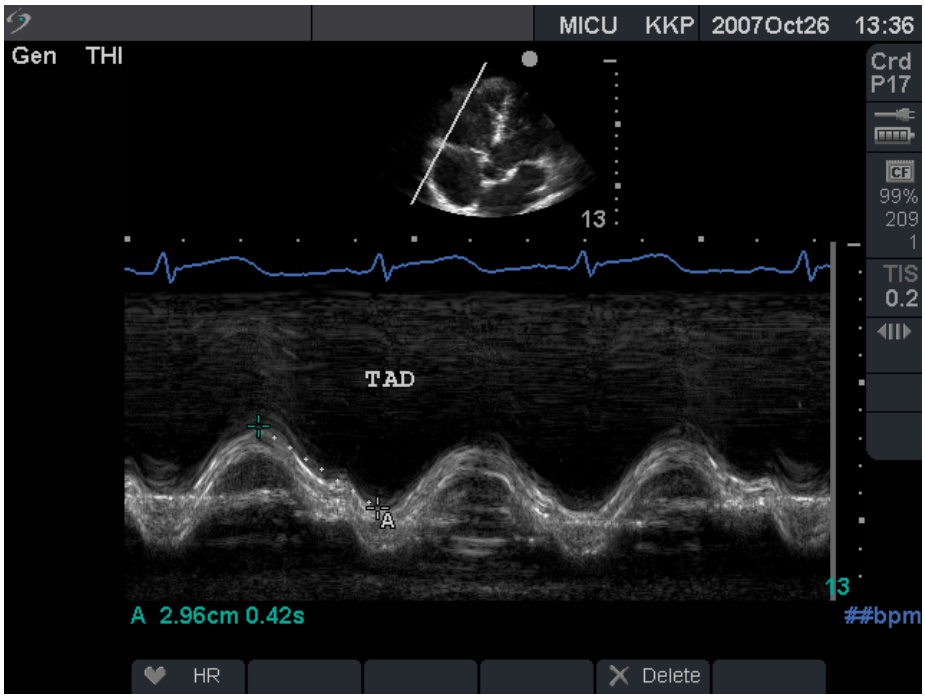

Fig.5 Tricuspid annular displacement indicating normal RV function

Tricuspid Annulus peak Systolic velocity (TAPSV)

Here again, the apical 4 chambered view is chosen, PWD is chosen and Tissue Doppler is activated. The TDI cursor sample volume is placed over the lateral tricuspid annulus in a similar manner to what has been previously described for the mitral valve in Tutorial 6. A doppler trace is obtained and frozen. Normally 2 diastolic excursions below the baseline and one systolic excursion above the baseline are noted. A caliper is used to measure the peak velocity of the systolic excursion. This value is the TAPSV. A TAPSV of <10cm/sec suggests RV dysfunction and correlates with RVEF <50% measured by Simpson's rule method with a sensitivity of 60% and a specificity of 90%.

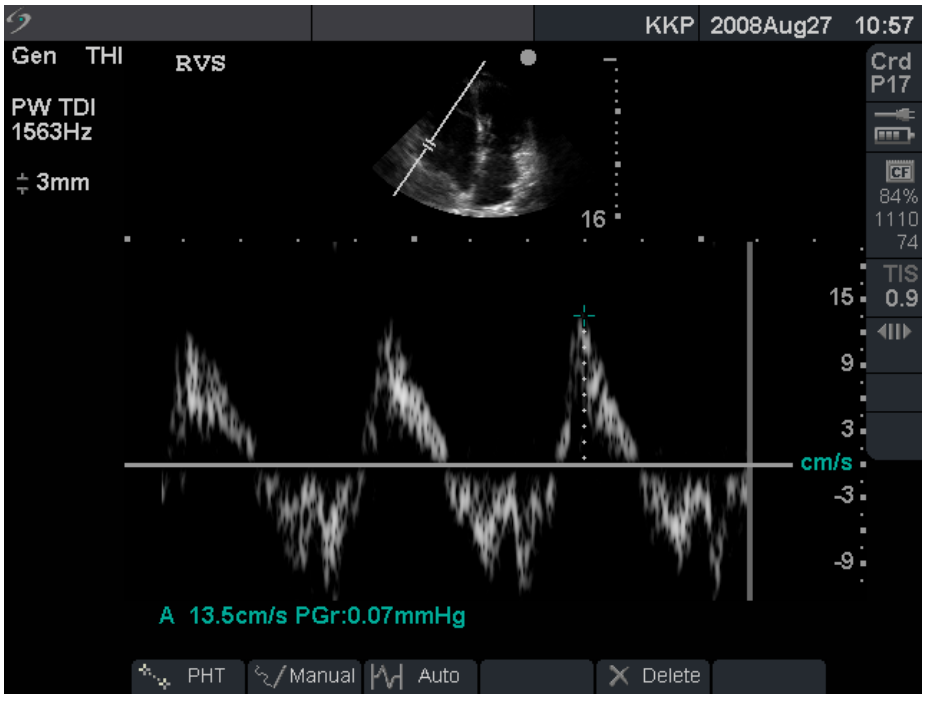

Fig.6 Tricuspid annulus peak systolic velocity

Assessment of Pulmonary Arterial pressures

Systolic pressure

The presence of at least a trivial tricuspid regurgitant jet is almost universal. In patients with right heart disease, this increases in severity. Measurement of the velocity off the tricuspid

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regurgitant jet velocity (v) enables the estimation of pressure gradient (TVpg) between the RV and the RA during systole using the simplified Bernoulli's equation (TVpg = 4v2). Since the RA pressure is measured from central lines in most critically ill patients, it is easy to arrive at the RV systolic pressure (RA pressure + TVpg). In mid systole, in the absence of RVOT or pulmonic stenosis, the pulmonary arterial pressure is the same as the RV systolic pressure.
The tricuspid regurgitation can be assessed in a number of views. The view that can be used most consistently for this purpose is the Apical 4 chambered view. In the A4C view, a color box is placed over the tricuspid valve and the presence of a turbulent jet flowing away from the probe is noted (see video). In a few patients, the TR jet is better visualized in the modified SAX (RV inflow-outflow view) or subcostal 4 chambered views.

Video 4. A broad jet of severe tricuspid regurgitation is seen

CWD is then activated and the CWD line is placed through the center of the jet. To get reliable velocity estimations, the angle of the jet should not be more than 20° out of alignment with the CWD line. A CWD tracing is then obtained and frozen. The peak velocity of the tricuspid jet is measured (see fig.7)

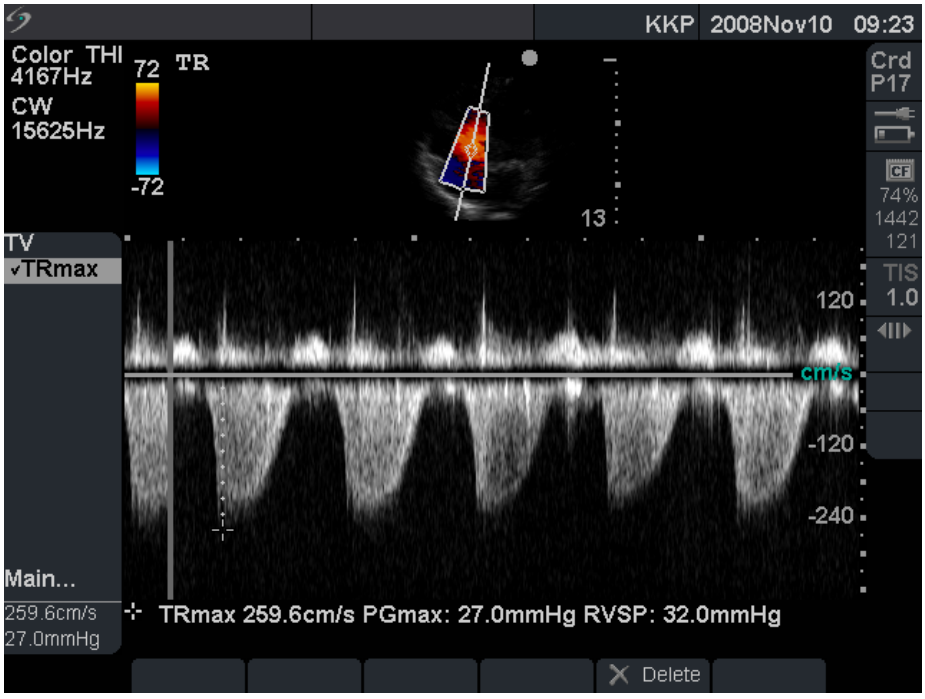

Fig.7 Measuring maximum tricuspid jet velocity on CWD trace

As seen in the above figure, most machines would automatically calculate the pressure gradient

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and would display the RV systolic pressure if the value of the RA pressure has already been entered (in this case RA pressure of 5mmHg + TVpg of 27mmHg gives us a RV or PA systolic pressure of 32mmHg).
Normal pulmonary arterial pressure in a person living at sea level has a mean value of 12-16 mm Hg. Pulmonary hypertension is present when mean pulmonary artery pressure exceeds 25 mm Hg at rest or 30 mm Hg with exercise.
Mean pulmonary artery pressure (mPAP) should not be confused with systolic pulmonary artery pressure (sPAP). A systolic pressure of 40 mm Hg typically implies a mean pressure more than 25 mm Hg.
Roughly, mPAP = 0.61•sPAP + 2.
Therefore, if PA systolic pressure is the only PA pressure estimated during an Echo study, A PA systolic pressure >40mmHg indicates pulmonary hypertension.

It is important to ensure that the Doppler gain is not set too high as this could lead to an overestimation of the jet velocity (see above figure).
When the tricuspid regurgitation jet is trivial and the CWD spectrum is suboptimal, an injection of agitated saline solution (see under "Technique of contrast echocardiography" above) into an arm vein enhances the tricuspid regurgitation velocity signal.
It is also important to remember that higher tricuspid velocities do not indicate more severe tricuspid regurgitation. The velocities are often lower in severe cases as the RA pressure is also high and the pressure gradient across the tricuspid valve therefore is smaller. Additionally, in conditions such as RV infarct and acute RV failure where severe TR is seen, the RV is not capable of producing very high RV systolic pressures, leading to a lower velocity jet. Severity of the tricuspid regurgitation is assessed by the area of the regurgitant jet, the width of the jet origin and how far back into the RA it extends.

Mean and diastolic Pulmonary arterial pressures

The mean and end diastolic pressures in the pulmonary artery are assessed by measuring peak and end-diastolic velocities of the pulmonary regurgitant jet.
First a short axis view at the level of the aortic valve is obtained. The probe is adjusted so that the RVOT and the pulmonary valve are visualized well. The CFI box is placed over the pulmonary valve. Pulmonary regurgitation appears as a small jet from the valve moving towards the probe. This is present in 85% of the normal population as well. A CWD cursor line is placed though the center of this jet and a CWD trace obtained and frozen.

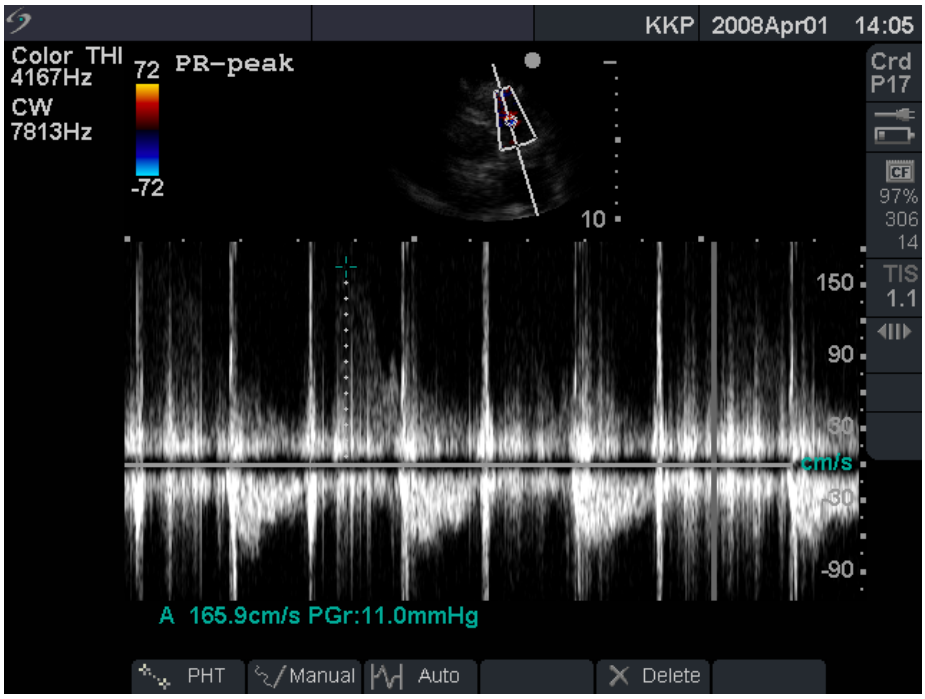

Fig.8 PR trace on CWD.: Note the notched appearance of the PR wave due to atrial contraction. The peak PR velocity is being measured

Unless severe, it is a faint signal above the baseline with a slow diastolic decay. Calipers are used to make 2 measurements, a peak velocity and velocity at end-diastole. Pressure gradients for both measurements are calculated automatically by the machine using the simplified Bernoulli's equation. The Diastolic and Mean PA pressures can be then calculated as follows.
Diastolic PA pressure (dPAP) = PRend diastolic PG + RA pressure
Mean PA pressure (mPAP) = PRpeak PG + RA pressure

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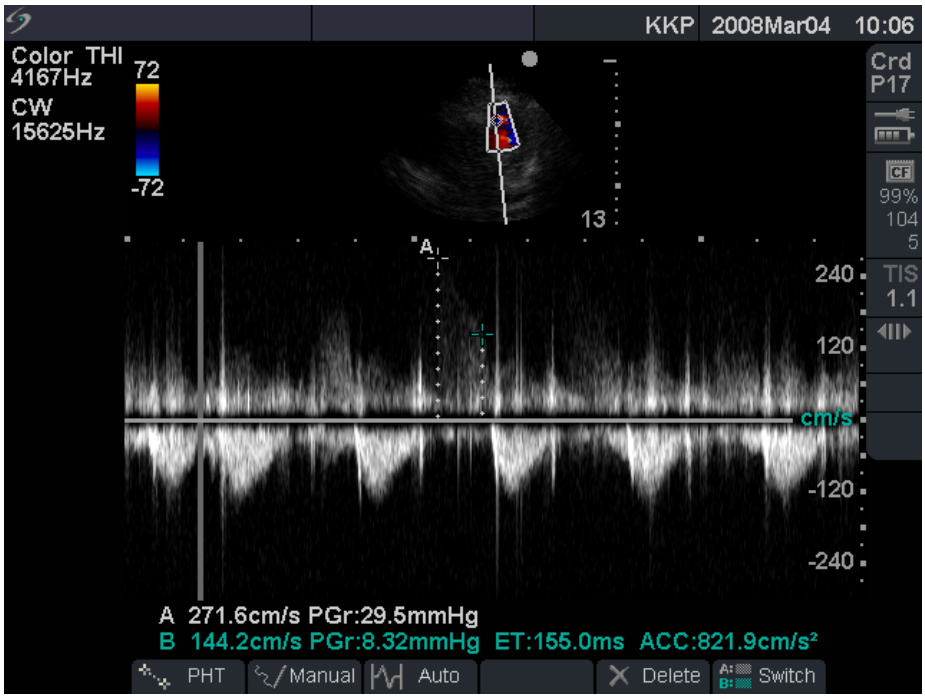

Fig.9 Peak and end-diastolic PR velocities being measured by 2 calipers.

In fig.9, the PRpeak PG is 29.5 and the PRend diastolic PG is 8.3mmHg. Since this patient had a RA pressure (CVP) of 15mmHg, the Mean PA pressure (mPAP) calculated is 44.5mmHg (29.5+15) and the Diastolic PA pressure (dPAP) calculated is 23.3mmHg (8.3+15).

Mean pulmonary arterial pressure can also be calculated from the acceleration time of the RVOT VTI.
It is also possible to calculate the Pulmonary vascular resistance (PVR) with an echo, though that would be out of the scope of this tutorial.

Identifying patterns of RV overload

The two major patterns of RV overload are pressure overload or volume overload.

Volume overload

Volume overload results from Tricuspid or pulmonary regurgitation, ASD, VSD and TAPVC. Volume overload results in enlargement of the Right ventricle and right atrium with little or no increase in free wall thickness. An analysis of the movement of the interventricular septum 9IVS) is important in identifying this form of overload.
Normally, the IVS is oriented and contracts such that it entirely forms a part of the left ventricle. During systole, it thickens and moves towards the center of the left ventricle and during diastole, it moves away from the center of the LV into the RV cavity. In RV volume overload, because the RV intracaviatry pressure is higher tn normal and the duration of systole longer, RV pressure exceeds LV pressure during end systole and early diastole, leading to the IVS being pushed into the LV. This may be maintained during the rest of the diastole. However, at the onset of systole, the sudden increase in LV pressure produced by LV contraction restores the normal transeptal pressure gradient, pushing the IVS towards the RV cavity. This results in IVS flattening and a D-shaped LV only during early diastole.

Pressure overload

Pressure overload can be acute or chronic.
Acute pressure overload is a result of massive pulmonary embolism or ARDS (both are forms of acute cor pulmonale - ACP) and is the commonest pattern of RV overload seen in critically ill patients. Echocardiographically, it is indistinguishable from a volume overload picture. RA and RV enlargement is associated with flattening of the IVS in diastole and the absence of RV free wall thickening. As the RV has not had time to hypertrophy, the peak systolic pressures generated rarely exceed 50mmHg in ACP.
Chronic pressure overload is a result of chronic lung diseases such as COPD and ILD, chronic thromboembolism or chronic pulmonary venous hypertension secondary to left heart valvular or myocardial pathologies. In addition to demonstrating RV enlargement, Thickening of the RV free wall is also apparent with increased trabeculation. Increase in interventricular septal thickness and trabeculation also occur but are difficult to make out echocardiographically. Because RV adaptation has taken place, the RV is able to generate very high RV systolic pressures, usually exceeding 50mmHg, sometimes exceeding LV pressures. The interventricular septum therefore is flattened into the LV cavity during the entire cardiac cycle.

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Because of the overlap of features with acute overload, it is difficult to diagnose acute on chronic cor pulmonale in the ICU using echocardiography, unless serial RV systolic pressures are available.
All forms of RV overload also affect the LV, reducing diastolic function and necessitating higher filling pressures.

Pulmonary Embolism

Pulmonary embolism is not uncommon in critically ill patients. It is a complication with a high morbidity and mortality and needs rapid diagnosis and specific treatment. The standard diagnostic algorithms for pulmonary thromboembolism may be difficult to follow in the ICU as they require the patient to be transported for CTPA or V/Q scans, which may not be feasible when they are very sick. Therefore, we may have to rely on the Echo to make a diagnosis before deciding on thrombolysis or catheter based interventions.
The echo picture of pulmonary embolism is that of acute pressure overload described above. In addition, there may be a right to left shunt through a patent foramen ovale. Typically, peak systolic pressures in the pulmonary artery do not exceed 50mmHg unless it has occurred on a background of chronic RV pressure overload.
One also needs to look for evidence of thrombi in transit, in the IVC (fig.9), RA, RV or main pulmonary artery (fig.10). These thrombi are highly mobile and have the appearance of popcorn or a snake (see videos).

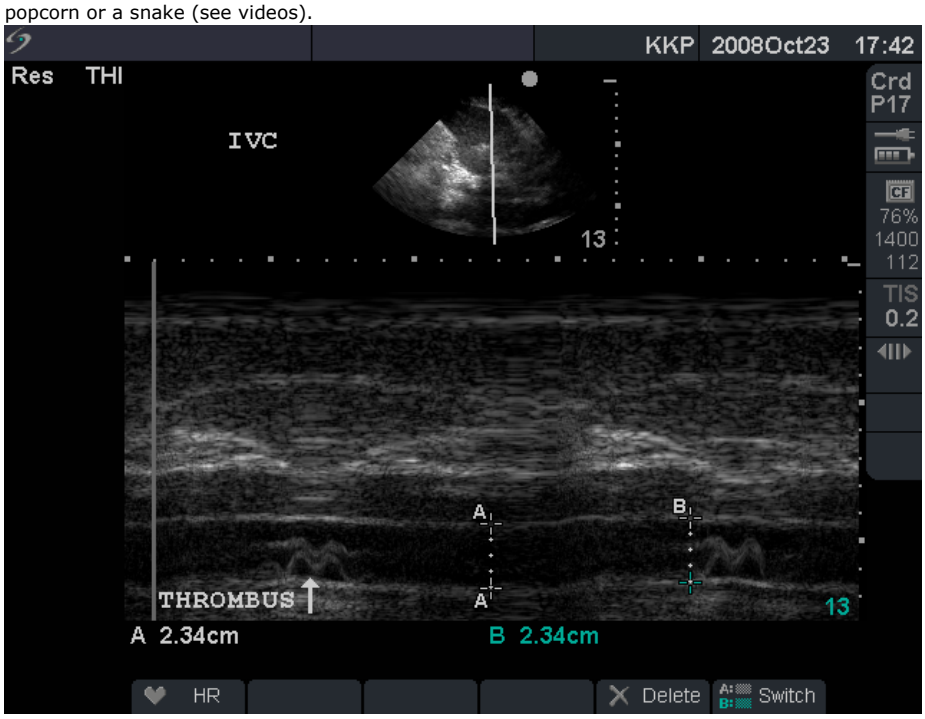

Fig.10 M-mode of IVC with thrombus within. Also note the lack of IVC variation with respiration

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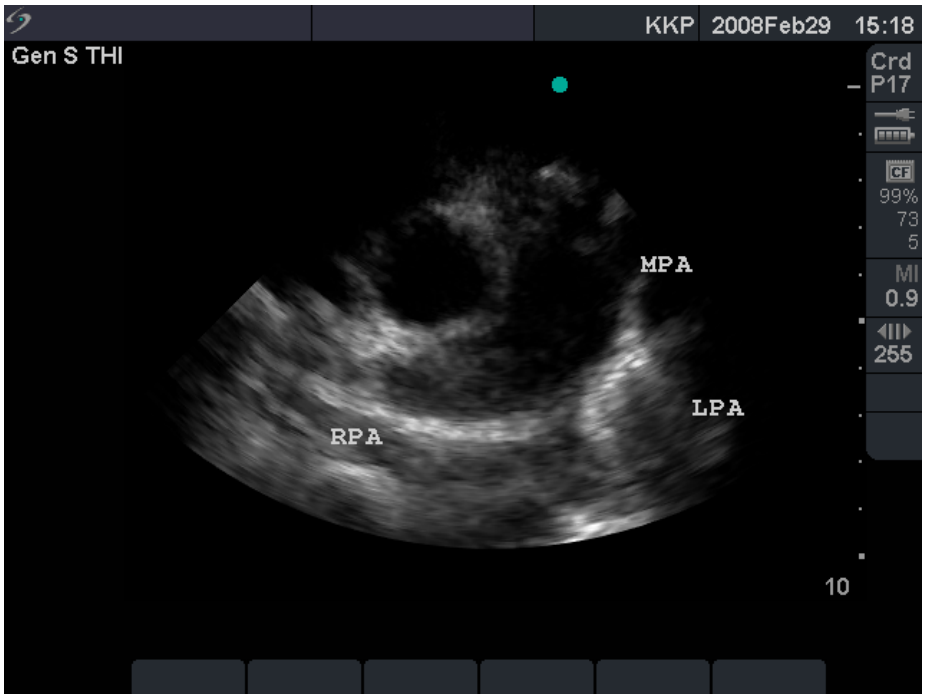

Fig.11 Visualization of Main pulmonary artery and its branches in the basal SAX view.: However, no thrombus is visualized.

Video 5. Short axis view showing mobile thrombus in the RA

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Video 6. Subcostal view showing mobile thrombus in the RA

Although they may sometimes be attached to the RA (see video),

Video 7. Thrombus attached to the auricle and wall of the right atrium

The mobile mass comes from en bloc embolization of venous thrombi cast. The presence of such visible thrombi should prompt urgent thrombolysis and the consideration for a catheter based intervention if required.
Therapeutic results are monitored by repeated echocardiography examinations. The next 2 videos demonstrate dissolution of an IVC thrombus and only residual fibrin strand are seen post thrombolysis with Streptokinase.

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Video 8. Residual strands of thrombus in the IVC, a longitudinal view

Video 9. Residual strands of thrombus in the IVC, a cross sectional view

The next assessment should be for RV function. The presence of RV dysfunction even in the absence of shock is considered an indication for thrombolysis by some.

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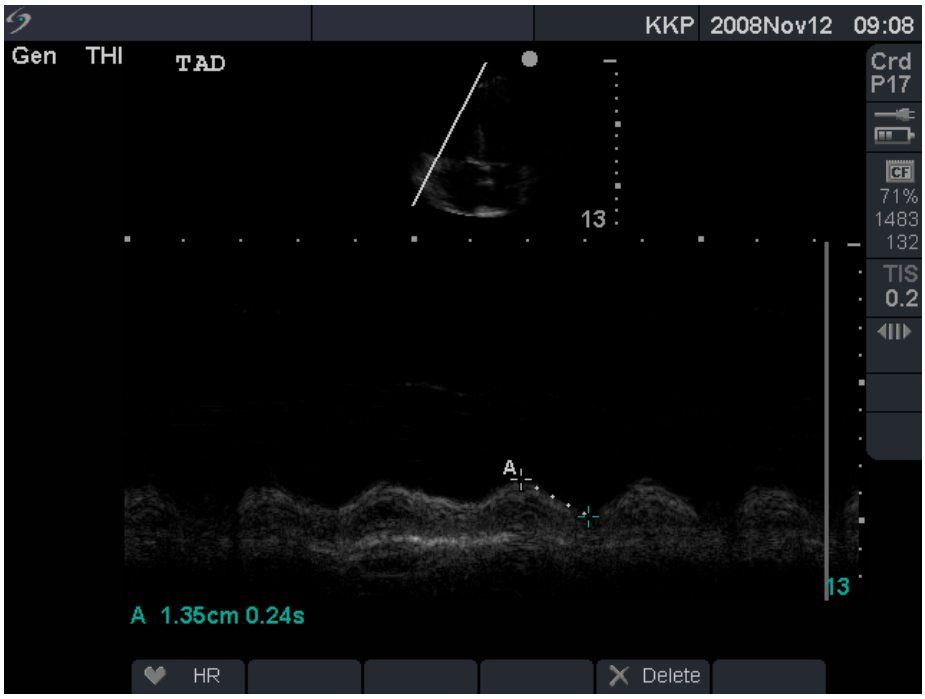

Fig.12 Reduced TAD in a patient with pulmonary embolism

Tutorial 8 - Assessment of pericardial disease

Assessment of Pericardial diseases

Although the purpose of the pericardium is mainly to protect the heart and provide lubrication, it also has important hemodynamic effects on the cardiac chambers. The nondistensible pericardium limits the acute distension of the ventricles and contributes to ventricular coupling: the distension of one ventricle alters the filling of the other, an effect that is important in the pathophysiology of cardiac tamponade and constrictive pericarditis. The pericardium is not seen normally on an echocardiogram neither is pericardial fluid. Therefore the visualization of either of these is pathological.

Pericardial effusion

Filling of the potential pericardial space with fluid or blood results in a pericardial effusion. When the volume of fluid is small, it can be seen as a black echo-free space present only posterior to the heart in the parasternal short and long axis view, and may be present only in the systolic phase (fig.1 and 2). When the volume of fluid is more than 25ml, an echo free space exists all around the heart throughout the cardiac cycle. When the amount of fluid is massive, the heart may have a swinging motion in the pericardial cavity (fig.3).

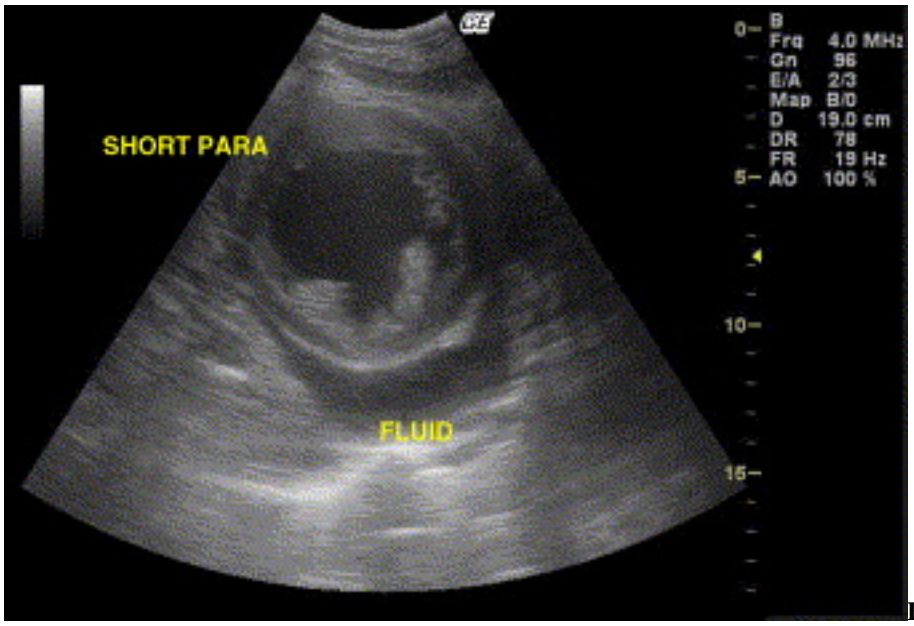

Fig.1 Posterior pericardial effusion in SAX view

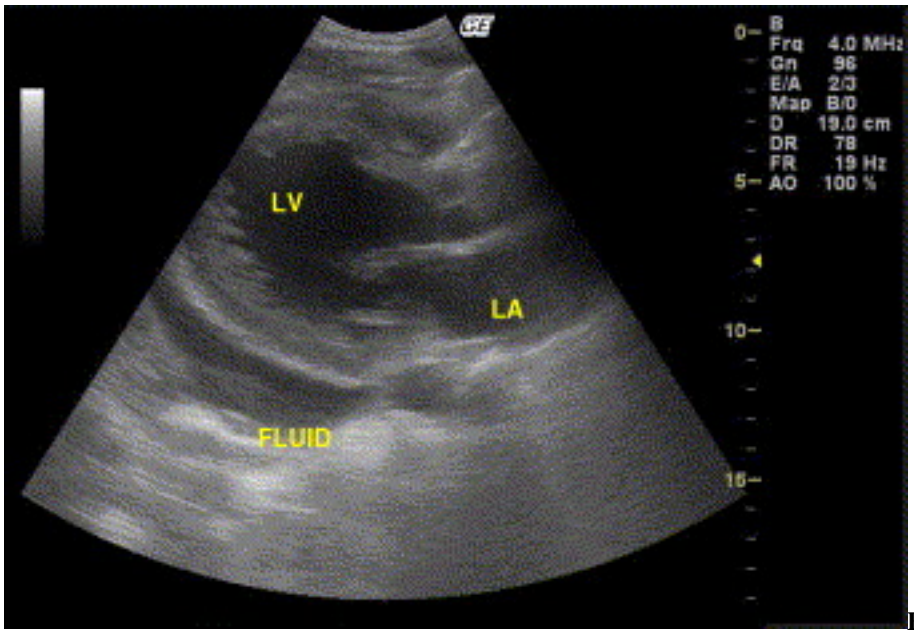

Fig.2 Posterior pericardial effusion in PLAX view

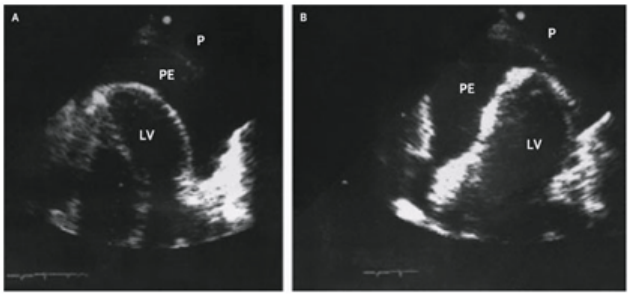

Fig.3 Large pericardial effusion with the heart swinging from one end to the other

While this fluid may be visible in all cardiac echo views, measurements of fluid thickness need to be made anteriorly and posteriorly on the PLAX or SAX views, apically in the A4C, A3C or A2C views and inferiorly in the subcostal view. Small effusions have an echo free space of <5mm, moderate sized effusions range between 5mm and 10mm and are circumferential and more than 10mm indicates a large effusion. Fluid adjacent to the right atrium is an early sign of pericardial effusion.

Pericardial versus pleural effusion

Pericardial effusion is usually located circumferentially. If the echo free space is seen only anteriorly, it is more likely an epicardial pad of fat or a pleural effusion. Posteriorly a pericardial effusion is anterior to the descending thoracic aorta whereas a pleural effusion is posterior to it. A pericardiaal effusion is rarely >4cms thick. If pericardial and pleural effusions co-exist, then a linear echo (the pericardium) separates them. A pleural effusion on the left side allows cardiac imaging from the back.
The other structure to differentiate from pericardial effusion is epicardial fat. Epicardial fat is tissue is more prominent anteriorly but may appear cicumferentially, thus mimicking an effusion. Fat is slightly echogenic and moves in concert with the heart, two characteristics that help to differentiate it from an effusion which is echolucent and motionless.

Cardiac Tamponade

The fluid in the pericardial sac can cause hemodynamic alterations depending on the volume of fluid and the speed at which it accumulates. The echocardiographic signs of tamponade are

2-D echo and M-mode
1.Diastolic right ventricular collapse
2.Right atrial collapse / inversion
3.IVC plethora with a lack of change with breathing

Doppler
1.Exaggerated respiratory variation in tricuspid and mitral inflows
2.Increased LVOT VTI variation
3.Exaggerated IVC flow variation

The first three are important to look for.

Right ventricular collapse

This occurs when the intrapericardial preesure exceeds the intraventricular pressure and occurs in early diastole. This can be observed in PLAX and A4C views, but being a rapid movement, may need to be resolved with M-mode through the RVOT or RV free wall in a PLAX view (see fig.4). The right ventricular outflow tract (RVOT) has the most compliance and is the first part of the RV to collapse. When the entire body of the RV collapses (see video), then it is an indicator of a more substantial elevation in intrapericardial pressure.
Although this sign is a relatively sensitive and specific marker for tamponade, RV diastolic collapse is sensitive to alterations in ventricular loading conditions and may not be seen in the presence of right ventricular hypertrophy.

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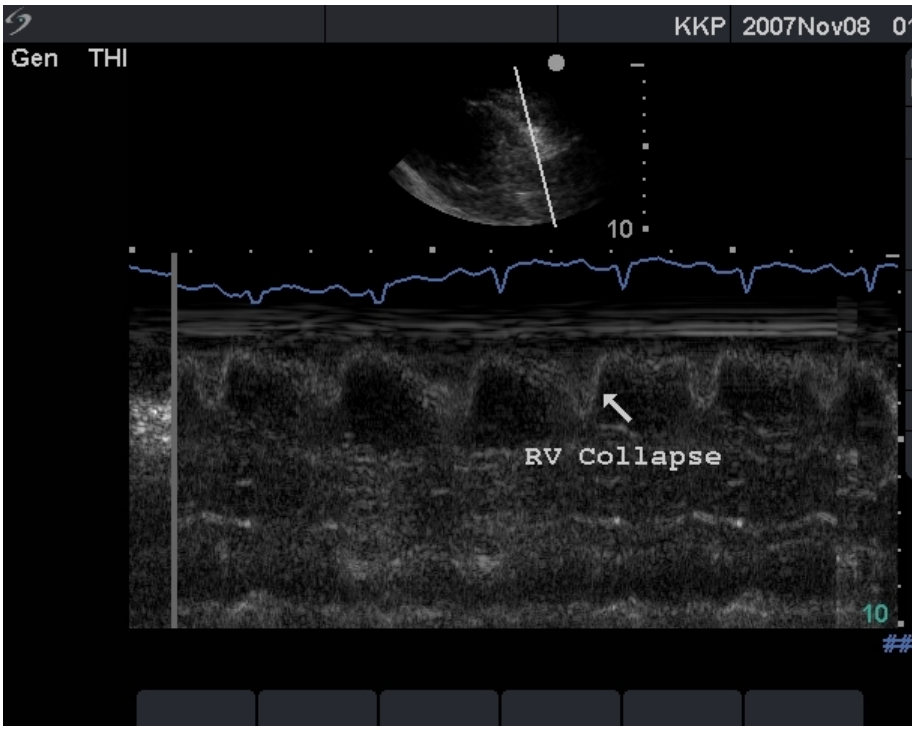

Fig.4 M-mode through the RVOT showing early diastolic collapse: This is a modification of the PLAX and the M-mode line has been placed through the RV free wall just proximal to the RVOT, which is where the maximal RV collapse was visualised on 2D

Right atrial collapse

The raised intrapericardial pressure also impedes atrial filling and causes the right atrium to remain collpapsed even after atrial systole. Atrial collapse therefore is a late diastole, early systole phenomenon. With increasing intrapericardial pressures, the atrium remains collapsed throughout diastole as well and buckles inward, reversing the normal wall curvature.
While this is best seen in the A4C and subcostal views, M-mode through the RA wall may be necessary to clearly identify the collapse, particularly when the heart rate is high.

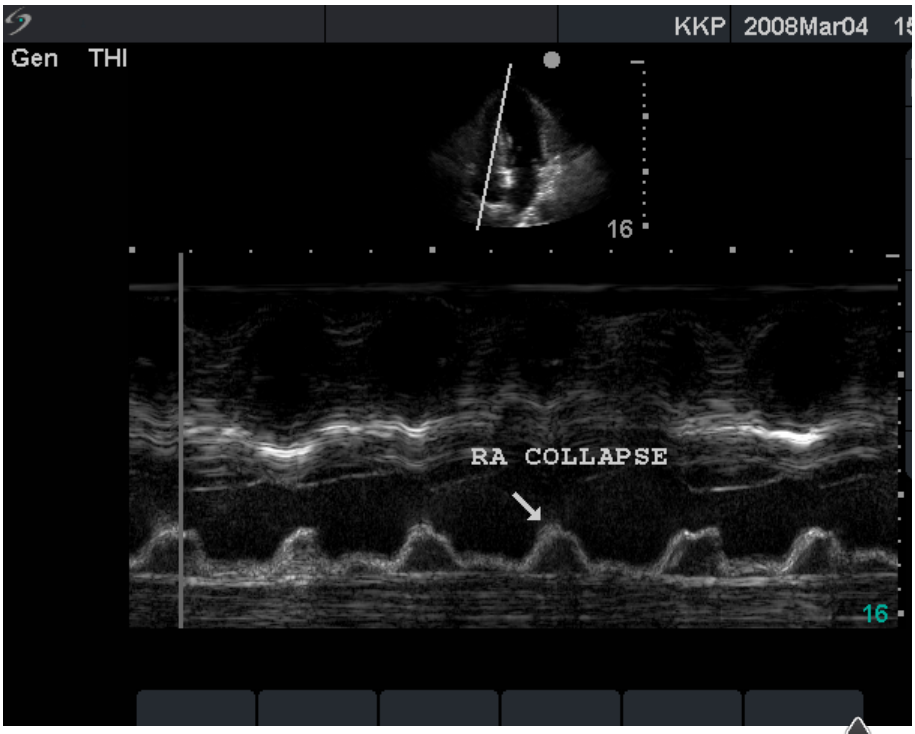

Fig.5 M-mode through RA in A4C showing end-diastolic and early systolic collapse: In this A4C view, a

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M-mode line has been passed through the right heart and the RA free wall to demonstrate its inward movement indicating early collapse of the RA

Right atrial collapse is virtually 100% sensitive for tamponade but less specific. Duration of this collpase exceeding one third of the cardiac cycle increases specificity without sacrificing sensitivity. Left atrial collapse is seen in about 25% of patients and is very specific for tamponade.

IVC plethora

Because of the elevated filling pressures of the right heart, the IVC becomes distended (>2cms diameter) and has less than 12% variation in diameter with respiration.

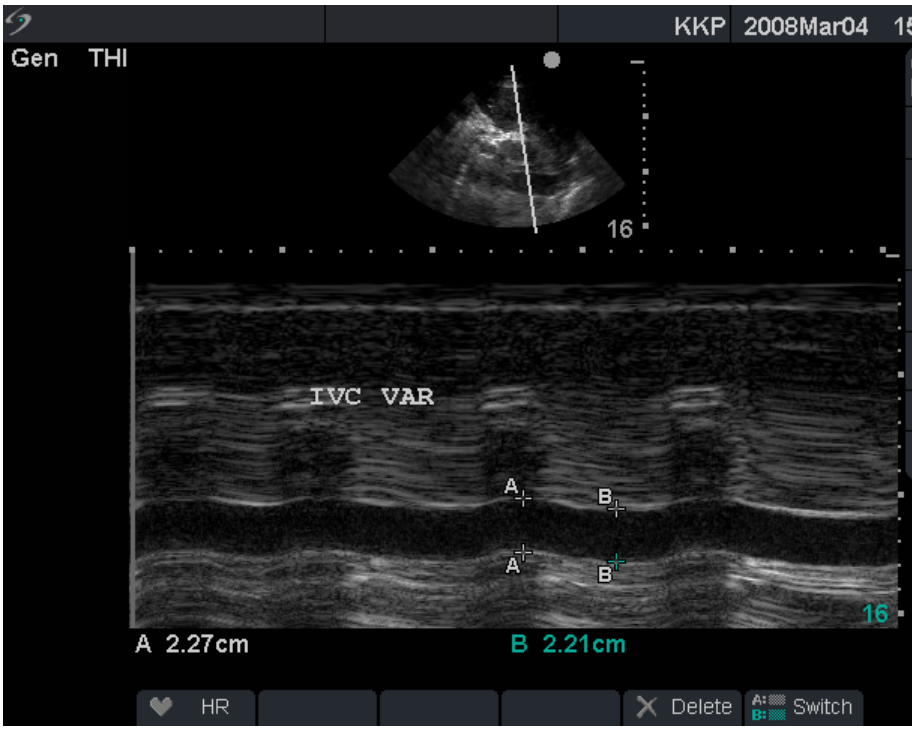

Fig.6 Distended IVC with only 2.7% variability in the same patient as fig.5

Doppler findings

Marked variations with respiration (>12%) in mitral and tricuspid inflow velocities (E and A) as well as LVOT and RVOT VTIs is commonly seen.

These changes may not be overtly manifest in the presence of a hypertrophic right ventricle as seen in pulmonary hypertension, thickening of the ventricular walls due to malignancy, overlying inflammatory response or overlying thrombus and in severe hypovolemia - the so called low pressure tamponade.

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Video 1. 2D and Doppler features of tamponade (Source: Youtube)

Video 2. RV collapse in tamponade

Echocardiography guided pericardiocentesis

While a detailed description of the procedure is out of the scope of this tutorial, a few key points are noted below
a) The procedure should be performed by a cardiologist or an intensivist with experience in the procedure
b) ECG monitoring is mandatory
c)A long 18 gauge catheter over needle can be used
d)The entry site could be just left of the xiphisternum or the apex. The site with the least distance from the skin to the pericardium as determined on the echo can be used.
e) The needle is advanced toward the left shoulder if the entry point is the xiphisternum or directly downward if it is the apex.
f) The needle should be advanced under direct visualization on the echo
g) If there is a doubt about having entered a cardiac chamber, agitated saline can be

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injected through the needle and this will delineate the pericardial space or cardiac chamber the tip is in
h) If a cardiac chamber has been entered, the catheter should be passed into it, locked and the patient should be transported urgently to the cardiac catheterization lab to plug the leak
I) Frond-like, band-like or shaggy intrapericardial echoes should alert one to the possibility of a difficult and potentially less therapeutic pericardiocentesis.
See this brief video on pericardiocentesis

Video 3. Steps of pericardiocentesis in brief (Source: Youtube)

Website supported by the VARTHINI AND ARUNACHALAM ENDOWMENT FUND

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Tutorial 9 - Lung ultrasound

Lung Ultrasound

Traditionally, air has been considered the enemy of ultrasound and the lung has been considered an organ not amenable to ultrasonographic examination. Visualizing the lung is essential to treating patients who are critically ill. The commonest investigation used to image the lung in the ICU is the bedside chest X-ray.
While the bedside roentgenogram is relatively inexpensive and is available even in most secondary hospital ICUs, it has a few limitations. It is difficult to ensure breath holding during the X-ray exposure and this leads to a reduction in the spatial resolution. The cassette is placed posteriorly and the X-ray beam originates anterior, at a shorter distance than recommended and quite often not tangentially to the diaphragmatic cupola, thereby hampering the correct interpretation of the silhouette sign. These problems lead to the incorrect assessment of pleural effusion, consolidation and the alveolar interstitial syndrome.
The chest X-ray is also not always useful for the diagnosis of a pneumothorax in a ventilated patient in the ICU. In such a patient the air in the pleural space tends to accumulate anterior to the lung in the supine position, causing it not to be seen on an AP view X-ray. In addition, mechanically ventilated lungs do not collapse even in the presence of a pneumothorax. For these reasons, X-rays have a sensitivity of only 53% in detecting pneumothoraces in such critically ill patients as compared to the gold standard of a CT scan of the chest. Add to these, the logistic difficulty of obtaining an urgent chest X-ray quickly, and it becomes clear that a faster, more reliable tool is needed to image the lung in the ICU.
While the CT scan of the chest is considered the gold standard for the imaging diagnosis of all the conditions listed so far, it is neither inexpensive nor available within the intensive care unit, necessitating potentially dangerous transport to the radiology department. In addition, it exposes the patient to high doses of ionizing radiation.
Ultrasound compares favourably with CT scan in the diagnostic ability for some disease conditions, most prominently pneumothorax, where it has a sensitivity of 92% compared to CT. In addition it is relatively cheap and is readily available at the bedside making it easier and faster to get an ultrasound imaging than a chest X-ray. For these reasons, ultrasound is fast becoming an essential part of the chest imaging armamentarium in the ICU.

Technique of lung ultrasound

A range of frequencies (4 to 12MHz) can be used to visualize the lungs. High frequencies are useful to look at the periphery of the lung with a high resolution as in looking for 'lung sliding' and other signs of pneumothorax, as well as studying lung comets. Lower frequencies help with the imaging of deep lung tissues as in looking at consolidation and pleural effusion. Hence a vascular probe is used for the assessment of pneumothorax, while the ultrasound probe is used for consolidation and pleural effusion.
In the supine position, the anterior and lateral lung areas can be easily scanned, but the patient may have to be turned to a lateral decubitus position for scanning posteriorly. Six regions, delineated by the anterior and posterior axillary lines should be systematically examined: upper and lower parts of the anterior, lateral and posterior chest wall.

When an ultrasound transducer is laid on a normal chest wall, the following is observed:

Static Image:

The probe is kept in the intercostal space with the marker pointing toward the head end of the patient. The ribs yield artefactual anechoic shadow images (black). In between the two ribs, there is a hyperechogenic line > 0.5cm deeper to the probe. This line is the interface between the soft tissues of the chest wall and the aerated lung the - "pleural" line. The air in normally aerated lungs stops progression of the ultrasound beam and hence the ultrasound "image" of the lung is composed of artifacts. These air artifacts arise from the pleural line. Two types of artifacts can be seen:

A lines: Horizontal, regularly spaced hyperechogenic lines representing reverberations of the pleural line. These are motionless and are artifacts of repetition. In two-thirds of normal lungs, this is the only artifact pattern that can be seen.

Video 1. Artifact pattern of normal lung (Bouhemad et al Critical Care 2007, 11:205 (doi:10.1186/cc5668))

B lines: Vertical narrow based lines arising from the pleural line to the edge of the ultrasound screen. The "comet-tail image" (Ultrasound Lung Comets, ULC) is a sonographic image detectable at the bedside with ultrasound probe positioned over the chest. It is defined as a hyperechogenic, coherent bundle with a narrow base spreading from the transducer to the further border of the screen. It extends **to the edge of the screen** (short comet-tail artifacts may exist in other regions), and arises only from the pleural line. These are also called by the descriptive term "comet tail artifacts". When several B lines are visible, the term used is "lung rockets".

In this context, 2 similar appearing artifacts should not be confused with B lines. Firstly, short, broad, ill defined, vertical comet tail artifacts arising from the pleural line but not reaching the distal edge of the screen are **not B lines**. These are called **Z lines** and and are found in normal persons as well as in those with pneumothorax (Fig.1). They are less echogenic than the pleural line, usually taper off at after 2-4 cms, do not erase A lines and do not move with lung sliding. Second, comet tail artifacts can be seen **superficial** to the pleural

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line in those with parietal emphysema or parietal echogenic multiple foreign bodies (shot gun pellets). These are called **E lines**.

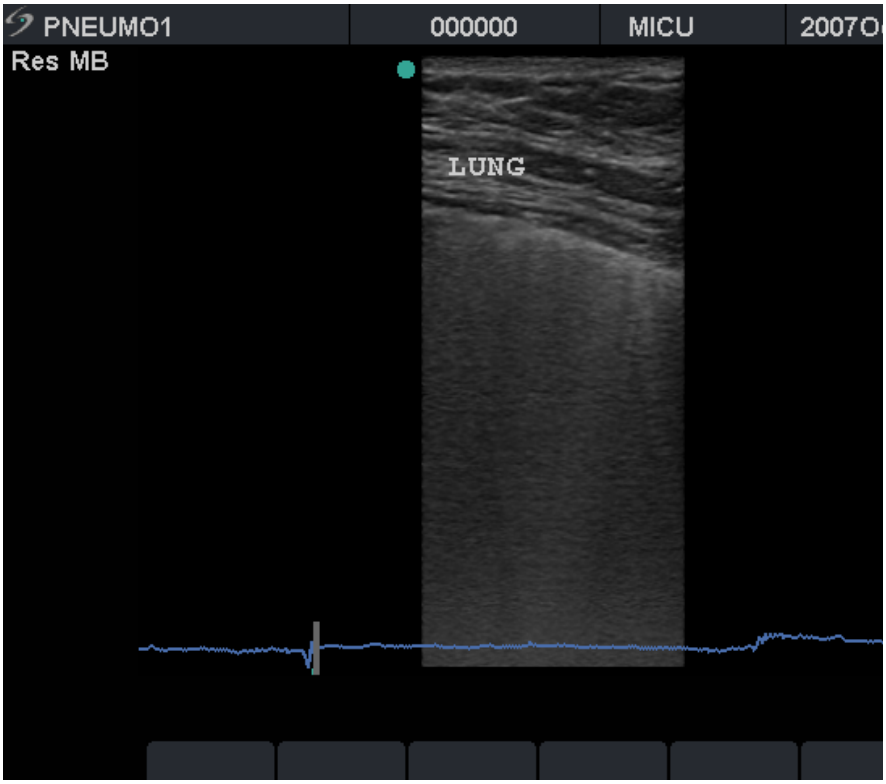

Fig.1 These short ill-defined comet tails are not B-lines

Mechanism of the artifact:

The comet-tail artifact appears when there is a marked difference in acoustic impedance between an object and its surroundings. The reflection of the beam creates a phenomenon of resonance. The time lag between successive reverberations is interpreted as a distance, resulting in a center that behaves like a persistent source, generating a series of very closely spaced pseudo-interfaces . The beam is "trapped" in a closed system, resulting in endless to-and-fro echoing . The figure below shows the mechanism. The path of the sound beam is shown as a function of time. When the beam meets the sub-pleural end of the thickened septum, it reflects indefinitely at a speed of 1,450 m/s, resulting in an artifact composed of all the micro-reflections. Each reflection of the beam is displayed on the screen behind the previous reflection. A distance of about 1 mm separates each reflection.

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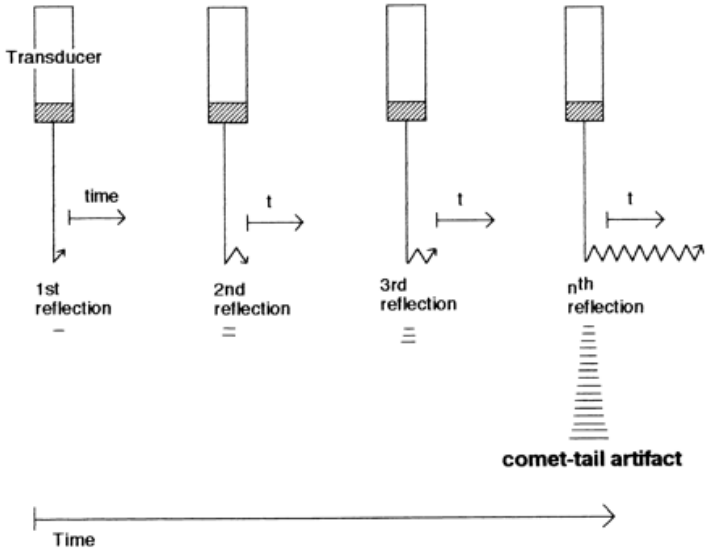

Fig.2 Mechanism of formation of an ultrasound lung comet

These interfaces yield, on the screen, a narrow-based laser-like ray extending to the edge of the screen. At the surface of the lung, the prominent element is air. Its acoustic impedance is very different from that of bone, parenchyma , and water. Bony tissues are not expected to be found at the surface of the lung. Normal lung contains predominantly air and little water, the comet-tail artifact described has the following features: it is related to a small water-rich structure, below the resolution of the ultrasound beam (which is about 1 mm), surrounded by air (resulting in a high impedance gradient). It is absent under normal conditions and present in alveolar-interstitial syndromes. This element has to be present at and all over the surface of the lung, and each element is separated from each other by an average distance of 7 mm. It is frequently found in the last intercostal space in normal subjects. Acute pulmonary edema as well as chronic interstitial disease cause "the artifact."

The classification is as follows:

Above Pleural Line: air, foreign bodies

Below Pleural Line:
Horizontal - A line
Vertical - comet tail
Short, ill defined - normal
Long well defined - B lines (originate from pleural line and go to the edge of the screen)
Single
Multiple - lung rockets

Dynamic Changes:

The pleural line "slides" (to and fro movement) with respiration. The movement is distinctive as the surrounding chest wall structures are still or move in an opposite direction to the lung (See first video). This is pleural / lung sliding. The sliding movement seen sonologically is the lung which moves on respiration. Its amplitude is greater at the base than at the apex where it may be imperceptible. The image best seen in an M mode as the superficial pareital layers are motionless and have a horizontal pattern of lines while the area deep to the pleural line appears "granular" as the motion of the pleural line is reflected all over this area. This is also known as the "seashore sign" (Fig.3).

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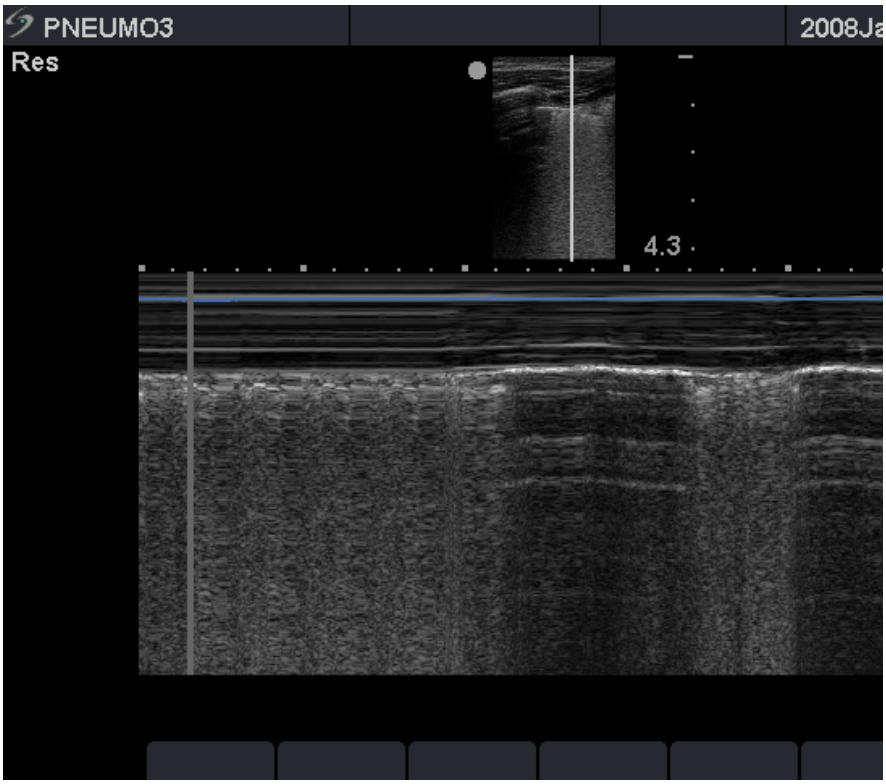

Fig.3 The granular pattern below the pleural line in the left half of the picture is lung parenchyma, while the horizontal lines above it indicate the chest wall.: The right half of the picture depicts artefacts due to movement of the probe - note the discontinuity of lines above the pleural line

In a sense, sonographic evaluation of the lungs involves analyses of true images as well as the artifacts.

Pneumothorax

In the supine patient, a free pneumothorax usually collects in the anterior and non dependent area. The signs are best with a high frequency probe. A probe > 5 MHz is advisable. High frequency linear (such as a vascular) probes will give a clearer picture.

a. **Absence of lung sliding**: This is a sign of pneumothorax. If lung sliding is present, pneumothorax can be ruled out. However, loculated posterior, mediastinal and apical pneumothoracies can be missed. For a complete examination, the probe must be placed along the anterior, lateral and posterior intercostals spaces and observation must include a whole respiratory cycle at each point. In an M mode, this will show absence of the normal granular pattern deep to the pleural line - the whole picture will show a number of horizontal lines (Fig. 4).

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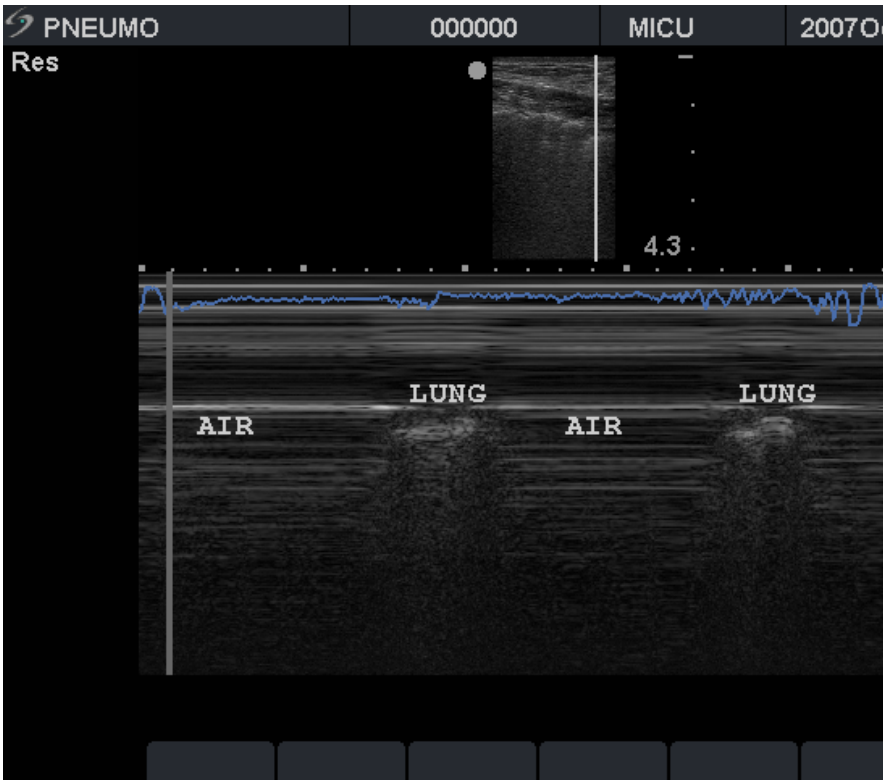

Fig.4 Horizontal lines have intermittently replaced the granular appearance of lung parenchyma suggesting a pneumothorax

b. **Absence of B-lines**: Although this is not specific for pneumothorax, the presence of B-lines rules out a pneumothorax. B-lines with absent lung sliding may be seen in lower lobe consolidations. Absent B-lines with lung sliding present may be seen in emphysema or hyperinflated lung states..
c. **The lung point**: Since the air in the pleural space moves anterior and the lung collapses to a dependent position posteriorly, there is a point, usually in the lateral regions where the lung and air may be visualized in the same view. On moving from anterior to lateral, a pneumothorax pattern gives way to a fleeting appearance of lung pattern in a particular location of the chest wall. When the above pattern is seen, a pneumothorax is likely. If there is a sudden change during respiration - a "pneumothorax" pattern which changes to a normal pattern during respiration - it signifies that the pleural air has been displaced elsewhere during lung expansion. On an M mode, this will be seen as parallel lines in one part of the screen with a sudden change to a granular pattern - the lung point (see Fig.2 and 3). The probe must be held motionless in one location to elicit this sign.

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Video 2. Lung point with motionless horizontal lines replaced by lung artefacts from the left on inspiration (Bouhemad et al Critical Care 2007, 11:205 (doi:10.1186/cc5668))

Video 3. Lung point with motionless horizontal lines of pneumothorax on left and lung with dense B lines moving in from the right

A suggested systemic sequential evaluation for pneumothorax is as follows:

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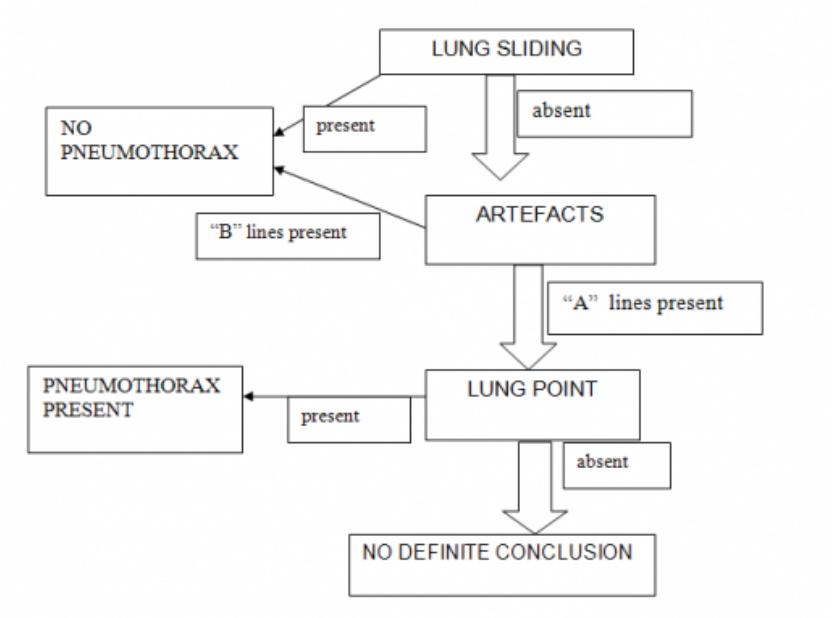

Fig.5 Algorithm for the ultrasound diagnosis of pneumothorax

With this approach, ultrasound can diagnose pneumothorax with a sensitivity of 92%, compared to CT scan. This is vastly superior to the sensitivity of a bedside chest X-ray. In addition the time taken for a complete ultrasound evaluation of the chest for air takes about 10 minutes, less than that taken to order a chest X-ray.

However, examination for a pneumothorax is also technically more challenging and the acquisition of skills for the same is the most difficult part of lung ultrasound training. The low incidence of pneumothorax as compared to consolidation or pleural effusion also contributes to a longer learning curve.

Pleural Effusion

Pleural effusions can be rapidly diagnosed and small effusions can be localized for aspiration at the bedside.

Effusions are looked for in the dependent lung areas delineated by the chest wall and the diaphragm. The standard ultrasound probe is used. The probe should be placed in the intercostal space with the long axis of the foot of the probe parallel to the adjacent rib.

The effusion appears as a hypoechoic (i.e. dark) and homogenous structure in the dependent areas which is present both in inspiration and expiration.

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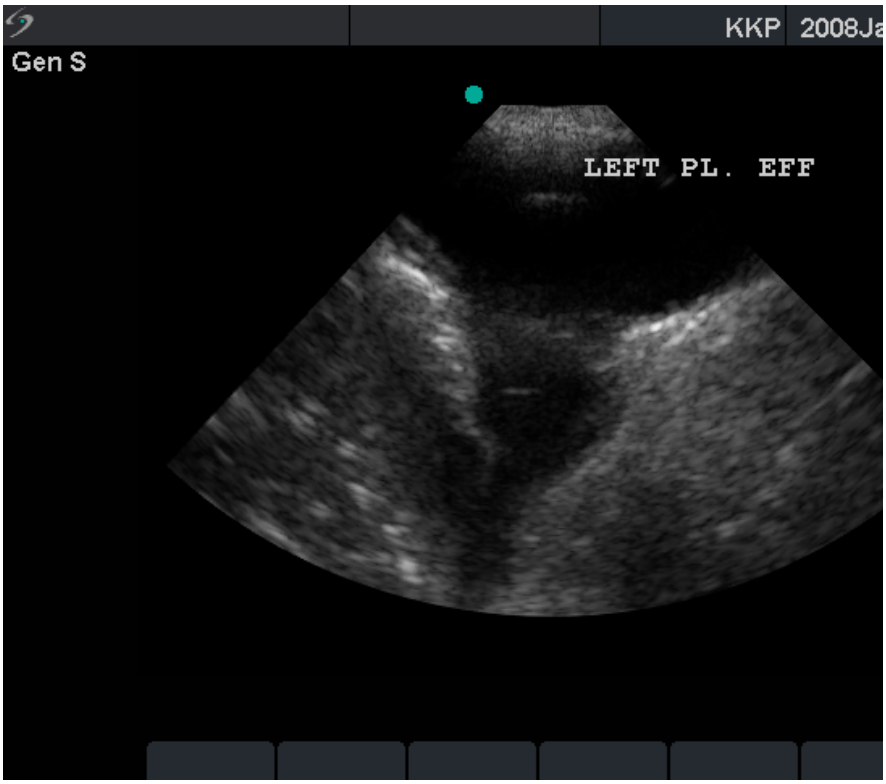

Fig.6 left sided pleural effusion with the spleen on the right and consolidated lung on the left

The diaphragm must be identified and the liver on the right and the spleen on the left should be visualized as these may rarely be confused with pleural fluid. The following must be kept in mind:
a.The image must be anatomic (not artefactual)
b.It must be located above the diaphragm (to avoid confusion with intraperitoneal fluid
c.Image must be bounded at the superficial surface by a straight line - the parietal pleura visible between the ribs and > 0.5cm deep
d.Image limited in depth by a regular line - the visceral pleura
e.Dynamic sign - the parietal pleural line is fixed while the visceral pleural line moved with the respiratory cycles. The interpleural distance decreases with inspiration (sinusoidal waveform on M mode). This inspiratory centrifugal shifting of the visceral pleura with decrease in apparent thickness of the effusion is known as the "sinusoid sign" and is specific for pleural effusion.
f. Identification of the lung behind the pleura is necessary before introducing a needle - it may be consolidated or aerated. In massive effusions, the lung will seem to swim in the effusion with frank undulations

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Video 4. Left pleural effusion with underlying heart and collapsed lower lobe (Bouhemad et al Critical Care 2007, 11:205 (doi:10.1186/cc5668))

It is difficult to measure the volume of pleural fluid accurately with ultrasound. If the depth of fluid is more than 5cms, then it is likely that there is more than 500ml of fluid. This interpleural distance can be measured in end inspiration or end-expiration and is left reliable on the left side.

Fibrin strands swimmimg in the fluid with undulations, debris or loculations suggest pus or blood. Other than this, the nature of pleural effusions cannot be accurately defined on an ultrasound.

Ultrasound is also useful to mark for and guide thoracocentesis. Pleural adhesions which hamper adequate drainage can be avoided (see video below). An assessment of adequacy of fluid for drainage can be made. It is suggested that an interpleural distance of at least 15mm, with effusion visible at the adjacent superior and inferior intercostal spaces is necessary in order to perform a safe pleural tap. It may be done laterally (in a supine patient) or posteriorly (in the lateral decubitus position). When an optimal location for tapping has been identified on the ultrasound, the position of the transducer on the skin is marked by an indelible marker. The probe is taken away and the drainage is performed at the marked point.

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Video 5. Pleural effusion with multiple thick septations and adherences (Bouhemad et al Critical Care 2007, 11:205 (doi:10.1186/cc5668))

For loculated pleural effusions, it is safe to drain it under direct visualization on the ultrasound. The ultrasound probe with some jelly is passed into a sterile sheath or glove and is used to visualize the needle tip during thoracocentesis and guide its entry into the locule. The details of the procedure are similar to the guided central venous cannulation described in the next tutorial.

Identification of pleural effusion is the easiest of the lung ultrasound skills to learn.

Consolidation

A standard ultrasound probe is used to image consolidations.
Water is a good transmitter of ultrasound and a consolidated lung is water rich. Alveolar consolidation usually reaches the lung surface. Collapsed lung segments can resemble consolidation sonologically. It appears as poorly defined hypoechoic lung tissue structure. In contrast, the tissue structure of normal lung cannot be seen. What is seen is the artifacts that arise at the pleural line.

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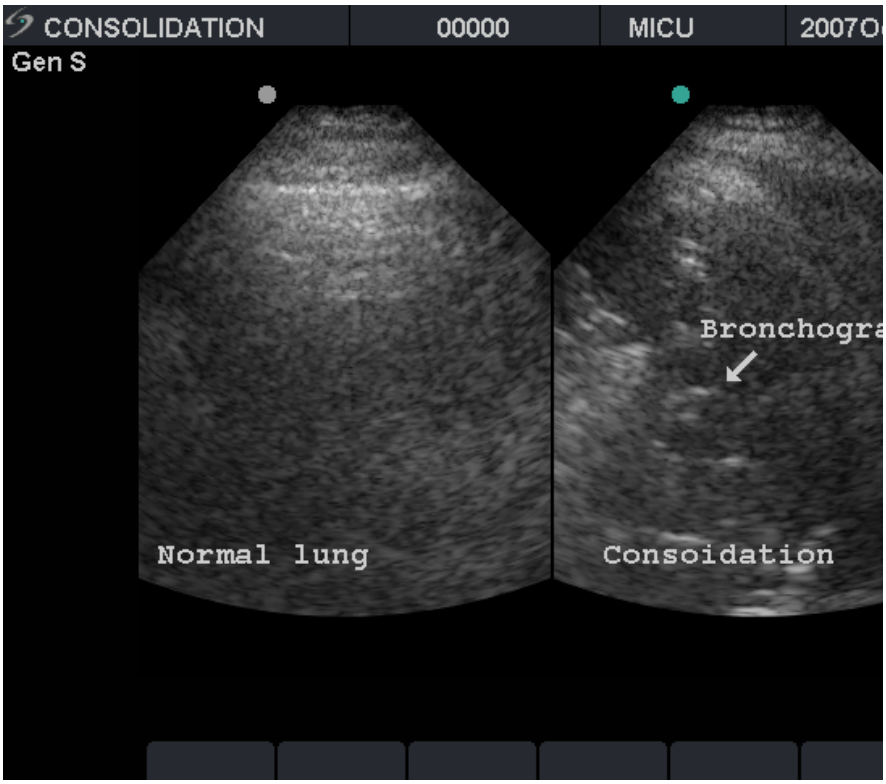

Fig.7 Comparing normal lung artifacts with consolidated lung

Within the consolidation, hyperechoic puntiform images can be seen corresponding to air in the bronchi - a so called ultrasound air bronchogram (figs. 7 and 8). These air bubbles can be seen to move in the bronchi during respiration. The size of a consolidation does not change with respiration, in contrast to a pleural effusion.

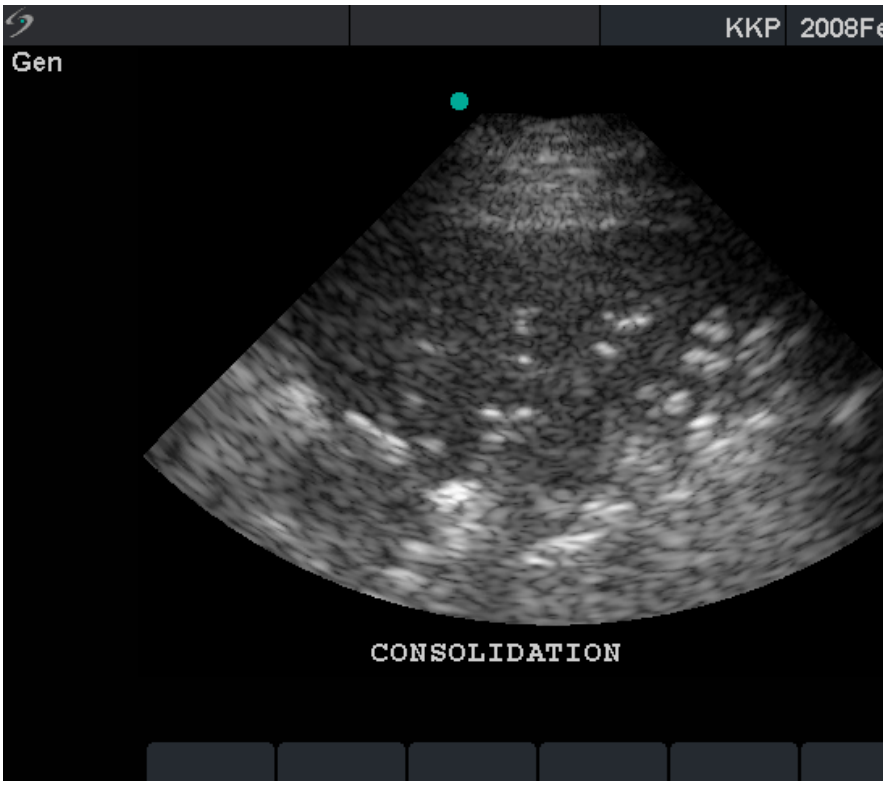

Fig.8 Consolidation with punctiform hyperechoic air bronchogram

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It is very easy to mistake the liver or spleen for consolidation and vice versa. Hence,the thorax should be demarcated sonologically from the abdomen by locating the diaphragm - usually at the mid clavicular line (Fig.9).

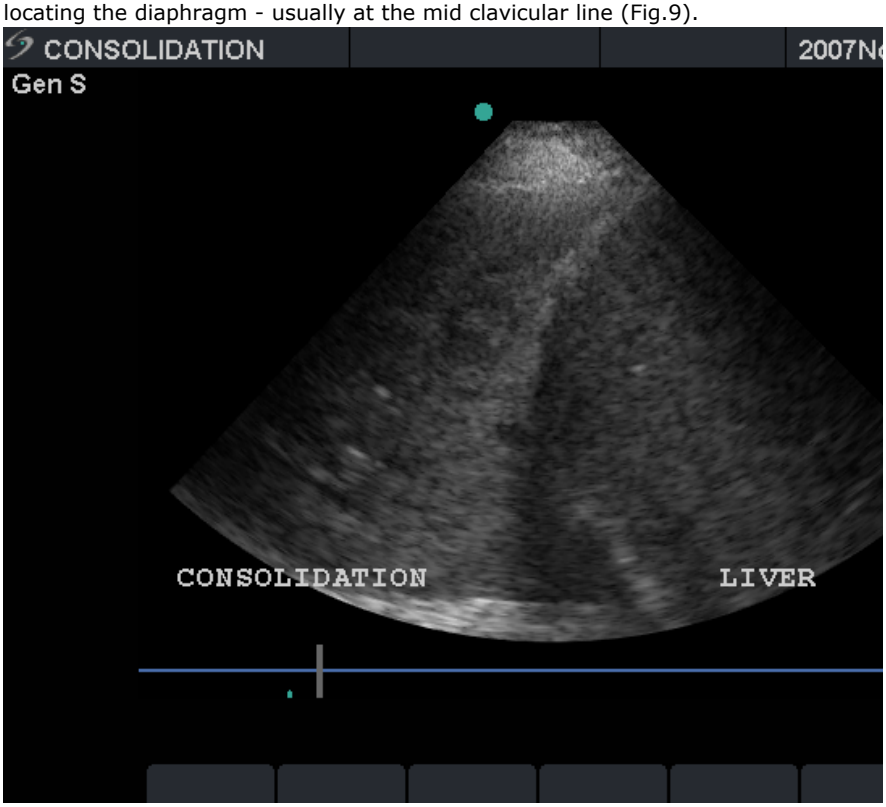

Fig.9 Right lower lobe consolidation on left and liver on right separated by the diaphragm

The following are needed to sonologically diagnose alveolar consolidation:

1.abnormal pattern should be in thorax (should be differentiated from the liver or spleen)
2.should arise from the "pleural line"
3.it should be a real image (not artefactual, like an aerated lung)
4.there should be a tissue like pattern (similar to liver echotexture)
5.anatomic boundaries must be present:
a.superficial boundary of consolidation should be at the pleural line or, if an effusion is present (and the consolidation is deeper to the effusion), at the deep boundary of a pleural effusion.
b.deep boundary of the consolidation may be irregular (aerated lung boundary) or regular (if whole lobe is consolidated).6.absence of sinusoid sign (see above under pleural effusion). In consolidation, caudal inspiratory movement (from left to right of the ultrasound screen) may be present or impaired but there will be no inspiratory centrifugal shift (from the bottom to the top of the screen, an axis called "core-to-surface axis").

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Video 6. Consolidated lower lobe with parapneumonic effusion (Bouhemad et al Critical Care 2007, 11:205 (doi:10.1186/cc5668))

Peripheral lung abscesses with pleural contact or embedded within a consolidation can also be seen with the ultrasound.

Video 7. A loculated abscess is seen embedded within a consolidated lung segment (Bouhemad et al Critical Care 2007, 11:205 (doi:10.1186/cc5668))

Ultrasound diagnosis of consolidation helps in clarifying the cause of respiratory failure, guiding lung aspiration or bronchoscopy, and assessing degree of aeration as a measure of effectiveness of therapy (PEEP effect or antibiotic

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effect on the consolidation)

Alveolar interstitial syndrome

A standard ultrasound probe in high resolution mode is used for this. It has been shown that multiple B-lines 7mm apart are caused by thickened interlobular septa representing interstitial edema (see first video), whereas B-lines 3mm or less apart are caused by ground glass areas characterizing alveolar edema (see second video). The number and intensity of B-lines increases with the degree of loss of aeration.

Video 8. Interstitial syndrome (Bouhemad et al Critical Care 2007, 11:205 (doi:10.1186/cc5668))

Video 9. Alveolar syndrome (Bouhemad et al Critical Care 2007, 11:205 (doi:10.1186/cc5668))

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It should be remembered that a few B-lines may be seen normally in lower dependent lung regions and this does not represent the alveolar interstitial syndrome.

Correlation with Extravascular Lung Water (EVLW):

The "lung rockets" image consists of multiple "tails" fanning out from the lung surface. It originates from water-thickened interlobular septa. Functionally, they are a sign of dysfunction of the alveolar-capillary membrane. They are probably the ultrasonic equivalent of Radiologic Kerley B lines. A patient with increased extravascular lung water (EVLW) has multiple comet tails fanning out from the lung surface originating from water-thickened interlobular septa. The comet-tail images appear when there is a marked difference in acoustic impedance between an object and its surroundings. The reflection of the beam creates a phenomenon of resonance. The time lag between successive reverberations is interpreted as a distance, resulting in a center that behaves like a persistent source, generating a series of very closely spaced pseudo-interfaces. A normal lung contains much air and little water on the lung surface, so with sonographic imaging, no dense structures are visible in normal subjects. The normal ultrasound lung pattern is characterized by roughly horizontal, parallel lines. whereas pulmonary interstitial edema yields roughly vertical, parallel lines. The comet-tail image is related to a small water-rich structure, below the resolution of the ultrasound beam surrounded by air, and this element has to be present at the surface of the lung. Subpleural interlobular septa thickened by edema perfectly combine all of these properties. The subpleural end of a thickened septum is too thin to be visualized by the ultrasound beam, but it is thick enough to "disturb" the beam and create a difference in acoustic impedance with the surrounding air.

Comet-tail images arising from the pleural line can be localized or disseminated to the whole lung surface. They are considered multiple when at least three artifacts are visible in a frozen image in one longitudinal scan with a distance < 7 mm between two artifacts. A positive study is defined as bilateral multiple comet-tail images, either disseminated (defined as all over the anterolateral lung surface) or lateral (defined as limited to the lateral lung surface). A negative study is defined as: an absence of comet-tail images (replaced by the horizontal line); isolated comet-tail images visible; or when multiple comet-tail images are confined laterally to the last intercostal space above the diaphragm. The sonographic examination is performed with patients in the supine position. The ultrasound scanning of the anterior and lateral chest is obtained on both the right and left hemithorax, the second to fourth (on the right side down to the fifth) intercostal spaces, and the parasternal to midaxillary line. In each intercostal space, the number of comet-tail images is registered at the parasternal, midclavicular, anterior, and mid axillary lines. The sum of the comet-tail images is added as an echo comet score of the extravascular fluid of the lung. Zero is defined as a complete absence of comet-tail images on the investigated area.

Using the above criteria, in a study published in 2005, the mean content of EVLW in a negative test result was below the normal limit of EVLW (< 500 ml). The sensitivity and specificity of the negative test result for detection of a content of EVLW < 500 ml were 90% and 89% respectively, whereas the sensitivity and specificity of the positive test result for detection of a content of EVLW > 500 ml (which is associated with pulmonary edema), were 90% and 86%, respectively. Finally, a positive test result had a sensitivity and specificity to detect an excess of EVLW below the threshold of alveolar edema of 87% and 89%, respectively. The pattern of rare, isolated comet tail images or ULCs confined laterally to the last intercostal space above the diaphragm is normal and must be considered as false-positive results.

Putting it all together - the BLUE protocol

A study published in 2008 evaluated the usefulness of lung ultrasonography in patients admitted with respiratory failure to the ICU. It concluded that lung ultrasound could help the clinician make a rapid diagnosis in patients with acute respiratory failure.

The lung ultrasonography compared results on initial presentation with the final

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diagnosis by the ICU team. Uncertain diagnoses and rare causes (frequency<2%) were excluded.

Three items were assessed:
1.artifacts (horizontal A lines or vertical B lines indicating interstitial syndrome),
2.lung sliding, and
3.alveolar consolidation and/or pleural effusion.
The results showed the following:
1.Predominant A lines plus lung sliding indicated asthma (n=34) or COPD (n=49) with 89% sensitivity and 97% specificity.
2.Multiple anterior diffuse B lines with lung sliding indicated pulmonary edema (n=64) with 97% sensitivity and 95% specificity.
3.A normal anterior profile plus deep venous thrombosis indicated pulmonary embolism (n=21) with 81% sensitivity and 99% specificity.
4.Anterior absent lung sliding plus A lines plus lung point indicated pneumothorax (n=9) with 81% sensitivity and 100% specificity.
5.Anterior alveolar consolidations, anterior diffuse B lines with abolished lung sliding, anterior asymmetric interstitial patterns, posterior consolidations or effusions without anterior diffuse B lines indicated pneumonia (n=83) with 89% sensitivity and 94% specificity.
The use of these profiles would have provided correct diagnoses in 90.5% of cases.
The Algorithm suggested (Bedside Lung Ultrasound in Emergency - BLUE protocol) is as follows:

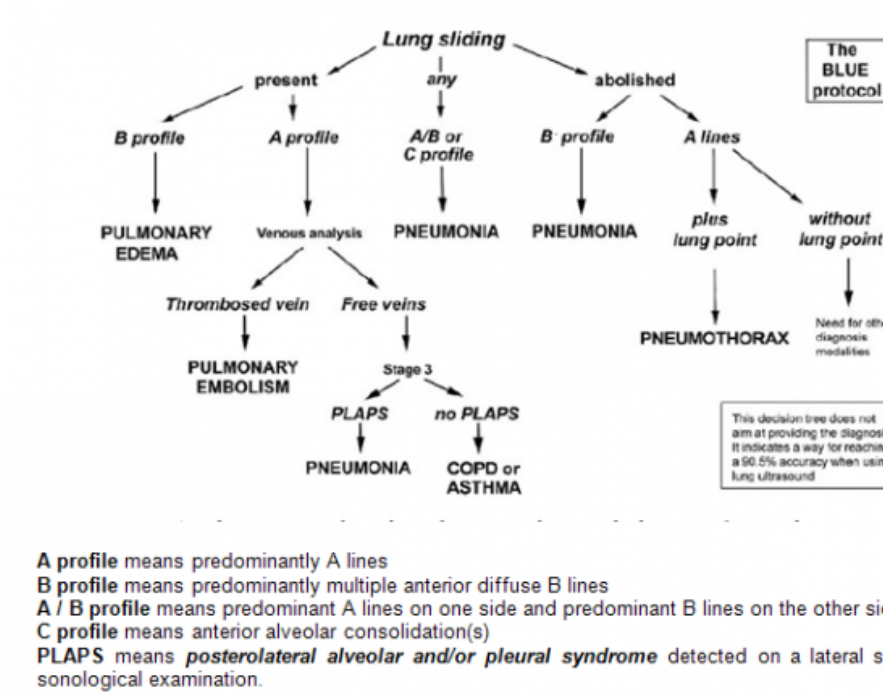

Fig.10 Decision tree indicating a way of reaching 90.5% accuracy with lung ultrasound: Lichenstein et al Chest July 2008 134:117-125

The procedure used a 5 MHz microconvex probe. It took less than 3 minutes for each study and was done within 20 minutes of admission. The authors comment that echocardiography completes the study in critically ill patients . However, echocardiography provides only indirect evidence of what is happening while lung ultrasonography provides a direct approach to respiratory failure.

Website supported by the VARTHINI AND ARUNACHALAM ENDOWMENT FUND

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Tutorial 10 - Vascular ultrasound

Vascular Ultrasound

Imaging of blood vessels is performed primarily for 2 reasons in the ICU. One is to detect deep vein thrombosis (DVT), commonly in the veins of the lower extremities. The other is to guide central venous and arterial cannulation.

Deep Vein Thrombosis

DVT is not uncommon in the ICU and it may often not be clinically obvious. It is important to rapidly diagnose DVT, allowing for immediate treatment with the reduced chances of pulmonary embolism Sometimes detecting a DVT strengthens a difficult diagnosis of pulmonary embolism. While venography and CT venography are more sensitive, Doppler ultrasound has been the most common modality of DVT diagnosis at the bedside.
Venous thrombosis can occur in the upper limbs and jugular veins too, but is most common in the lower limbs. Hence the focus.
The traditional radiological approach of systematically scanning the entire venous tree of both lower limbs is meticulous, but, it needs a lot of time and skill to perform. This examination has been adapted for use by the bedside in the ICU.

Locations studied

1.**Common femoral vein**, just below the inguinal ligament
The artery is located lateral to the vein here. Sometimes, the junction of the superficial with the deep femoral vein may be visible at this level.
2.**Deep femoral vein**, at the mid-thigh
The artery and vein are located much deeper at this level and an increase in the depth settings of the image may have to be made.
3.**Popliteal vein**, in the popliteal fossa
The popliteal artery and vein lie next to each other in the upper part of the popliteal fossa. If the trifurcation of the politeal vein is seen or if any smaller vessels are sen around the two main ones, then the probe is probably too distal and needs to be moved up.

Probe used

The high frequency linear array vascular probe with a frequency range of 7 to 12MHz is used for DVT studies. A lower frequency may be

required on overtly obese or edematous lower limbs. CFI and PWD capabilities should be available with the probe.

Patient and Probe positioning

The supine position with neutral positioning of the lower limb is adequate for imaging of the common femoral vein at the inguinal ligament, while the limb must be externally rotated and mildly flexed at the knee to allow imaging of the other 2 locations.
The probe is placed tranversely, i.e. the long axis of the probe foot is perpendicular to the long axis of the vessel being studied.
While no angulation of the probe is necessary for 2D imaging, CFI and PWD provide accurate information when the probe is slightly angulated to face toward or away from the direction of blood flow.

Procedure

Identifying the vein and artery

On the 2D image the vein and artery appear as a hypo or anechoic lumen surrounded by an echogenic wall. The artery and the vein can be differentiated based on

surrounded by an echogenic wall. The artery and the vein can be differentiated based on

1.**Location** - for example, just below the inguinal ligament, the artery is normally placed lateral to the vein. Anatomical aberrations are possible, however and this is not to be depended on.

2.**Shape and pulsatility** - the artery is round while the vein is ovoid. The artery is pulsatile whereas the vein is usually not.

3.**Compressibility** - On applying downward pressure vertically over the vessels, the muscular wall of the artery resists deformation and stays open, while a non-thrombosed vein gets compressed and the walls meet each other.

4.**Color flow imaging** - Angulating the transducer slightly downwards and selecting the CFI brings up the color box, which should be placed to cover both visualized vessels. The artery shows an intermittent pulsatile flow with color alaising, whereas the vein shows a gradually undulating continuous flow. Do not go by the color of the flow (eg. Blue for vein, red for artery), as this can be changed by changing the direction of angulation of the probe.

5.**Pulsed wave Doppler** - Selecting the PWD and placing the cursor at the center of the vessel and obtaining a trace will show a steady gradually changing flow in the vein and sharply accelerating, pulsatile flow in the artery.

Looking for thrombosis

1.**Compressibility**

Once the vein is identified, downward pressure is applied to the ultrasoud transducer until the vein collapses completely on the ultrasound screen. If the vein is not collapsing and the artery is starting to deform, consideration must be given to the posibility of thrombus within the veins lumen. The amount of pressure required to collapse the vein will differ from patient to patient, and with experience, the sonologist will be able to ascertain if enough pressure has been applied. Care must be taken because downward pressure at the wrong angle or down the wrong vector can greatly decrease the actual pressure felt by the vein and make it appear uncollapsible. As the vein lumen disappears and the walls meet each other, pressure on the transducer is relaxed and the vein allowed to take its normal shape.

Compressibility indicates a lack of thrombus in the vein at the location being compressed, and hence for a complete study, the entire vein will have to be sequentially compressed along its entire length. However, it is enough to limi

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oneself to performing this test at the three designated locations described above and use other techniques to assess the rest of the segments.

2.**Phasicity**
The probe is slightly angulated downwards and a PWD cursor is placed in the center of the lumen of the vein. A PWD tracing is then obtained and observed for any significant variation in the flow velocity (Fig.1). The tracing shows flows below the baseline, but this may change if the probe is angulated upwards. Two variations are normally noticed, cardiac and respiratory. Variations with respiration are the more marked of the two. In spontaneously breathing patients, there is an increase in flow with inspiration, while in mechanical ventilation, the reverse is true.

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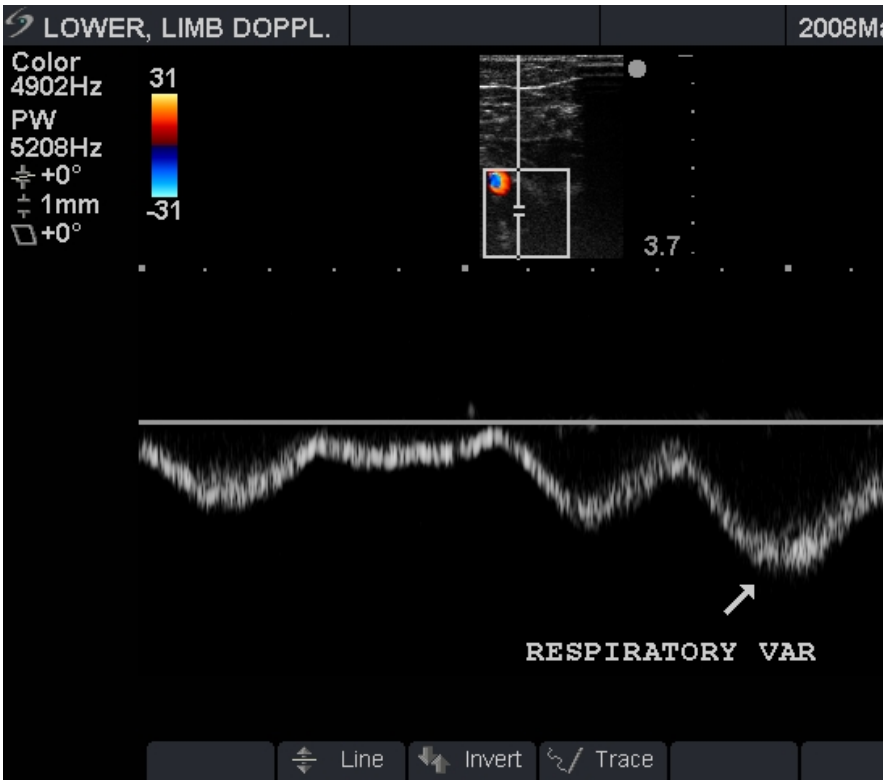

Fig.1 Phasicity of flow with respiration

The absence of such variation suggests a thrombosis above the point in the vein being tested. For example, if an absence of phasicity was noticed in the common femoral vein, then an iliac or IVC thrombus should be suspected.

3.**Augmentation**
With the PWD still in place in the vein lumen, a PWD trace is obtained and the calf muscles are gently squeezed. This normally results in a surge of blood flowing through the vein and a peaking of the plow velocity trace (Fig.2).

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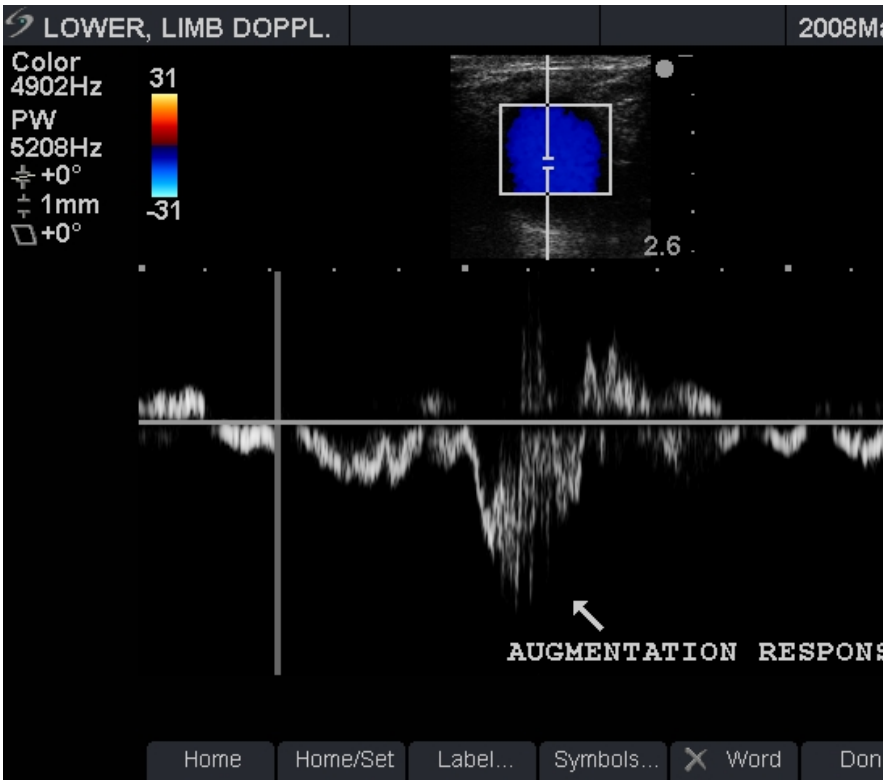

Fig.2 Augmentation response to calf compression, with noise due to movement

If this response is absent, it suggests thrombosis between the point being studied and the calf veins. It is important that the sqeezing of the calf is not done too close to the probe, as this may result in spiky noise artefacts on the PWD trace

4.**Direct visualization of thrombus**
Sometimes the thrombus may be visible as a slightly echogenic mass seen inside the vein lumen. It may be sessile, fixed to the wall or be floating in the flow tethered at one point to the wall. As a clot becomes chronic and gets organized, it becomes more firmly attached to the wall and appears more echogenic. The degree to which the lumen is obstructed is variable.

It is important not to do the compressibility and augmentation tests when a thrombus is visualized as it may result in its embolization.

Upper limb and Jugular thrombosis
While less common that the femoral veins, central line associated thrombosis of the brachial, subclavian and jugular veins are sometimes encountered. The principles of identifying the vein, compressibility, phasicity and direct visualization of thrombus can be applied to these veins as well.

Ultrasound guided Vascular Access

Ultrasound can be used to guide both arterial and venous cannulation. Traditional approaches depend on anatomical landmarks to guide safe and successful cannulation. Therefore, in this approach successful cannulation depends on a knowledge of surface anatomy, an expectation that the anatomy will be normal and personal experience. However, the fact that patient anatomy does not always follow the rules and even if it does, the possibility of a thrombosis in the vein make needle malposition with its attendant complications a real possibility. The incidence of complications depends on patient factors such as short neck, obesity, scarring, cachexia and coagulopathy, operator experience, number of attempts and urgency of insertion.
The fundamental concept behind ultrasound guidance is that if the anatomy and

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patency of the vein can be visualized, the success of cannulation increases and complications related to multiple needle passes or aberrant anatomy can be avoided.
There is mounting evidence that using ultrasound guidance for venous cannulation reduces arterial puncture rates, counter-intuitively reduces time to cannulation and bridges the complication rate gap between inexperienced and experienced operators.
While ultrasound guidance is used most commonly for Internal Jugular and Femoral venous cannulation, it can be used for Subclavian veins as well. Most evidence exists only for Internal Jugular venous cannulation.

Techniques of ultrasound

There are different ways in which ultrasound can be used to guide vascular access

Landmark method
Doppler guided
2D or B-mode
X marks the spot
Real time
2 hand technique
3 hand technique

Landmark method

This is similar to unguided access, except that an ultrasound is done prior to the procedure to ensure that the vein is patent and the anatomy normal. No marking of the vein is done

Doppler guided

In this method, a Doppler probe (not a visual ultrasound) is placed over the area of interest and the artery and vein are identified by the pattern of audio output from the Doppler machine. There is no visual correlation. This is less effective than 2D in preventing arterial punctures and takes longer to localize the vein.

2D or B-mode
X marks the spot

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Tutorial 11 - Abdominal ultrasound

Abdominal Sonology in the ICU

While ultrasound has always been an important imaging modality in the management of abdominal pathology, its use in the ICU is focussed on a few specific disease entities. A comprehensive, detailed ultrasound examination is often not urgent and can be requested from the radiology service. Critical care ultrasound should serve to answer specific questions which would have an immediate impact on the care of the patient. It has been shown that bedside ultrasound studies in the ICU helps to avoid more elaborate imaging studies.

The following organs / areas are usually imaged as part of an ICU abdomen scan.

- Liver
- Gallbladder
- Kidneys
- Peritoneal cavity
- Spleen and pancreas

All these scans are done using the standard abdominal convex probe or in some machines, the echo probe with abdominal settings, both of which have frequencies between 2 and 5 MHz.

Liver

The 2 lobes of the liver should be evaluated separately in 2 planes.

The right lobe is imaged by first placing the probe just below the costal margin on the mid-clavicular line with the marker dot pointing towards the head end of the patient. The probe is angled slightly to point the beam cephalad. The depth of the image should be adjusted to ensure visualization of the entire lobe. This is a longitudinal view of the left lobe. The liver will be seen to be bounded by the diaphragm postero-superiorly (i.e in the lower part of the image) separating it from lung. The probe must be rotated clockwise 90° to obtain a transverse scan and complete the imaging of the lobe in both planes.

The left lobe is imaged by first placing the probe on the midline of the epigastrium midway between the xiphoid and the umbilicus, with the marker dot pointing towards the head end of the patient. The probe is angled slightly to point the beam cephalad. The depth of the image should be adjusted to ensure visualization of the entire lobe. The liver will be seen to be bounded by the diaphragm postero-superiorly (i.e in the lower part of the image) separating it from the heart. As with the right lobe, the probe must be rotated clockwise 90° to complete the imaging in both planes.

Size:

The normal dimension of the right lobe from lower border to the postero-superior aspect (a vertical caliper measurement on either right lobe view) is 13 to 15 cms. It is labelled as hepatomegaly when this dimension is more than 17 cms. The size of the left lobe is more varied. Clinically, it is said to be enlarged if the lower border extends lower than the midpoint between the xiphoid notch and the umbilicus. Cirrhosis and fulminant hepatitis are 2 conditions, where the liver is shrunken (ultrasound liver span of less than 13 cms). The right and the

left lobes enlarge equally in most diffuse diseases except early cirrhosis, where the left lobe may be hypertrophied while the right lobe may be shrunken. Focal pathologies like abscesses and tumors cause enlargement of the lobe that is involved.

Echotexture:

The liver should be homogenous and moderately echogenic throughout. Usually, the echogenecity should be the same as the adjacent right kidney. However, in critically ill patients, renal dysfunction is common and renal echogenicity may not be normal. Hence it cannot always be used as a standard to compare hepatic echogenicity against. Decreased hepatic echogenicity is seen in acute hepatitis, while fatty liver and alcohol related changes cause the parenchyma to have a hyperechoic appearance. Coarse echotexture can be seen in cirrhosis.

Surface:

The surface of the normal liver is smooth and the capsule is seen as a thin echogenic line. Biconvex anechoic, hypoechoic or hyperechoic structures just below the capsule may indicate subcapsular blood or pus. Irregularity of the surface with blunting of the edge is usually seen in cirrhosis. isolated humping of the surface may be due to an intrahepatic tumor or a regenerating nodule.

Intrahepatic structures:

A hyperechoic rounded or triangular structure may be seen appearing to divide the right and left lobes in a transverse scan of the liver. This is the Falciform ligament. It can also be seen sometimes on a longitudinal scan as a highly echogenic sickle shaped structure. The main lobar fissure and the ligamentum teres are other echogenic collagenous structures that can be visualized.

Numerous hypoechoeic structures are seen within the liver parenchyma. These include biliary radicals and vessels - Hepatric veins, branches of portal vein and hepatic artery. Some may appear round, being seen in cross section, while others may be visualized in longitudinal section. Biliary intrahepatic ducts are typically much smaller than the portal venous radicals that accompany them. Color flow imaging can be used to differentiate vessels from biliary radicals.

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Tutorial 12 - Miscellaneous uses

Although the uses of ultrasound detailed in the last 11 tutorials covers most of its utility in the ICU, there remain a few less common applications that we will discuss in this tutorial.

Detection of raised intracranial pressure (ICP)

It is possible to detect raised ICP by measuring the Optic Nerve Sheath Diameter (ONSD) using ultrasound in the ICU. The optic nerve is enclosed in a meningeal sheath all the way upto its insertion into the globe of the eye. This sheath is an extension of the meningeal covering of the brain and the CSF space enclosed between the nerve and the sheath is in continuity with the CSF around the brain. When CSF pressure is increased, this pressure is transmitted to the CSF space in the Optic Nerve Sheath and causes it to distend. This distension can be detected as a change in the diameter of the sheath and can be measured on imaging techniques like MRI, CT and ultrasound.

The high frequency (8-13 MHz) probe is used for this. The optic nerve is viewed in 3 planes - the axial planes (saggital and transverse) and coronal plane.

The sonographic optic nerve sheath diameter, ONSD (but not optic nerve diameter, OND) is strongly related to ICP. Additionally, changes in optic nerve sheath diameter (ONSD) are strongly related to changes in ICP. The measure of the distension of the sheath surrounding the optic nerve (but not of the nerve itself) can be used to detect elevated ICP in neuro-ICU patients.

Axial planes

The probe is placed over the closed eyelid with the patient in supine position. Use small quantities of ultrasound gel to prevent it from getting into the eyes of the patient. The probe is held vertically over the eye and the beam is directed posteriorly towards the optic disc and nerve. A good view of the nerve often necessitates moving the probe slightly to the temporal side and angulating the beam a little nasally.

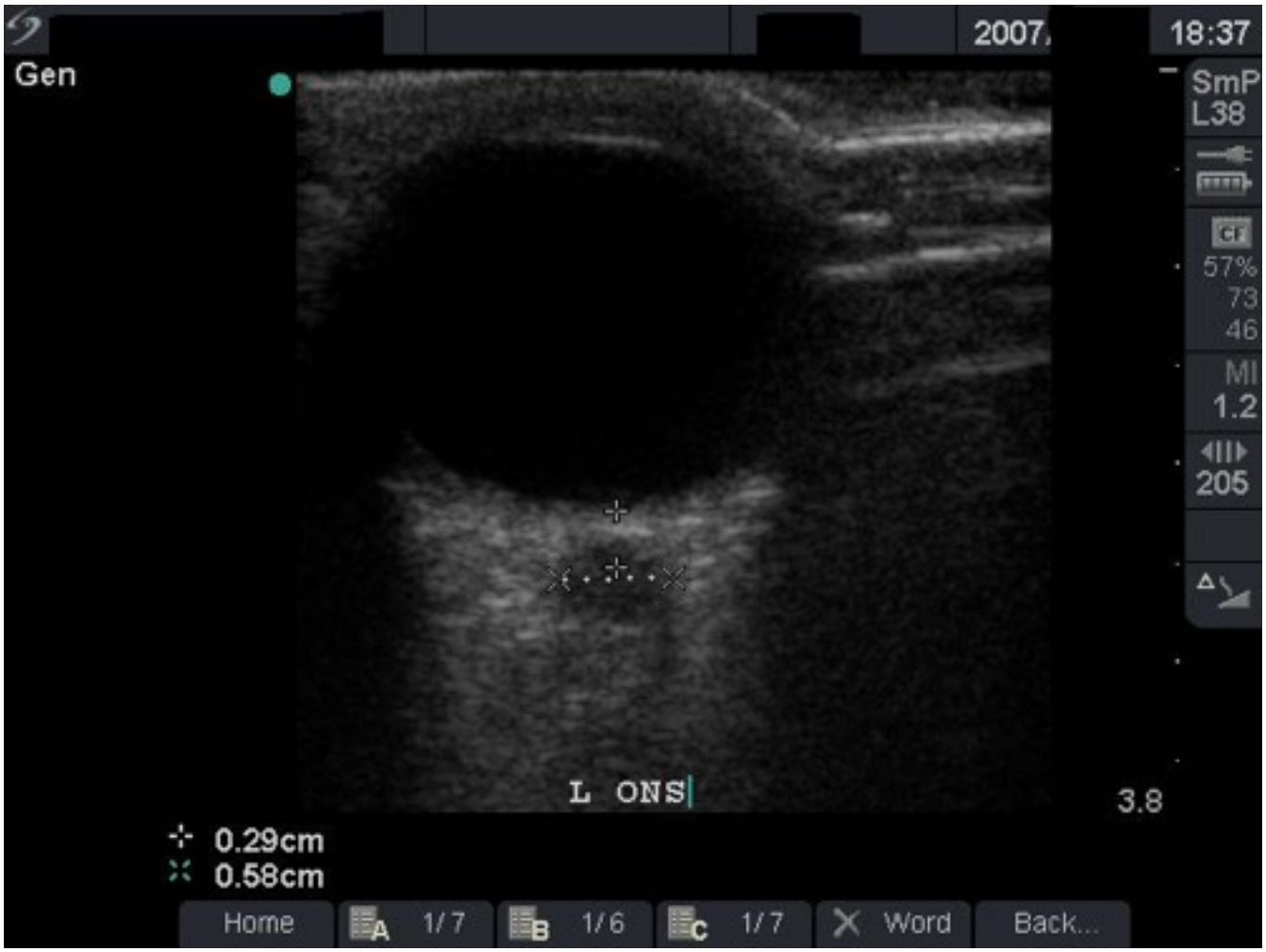

Fig. 1. Meaasuring ONSD with electronic calipers

2D mode is used and the field depth is reduced to 4 cms. The normal sonographic aspect of the optic nerve is from center to peripheral: hypoechogenic nerve fibers closely surrounded by the echogenic pia mater; the subarachnoid space appears anechogenic or hypoechogenic and is surrounded by hyperechogenic dura mater and periorbital fat. The OND can be measured as the distance inside the pia mater, and the ONSD as the distance inside the dura mater. Without zoomong in on the optic nerve, OND and ONSD are measured 3 mm behind the globe, using an electronic caliper and an axis perpendicular to the optic nerve. Two measurements are made for each optic nerve: one in the sagittal plane and the other in the transverse plane, by rotating the probe clockwise. The mean of value obtained for both eye is taken as the OND and the ONSD values.

Coronal plane

The probe is placed over the lateral angle of the eye with the marker facing cephalad. The beam is focussed posteriorly and medially until the nerve sheath is seen as an oval structure behind the globe. The shortes t diameter of this oval represents the coronal view diameter of the nerve sheath. This is an oblique coronal section as a true coronal view is technically not possible because of the intervening lateral wall of the bony orbit. the average values of the axial and coronal section diameters can be taken.

In a study published in 2007, done on patients with head injury (excluding those with ocular trauma), the best cut off value (using an ROC curve) for ONSD predicting elevated ICP was 5.9mm. In the same study, it was found that if the ONSD was less than 5.7mm, its sensitivity and negative predictive value were 100%. In another study published in 2008, it was found that in sedated neurocritical care patients, non-invasive sonographic measurements of ONSD correlated with invasive ICP, and the probability to have raised ICP if ONSD was less than 5.86 mm was very low.

Artifact

It is important to be aware that sometimes, an edge artifact may be seen as 2 diverging black areas on either side of the nerve sheath. Including this in the measurement of ONSD will lead to an erroneous prediction of raised intracranial pressure.

Airway management

Pre-intubation

Ultrasound can be used to assess the anticipated difficulty in intubation of obese and sleep apnoeic individuals by measuring the thickness of fat tissue in the anterior neck region with a high frequency probe. In patients presenting with airway obstruction, tumors, abscesses or epiglottitis can be diagnosed on sonography.

Confirmation of endotracheal tube placement

It has been established that auscultating breath sounds is not a very accurate method of ensuring correct placement of the endotracheal tube in the trachea after intubation. Ultrasound, albeit using indirect evidence can do tha same more accurately.

Two assessments need to be made on each side after intubation.

1. Assessment of lung sliding
2. Assessment of diaphragmation excursion

Assessment of lung sliding

The to and fro movement of the visceral pleura can be assessed with a routine ultrasound or high frequency probe as described in tutorial 9.

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