Echocardiography in Intensive Care: The Basics. Part II

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ABSTRACT
Objective: To review the current status of echocardiography in critically ill patients with special reference to the advantages and disadvantages of the transthoracic and transoesophageal approaches.

Data sources: A review of articles published in peer reviewed journals from 1976-1999 and identified through a MEDLINE search on echocardiography.

Summary of review: Echocardiography is a rapidly evolving field which is relatively new to intensivists. Both transthoracic echocardiography (TTE) and transoesophageal echocardiography (TOE) are extremely useful in managing critically ill patients. In intensive care practice echocardiography is used to evaluate clinical syndromes such as unexplained hypotension, search for source of sepsis or source of emboli, as well as haemodynamic assessment and monitoring. TOE although somewhat invasive, provides superb images which often assist in both diagnosis and improved treatment.

Conclusions: Echocardiography often provides useful information in critically ill patients. Intensivists should familiarise themselves with this new technology and if possible become skilled practitioners of this exciting technique. The care of critically ill patients will benefit from its widespread use. (Critical Care and Resuscitation 1999; 1: 296-310)

Key Words: Echocardiography, transoesophageal echocardiography, transthoracic echocardiography, intensive care, critical care

INDICATIONS FOR ECHOCARDIOGRAPHY
The indications for echocardiography broadly fall into three categories (Table 1):
1) diagnosis of specific cardiac/aortic pathology (usually guided by clinical suspicion),
2) haemodynamic assessment/monitoring of cardiac function, and
3) others - such as evaluation of patients with atrial fibrillation for the presence of left atrial clot prior to cardioversion.

In intensive care practice echocardiography is used to evaluate ‘clinical syndrome(s)’ that suggest a diagnosis or more often a number of possible diagnoses. For example, hypotension in a patient with acute myocardial infarction suggests severe left ventricular dysfunction associated with a major regional wall motion abnormality. Nevertheless, the echocardiographic examination might reveal a ruptured papillary muscle causing torrential mitral regurgitation, or a ventricular septal defect. Some common syndromes in which echocardiography is especially useful are listed in table 2. The most important in the management of critically ill patients is the rapid diagnosis and assessment of unexplained hypotension. As a general rule, unless there is gross haemodynamic instability or some other overriding reason, the echocardiographic examination should be structured around a ‘basic’ set of 2-D views. This applies to both TTE and TOE.

It should however, be appreciated that a truly ‘comprehensive’ echocardiographic examination is impractical in most critically ill patients. Indeed, once the echocardiographic diagnosis has been made, the patient’s condition may be so grave that immediate treatment takes precedence over any further examin-
### Table 1. Indications for Echocardiography

#### Diagnosis

**Valvular heart disease** (native or prosthetic valves)
- Stenosis/regurgitation
- Infective endocarditis

**Left ventricular (and/or right ventricular) function**
- Systolic - Regional wall motion abnormality (myocardial infarction/stunning)
  - Global (cardiomyopathy/stunning)
- Diastolic function
- Cardiomyopathy - dilated
  - hypertrophic
  - restrictive

**Pericardial disease**
- Pericardial effusion
- Cardiac tamponade
- Constriction
- Tumour (rare)

**Cardiac masses (thrombus, tumours and vegetations)**
- Clot - atrial (left > right)
  - ventricular (usually left)
- Tumours (e.g. left atrial myxoma)
- Vegetations - commonly involve valves

**Pulmonary embolism**

**Aortic disease**
- Aortic dissection
- Traumatic rupture
- Aortic atherosclerosis
- Others e.g. coarctation, aneurysm of sinus of valsalva

**Congenital heart disease/miscellaneous**
- Ventricular septal defect - congenital
  - acquired (acute myocardial infarction)
- Atrial septal defect
- Patent ductus arteriosis
- Ebstein’s anomaly
- Other e.g. Tetralogy of Fallot

**HAEMODYNAMIC ASSESSMENT/MONITORING** (mainly left ventricle)

**LV systolic function**
- preload - end diastolic volume/end-diastolic area
- contractility - visual assessment - global/regional
  - left ventricular ejection fraction/fractional area of change
  - stroke volume/cardiac output

**RV systolic function**

**Diastolic function**
- Mitral flow velocity profiles
- Pulmonary venous flow velocity profiles

**MISCELLANEOUS**
- Exclusion left atrium/left atrial appendage clot prior to DC cardioversion
Table 2. Indications for echocardiography in the intensive care unit according to clinical syndrome

<table>
<thead>
<tr>
<th>Clinical Syndrome</th>
<th>Findings</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>HYPOTENSION</td>
<td></td>
<td></td>
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<tr>
<td>Acute myocardial infarct</td>
<td></td>
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<tr>
<td>- no murmur</td>
<td>LV RWMA(s)</td>
<td>Usually severe ↓ LV systolic function.</td>
</tr>
<tr>
<td></td>
<td>RV RWMA</td>
<td>RV ↓ &gt; LV ↓.</td>
</tr>
<tr>
<td></td>
<td>Hypovolaemia</td>
<td>‘Empty’ LV cavity, systolic function largely preserved.</td>
</tr>
<tr>
<td>- new murmur</td>
<td>Papillary muscle rupture/severe MR</td>
<td>‘Good’ LV function with MR.</td>
</tr>
<tr>
<td>- rarely</td>
<td>LV pseudoaneurysm</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cardiac rupture/tamponade</td>
<td></td>
</tr>
<tr>
<td>Cardiothoracic surgery</td>
<td>Cardiac tamponade</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(New) RWMA</td>
<td>May be due to graft occlusion, air embolism.</td>
</tr>
<tr>
<td></td>
<td>Global</td>
<td>Stunning or long standing cardiomyopathy.</td>
</tr>
<tr>
<td></td>
<td>Valvular dysfunction</td>
<td>Often long standing but may be caused or worsened by surgery e.g. ischaemia to papillary muscle of mitral valve.</td>
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<tr>
<td></td>
<td>Dynamic LVOT obstruction</td>
<td>Inotropes/IABP/hypovolaemia worsens LVOT obstruction.</td>
</tr>
<tr>
<td>Trauma</td>
<td>Hypovolaemia</td>
<td>‘Empty’ LV with vigorous contraction.</td>
</tr>
<tr>
<td></td>
<td>Cardiac contusion</td>
<td>RWMA (RV &gt; LV).</td>
</tr>
<tr>
<td></td>
<td>Valvular injury</td>
<td>Most common aortic valve (AR) or mitral valve (MR), occasionally tricuspid valve (TR).</td>
</tr>
<tr>
<td></td>
<td>VSD/ASD</td>
<td>Occasionally.</td>
</tr>
<tr>
<td></td>
<td>Cardiac tamponade</td>
<td>More common in penetrating chest injuries.</td>
</tr>
<tr>
<td></td>
<td>Ruptured thoracic aorta</td>
<td>90% at isthmus of aorta.</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Often normal LV systolic function</td>
<td>Possible global/regional LV depression.</td>
</tr>
<tr>
<td></td>
<td>with ‘empty’ LV</td>
<td>TOE more sensitive than TTE for imaging vegetations/abscess.</td>
</tr>
<tr>
<td>‘Isolated’ hypotension</td>
<td>Globally ↓ or RWMA</td>
<td>Cardiomyopathy or stunning.</td>
</tr>
<tr>
<td>(‘hypotension ?cause’)</td>
<td>Valvular dysfunction</td>
<td>Usually chronic, occasionally acute e.g. ruptured papillary muscle of mitral valve.</td>
</tr>
<tr>
<td></td>
<td>Dynamic LVOT obstruction</td>
<td>Pulmonary embolism - usually dilated right heart sometimes with clot in a proximal pulmonary artery.</td>
</tr>
<tr>
<td></td>
<td>Acute cor pulmonale</td>
<td></td>
</tr>
<tr>
<td>SEPSIS (?source)</td>
<td>Vegetations, regurgitation ± abscess</td>
<td>Infective endocarditis until proven otherwise.</td>
</tr>
<tr>
<td></td>
<td>Normal TOE examination</td>
<td>Virtually excludes infective endocarditis.</td>
</tr>
</tbody>
</table>
### Table 1. (continued)

<table>
<thead>
<tr>
<th>Clinical Syndrome</th>
<th>Findings</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>SYSTEMIC EMBOLI (?source)</td>
<td>• LA/LAA clot</td>
<td>Usually enlarged LA and atrial fibrillation. TOE usually required for diagnosis.</td>
</tr>
<tr>
<td></td>
<td>• LV clot</td>
<td>Usually associated with RWMA or global LV decrease.</td>
</tr>
<tr>
<td></td>
<td>• Aortic atherosclerotic plaques</td>
<td>TOE essential for diagnosis.</td>
</tr>
<tr>
<td></td>
<td>• Vegetations - aortic or mitral valve</td>
<td>?Sepsis.</td>
</tr>
<tr>
<td></td>
<td>• Clot - prosthetic aortic or mitral valve</td>
<td>Associated prosthetic valve dysfunction.</td>
</tr>
<tr>
<td></td>
<td>• Patent foramen ovale with paradoxical embolism</td>
<td>RA pressure &gt; LA pressure (e.g. IPPV with high PEEP). TOE (bubble contrast) usually necessary for diagnosis.</td>
</tr>
<tr>
<td></td>
<td>• Tumour (e.g. LA myxoma)</td>
<td>Uncommon.</td>
</tr>
<tr>
<td>PULMONARY OEDEMA (?cause)</td>
<td>• ↓ LV systolic/diastolic function</td>
<td>Occasionally normal systolic but abnormal diastolic LV function.</td>
</tr>
<tr>
<td></td>
<td>• Valvular dysfunction (MR, MS, AR, AS)</td>
<td>Note, if flail leaflet/ruptured papillary muscle suspected then TOE indicated.</td>
</tr>
<tr>
<td></td>
<td>• Intracardiac shunt</td>
<td>Suggests non-cardiac cause e.g. ARDS.</td>
</tr>
<tr>
<td></td>
<td>• Normal</td>
<td>Infers moderate to large clot burden.</td>
</tr>
<tr>
<td>DYSPNOEA/HYPOXIA WITHOUT PULMONARY OEDEMA (dyspnoea ?cause)</td>
<td>• Pulmonary embolism (dilated right heart chambers ± clot in pulmonary artery)</td>
<td>Transient - infers ischaemia (stunning), permanent indicates MI.</td>
</tr>
<tr>
<td></td>
<td>• Cardiac tamponade</td>
<td>TOE more sensitive than TTE.</td>
</tr>
<tr>
<td></td>
<td>• Miscellaneous e.g. chronic cor pulmonale, constrictive pericarditis, intracardiac shunt</td>
<td>Moderate to large embolus.</td>
</tr>
<tr>
<td>CHEST PAIN OF UNCERTAIN AETIOLOGY (chest pain ?cause)</td>
<td>• RWMA</td>
<td>Effusion often too small to diagnose with echocardiography.</td>
</tr>
<tr>
<td></td>
<td>• Dissecting aortic aneurysm (intimal flap, true/false lumen)</td>
<td>Usually other clinical signs of stenosis.</td>
</tr>
<tr>
<td></td>
<td>• Pulmonary embolism (dilated right heart chambers ± clot in pulmonary artery)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Pericarditis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Aortic stenosis</td>
<td></td>
</tr>
</tbody>
</table>

AR = aortic regurgitation, AS = aortic stenosis, ASD = atrial septal defect, IABP = intra-aortic balloon pump, LA = left atrium, LAA = left atrial appendage, LV = left ventricle, LVOT = left ventricular outflow tract, MI = myocardial infarction, MR = mitral regurgitation, MS = mitral stenosis, RA = right atrium, RV = right ventricle, RWMA = regional wall motion abnormality, TR = tricuspid regurgitation, VSD = ventricular septal defect.

**Valvular stenosis**

Narrowing or stenosis of any heart valve obstructs blood flow, increasing velocity and causing a pressure gradient across the valve. Evaluation of valvular stenosis requires:

1. imaging of the valve to define the morphology and mobility of the valve cusps,
2. some quantification of the degree of stenosis, and,

...
3) the effect of the pressure overload on relevant cardiac chambers.

Pressure gradients cannot be measured directly by echocardiographic techniques. Transvalvular pressure gradients (ΔP) can however be estimated by Doppler techniques. Using the simplified Bernouilli equation (ΔP = 4V², where V is the velocity of blood flow) accurate and reproducible estimations of maximum and mean pressure gradients can be obtained. Note that in conditions of low cardiac output pressure gradients will be low, thereby underestimating the severity of stenosis.

Aortic stenosis. Aortic stenosis should be excluded in critically ill patients presenting with sudden collapse, cardiogenic shock, or pulmonary oedema of uncertain aetiology. The most common cause of valvular aortic stenosis is degeneration and calcification of the valve apparatus. Thickening/calcification of relatively immobile valve leaflets cause left ventricular hypertrophy (pressure overload).

Severe aortic stenosis usually causes:

1) increased peak aortic velocity (> 4m/sec),
2) mean pressure gradient > 35mmHg, and
3) aortic valve area < 0.8cm².

The aortic valve area is estimated using the continuity equation, which based on the principle: if the volume of blood flow upstream from an orifice is known, then measuring the velocity of blood immediately downstream from the orifice allows calculation of its cross sectional area. According to the formula: A₁ x V₁ = A₂ x V₂

Where
A₁ = LVOT cross sectional area
V₁ = LVOT velocity time integral
A₂ = aortic valve cross sectional area
V₂ = aortic velocity time integral.

The equation can be rearranged:

A₂ = \( \frac{A₁ \times V₁}{V₂} \)

The ratio of LVOT velocity/aortic valve velocity provides a measurement of aortic stenosis severity which is relatively independent of cardiac output. A velocity ratio of < 0.25 indicates an aortic valve area of < 1.0cm² i.e. at least moderate aortic stenosis. Accurate maximum aortic jet velocity is best obtained by TTE. TOE allows planimetry (tracing) of the aortic valve orifice in the short axis view and this has been shown to correlate well with catheter derived aortic valve area.

Mitral stenosis. Significant mitral stenosis can occasionally contribute to haemodynamic instability in critically ill patients. Dyspnoea may have been present for many years so the diagnosis has usually been made prior to admission to intensive care. Mitral stenosis produces typical 2-D images of thickened and fused mitral valve leaflets with diastolic doming resulting in a ‘hockey stick’ appearance of the anterior leaflet together with an enlarged left atrium.

In the short axis view the mitral valve appears as a ‘fish mouth’ orifice the area of which can be planimeted to estimate the mitral valve area. Mitral stenosis severity can be assessed by Doppler techniques:

1) the peak velocity across the mitral valve is usually increased (> 1.3m/sec),
2) a significant transmitral pressure gradient (mean pressure gradient > 12mmHg suggests severe stenosis), although the pressure gradient can be quite small when cardiac output is low,
3) mitral valve area - the pressure half time method \( (P_{1/2})^2 \) is commonly used.

This is based on the principle that the rate of pressure decrease across a stenotic orifice is determined by its cross sectional area i.e. the smaller the orifice the slower the rate of fall of pressure. The \( P_{1/2} \) is the time it takes for the maximum pressure gradient to halve. The mitral valve area as estimated by the \( P_{1/2} \) method should always be compared to the valve area as estimated by another technique such as planimetry of the mitral valve orifice. Pulmonary artery pressure can be estimated by Doppler techniques.

Tricuspid stenosis and pulmonary stenosis. These disorders can also be diagnosed and quantified using similar echocardiographic techniques.

Valvular regurgitation

Evaluation of valvular regurgitation requires:

1) assessment of the valve morphology,
2) estimation of severity of regurgitation,
3) effects of volume overload on relevant cardiac chambers.

Mild valvular regurgitation of the mitral, tricuspid and pulmonary valves is common (70-90%) in normal individuals. Trivial aortic regurgitation is found in only about 5% of ‘normal’ examinations.

Aortic regurgitation. 2-D echocardiography may show the cause of the aortic regurgitation, for example
valve destruction caused by infective endocarditis or a
dilated aortic root with aortic dissection. Colour-flow
doppler imaging allows some grading of severity of
regurgitation by comparing the width of the regurgitant
jet to the LVOT area (Figure 1).

![Figure 1. Severe aortic regurgitation; aliased jet occupies entire left
ventricular outflow tract (TOE)](image)

If the jet occupies more than 60% of the LVOT area
then severe aortic regurgitation is probably present. The
pressure half-time measurement can also be used to
assess severity; a pressure half-time less than 250msec
infers severe regurgitation. Diastolic flow reversal
downstream in the aorta and great vessels occurs when
regurgitation is severe.

Mitral regurgitation. Acute mitral regurgitation may
be a catastrophic event causing overwhelming pulmonary
oedema and cardiogenic shock. Chronic mitral regur-
gitation even when severe usually causes few symptoms until
the left ventricle (LV) fails. 2-D and Doppler
echocardiography can diagnose mitral regurgitation
(Figure 2) and define the underlying anatomical aetiology
for the regurgitation, for example papillary muscle
rupture, infective endocarditis, annular dilatation etc.

Echocardiography is useful in differentiating acute
from chronic mitral regurgitation: acute mitral regurgita-
tion (e.g. papillary muscle rupture), the LV is normal in
size and LV function hyperdynamic and the left atrium is
not dilated; chronic mitral regurgitation causes enlarge-
ment of the left atrium and sometimes of the LV although
initially LV function is well preserved.

Severity of mitral regurgitation is assessed by colour
Doppler imaging of the regurgitant jet in several planes:
regurgitation jet area/left atrial area ratio > 40% suggests
severe mitral regurgitation as does systolic flow reversal
in the pulmonary veins. TOE images only part of the left
atrium and therefore jet area/left atrial ratio may be
inaccurate. A semiquantitative grading system based on
jet area may be useful. In difficult cases other techniques
such as measurement of proximal isovelocity surface area
(PISA) can be used. These methods are somewhat tedious
and prone to error although in modern echocardiography
machines measurement of the PISA has been greatly
simplified. Recently it has been demonstrated that colour
flow mapping of the narrowest cross sectional area of the
regurgitant jet (vena contracta) accurately estimates the
severity of the mitral regurgitation and this technique can
be done in < 1 minute.

![Figure 2. Severe mitral regurgitation secondary to flail posterior mitral
valve leaflet (TOE)](image)

Tricuspid regurgitation and pulmonary
regurgitation. These disorders can also be diagnosed
and evaluated using similar principles, for example
severe tricuspid regurgitation causes systolic flow
reversal in the hepatic veins.

INFECTIVE ENDOCARDITIS

Exclusion of endocarditis is one of the most frequent
indications for a comprehensive echocardiographic
examination in intensive care. Untreated and undiagno-
sed infective endocarditis is a devastating disease. The
diagnosis of infective endocarditis requires integration
of clinical, microbiological and echocardiographic
information. An independent, chaotically moving, mass
tached to a heart valve in a septic patient is
almost certainly a vegetation (Figure 3, Figure 4).
In other situations clot, tumour, marantic vegetation or some other echocardiographic abnormality may mimic endocarditis. In a patient with endocarditis echocardiography can:

1) image the vegetation,
2) diagnose complications such as paravalvular abscess, fistula etc.,
3) examine underlying valve morphology e.g. bicuspid aortic valve, perforated mitral valve leaflet etc.,
4) diagnose and assess severity of associated valvular regurgitation,
5) assess cardiac function,
6) image other heart valves.

Large vegetations (> 10mm) increase the risk of embolic events.\textsuperscript{7,8} Detection of vegetations is better with TOE compared with TTE (TOE 90-100% vs TTE 40-80%).\textsuperscript{7,9,10}

About 25% of patients with Staphylococcus aureus sepsicaemia have infective endocarditis even in the absence of obvious clinical signs. TOE is essential for diagnosis and detection of associated complications.\textsuperscript{11}

Suspected prosthetic valve endocarditis mandates TOE and serial examinations are often necessary.

PROSTHETIC VALVES

Echocardiographic examination of prosthetic valves requires knowledge of the various types of valves (bioprosthesis, homograft, mechanical). Blood velocity is nearly always increased through normal prosthetic valves. Trivial regurgitation is characteristic of most mechanical valves. Such jets are always small, may be multiple and occur within the sewing ring of the valve.

Paravalvular leaks are always abnormal and associated with dehiscence; infective endocarditis should always be considered. A characteristic rocking motion of the valve is diagnostic of dehiscence. TOE is superior to TTE for assessing mitral regurgitation in patients with prosthetic mitral valves as acoustic shadowing by the mechanical valve of the left atrium causes poor imaging.

VENTRICULAR FUNCTION, HAEMODYNAMIC MONITORING AND ASSESSMENT

In critically ill patients echocardiography, especially TOE, has evolved as an independent approach in the assessment of cardiovascular haemodynamics.\textsuperscript{9,12,13} Cardiac systolic function is directly visualised and other parameters can be directly measured. On occasions feedback from the echocardiographic images to the intensivist can be virtually instantaneous.

Left ventricle

Echocardiography allows accurate measurements of LV dimensions and wall thickness.

Systolic function

**Preload**

The assessment of left ventricular end diastolic volume (LVEDV) can be crucial in critically ill patients. 2-D echocardiography provides both qualitative and quantitative estimation of preload. LV end systolic cavity obliteration infers significant hypovolaemia and may precede changes in blood pressure.\textsuperscript{14} Although a presumptive diagnosis of hypovolaemia can be made, the other causes of end systolic cavity obliteration must be excluded including decreased systemic vascular resistance, severe mitral or aortic regurgitation, or ventricular septal defect. Integrating the clinical picture
together with colour flow Doppler and if necessary quantitative assessment of preload, differentiates between the various diagnoses. For example in a patient with a ruptured papillary muscle the LV is usually hyperdynamic and on occasions the walls may ‘kiss’ at end systole; nevertheless severe mitral regurgitation will be obvious on colour flow Doppler.

**End diastolic area (EDA)** Measurement of LV end diastolic area at the level of the papillary muscles in the short axis view provides a quick and easy estimate of LV filling (normal = 15-34cm$^2$) even in patients with chronic LV dysfunction. This method correlates well with LV volume determined by radionuclide studies.

**Left ventricular end-diastolic volume.** More precise assessment of LV volume requires multiple imaging planes and axes. However, these methods are time consuming and infer three-dimensional volume from two-dimensional imaging. The recent development of three-dimensional echocardiography will allow more rapid and accurate computation of left ventricular volume.

**Afterload**

End systolic wall stress [where wall stress = (pressure x radius)/(2 x wall thickness)] determined by echocardiography provides an index of LV afterload. This can be calculated using both echocardiographic and systolic arterial pressure measurements and correlates well with micromanometer LV pressure recordings.

**Contractility**

Assessment of LV contractility in a patient cannot be independent of loading conditions. Nevertheless 2-D and Doppler echocardiography provides global and regional estimates of ventricular performance.

**Global contractility.** Qualitative estimation of systolic function (‘eye balling’) can be performed quickly by an experienced observer.

**Ejection fraction.** In clinical echocardiography the left ventricular ejection fraction (LVEF) is one of the most frequently used parameters to assess global LV systolic function (normal $\geq$ 50%).

$$\text{LVEF}\% = \frac{\text{LVEDV} - \text{LVESV}}{\text{LVEDV}} \times 100$$

Where,

- LVEDV = left ventricular end diastolic volume
- LVESV = left ventricular end systolic volume

The endocardial border is traced out during systole and diastole.

**Fractional area of change (FAC).** The LV fractional area of change (normal $> 37\%$) is a reasonable approximation of LV ejection fraction. Its major advantage is the speed and ease of measurement. Fractional area of change is measured at the level of the papillary muscle in the short axis view:

$$\text{FAC} = \frac{\text{EDA} - \text{ESA}}{\text{EDA}}$$

Where,

- EDA = end diastolic area
- ESA = end systolic area

**Automatic border detection (ABD).** An automated analysis system is now available which continuously tracks the endocardial borders and provides real time continuous display of LV end diastolic area (EDA) and end systolic area (ESA) (Figure 5).

![Figure 5. Automated border detection (from TTE: apical 4-chamber view). Left ventricular (LV) cavity enclosed in a ‘region of interest’. Real time LV end-diastolic, end-systolic volumes and ejection fraction are displayed.](image-url)

These automated estimates have been shown to be virtually identical to expert laboratory measurements. ABD should represent a real advance in monitoring of critically ill patients.

**Regional function.** Echocardiography may be the ideal diagnostic tool with which to detect myocardial ischaemia: ischaemic segments or regional wall motion abnormalities (RWMA) are easily detectable.

There is a very close temporal relationship between the onset of myocardial ischaemia and the development of a RWMA. To improve the characterisation of the RWMA, various ‘semi-quantitative’ scoring systems are available. A wall motion index representing 16 segments of the LV is widely used. The use of echocardiographic contrast agents may aid in differen-
Stoke volume and cardiac output. Doppler derived blood flow velocities can be used to quantify cardiac output. The technique is based on the principle that the velocity-time integral (VTI) of blood flow multiplied by the cross sectional area (CSA) of its orifice yields an estimate of stroke volume (SV = CSA x VTI). Investigators have measured stroke volume at different sites including LVOT, pulmonary artery and mitral valve. Correlations between echocardiographic and thermodilution derived cardiac output have yielded mixed results. Obtaining the necessary 2-D and Doppler information can be time consuming compared to the ease with which more robust data on cardiac output is available using the pulmonary artery catheter.

Diastolic function
Abnormalities of diastolic function may precede systolic dysfunction. Assessment of diastolic function is complex and it should be emphasised that abnormal diastolic filling patterns are not specific for any pathologic process. Thus Doppler diastolic function assessment should be interpreted in conjunction with 2-D echocardiographic findings. An attempt to evaluate diastolic function using diastolic filling profiles can be made utilising: 1) mitral flow velocity profile - E velocity (early diastolic filling), A velocity (atrial systole), E/A ratio, deceleration time (slope of early diastolic deceleration), isovolumetric relaxation time (IVRT); 2) pulmonary venous flow profile: S-wave (systolic forward flow), D-wave (diastolic forward flow), AR-wave (atrial reversal). In the clinical setting evaluation of diastolic LV function is often difficult and requires expert echocardiographic knowledge and training.

Right ventricle
Evaluation of right sided heart function is important in critically ill patients.

Chamber size: The normal shape of the right ventricle is a crescent, curving in front of, and concave towards, the LV. A combination of image planes is necessary to assess right ventricular size. Ventricular dilation may be due to right sided volume overload secondary to conditions such as tricuspid regurgitation, pulmonary regurgitation, or atrial septal defect. Colour flow Doppler and occasionally venous injection of a contrast agent such as agitated saline will help distinguish the aetiology.

Wall thickness: Right ventricular hypertrophy (due to pressure overload) leads to increased thickness of the right ventricular free wall.

Systolic function: Right ventricular end diastolic volume (RVEDV) assessment is difficult because the shape of the right ventricular and the presence of prominent trabeculations make the endocardial border difficult to outline. Right ventricular dysfunction, often due to pulmonary hypertension, is especially problematic following orthotopic heart transplantation and is one of the leading causes of death in the first month. TOE is now considered essential for assessing right ventricular dysfunction following cardiac transplantation.

Contractility: Detection of regional wall motion abnormalities are sensitive and specific markers of right ventricular ischaemia or infarction. The diagnosis of cardiac contusion which occurs commonly after blunt chest trauma is often unrecognised. This diagnosis can be readily made by TOE.

Pulmonary artery pressure estimate: This is an important and routine part of assessing right ventricular function. The simplified Bernoulli equation is used: systolic pulmonary artery pressure = 4V^2_tr + RAP (or CVP). V_tr is the tricuspid regurgitation velocity measured by Doppler, and RAP is right atrial pressure (or CVP). Some tricuspid regurgitation is present in over 90% of patients.

CARDIOMYOPATHIES
A simple classification of the cardiomyopathies includes, dilated, hypertrophic and restrictive.

Dilated cardiomyopathy
All echocardiographic features of dilated cardiomyopathy are non-specific, nevertheless, there is characteristic enlargement of all four chambers with reduced global systolic function especially of the LV. Ejection fraction and fractional area of change are uniformly decreased.

Due to the increased LV end diastolic volume, stroke volume and cardiac output may be preserved at rest. Significant mitral regurgitation, secondary to annular dilation and poor coaptation of the mitral leaflets may be present. Diastolic dysfunction is not usually a prominent feature. Pulmonary artery pressure as estimated from the velocity of the tricuspid regurgitant jet is usually elevated.
Hypertrophic cardiomyopathy

In critically ill patients the diagnosis, unless already known, cannot be made without the assistance of echocardiography. Asymmetrical septal hypertrophy (ASH), the most common feature can be readily identified and quantified. A variety of other hypertrophic patterns may occur. In the elderly a normal proximal septal bulge (‘sigmoid septum’) should not be confused with true hypertrophic cardiomyopathy.

Left ventricular outflow tract obstruction. During systole, the anterior leaflet (occasionally both leaflets are involved) of the mitral valve moves anteriorly toward the interventricular septum (SAM) and may obstruct the LVOT. The LVOT obstruction is dependent on loading conditions and may be absent at rest. Indeed, the dynamic nature of the LVOT obstruction is one of the hallmarks of this condition. The systolic anterior motion of the leaflet distorts the mitral valve causing mitral regurgitation. Colour flow (and PW) Doppler not only localise the site of LVOT obstruction but also quantify the degree of mitral regurgitation. The severity of LVOT obstruction can be estimated by CW Doppler. Although the degree of obstruction may vary, a gradient \( \geq 50\text{mmHg} \) indicates significant LVOT obstruction.

Dynamic LVOT obstruction without asymmetrical septal hypertrophy.\(^{34}\) Dynamic LVOT obstruction with SAM can occur whenever a hyperdynamic state exists, especially in hypovolaemia. The haemodynamic effects of this condition are identical to the LVOT obstruction of hypertrophic cardiomyopathy and may cause cardiogenic shock. Unexplained persistent hypotension and/or low cardiac output in critically ill patients will often result in initiation of inotropic therapy. Dynamic LVOT obstruction may be precipitated or worsened by positive inotropic or vasodilator therapy. Indeed intra-aortic balloon pump, by reducing afterload, will have a deleterious effect.

A paradoxical haemodynamic response to conventional therapy is an indication for TOE in critically ill patients. The LVOT gradient can usually be abolished by ensuring adequate volume replacement and avoiding positive inotropic agents and increasing afterload by administration of an agent such as phenylephrine (a pure \( \alpha \)-agonist).

Restrictive cardiomyopathy (e.g. amyloid)

Echocardiographic findings include small thick walled ventricles with abnormal diastolic function and normal systolic function. Doppler echocardiography demonstrates a characteristic LV filling pattern. Differentiation from constrictive pericarditis is often difficult and best left to experts.

Pericardial disease

Pericardial effusion and tamponade

Cardiac tamponade is a true and reversible cardiac emergency. Pericardial fluid can be recognised on 2-D as an ‘echo free’ space around the heart (Figure 6a).

Whenever a pericardial effusion is imaged the possibility of tamponade should be considered. When the pressure in the pericardial sac exceeds the pressure within the cardiac chambers, cardiac tamponade occurs with impaired cardiac filling and low cardiac output. 2-D echocardiographic features of cardiac tamponade include right atrial collapse during ventricular systole, right ventricular diastolic collapse and occasionally there is left atrial collapse. Doppler echocardiography demonstrates exaggerated increase in tricuspid and decrease in mitral valve diastolic flow with inspiration.

Cardiac tamponade occurring after cardiothoracic surgery can be due to clot which is ‘echo dense’ and there is little or no ‘echo free’ space around the heart (Figure 6b). Localised tamponade, for example of the right atrium, is not uncommon in such patients. Not surprisingly standard echocardiographic criteria such as chamber collapse, are unreliable in detecting tamponade after cardiac surgery. The presence of \( \geq 1 \text{cm} \) of pericardial separation (fluid/clot) in a patient with unexplained clinical deterioration (hypotension, decreased cardiac output) appears to be sensitive in detecting tamponade.\(^{35}\) When tamponade is excluded other causes of hypotension such as unsuspected hypovolaemia, ventricular dysfunction, LVOT obstruction may be diagnosed. Indeed, unnecessary reoperation may be prevented in some instances.\(^{36}\)
Echocardiography is of value in the diagnosis of constrictive pericarditis. There is often thickening of the pericardium which may be imaged with 2-D echocardiography. Doppler studies of the tricuspid, mitral and pulmonary veins may show typical findings although differentiating constriction from restriction may be problematic.

CARDIAC MASSES

Before any diagnosis is made of a cardiac mass, it is essential to rule out pseudo-masses, i.e. artefacts or normal cardiac structures, for example, the eustachian valve may be mistaken for an intracardiac mass. TOE is usually more sensitive than TTE. Intracardiac masses include thrombi, tumours and vegetations.

Thrombi

Left atrial clot is much more common than right atrial clot. TOE is vastly superior to TTE as left atrial clot is usually located in or near the left atrial appendage (Figure 7) which is poorly visualised with TTE. Spontaneous echo contrast or echo ‘smoke’ is considered to be a precursor of clot formation and thromboemboli.13 LV clot (Figure 8) nearly always occurs in the setting of a regional wall motion abnormality or severe global depression of LV systolic function. TTE is more sensitive than TOE for detection of LV apical clot.

Tumours

Left atrial myxoma is by far the most common cardiac tumour and can be imaged by TTE although superb images can be obtained with TOE. The tumour is usually attached by a pedicle to the interatrial septum. During diastole the tumour may obstruct the mitral valve orifice. Rarely other tumours, primary or metastatic, may be visualised.
Critical Care and Resuscitation 1999; 1: 296-310  K. D. DONOVAN, ET AL

Cooperative Study Group for Echocardiography confirmed the high sensitivity (99%) and specificity (98%) of TOE in diagnosing aortic dissection when compared with thoracic CT and angiography. 2-D echocardiography will reveal the intimal flap. Colour flow Doppler is useful in differentiating the true from the false lumen, identifying the entry point and diagnosing the presence and extent of aortic regurgitation (Figure 9).

Figure 9. Aortic dissection with intimal flap separating the true lumen from the false lumen. Colour flow Doppler demonstrates flow only in the true lumen (TOE).

**Intramural haematoma (IMH).** Intramural haematoma represents a variant of aortic dissection and may be an early finding in patients who develop classical aortic dissection or rupture. It cannot be diagnosed by angiography as there is no intimal flap. 2-D echocardiography identifies IMH as a (> 0.7cm) circular or crescentric thickening of the aortic wall, with central displacement of intimal calcification. There may be a thrombus-like echo pattern of the aortic wall.

*Traumatic aortic rupture.* In patients with blunt chest trauma a widened mediastinum on chest x-ray should always arouse suspicion of a ruptured aorta. In critically ill patients however, mediastinal widening is difficult to assess and lacks specificity. The bedside availability of TOE and avoidance of contrast injection make it a useful screening test for aortic rupture in this situation. In one of the largest trials to date, 101 patients with suspected aortic trauma underwent both TOE and aortography; the sensitivity and specificity of TOE was 100% and 98% respectively.

The echocardiographic signs of aortic injury may include an intimal flap, aortic wall haematoma, aortic occlusion and fusiform aneurysm. It must be emphasised that echocardiographic assessment for aortic injury requires an experienced operator as other studies have demonstrated less favourable results in inexperienced hands.

*Aortic atherosclerosis.* TOE allows imaging of aortic plaques which may be layered and immobile or pedunculated and liable to embolise systemically. On occasions the aetiology of stroke, renal failure or peripheral ischaemia in critically ill patients may become apparent after echocardiographic examination of the aorta. Although optimal treatment of complex mobile plaque is currently unknown, recognition of its presence may lead to avoidance of procedures involving catheterisation of the aorta.

**PULMONARY EMBOLISM**

Acute obstruction of the pulmonary vasculature by pulmonary embolism usually causes hypoxia and cardiovascular collapse. Although there are many other potential causes of this common syndrome in critically ill patients, an echocardiographic examination can usually differentiate between them.

Massive pulmonary embolism (i.e. embolism involving two or more lobar arteries) invariably causes acute cor pulmonale. The right ventricle is acutely overloaded causing distension and ‘rounding’ of the right ventricle with the interventricular septum bulging into the LV cavity resulting in LV diastolic dysfunction. The LV cavity is usually small and appears ‘under-volumed’. Significant right ventricular hypertrophy is absent. Pulmonary hypertension is invariable and its severity can be estimated from the tricuspid regurgitant jet. Clot can also on occasions be directly imaged with TOE in the right heart cavities, main pulmonary artery, or right or left branches. Visualisation of a clot is not a rare event (Figure 10).

Echocardiography is both sensitive and specific for the diagnosis of massive pulmonary embolism. Jardin, for example, investigated 104 patients by echocardiography for suspected pulmonary embolism. Acute cor pulmonale was diagnosed in 75 patients with confirmation of diagnosis at angiography in 74 of 75 patients. Of the remaining 29 patients without echocardiographic signs of acute cor pulmonale, 5 patients had submassive pulmonary embolism on angiography and 24 had a normal pulmonary angiogram. It should be appreciated that a normal echocardiogram does not exclude a small pulmonary embolus because it does not cause significant haemodynamic perturbations. Other diagnostic procedures may be appropriate.
CONGENITAL HEART DISEASE

Echocardiography is essential for the evaluation of patients with known or suspected congenital heart disease. It is however, unusual for adult patients to present to intensive care with undiagnosed haemodynamically significant congenital heart disease. Moreover the echocardiographic assessment of complex congenital heart disease is best left to an expert cardiologist with an interest in this field. Relatively simple congenital heart abnormalities such as atrial septal defect (Figure 11), ventricular septal defect, patent foramen ovale, patent ductus arteriosis, coarctation of the aorta, can be diagnosed by the intensive care echocardiographer. Similarly non-congenital ventricular septal defect, for example complicating acute myocardial infarction or trauma is easily diagnosed using either TTE or TOE.

Figure 10. Clot in the right pulmonary artery (TOE).

Figure 11. Large atrial septal defect. (TOE).

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