Echocardiography and the critically ill patient

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Summary Echocardiography provides valuable diagnostic information in the assessment and management of the critically ill patient on the intensive care unit (ICU). Transthoracic echocardiography (TTE), through the advent of portable hand-held echocardiography devices can provide the cardiologist, the intensivist or the sonographer with a rapid non-invasive and safe assessment of the cardiovascular status of the patient on ICU. Recent advances in technology such as harmonic imaging, digital image acquisition and contrast echocardiography have made for improvements in image quality. This has extended the use of bedside echocardiography from not only assessing the cardiac status, but also to diagnose or exclude complications of procedures and therapies commonly used in ICU. However, in common with other investigations, the information obtained from bedside echocardiography should be interpreted in the context of other relevant investigations and clinical findings. As more and more critical care physicians are getting trained in performing bedside echocardiography, they should be aware of different techniques that can be used while performing the study. Here, we review the application of echocardiography in various clinical diagnoses encountered in ICU and the interpretation of findings in echocardiography.

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Introduction

Echocardiography has emerged as an important diagnostic tool in the management of the critically ill patient on the intensive care unit (ICU). Transthoracic echocardiography (TTE) is simpler and safer to perform in the ICU and is less time consuming when compared to transesophageal echocardiography (TEE), which was once considered the principle diagnostic tool for the assessment of the ICU patient. With new advances in technology, such as harmonic imaging, digital image acquisition and the use of contrast agents for endocardial enhancement, TTE can provide important information that can aid in the diagnosis and management of the patient in the ICU.

With the advent of new portable hand-held echocardiography devices, any suitably trained person (whether they be an intensivist, cardiologist or sonographer) are now able in a relatively short period of time to undertake a point-of-care echocardiographic study. Obviously these bedside echocardiographic studies have their own...
limitations in terms of image quality and functional limitations of the hand-held machines, which the physician should be aware of, when interpreting the study. In addition, the ability to undertake an echocardiographic study in the average ICU patient is hampered by sub-optimal positioning of the patient, the inability of the patient to cooperate with the study, limited access to the traditional echocardiography 'windows' (due to intravenous lines and chest drains), the use of inotropes and the fact that the patients is intubated. Also these bedside studies should be employed as a means of stabilizing the ICU patient such that further definitive information can be obtained by performing a more detailed departmental echo.

However, in common with other investigations, the information obtained from bedside echocardiography should be interpreted in the context of other relevant investigations and clinical findings. This article will review the potential uses of bedside echocardiography in the assessment and management of critically ill patients on the ICU (see Table 1).

Assessment of left ventricular (LV) function

The assessment of LV function is the most common reason for performing bedside echocardiography in the ICU. LV systolic function can be assessed using echocardiography by measuring ejection fraction (EF—normal range 55–75%), fractional shortening (FS—normal range 30–42%) and cardiac output (CO). The relevant LV dimensions can be obtained from the parasternal long axis view and EF and FS can be calculated using the formula in Box 1. However, quantification of EF and FS has its limitations particularly, when accurate LV volumes and dimensions cannot be obtained due to suboptimal imaging.6 In this scenario, a visual estimate of global LV systolic function can be determined and graded normal LV systolic function or mild, moderate and severe impairment of LV systolic function.7

Estimation of stroke volume by the modified Simpson method (which requires accurate delineation of the endocardium in systole and diastole) is technically difficult in the critical care setting. It is far more practical and amenable to estimate stroke volume and CO, using Doppler-derived instantaneous blood flow velocity through a conduit with a known cross-sectional area (CSA). Using the LV outflow tract (LVOT) as the conduit is probably the most reliable and most commonly used application of this principle.8 The LV stroke volume is obtained by measuring the CSA of the LVOT multiplied by the transaortic flow velocity time integral (VTI) derived by spectral doppler tracing (see Box 2). The LVOT diameter is best measured in the parasternal long axis view 1 cm below the aortic valve and the transaortic VTI is obtained in the apical 5-chamber view.

The ability to assess the LV for regional wall motion abnormalities (RWMA) in the critically ill patient is essential particularly after a suspected myocardial ischemic event either in the perioperative patient, or in the patient who has

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**Table 1** Echocardiography in the critically ill patient.

<table>
<thead>
<tr>
<th>Haemodynamic information</th>
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<tbody>
<tr>
<td>Hypotension</td>
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<tr>
<td>Assess volume status</td>
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<tr>
<td>Left ventricular (LV) systolic function</td>
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<tr>
<td>Regional wall motion abnormality</td>
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<tr>
<td>Global dysfunction</td>
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<td>Transient dysfunction</td>
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<td>Left ventricular diastolic function</td>
<td></td>
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<tr>
<td>Right ventricular function</td>
<td></td>
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<tr>
<td>Outflow tract obstruction</td>
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<tr>
<td>Valvular stenosis/regurgitation</td>
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<tr>
<td>Pericardial effusion/tamponade</td>
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<tr>
<td>Hypoxia</td>
<td></td>
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<tr>
<td>Right ventricular function</td>
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<tr>
<td>Right ventricular pressure</td>
<td></td>
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<tr>
<td>Intracardiac shunts</td>
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<tr>
<td>Pulmonary embolus</td>
<td></td>
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<tr>
<td>Excluding infection</td>
<td></td>
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<tr>
<td>Infective endocarditis</td>
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**Box 1** Calculating ejection fraction and FS from LV dimensions obtained from echocardiography.

\[
\text{Ejection fraction} = \frac{(\text{End-diastolic dimension})^3 - (\text{End-systolic dimension})^3}{(\text{End-diastolic dimension})^3} ,
\]

\[
\text{Fractional shortening} = \frac{\text{End-diastolic dimension} - \text{End-systolic dimension}}{\text{End-diastolic dimension}}.
\]
acutely deteriorated haemodynamically. This involves dividing the left ventricle into 16 segments (see Fig. 1) each of which is then graded according to their movement, as follows:9

1 = normokinesia,
2 = hypokinesia,
3 = akinesia,
4 = dyskinesia,
5 = aneurysmal.

From this, a wall motion score index (normal index is 1) can be calculated as follows:

Wall motion score index = \( \frac{\text{Sum of all segment score}}{\text{Number of segments scored}} \).

**Box 2** Estimating stroke volume and cardiac output by measuring the cross-sectional area of the LV outflow tract and the transaortic flow velocity time integral.

Cross-sectional area (CSA) = \( \pi r^2 = \pi (D/2)^2 \)

CSA = \( D^2 \times 0.785 \) (D is the measured LVOT diameter in cm)

LV stroke volume = CSA \times VTI

Cardiac output (CO) = Stroke volume \times Heart rate

**LV diastolic function**

LV diastolic dysfunction is commonly seen in patients with hypertension, coronary artery disease, cardiomyopathy and many forms of valvular heart disease, and is a potential cause of pulmonary edema in patients with documented normal LV systolic function. Although there are number of ways for evaluating LV diastolic function, the commonly used methods in routine practice include Doppler assessment of mitral inflow, the pulmonary venous flow pattern and tissue Doppler imaging of the mitral annulus, using the apical 4-chamber view, with classical patterns portraying varying degree of LV diastolic dysfunction (see Fig. 2). Note that the pseudonormal mitral inflow trace can be distinguished from a normal mitral inflow trace, by examining the pulmonary venous flow, which should display atrial reversal.

**Assessment of right ventricular (RV) function**

RV dysfunction is common in critically ill patients and its pathological role is underestimated in these patients. RV dysfunction can result from pressure or volume overload of RV. The two common causes for acute RV dysfunction are massive pulmonary embolus (PE) and acute respiratory distress syndrome (ARDS).10 Adequate assessment of RV function is needed in this condition, as the findings may
Altered therapy and is of prognostic value. Acute RV dysfunction may also be due to acute RV infarction, acute sickle cell crisis and sepsis.

Normal RV size is approximately two-thirds that of the left ventricle, and so RV dilatation should be easy to gauge. The qualitative assessment of RV systolic function can be done by visualizing the RV in multiple views (parasternal long axis, RV inflow tract view, apical 4-chamber view and subcostal). Abnormal RV wall motion occurs in inferior myocardial infarction and pulmonary hypertension. Interventricular septal movement can be used to assess RV dysfunction and differentiate volume overload from pressure overload of the RV. Septal flattening is common in RV dysfunction and if the septal distortion is only visualized during diastole, it is most likely due to volume overload, whereas in pressure overload, the septal flattening is usually present in both systole and diastole. Quantitative assessment of RV dysfunction such as RV wall thickness, RV fractional area change and long axis function by tissue Doppler imaging and myocardial performance index (MPI) are difficult in the critical care setting.

![Diagram of LV diastolic dysfunction using echocardiography by analyzing the mitral valve inflow pattern and pulmonary venous flow](image)

**Figure 2** Assessing for LV diastolic dysfunction using echocardiography by analyzing the mitral valve inflow pattern and pulmonary venous flow.

It is possible to estimate the RV systolic pressure (RVSP) from the addition of right atrial pressure (RAP) to pulmonary artery systolic pressure (PASP). PASP can be quantified from the peak velocity of the tricuspid regurgitation (TR) jet velocity, in the apical 4-chamber (using the Bernoulli equation). RAP can be estimated from the diameter of the inferior vena cava (IVC), visualized in the subcostal view, and the degree to which it collapses with inspiration (Box 3).

**Pulmonary embolus**

TTE can be used to detect acute RV dilatation and dysfunction in massive PE. One study reported that patients presenting with acute PE displayed a distinct regional pattern of RV dysfunction with akinesia of the mid-free wall but with normal motion at the apex. This finding of regional RV dysfunction was found to have a sensitivity of 77% and a specificity of 94% for the diagnosis of acute PE. It may also be possible to visualize the thrombus directly in the main pulmonary artery,
although TOE is more useful in visualizing the thrombi lodged in main and right pulmonary artery (see Table 2).

**Box 3** Estimating the right ventricular systolic pressure from the pulmonary artery systolic pressure (PASP) and right atrial pressure (RAP). The PASP is estimated from the peak velocity of the tricuspid regurgitant (TR) jet. The RAP is estimated from the diameter of the inferior vena cava (IVC), and its collapse in inspiration.

\[ \text{RVSP} = \frac{\text{PASP} + \text{RAP}}{2} \]

<table>
<thead>
<tr>
<th>RAP (mmHg)</th>
<th>IVC diameter (cm)</th>
<th>Collapse on inspiration (%)</th>
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<tbody>
<tr>
<td>&lt;5–10</td>
<td>&lt;2</td>
<td>100</td>
</tr>
<tr>
<td>5–10–15</td>
<td>&lt;2</td>
<td>&gt;50</td>
</tr>
<tr>
<td>10–20</td>
<td>&gt;2</td>
<td>25–50</td>
</tr>
<tr>
<td>15–20</td>
<td>&gt;2</td>
<td>&lt;25</td>
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**Table 2** Echocardiographic signs used in the diagnosis of acute pulmonary thromboembolism.

1. Direct visualization of thrombus in the right sided chambers or the pulmonary artery
2. Right ventricular dilatation
3. Reduced right ventricular function
4. Reduced left ventricular cavity size
5. Dilated pulmonary arteries
6. Abnormal septal motion/systolic flattening of the septum
7. Significant (moderate to severe) TR
8. Increased velocity of TR jet
9. Dilatation of IVC

Pericardial effusion and cardiac tamponade

Pericardial effusions are very common in ICU patients particularly after cardiac surgery on the cardiothoracic ICU. The parasternal long and short axis view, apical view and subcostal view will usually reveal the effusion. In many critically ill patients, the subcostal view will often be the only adequate window to detect pericardial effusion. It should be emphasized that cardiac tamponade is a clinical diagnosis and echocardiography may suggest a hemodynamic abnormality that may be the substrate for tamponade, but this alone will not establish the diagnosis of tamponade. The signs of tamponade are directly due to actual elevation of intrapericardial pressure. The RV free wall collapse is seen in early diastole in parasternal view and right atrial wall collapse is seen in late diastole in the apical view.

Doppler evaluation of mitral and tricuspid inflow can also be used to assess tamponade. In normal circumstances, the peak velocity of mitral inflow varies by no more than 15% with respiration and tricuspid inflow by no more than 25%. In cardiac tamponade, respiratory variation in filling is exaggerated above these thresholds. The earliest feature of tamponade to be noted is this exaggerated respiratory variation of tricuspid inflow. Subsequent to this, exaggeration in mitral inflow patterns can be noted. This is followed by right atrial collapse and RV collapse is noted at a later stage. Superior vena caval and hepatic vein flows can also reflect the elevated intrapericardial pressure. Normally, venal caval flow is continuous and occurs in both systole and diastole. In the presence of elevated intrapericardial pressure, flow during diastole is truncated and majority of flow into the heart occurs during ventricular systole.

In some instances these changes may not be seen, the commonest example being in the patient with significant RV hypertrophy, usually due to pulmonary hypertension. The thick non-compliant RV wall is not compressed by the relatively modest elevation in pericardial pressure seen in early diastole and both clinical and echocardiographic signs of compromise may be minimal or absent. Ventricular wall thickening due to malignant infiltration, an overlying inflammatory response or an overlying thrombus in hemorrhagic pericarditis may also have the same effect.

Assessment of valvular function

Significant valvular abnormalities can be present in the critically ill patient without being recognized. Precise evaluation of valvular pathologies needs to be identified in ICU. The common indications for bedside echocardiography in this group of patients are mitral and aortic valve disease, excluding native infective endocarditis (IE) and for excluding prosthetic valve endocarditis.

In the critical care setting, TTE can provide valuable information concerning valvular integrity...
and function, but it may be suboptimal and not sensitive for detecting vegetations of endocarditis, or in the evaluation of a dysfunctional mitral valve and the assessment of most prosthetic valves. In this regard, TEE is superior for assessing valvular function. In some cases, TTE may provide better imaging than TEE for evaluation of the anterior structures such as the aortic valve and also for doing Doppler measurements.

Aortic stenosis

The aortic valve is first visualized in the parasternal view for thickening and shortening in case of aortic stenosis. All three leaflets can be visualized in short axis view and their mobility assessed. Doppler assessment of flow through aortic valve can be obtained in apical view and peak and mean velocity measured across the valve with CW Doppler. The peak gradient across the valve can be derived from the equation below and thus the severity of aortic stenosis can be assessed as follows:

\[
\text{Peak pressure gradient (mmHg)} = 4(\text{peak velocity})^2.
\]

The aortic valve area can be assessed using the continuity equation, by measuring the LVOT area (as described before) and obtaining the mean velocity of flow through LVOT with PW Doppler, and aortic mean velocity by CW Doppler. The aortic valve area can be calculated by the equation below. In patients with impaired LV systolic function, it is imperative to calculate the aortic valve area when assessing the severity of aortic stenosis, as the peak pressure gradient across the stenotic aortic valve may well be reduced.

\[
\text{Aortic valve area (cm}^2) = \frac{\text{LVOT CSA \times LVOT VTI}}{\text{Aortic VTI}}.
\]

Aortic regurgitation

Aortic regurgitation is detected and quantified by both color flow and CW Doppler. The regurgitant jet can be detected by color flow in the parasternal long axis view as a blue color coded jet. On the apical view, the direction of aortic regurgitation appears red, as the flow is towards the transducer. AR can be quantified by four main methods:

1. Color flow Doppler,
2. CW spectral Doppler trace of the regurgitant jet,
3. PW spectral Doppler trace of flow in descending aorta,
4. LV volume overload.

Mitral valve

Assessment of the mitral valve by transthoracic echo in the ICU setting is difficult and may underestimate the severity of mitral valve disease. TEE provides better imaging for the assessment of the mitral valve in ICU patients, because of its close anatomic proximity to the esophagus. Mitral valve leaflet opening and prolapse can be noted in a parasternal long axis view and in short axis view. M-mode across the mitral valve leaflet can show the opening of the valve clearly and can be used to diagnose mitral stenosis. Mitral valve area (MVA) can be assessed commonly by two methods:

1. Valve planimetry can be done in a short axis view, by tracing around the inside edges of the MV leaflets when they are open to the maximum.
2. It can also be assessed by measuring the pressure half time (PHT) on the E wave of the LV inflow jet, with PW Doppler along the inflow. MVA (cm²) = \(220/\text{PHT}\). Measurement of LA size will give additional information in mitral valve disease.

Assessment of MR by color flow doppler may not be accurate and lead to underestimation of MR severity, especially with the hand-held machines. TEE will provide a much more precise evaluation of the degree of MR and often provides information as to the etiology of MR. The diagnosis of acute severe MR is a surgical emergency, and so the threshold to perform a TEE if this is suspected should be low.

Infective endocarditis

IE is not uncommon in the ICU, either as a presenting diagnosis or a complication of concurrent sepsis. IE has been reported to be the second most common indication for performance of echocardiography in ICU. As the consequence of endocarditis may be potentially devastating and possibly fatal, it is important to diagnose IE early and treat appropriately. The echocardiographic features typical of IE are an oscillating intracardiac mass on a valve or supporting structure or an iatrogenic device, intracardiac abscesses, new partial dehiscence of a prosthetic valve or new
valvular regurgitation. There is no single character on the echocardiogram that will conclusively identify a mass as vegetation. The ability to detect a vegetation depends on several factors including vegetation size, location, the presence of underlying heart disease, image quality and instrument settings. Different echo window should be used, and Doppler flow mapping should be performed to identify any associated valvular regurgitation. Vegetations must be at least 3–6 mm in size to be reliably seen by TTE, even though masses of 2 mm have been reported.

The sensitivity of TTE for diagnosing endocarditis has been reported to be 58–62%. The sensitivity of TEE is superior to TTE and should be used in cases where clinical suspicion persists despite TTE being negative.

Intracardiac shunt

Intracardiac or intrapulmonary shunts should be suspected in cases of unexplained embolic stroke or refractory hypoxemia. Common causes of right to left shunts are atrial septal defects (ASD) and a patent foramen ovale (PFO). A PFO is present in 25–30% of normal population. In cases where RA pressure is greater than LA pressure such as ARDS, pulmonary embolism and RV infarction and severe TR, there will be increased right to left shunting through the defect, causing significant hypoxemia. Ventricular septal defects can also complicate patients presenting with an acute myocardial infarction and leads to haemodynamic instability.

Color flow imaging can usually detect the larger shunts, but contrast 'bubble' study is often needed to detect smaller ones. A contrast study should always be performed when evaluating patients with unexplained embolic stroke or refractory hypoxemia. Approximately 0.5 mL air is mixed with 10 mL normal saline solution, which is then agitated back and forth between two syringes connected by a three-way stopcock and injected into the venous circulation, immediately following the release phase of a Valsalva manoeuvre. In the absence of a shunt, only minimal amount of contrast should be expected to be seen in left sided cavities. If an intracardiac shunt is present, contrast in the left sides of the heart will be observed immediately after right side opacification and the contrast will be seen going through the interatrial septum.

The application of echocardiography on the ICU

The value of immediate bedside echocardiography for aiding in the diagnosis of acute haemodynamic disturbances has been well demonstrated in both the ICU and the emergency department. With the introduction of new hand-held echocardiographic machines, they can be used as an adjunct to the physical examination. This should be directed to answer a specific clinical question and should be shorter than the traditional echocardiogram, that is the echocardiographic study is a point-of-care assessment. However, one should only view this as an extension of the physical examination and it should not be compared with the quality of high-end machines that can be undertaken in the department. A recent study by Gorcsan et al. investigated the utility of hand-held echo when specifically used as an extension of the physical examination. The hand-held echo demonstrated an excellent close overall agreement (92–100%, r = 0.91–0.96) for estimation of EF, LV hypertrophy, RWMA and pericardial effusion when compared to an echocardiographic study using a full-size echocardiographic system. The authors of this study concluded that use of 'goal-directed hand-held echocardiography has the potential to influence bedside patient treatment decisions and expedite healthcare'.

In another study, it was reported that the accuracy of the hand-held machines with respect to two-dimensional imaging remains very good in the critically ill patient when compared to standard echocardiography machine, but that information provided from hand-held color Doppler imaging should be interpreted cautiously in this patient population.

In most hospitals, particularly district general hospitals, the provision of 24 h echocardiography by either a cardiologist or sonographer is not feasible. In this regard, studies have reported the successful undertaking of bedside echocardiography by suitably trained intensivists. However, adequate training is essential and this must be individualized to the specific needs and application of the user. There is no specific accreditation available from the American or British society of echocardiography in this scenario, but there are number of courses available to equip the intensivist in performing bedside echocardiography on the ICU.

Conclusion

TTE has been accepted as one of the safest imaging modalities to assess patients in ICU. This investigation provides a powerful adjunctive tool to the physical examination and can be used to guide in the management of patients in the critical care setting. It can also be used to exclude underlying
cardiac abnormalities in patients admitted to the ICU with a non-cardiac condition. Certain complications of procedures and therapy given in ICU can also be assessed by bedside TTE.

With hand-held echo machines readily available, bedside echocardiography will become an indispensible tool in the management of patients on the ICU. There may be a graduated progression of the use of hand-held device from cardiologist to intensivist in future. However, there should be adequate training and this should be individualized to the specific needs of the operator. In addition, the limitations of bedside echocardiography should be appreciated and the findings from a study interpreted in the context of other investigative findings. The successful use and dissemination of hand-held machine will not rest simply on the size of the instrument, but on the individual user and their understanding of and response to the information imparted.

References