REVIEW ARTICLE

Transesophageal Echocardiography in the Intensive Care Unit

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ABSTRACT

Management of shock in the intensive care unit involves advanced hemodynamic monitoring. Invasive monitoring with central venous lines and pulmonary artery catheters may be inadequate in guiding therapy and improving outcomes. Echocardiography is a reasonably-safe, minimally-invasive diagnostic technique that provides rapid bedside evaluation of ventricular filling and function. While transthoracic echocardiography is the method of choice initially, images can be suboptimal in up to a third of intensive care patients. Transesophageal echocardiography is then required to better evaluate the cause of hemodynamic instability. In addition, transesophageal echocardiography can be used to diagnose other causes of hemodynamic failure (for e.g., pericardial tamponade, pulmonary embolism and left ventricular outflow tract obstruction) and to diagnose intracardiac shunt. Echocardiography is also vital in diagnosing the cause and guiding management in patients with cardiac arrest. Specific training is required for physicians in order to achieve competence in probe insertion, completion of a comprehensive examination and interpretation of the images. In this article, we provide an overview of the indications and complications of the technique and training pathways for the intensivist, followed by transesophageal echocardiography-guided hemodynamic assessment and diagnosis of specific cardiac disorders commonly encountered in the intensive care unit.

Keywords: Transesophageal echocardiography, Hypotension, Intensive care unit, Preload, Contractility, Pericardial tamponade, Left ventricular outflow tract obstruction, Aortic dissection, Blunt chest trauma.

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INTRODUCTION

Early and aggressive management of tissue hypoperfusion is vital in critically ill patients. Hypoperfusion can manifest as hypotension, tachycardia and cold extremities, and as evidence of end-organ ischemia: altered mental state, oliguria, tachypnea, ST-segment depression on electrocardiogram, elevated lactate or base deficit and decreased venous oxygen saturation.

Management of shock depends on its etiology. Although hypovolemic, cardiogenic and distributive shock are commonly treated by fluid resuscitation, inotropes and vasopressors respectively, most critically ill patients suffer from a combination of different etiological and patho-physiological processes simultaneously. Hemodynamic monitoring is therefore helpful in tailoring therapy to the patient's individual hemodynamic state. Central venous pressure and pulmonary capillary wedge pressure have been shown to be inadequate in predicting fluid-responsiveness in critically ill patients, and therapy based on these parameters has not been shown to improve outcomes.[1] While respiratory variation in cardiac output measured by systolic pressure variation and pulse pressure variation is highly predictive of the fluid-responsiveness, the utility of these parameters is limited to mechanically ventilated patients in sinus rhythm.

Transesophageal echocardiography (TEE) is a minimally-invasive diagnostic technique that provides rapid bedside evaluation of ventricular filling and function. In addition, echocardiography can be used to diagnose infective endocarditis, pericardial tamponade, pulmonary embolism, left ventricular outflow tract obstruction, aortic dissection and myocardial contusion, all of which can contribute to hemodynamic failure. TEE can also be used to evaluate the presence and impact of intracardiac shunt in patients with unexplained hypoxemia.

When utilized, echocardiography has a significant impact on the management of up to 50% of patients in the intensive care unit.[2] For example, the finding of ventricular dysfunction in patients with septic shock often alters the combination of vasoactive agents used in therapy. TEE can also be used to establish the diagnosis and guide specific management in patients with cardiac arrest.[3]

INDICATIONS, CONTRAINDICATIONS AND COMPLICATIONS

Initial bedside cardiac evaluation of critically ill patients is best performed using transthoracic echocardiography (TTE). However, TTE images can be suboptimal in up to a third of these patients because of difficulty with positioning, mechanical ventilation, chronic obstructive pulmonary disease, obesity, surgical drains and dressings or subcutaneous emphysema in trauma patients. TEE is then required to better evaluate the cause of hemodynamic instability. TEE is also better suited for the evaluation of valvular pathology and cardiac source of emboli and for imaging of the thoracic aorta. Various organizations have published guidelines for the use of TEE.[4,5] Based on these guidelines, TEE should be used when diagnostic information that is expected to alter management cannot be obtained by TTE or other modalities in a timely manner (Table 1).

Table 1: Indications for TEE in the ICU

Acute hemodynamic instability with inadequate TTE images. Assessment for structures and pathologies best evaluated by TEE:
• Aortic dissection
• Endocarditis
• Prosthetic valve structure and function
• Intracardiac shunt
• Intracardiac thrombus (e.g. in the left atrial appendage)

Absolute contraindications to TEE are as follows: known or suspected presence of esophageal stricture or mass, esophageal laceration or perforation, esophageal diverticulum and an unstable cervical spine. Relative contraindications include esophageal varices, large diaphragmatic hernia and mediastinal irradiation.

TEE is a fairly safe procedure,[6] with a minor complication rate of 0.2% (1 in 500 patients). Most complications are related to probe insertion: oropharyngeal, esophageal or gastric trauma, odynophagia, dental trauma and endotracheal tube malpositioning. Major complications (e.g. esophageal perforation) have been reported in one per 2,300 to 7,200 patients.

Training

Specific TEE training is required for physicians in order to achieve competence in probe insertion, completion of a comprehensive examination and interpretation of the images. TEE training for intensivists with no previous training in echocardiography has been investigated.[7,8] Intensivists were able to complete a basic TEE examination in about 15 minutes and their interpretations of ventricular volume and function correlated with experts 90% of the time after didactic sessions and an average of 30 supervised examinations. These data suggest that reliable hemodynamic diagnosis in intensive care is possible without sophisticated echocardiographic techniques and cardiology expertize. The WINFOCUS group[9] outlined the specific requirements of echocardiography training for intensivists, and conducts workshops around the world. Of their three proposed levels of expertise, intensivists should achieve a basic level of expertise (level 1) to diagnose the most common and important causes of hemodynamic instability in critically ill patients.

While the usual goal for TEE exams in the ICU is to address the cause of acute hemodynamic instability, a comprehensive evaluation should be performed on every patient[10] to avoid missing unexpected findings. Various approaches to examination have been proposed.[11] Like all diagnostic modalities, the benefits of a TEE exam depend on clinical suspicion, practitioner expertize and continued monitoring. The newly available ClariTEE probe (ImaCor, Uniondale, NY, USA), which is 5.5 mm in diameter and has been approved to remain in patients for up to 72 hours, allows continuous monitoring in critically ill patients.[12] However, this probe only provides monoplane scanning, making a complete assessment difficult.

TEE evaluation in critically ill patients should include assessment of ventricular filling and function, along with investigation for specific pathologies, such as significant valvular abnormalities, LV outflow tract obstruction and pericardial tamponade.

Preload

Preload assessment can be done through echocardiography by various methods. The transgastric short axis view is useful in estimating left ventricular dimensions in different stages of the cardiac cycle. Left ventricular end-diastolic area (LVEDA) correlates well with left ventricular end-diastolic volume (LVEDV). A single-snapshot assessment of static preload parameters like LVEDA and LVEDV are susceptible to the same pitfalls as CVP and PCWP. However, the trends in these parameters, particularly in response to a fluid challenge, would be far more reliable. Patients with LV failure are likely to have supra-normal or high-normal LVEDA at baseline, hence 'normal' parameters would be unreliable in decision-making for hypovolemia. Serial measurements analyzed overtime and in correlation with the clinical picture allow for better understanding of optimal LVEDA for individual patients. Area measurements can be performed subjectively, manually, or with the aid of automated software. End-systolic obliteration of the LV cavity (Fig. 1) is a highly sensitive, but not specific, marker of severe hypovolemia.

IVC size and collapsibility with respiration[13] can be used to predict right atrial pressure and fluid responsiveness in spontaneously breathing patients. A collapsed IVC predicts hypovolemia, while a distended IVC that does not vary with respiration predicts a patient will not be fluid responsive. In mechanically ventilated patients, a small vena cava reliably excludes the presence of elevated right atrial pressures, while IVC dilation is much more common and difficult to interpret.

Doppler evaluation of the ratio of peak early transmitral flow (E wave) to peak mitral annular tissue velocity during passive ventricular filling (E' wave) can be used to predict LV filling pressure (Figs 2 and 3). An E/E' < 8 predicts normal filling pressure, while E/E' >15 predicts elevated LV filling pressures.

Systolic Ventricular Function

Critically ill patients can develop poor systolic function because of myocardial ischemia or infarction, sepsis-induced myocardial depression or stress-induced cardiomyopathy,

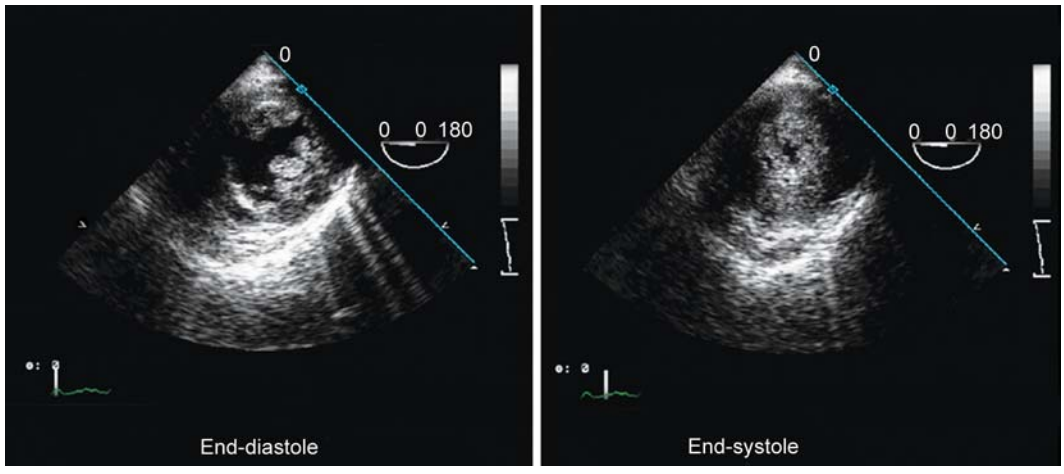


Fig. 1: Transgastric short axis view of the left ventricle at the level of the papillary muscles in end-diastole and in end-systole. Notice the end-systolic LV cavity obliteration (also called 'kissing papillary muscle sign'), which is a highly sensitive, but not specific, marker of severe hypovolemia. The small LVEDA does indicate hypovolemia

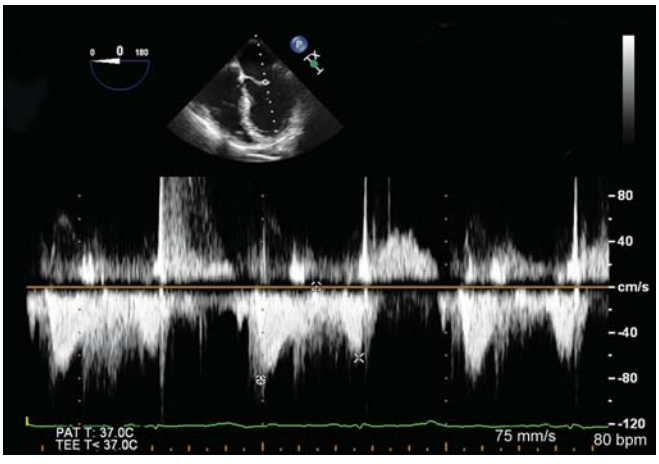


Fig. 2: Continuous wave Doppler of mitral inflow measured in the midesophageal four-chamber view, to estimate the peak velocity of early transmitral filling (E) and the contribution of the atrial contraction (A). The slope of decline of the E wave is also measured (deceleration time, or DT)

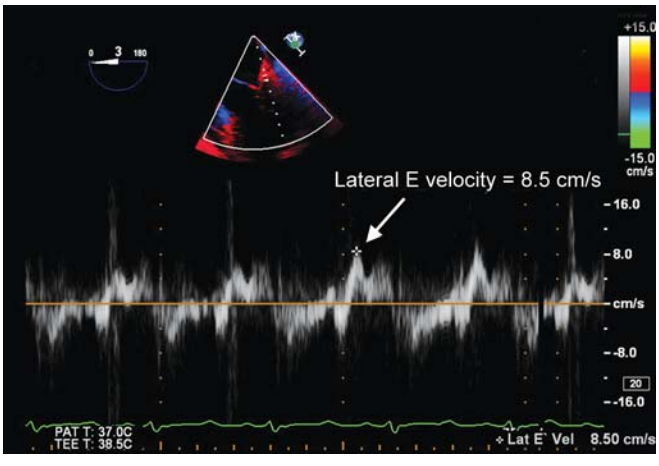


Fig. 3: TDI of the lateral mitral annulus, measuring the peak velocity (E') for early passive ventricular filling

or may present with pre-existing systolic dysfunction. Distinguishing between a depressed state of contractility and a hyperdynamic state is essential in tailoring hemodynamic therapy in the critically ill patient. TEE provides several methods to analyze ventricular contractility. While a single technique may provide some insight as to the overall systolic function of the ventricle, the use of multiple echocardiographic methods in concert with other clinical data affords a more robust estimation of true cardiac systolic function. The left and right ventricles (RV) are radically different in both structure and function and the echocardiographic assessment of each will be discussed separately.

Common methods for evaluation of left ventricular systolic function can be assessed by evaluating the ejection fraction (LVEF), and by measuring LV stroke volume, mitral annular plane excursion, myocardial performance index and by the characteristics of the mitral regurgitation jet.

Left Ventricular Ejection Fraction

LVEF is routinely used in clinical practice to estimate myocardial contractility. While LVEF of 55 to 75% is considered normal and may be encouraging, one cannot assume adequate forward flow and ultimately tissue perfusion based solely on a value in the normal range. Factors, such as mitral regurgitation, decreased preload, inotrope administration and decreased afterload can result in a normal LVEF in the absence of adequate systolic function. Despite its shortcomings, LVEF remains an important component in the evaluation of LV systolic function.

The most widely used 2D method for the estimation of LVEF is the biplane method of disks, also known as the modified Simpson's rule. This method relies on the attainment of two long axis views of the left ventricle—the midesophageal two- and four-chamber views. Computer software then constructs a stack of elliptical disks, the summation of which provides an estimate of LV volume.

Using the resulting end-diastolic volume (EDV) and end-systolic volume (ESV), an ejection fraction is calculated using the formula:[14]

Ejection fraction = (EDV – ESV)/EDV

Accuracy using the biplane method of disks depends on the acquisition of nonforeshortened views of the LV. Obtaining such views using TEE, though more technically challenging, is comparable to those obtained using TTE.[15]

With the introduction of three-dimensional (3D) echocardiography, plane-positioning errors including foreshortening can be avoided. Unlike two-dimensional (2D) techniques, such as the biplane method of disks, 3D echocardiography (Fig. 4) determines ventricular volumes without the need for geometric assumptions. This technique's accuracy, when compared to that of magnetic resonance imaging, has been well established.[14] Visual estimation of ejection fraction by an experienced echocardiographer is comparable to quantitative methods, including the biplane method of disks discussed above.[16]

The two- and one-dimensional surrogates for ejection fraction are fractional area change (FAC) and fractional shortening (FS). FAC is calculated by measuring the end-diastolic and end-systolic LV area on still 2D images of the transgastric short axis view (Fig. 5). Normal FAC is 55 to 65%. FS is calculated by measuring the end-diastolic and end-systolic LV diameter on M-mode imaging of the transgastric short axis view. Normal FS is 25 to 45%.

FAC = (LV end-diastolic area – LV end-systolic area)/LV end-diastolic area.

FS = (LV end-diastolic diameter – LV end-systolic diameter)/LV end-diastolic diameter.

Stroke Volume Measurement

Left ventricular function can also be assessed by echocardiographic measurement of stroke volume.

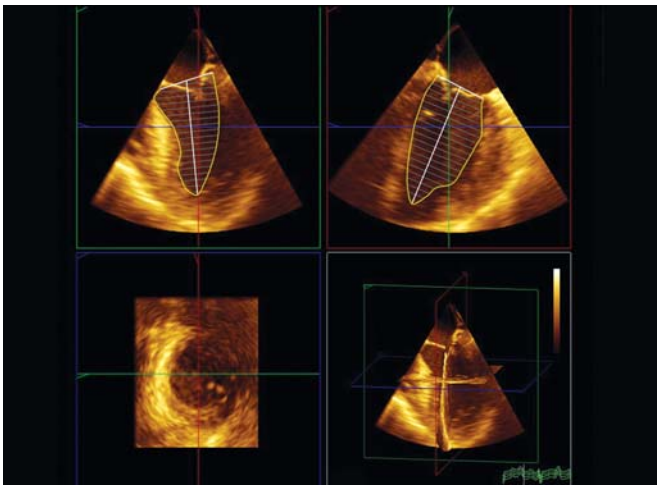


Fig. 4: LV ejection fraction measurement using 3D echocardiography. The LV is traced in the midesophageal four-chamber and mid-esophageal two-chamber views in end-diastole and in end-systole

Stroke volume = Cross-section area × Velocity time index

The cross-sectional diameter of the left ventricular outflow tract (LVOT) is measured in the midesophageal long axis view (Fig. 6A), and used for calculation of the cross-sectional area of the LVOT with the assumption that the LVOT is a circular structure. This measurement is critically important, because the LVOT diameter is squared to calculate area. However, it has been proven by 3D echocardiography, multidetector computer tomography and magnetic resonance imaging that the LVOT is in fact elliptical, and not circular.[17] The flow through the LVOT is measured using pulse-wave Doppler measurement in the deep transgastric long axis view or the transgastric long axis view (Fig. 6B). Accurate alignment with the flow in the LVOT is vital. Despite these limitations, the calculation of stroke volume can be a reliable tool if performed precisely. Flow and area measurements at the mitral annulus or at the pulmonic valve can also be used for stroke volume calculation.

Mitral Annular Plane Excursion

Contraction of longitudinally aligned fibers of the left ventricle results in mitral annular motion toward the apex. Referred to as mitral annular plane systolic excursion (MAPSE), the characteristic motion of the mitral annulus is measured using M-mode echocardiography directed across the mitral valve annulus in the four-chamber view (Fig. 7). A decrease of MAPSE measurements correlates with impaired systolic ventricular function. MAPSE has been shown to be an acceptable surrogate for assessment of ejection fraction and achieves results comparable with other methods of EF estimation, including cardiac magnetic resonance imaging and 3D echocardiography.[18]

MAPSE may be particularly well suited for the critical ill patient as the measurements are easily and rapidly obtained and can be derived from poor-quality images which may not be suitable for interpretation by other methods.

Similarly, tissue Doppler imaging (TDI) of the mitral annulus to measure peak systolic longitudinal velocity (Sa) done in the midesophageal four chamber or two chamber views can be used to estimate global LV systolic function (Fig. 8). Sa values > 7.5 cm/s are associated with normal global LV function while systolic velocities <5.5 cm/s indicate LV failure. Mitral annular calcification and prosthetic valves and rings can make estimation of mitral annular excursion and velocity difficult.

Myocardial Performance Index (Tei Index)

The ratio of total LV isovolumic time to ejection time has been proposed as a global measure of systolic and diastolic

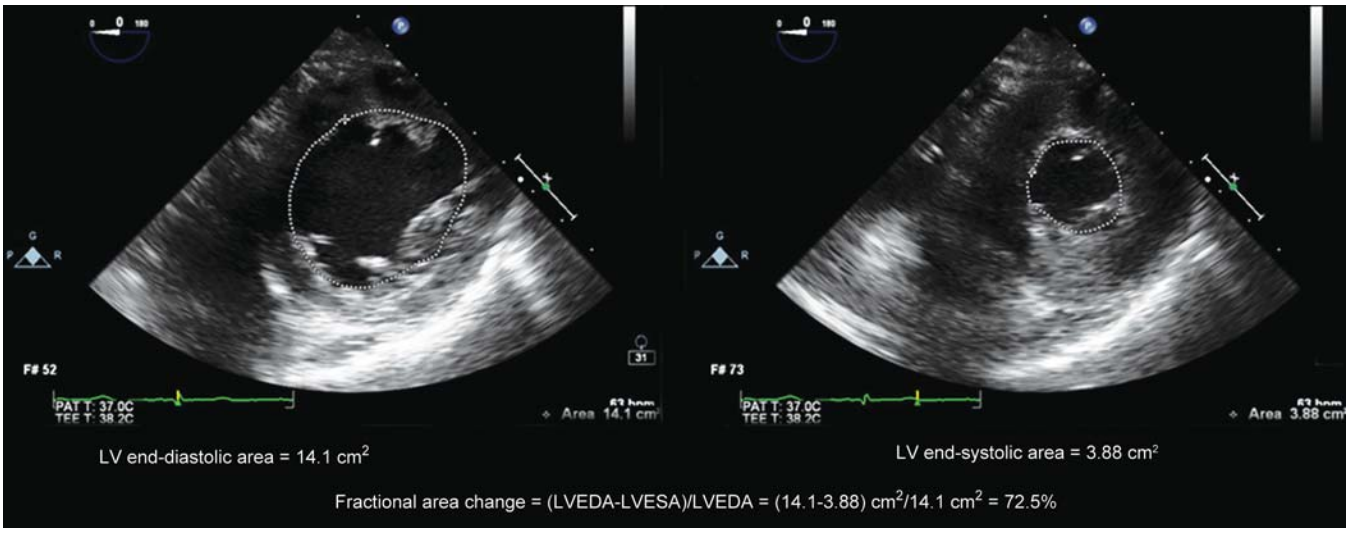
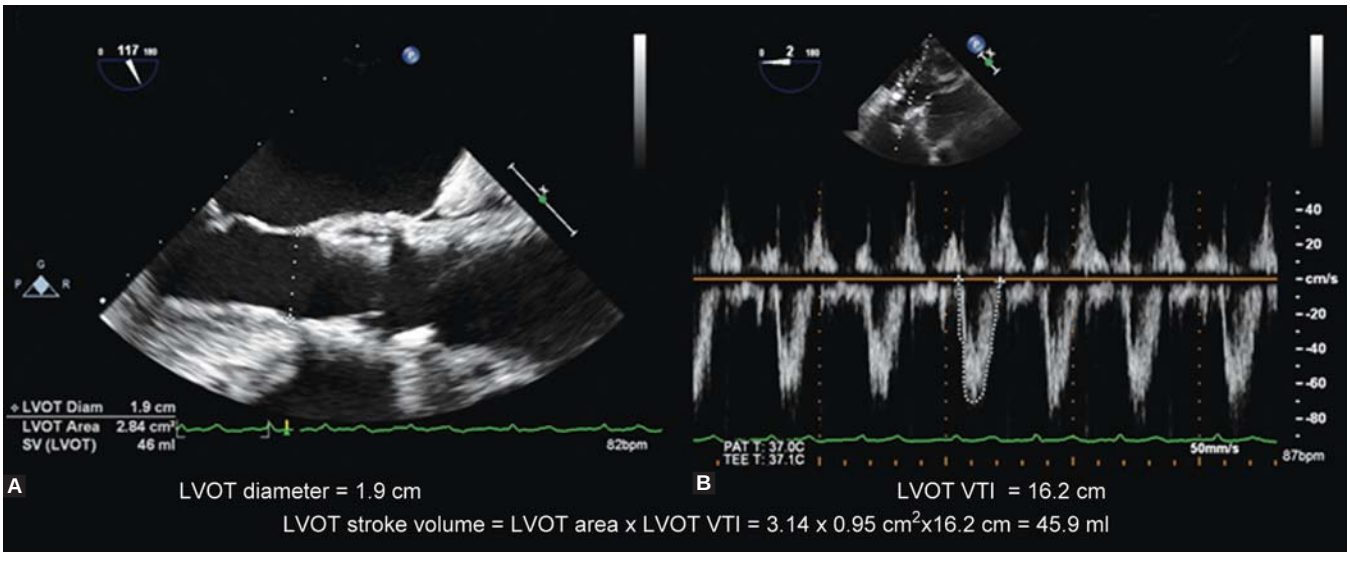


Fig. 5: Left ventricular systolic function measured by FAC of the left ventricular area on transgastric mid-short axis views of the left ventricle in end-diastole and end-systole in 2D



Figs 6A and B: Left ventricular stroke volume measurement by calculation of LVOT velocity time-integral (VTI) on deep transgastric long axis view and LVOT diameter on the midesophageal long axis view

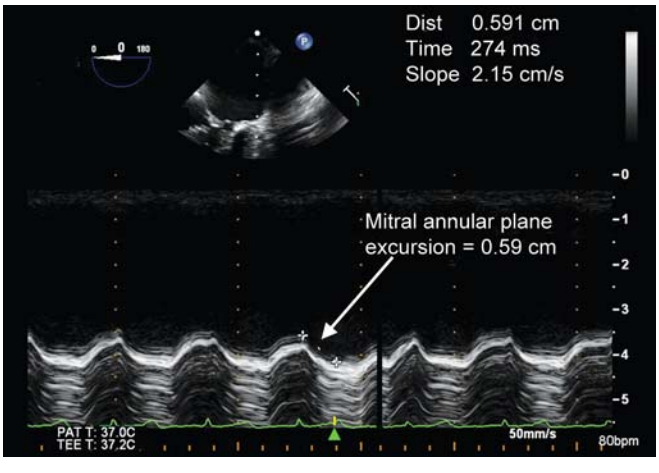


Fig. 7: Mitral annular plane systolic excursion measured on M-mode echocardiography of the lateral mitral annulus on midesophageal four-chamber view

function. The myocardial performance (or Tei) index (MPI) is calculated as the sum of the duration of the isovolumic relaxation (IVRT) and contraction (IVCT) times, divided by the systolic ejection period (SEP):

$$MPI = (IVRT + IVCT)/SEP$$

The normal MPI is about 0.4 with values >0.6, indicating ventricular dysfunction either due to systolic dysfunction (with a prolonged IVCT) and/or diastolic dysfunction (prolonged IVRT). Although the MPI is not affected by abnormal ventricular geometry or changes in heart rate, it is affected by preload and afterload. MPI can be calculated using time intervals measured from conventional Doppler or, preferably, from tissue Doppler recordings.

Mitral Regurgitation Jet Analysis

In the presence of mitral regurgitation, continuous wave Doppler interrogation of the regurgitant jet allows for estimation of LV systolic function. A rapid rise of velocity

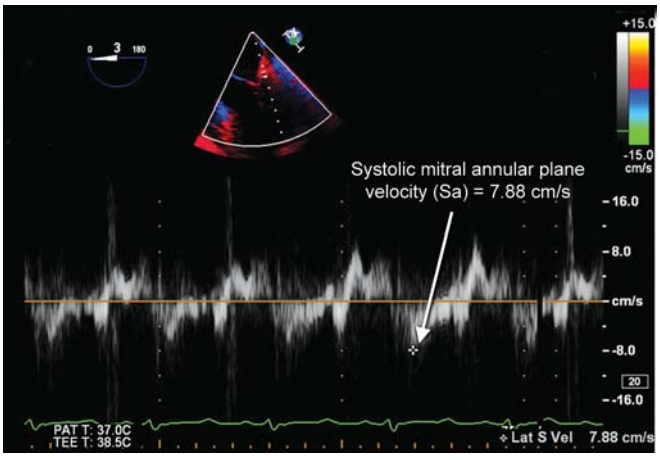


Fig. 8: TDI of the lateral mitral annulus, measuring the peak velocity (Sa) for the systolic phase of the cardiac cycle

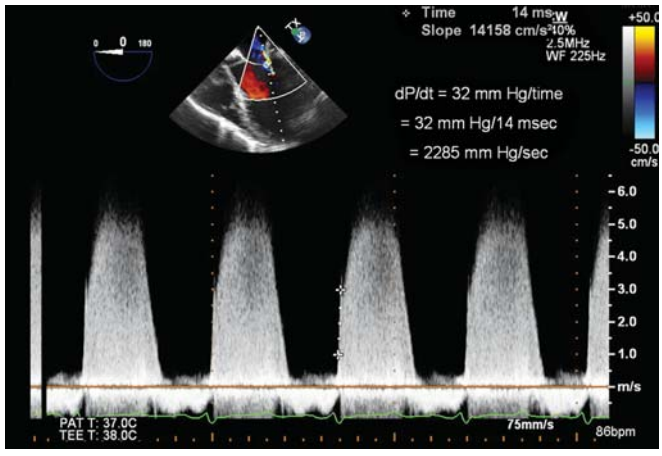


Fig. 9: Measurement of the rate of rise of left ventricular pressure by continuous wave Doppler estimation of dP/dt on the mitral regurgitation jet

from 1 to 3 m/s indicates normal systolic function, while reduced LV systolic function results in a slower rate of increase in LV systolic pressure and hence a slower rate of rise for the velocity of mitral regurgitant jet. The mitral regurgitant velocity is converted to the corresponding pressure gradient per the Bernoulli equation ($P = 4v^2$), and the rate of rise of ventricular pressure (dP/dt) is calculated as follows:

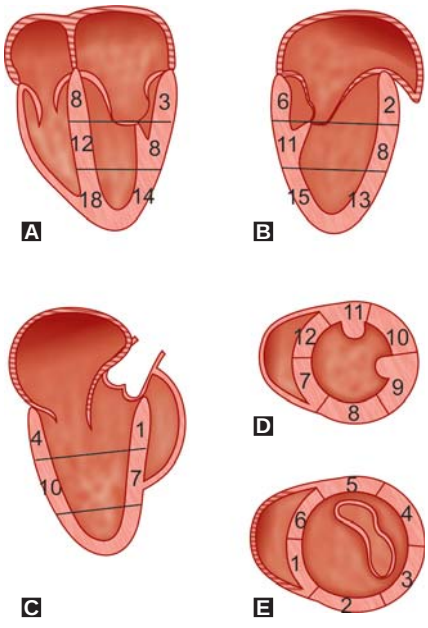
$$dP/dt = [4(3)^2 - 4(1)^2]/\text{time interval}$$
$$= 32 \text{ mm Hg/time interval}$$

The time interval measured is the time between the mitral regurgitant jet velocity at 1 and at 3 m/s (Fig. 9). Values of <1,000 mm Hg/sec for dP/dt and >32 msec for the time interval reflect decreased LV contractility.

Regional Left Ventricular Function

A method commonly used in TEE is a 16-segment model which divides the LV into 6 basal, 6 midcavity, and 4 apical segments[10] (Figs 10A to E). In an effort to standardized segmentation across imaging modalities, an alternative 17-segment model which assigns the apical cap as the 17th segment has been advocated;[19] however, for evaluation of regional wall motion the 16-segment method is considered appropriate as the normal apical cap does not move. Five echocardiographic views are necessary to adequately assess regional wall motion. These include three views from the midesophageal position—the four-chamber, two-chamber and long axis views; and two views from the transgastric position—the mid and basal short axis views.[10]

Despite variability among individuals, myocardial segments can be assigned to one of the three main coronary artery territories using the 17-segment model.[19] In general, the anterior and septal walls are perfused by the left anterior descending artery, the inferior wall by the right coronary artery, and the posterior and lateral walls by the circumflex artery.



Basal segments	Mid segments	Apical segments
1 = Basal anteroseptal	7 = Mid anteroseptal	13 = Apical anterior
2 = Basal anterior	8 = Mid anterior	14 = Apical lateral
3 = Basal lateral	9 = Mid lateral	15 = Apical inferior
4 = Basal posterior	10 = Mid posterior	16 = Apical septal
5 = Basal inferior	11 = Mid inferior	
6 = Basal septal	12 = Mid septal	

Figs 10A to E: 16-segment model of the left ventricle. 16-segment model of the left ventricle. (A) Four-chamber views show the three septal and three lateral segments. (B) Two-chamber views show the three anterior and three inferior segments. (C) Long axis views show the two anteroseptal and two posterior segments. (D) Mid short axis views show all six segments at the mid level. (E) Basal short axis views show all six segments at the basal level[10]

Characteristics of normal systolic myocardial function included both motion toward the center of the ventricular cavity and thickening of the ventricular wall. Wall thickening abnormalities may more accurately represent areas of dysfunction caused by ischemia, whereas regional wall motion may overestimate the extent of regional myocardial dysfunction. Shanewise et al recommend a

5-point grading scale based on qualitative visual assessment to describe the systolic function of myocardial segments as follows: 1 = normal (>30% thickening), 2 = mildly hypokinetic (10-30% thickening), 3 = severely hypokinetic (<10% thickening), 4 = akinetic (does not thicken), 5 = dyskinetic (moves paradoxically during systole).[10]

Emerging Modalities

2D speckle-tracking echocardiography (2D STE, Fig. 11) is able to evaluate characteristic of myocardial function essentially independent of the angle of incidence from the ultrasound beam. From the noise seen in grayscale B-mode images, 2D STE filters the meaningful speckles associated specifically with myocardial motion to measure parameters of myocardial mechanics. 3D STE achieves similar goals without the limitations inherent with 2D modalities, such as foreshortening and with the ability to track speckle motion regardless of direction within the scanning volume. While some emerging modalities demonstrate promise, further refinement and validation is necessary prior to their adoption into routine clinical practice.[20]

Right Ventricular Systolic Function

As a result of the crescent-shaped morphology of the RV and the lack of standard classification methods, RV systolic function is typically estimated based on a qualitative assessment. As RV enlargement typically coincides with dysfunction, estimation of RV size obtained from the midesophageal 4-chamber view should be part of every evaluation of the RV. Enlargement of the RV is supported by a midventricular diameter equal to or greater than that of the LV. Another indication of enlargement includes the relationship between the apical termination of the RV and the true apex of the heart (Fig. 12). In the 4-chamber view of a normal heart, the true apex is formed exclusively by the LV. An enlarged RV may extend to the apex of the heart, whereas a severely enlarged RV may extend beyond the LV, essentially becoming the new apex of the heart.[14] Unlike the predominately circumferential configuration of LV lateral wall fibers, the fibers of the RV free wall are longitudinally and obliquely arranged.[21] As a result, displacement of the tricuspid annulus toward the apex is a distinctive feature of RV motion visualized in the 4-chamber view and an important indication of RV systolic function. Traditionally, tricuspid annular plane systolic excursion (TAPSE), measured using M-mode across the tricuspid annulus in the 4-chamber view, has been employed to translate this distinct motion into a meaningful representation of RV systolic function. Due to M-mode beam misalignment, the calculation of RV longitudinal shortening fraction using speckle tracking echocardiography may more accurately represent RV systolic function.[22]

Diastolic Function

Symptomatic heart failure can occur in patients with impaired ventricular relaxation, despite adequate systolic function. Diastolic heart failure should be suspected when ventricular filling pressures are elevated. Myocardial ischemia and left ventricular hypertrophy because of hypertension or aortic stenosis can lead to diastolic dysfunction in critically ill patients.

Echocardiographic evaluation of diastolic function can be done by Doppler evaluation of transmitral flow and pulmonary venous flow, tissue Doppler evaluation of mitral annular velocity and color M-mode evaluation of flow propagation through the LV during diastole (Figs 2 and 3). Patients are graded to have normal diastolic function, impaired relaxation, pseudonormal pattern or restrictive filling patterns.[23]

In the presence of impaired diastolic relaxation, elevated filling pressures are often required to maintain adequate ventricular filling. At the same time, these patients have a limited ability to handle hypervolemia. Therefore, closer

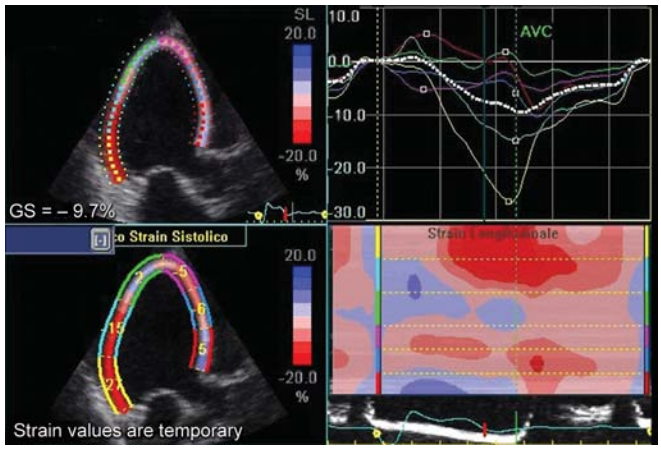


Fig. 11: Speckle tracking

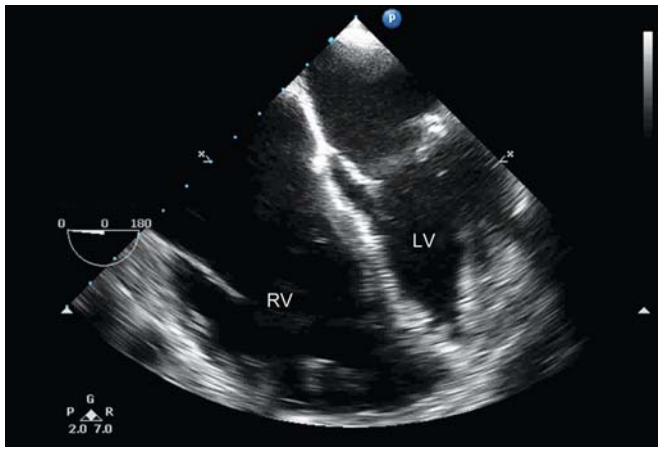


Fig. 12: Right ventricular failure leading to dilation of the RV. Notice the RV/LV ratio of >0.6, and the extension of the RV to the apex of the heart

monitoring of volume and pressure indices is required to maintain optimal fluid status.

Afterload

Low afterload should be suspected when the patient has a low mean arterial pressure, elevated filling pressures and good contractility. Adequate LVEDV with endsystolic collapse of the LV cavity is an echocardiographic sign that may suggest a hyperdynamic ventricle, or extremely low afterload.

Severe Valvular Abnormalities and Infective Endocarditis

Both acute and chronic valvular abnormalities might be present in critically ill patients. Patients with acute respiratory failure should be evaluated for new-onset mitral regurgitation and heart failure to exclude a cardiac cause. Similarly, patients with blood stream infections, embolic events or unexplained fever should be evaluated for infective endocarditis (Fig. 13). Echocardiographic features of severe valvular abnormalities are described in the American Society of Echocardiography guidelines (Available from: www.asecho.org; clinical information guidelines and standards, Accessed Sept 27, 2012).

Pericardial Tamponade

Acute collection of a relatively small amount of pericardial fluid can lead to tamponade and hemodynamic compromise, while more chronic, slowly accumulating collections are better tolerated. Common causes of cardiac tamponade in the ICU include myocardial or coronary perforation secondary to a catheter-based intervention, postcardiac surgery, ascending aortic dissection, chest trauma, anticoagulant therapy, malignant states, infective or uremic pericarditis, and as a complication of myocardial infarction.

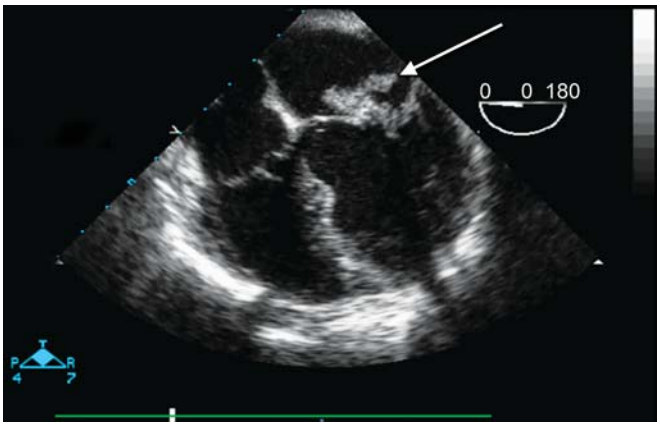


Fig. 13: Infective endocarditis of the mitral valve, with large vegetations (white arrow) attached to the anterior mitral leaflet

Increase in pericardial pressure causes decreased filling of cardiac chambers, leading to decreased cardiac output. Clinically, patients have increased filling pressures (central venous pressure, pulmonary artery diastolic pressure and pulmonary capillary wedge pressure).

Fluid collection along the dependent area or completely surrounding the heart along with septal shift and early-diastolic collapse of the RV provides 2D echocardiographic evidence of pericardial tamponade (Figs 14A and B). Late-diastolic right atrial collapse that persists through more than a third of the cardiac cycle is a very specific sign of tamponade. Increased respiratory variation in mitral and tricuspid inflow on Doppler echocardiography is also a sign of tamponade, though these might be confounded in the ICU patient by mechanical ventilation, respiratory distress, bronchospasm, pleural effusions and arrhythmias.

Loculated pericardial collections can cause selective compression of one or more cardiac chambers in postcardiac surgical patients and can cause hemodynamic compromise, necessitating a thorough evaluation.[24]

It is important to remember that the diagnosis of tamponade is most often made clinically, and that treatment in an unstable patient should not be delayed for echocardiographic diagnosis.

Pulmonary Embolism

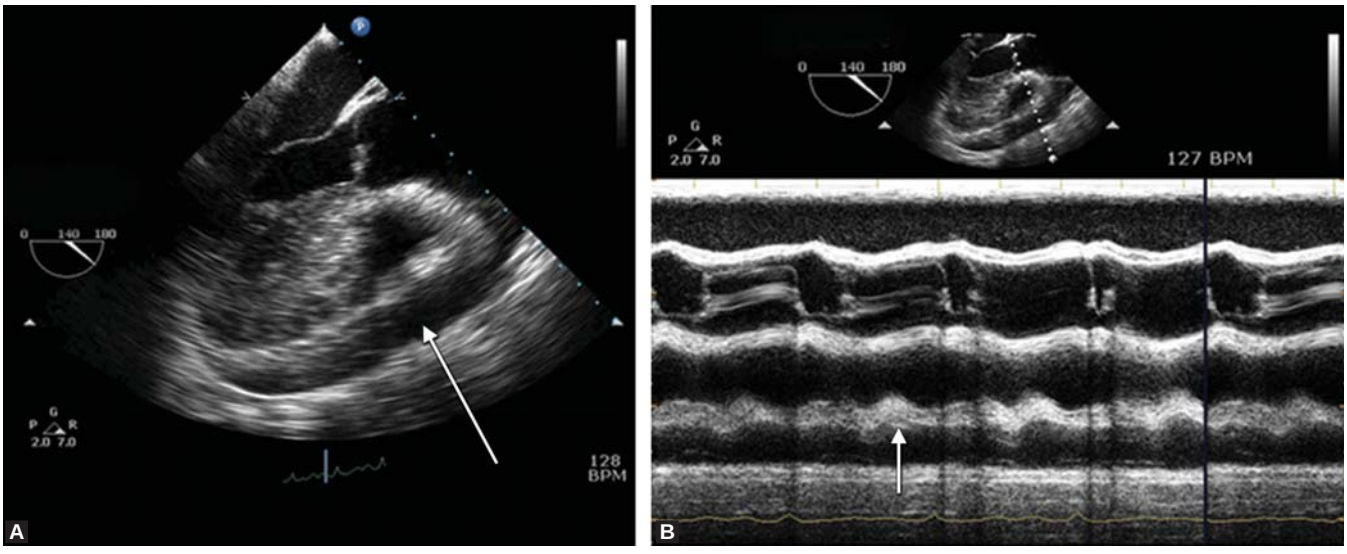
Trauma, prolonged immobility and hypercoagulable disease states (for example, cancer, trauma, postmajor surgery) predispose ICU patients to pulmonary thromboembolism.

Obstruction of the pulmonary artery leads to right ventricular strain, dilation and ultimately failure. These manifest as low cardiac output, elevated right atrial pressure, hypoxia and elevated arterial to end-tidal CO_2 gradient because of dead space ventilation.

TEE has 70 to 80% sensitivity and 80 to 97% specificity in diagnosing pulmonary embolism.[25,26] Since, the left pulmonary artery and distal pulmonary artery branches cannot be visualized on TEE (Fig. 15), the thrombus itself might not be seen. Diagnosis therefore relies on clinical suspicion along with echocardiographic signs of severe acute cor pulmonale, which are nonspecific for PE. These include dilation and failure of the RV, tricuspid regurgitation with elevated peak flow velocity, dilation of the right atrium with dilated inferior vena cava and minimal respiratory variation, and flattening of the interventricular septum (D-shaped left ventricle) due to volume and pressure overload of the RV. Right ventricular dysfunction sparing the apex is highly suggestive of PE.[27]

LVOT Obstruction

Left ventricular outflow tract obstruction (LVOTO) occurs when narrowing of the left ventricular outflow tract, between



Figs 14A and B: Pericardial tamponade. (A) Midesophageal long axis view of the pericardial effusion (white arrow). (B) M-mode interrogation of right ventricular wall. Notice the RV diastolic collapse (white arrow) on M-mode echocardiography indicating tamponade

the interventricular septum and the anterior leaflet of the mitral valve during the systolic ejection phase, impedes the left ventricular ejection. LVOTO can develop in patients with a hypertrophied left ventricle due to uncontrolled hypertension, aortic stenosis or hypertrophic obstructive cardiomyopathy, and after mitral valve repair due to undersized annuloplasty or excessive posterior leaflet length. A small hyperdynamic ventricle causes a Venturi effect and systolic anterior motion (SAM) of the anterior mitral leaflet and LVOTO. Echocardiographic features of LVOTO include 2D visualization of SAM of the anterior mitral valve leaflet causing obstruction and turbulence in the LVOT on color Doppler (Figs 16 and 17). A gradient can also be quantified across the LVOT on continuous wave Doppler with a characteristic late-peaking profile.

LVOTO is best seen in the midesophageal LVOT long axis view at 120°. The gradient across the LVOT can be quantified in the transgastric LV long axis view, the deep transgastric 4-chamber view and occasionally in the midesophageal four-chamber view. Once LVOTO is diagnosed, resuscitation with volume and vasopressors should be promptly initiated, along with discontinuation of inotropic agents and initiation of beta-blockade. Echocardiography plays a key role in diagnosing LVOTO, with a dramatic effect on therapy.[28]

Unexplained Hypoxemia

Hypoxemia due to intracardiac right-to-left shunting can occur with a PFO, ASD or VSD, in the presence of elevated right-sided pressures. Intracardiac shunting should be suspected in the presence of an unexplained embolic stroke or refractory hypoxemia out of proportion to the severity of pulmonary disease. Up to 25% of the adult population has a PFO. Ordinarily, PFOs allow minimal shunting. However, elevation of right atrial pressures in the ICU due to pulmonary hypertension, severe tricuspid regurgitation or right ventricular infarction can cause significant right-to-left shunting with hypoxemia.[29]

The interatrial septum can be evaluated best in the midesophageal aortic valve short axis view and in the midesophageal bicaval view. The presence of a shunt can be detected on color Doppler (Fig. 18) and by contrast (agitated-saline) injection. Valsalva maneuver in a spontaneously breathing patient, or sudden release of sustained airway pressure at the time of contrast injection in a ventilated patient, increases right-to-left shunt and improves detection. Immediate appearance of contrast on the left side after right-sided opacification indicates an intracardiac shunt. A longer delay in contrast appearance on the left side (>3-5 cardiac cycles) indicates an extracardiac shunt, as in arteriovenous fistulae with

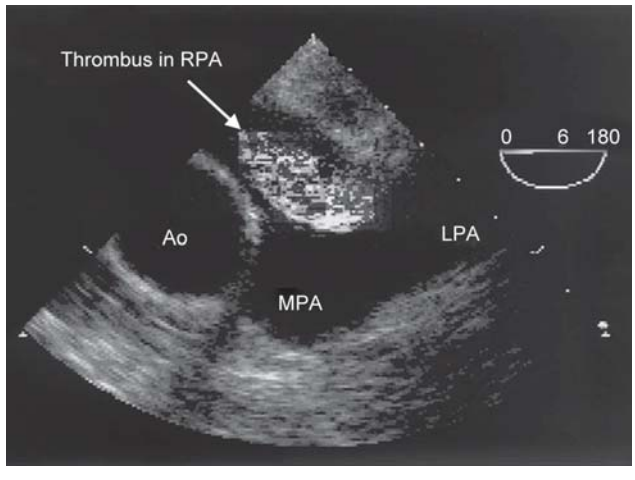


Fig. 15: Clot in the main pulmonary artery [RPA: Right pulmonary artery; NP: Is not relevant to the image, and can be blacked out. Most likely, this is hp, as in Hewlett Packard, the device manufacturer; AO: Aorta; MPA: Main pulmonary artery; LPA: Left pulmonary artery]

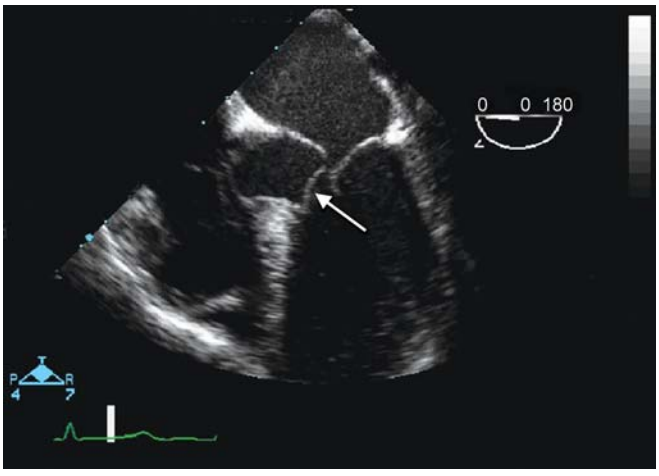


Fig. 16: SAM of the anterior mitral leaflet (white arrow) in systole, causing left ventricular outflow tract obstruction

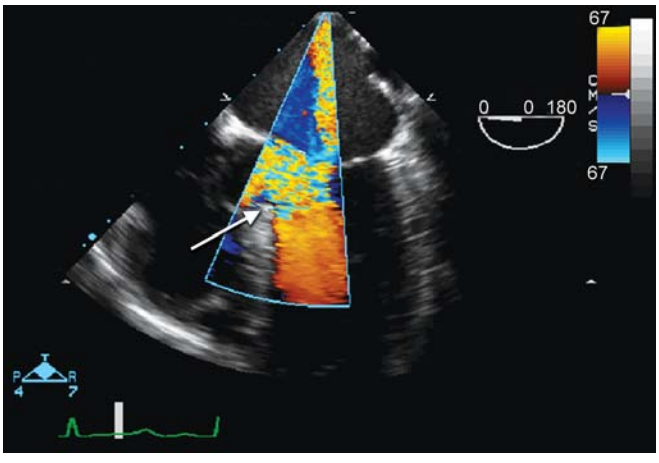


Fig. 17: Left ventricular outflow tract obstruction causing turbulence on color Doppler (white arrow)

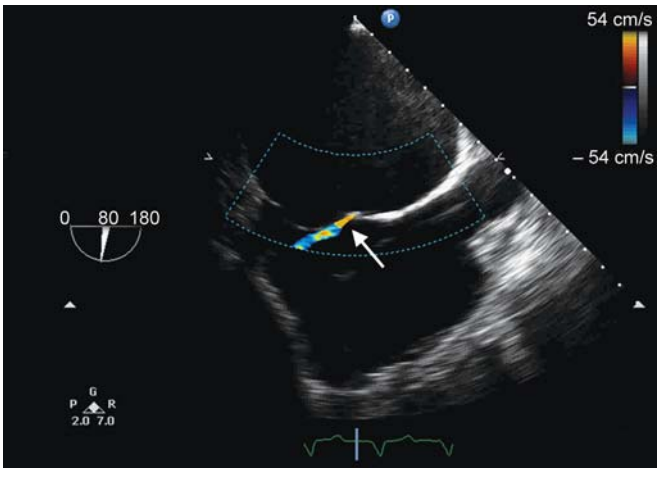


Fig. 18: Midesophageal bicaval view demonstrating a patent foramen ovale with right to shunt (white arrow) on color Doppler

end-stage liver disease (hepatopulmonary syndrome), with contrast entering the left atrium through the pulmonary veins.

Aortic Dissection

Aortic dissection may involve the ascending aorta (Stanford type A) or the arch and/or descending aorta (Stanford type B) or both. Severe hypertension, connective tissue disorders, trauma and aortic cannulation in cardiac surgery or catheterization can lead to aortic dissection. TEE has 99% sensitivity and 98% specificity in the diagnosis of aortic dissection,[30] with a quick bedside examination. Visualization of the dissection flap on TEE is needed to diagnose aortic dissection (Figs 19 and 20). To avoid misdiagnosis due to artifacts, the dissection flap should be visualized in more than one imaging plane. TEE features of artifacts include displacement parallel to aortic walls, similar blood flow velocities on both sides, over imposition of blood flow angle with the aortic wall >85° and thickness >2.5 mm.[31] The flap should be followed along the length of the aorta to define the extent of the dissection, and to identify the location of the intimal tear if possible. TEE should also be used to detect the presence, severity and mechanism of aortic insufficiency, evaluate for coronary involvement, estimate ventricular function and identify pericardial effusion and tamponade.

The root of the aorta can be visualized in the midesophageal aortic valve long axis and short axis views, and the proximal ascending aorta in the midesophageal ascending aorta short and long axis views by pulling the probe slightly out. The aortic arch and descending thoracic aorta are visualized by turning the probe further to the left, and advancing deeper. The distal ascending aorta and the proximal aortic arch are not visualized on TEE because of the interposition of the airway between the esophagus and the aorta.

Blunt Chest Trauma and Myocardial Contusion

Blunt chest trauma with high-speed sudden deceleration is associated with cardiac contusion, traumatic aortic disruption and aortic dissection, as well as disruption of other major vessels and the mitral apparatus. Traumatic aortic disruption usually occurs at the isthmus or the ascending aorta proximal to the origin of the great vessels. These severe, life-threatening injuries require prompt diagnosis and treatment, and TEE provides an accurate, rapid and safe method of assessment.[32]

Right ventricular wall motion abnormality without signs of infarction on electrocardiogram, along with increased diastolic wall thickness and increased ventricular wall brightness, are echocardiographic signs of traumatic myocardial contusion. Traumatic disruption of the aorta presents as loss of aortic continuity, intramural hematoma, an increased distance between the probe and the aorta, a double-contour sign where the aortic wall appears thicker, and mediastinal and pleural fluid collections. As with dissection, the distal ascending aorta and the proximal arch

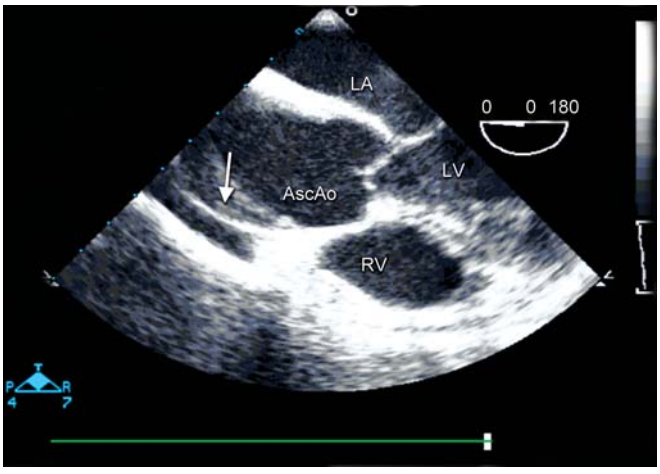


Fig. 19: Midesophageal four-chamber view, with the probe withdrawn slightly to visualize the aortic valve and proximal ascending aorta, showing aortic dissection with hematoma in the false lumen (white arrow) of the ascending aorta (LA: Left atrium; AscAO: Ascending aorta; LV: Left ventricle; RV: Right ventricle)

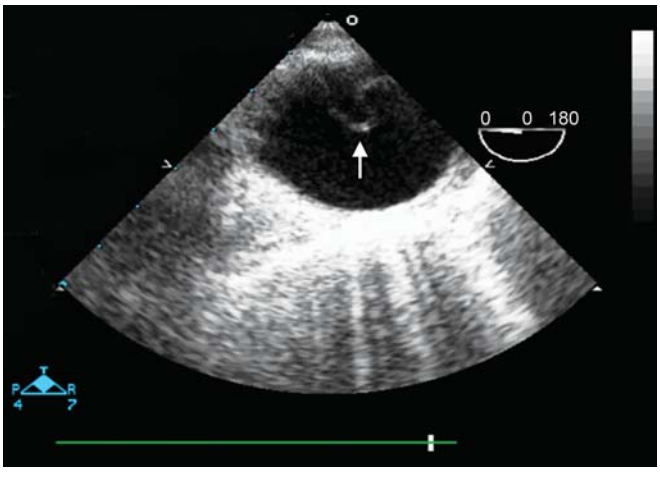


Fig. 20: Midesophageal short-axis view of the descending thoracic aorta demonstrating aortic dissection. The white arrow points to the intimal flap

are not visualized on TEE. TEE can also identify associated pericardial and pleural effusions in chest trauma victims.

CONCLUSION

TEE is a user-dependent, relatively safe and extremely useful diagnostic tool that can help tailor therapy for hemodynamically unstable, critically ill patients. This modality can be used to estimate ventricular volume and function, and diagnose specific valvular abnormalities, pericardial tamponade, pulmonary embolism, LV outflow tract obstruction, aortic dissection and trauma-related cardiac injury. Specific training in TEE is essential in acquisition of this skill, and continuing education is necessary to maintain proficiency.

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