Echocardiography in the Intensive Care Unit

a report by

Achikam Oren-Grinberg, MD, MS1 and Daniel Talmor, MD, MPH2

1. Instructor in Anesthesia; 2. Associate Professor of Anesthesia, Department of Anesthesia and Critical Care Medicine, Beth Israel Deaconess Medical Center and Harvard Medical School

Echocardiography has evolved to become a widely used and versatile modality for diagnosis and monitoring of critically ill patients. As such, its use has expanded from the cardiology suite into the emergency room, peri-operative period, and intensive care unit (ICU). Echocardiography provides both anatomical and functional information about the heart, with the ability to assess systolic and diastolic function, cavity size, and valvular function.

Basic Terminology of Echocardiography Techniques

In order to reach a diagnosis or decide on a management plan, a sonographer must utilize different echocardiographic imaging techniques and hemodynamic modalities. The following is a list of the basic techniques used during an echocardiographic study.

2D Echocardiography

2D echocardiography is the backbone of the echocardiographic exam. Using 2D, a complete visualization of the beating heart is achieved by displaying anatomical structures in realtime tomographic images. By aiming the ultrasound probe at the heart, precisely oriented anatomical ‘slices’ are obtained. The information acquired includes cardiac chamber size, global and regional systolic function, and valvular anatomy.

M-mode Echocardiography

M-mode or motion-mode images are a continuous 1D graphic display that can be derived by selecting any of the individual sector lines from which a 2D image is constructed. It is useful for quantification of myocardial wall and chamber sizes, which in turn can be used to estimate left ventricular (LV) mass and chamber volume, respectively. In addition, since it has high temporal resolution, M-mode is helpful in assessing the motion of rapidly moving cardiac structures such as cardiac valves.

Doppler Echocardiograph

Doppler echocardiography is used to supplement 2D and M-mode echocardiography. It can provide functional information regarding intra-cardiac hemodynamics: systolic and diastolic flow, blood velocity and volume, severity of valvular lesions, location and severity of intracardiac shunts, and assessment of diastolic function. The four types of Doppler modality used are continuous-wave (CW), pulsed-wave (PW), color flow mapping (CFM), and tissue Doppler. CW Doppler is used for measuring high-pressure-gradient/high-velocity flows, such as those seen in aortic stenosis. When using CW Doppler, the ultrasound probe continuously transmits and receives sound waves. PW Doppler can evaluate higher flows, but does so at the expense of spatial specificity. PW Doppler is used for measuring lower-pressure-gradient/lower-velocity flows, such as those seen in mitral stenosis. In this mode, the ultrasound probe sends out a pulse of sound and then waits to receive reflected waves. CFM is useful for screening valves for stenosis or regurgitation, quantifying the degree of valvular regurgitation, imaging systolic and diastolic flow, and detecting intracardiac shunts.

Assessment of Ventricular Systolic Function

Qualitative Method

Assessment of Ejection Fraction

Systolic dysfunction of either ventricular chamber must be considered in every unstable patient. Most commonly, assessment of systolic function is performed using the qualitative method, whereby the interpreting physician assesses ventricular function using 2D images. When using 2D imaging, two of the most important questions regarding hemodynamic instability can be rapidly answered: are the ventricles contracting well and are they adequately filled? This should be assessed using multiple tomographic planes, and attention must be paid to obtaining adequate endocardial definition. Normal ventricular contraction consists of simultaneous myocardial thickening and endocardial excursion toward the center of the ventricle. It is important to look for this myocardial thickening: infarcted myocardium may be pulled inward by surrounding normal myocardium. For qualitative assessment of overall systolic function, the echocardiographer integrates the degree of wall thickening and endocardial motion in all tomographic views and reaches a conclusion about overall LV systolic function and ejection fraction (EF). This method of EF

Achikam Oren-Grinberg, MD, MS, is a Staff Anesthesiologist and Intensiv Care Physician at Beth Israel Deaconess Medical Center in Boston, Massachusetts. His main academic and clinical interests include the utilization of ultrasound—including echocardiography and lung ultrasound—in the management of critically ill patients. Dr Oren-Grinberg established the echocardiography program in the intensive care units of Beth Israel Deaconess Medical Center, where intensivists perform and manage hemodynamically unstable patients with echocardiography.

Daniel Talmor, MD, MPH, is Director of Trauma Anesthesia and Critical Care in the Department of Anesthesia, Critical Care, and Pain Medicine at the Beth Israel Deaconess Medical Center in Boston, Massachusetts and an Associate Professor of Anesthesia at Harvard Medical School. His fields of research include intensive care outcomes and the optimal delivery of mechanical ventilation. He has lectured extensively nationally and internationally on subjects related to both critical care and disaster medicine. He has received many awards, honors, and grants for this work.

E: dtalmor@bidmc.harvard.edu
Critical Care

Figure 1: Schematic Representation of the Left Ventricular Outflow Tract as a Cylinder

A: To calculate the volume of a cylinder, the base is multiplied by its height. In the left ventricular outflow tract (LVOT), the cylinder’s base is the LVOT cross-sectional area (CSA), which is calculated from the LVOT as a 2D2 measure. B: Since blood flow through the cardiac system is pulsatile, the instantaneous velocities during the ejection phase should be sampled and then integrated. Velocity time integral (VTI) is the sum of the instantaneous velocities, which is equal to the area enveloped by the Doppler velocity profile. The distance the average blood cell travels during systole is calculated automatically by the echocardiographic computer as an integral of the VTI area under the curve. AV = aortic valve; D = diameter.

Figure 2: Transesophageal Echocardiogram Measurement of Cardiac Output

A: Mid-esophageal long-axis view measuring left ventricular outflow tract (LVOT) diameter of 1.9 cm. B: Deep transgastric view. A pulse-wave Doppler samples the LVOT at the level at which the diameter of the LVOT was measured. Velocity time integral (VTI) is calculated by the computer by tracing the outer envelope of the spectral signal and is determined to be 19.4 cm. Stroke volume (SV) is the product of the cross-sectional area (CSA) and VTI: 2.83 cm^2 x 19.4 cm = 55 cm^3. Cardiac output (CO) is then calculated by multiplying the calculated SV by the heart rate (HR): CO (cm^3/min) = SV x HR.

This approach to SV and CO calculations has shown very good correlation with thermodilution-derived CO measurements. There are, however, several potential sources of error. First, CSA determination often leads to the greatest source of error. When using any diameter for CSA determination, any error in measurement will be squared, as CSA = aD/2. This translates to a 20% error in calculation of CO for each 2 mm error when measuring a 2 cm-diameter LVOT. Studies have shown that while the Doppler velocity curves can be recorded consistently with little inter-observer measurement variability (2–5%), the variability in 2D LVOT diameter measurements for CSA is significantly greater (8–12%).

Second, the Doppler signal is assumed to have been recorded at a parallel or near-parallel intercept angle, called β, to blood flow. The Doppler equation has a cos β term in its denominator. With an intercept angle of 0°, the cos β term equals 1. Deviations of up to 20° in intercept angle are acceptable since only a 6% error in measurement is introduced. Third, velocity and diameter measurements should be made at the same anatomical site. When the two are measured at different places, the accuracy of SV and CO calculations is reduced. Finally, while the pattern of flow is assumed to be laminar, in reality the flow profile is parabolic. This has some impact on velocity-based calculations. However, in routine clinical practice this factor is of little significance and can essentially be ignored.

Assessment of Fluid Responsiveness

One of the most challenging and crucial tasks in the management of a hemodynamically unstable patient is to accurately predict whether the patient would benefit from fluid therapy. Overhydration may lead to pulmonary edema, hypoxia, and worst outcome, and therefore should be avoided. Fluid responsiveness is best assessed by following the dynamic parameter approach (SV variation [SVV] or pulse pressure variation), and can be easily performed with echocardiography. The increase in intrathoracic pressure during the inspiratory phase of positive-pressure ventilation (PPV) leads to simultaneous but different physiological effects on the left and right sides of the heart. In the left side, SV increases as blood is pushed forward out of the pulmonary veins into the LV. In the right side, RV inflow decreases secondary to compression of the inferior vena cava. At the beginning of the exhalation phase, SV decreases since both the pulmonary veins and the RV are relatively ‘empty.’ This is a normal phenomenon in all patients ventilated with PPV, and is known as reversed pulsus paradoxus. In hypovolemic patients this phenomenon is further

stroke volume (SV) is the product of the cross-sectional area (CSA) and VTI: SV = CSA x VTI. CO can then be easily derived by multiplying the calculated SV by the heart rate (HR): CO (cm^3/min) = SV x HR.

estimation is of great clinical utility and can be performed with good correlation to quantitative measurements. There are, however, a few potential pitfalls to 2D assessment of EF that must be considered: accurate assessment requires satisfactory endocardial border definition—qualitative EF estimation becomes inaccurate when the endocardium is inadequately defined; accurate estimation of EF depends on the experience of the echocardiographer; and in asynchronous contraction (paced-rhythm, conduction defects, etc.), assessment of EF is more difficult.

Quantitative Assessment of Left Ventricular Systolic Function

Quantitative methods of assessing LV systolic function include the volumetric method utilizing geometrical models and the disc method (Simpson’s rule). Both are limited by the detection of endocardial borders. The explanation of the theory of these methods is beyond the scope of this article.

Calculation of Cardiac Output

Doppler spectral profiles can be used to quantitatively evaluate LV function. This evaluation of LV systolic function is based on calculation of the stroke volume (SV) and cardiac output (CO). SV—the volume of blood ejected during each cardiac cycle—is a key indicator of cardiac performance. SV is calculated using PW Doppler to measure the instantaneous blood velocity recorded during systole from an area in the heart where a cross-sectional area (CSA) can be easily determined. Any cardiac chamber or structure that has a measurable CSA may be used, for example mitral valve annulus, right ventricular outflow tract (RVOT), and tricuspid annulus. Despite this, the LV outflow tract (LVOT) is most commonly used because its cross-section is essentially a circle. Assuming a circular geometry, the LVOT can be thought of as a cylinder, as such, its volume is calculated as the base multiplied by its height. By measuring the diameter (D) of the LVOT, the CSA is calculated as aD^2 (see Figures 1 and 2). By tracing the outline of the PW Doppler profile, the echocardiographic computer can calculate the integral of velocity by time or the velocity time integral (VTI) (see Figures 1 and 2). The VTI is the distance (commonly referred to as the stroke distance) that the average red cell has traveled during the systolic ejection phase. SV (cm^3) is then calculated by multiplying the VTI (stroke distance in cm) by the CSA (cm^2) of the conduit (i.e. LVOT, aorta, mitral valve annulus, pulmonary artery) through which the blood has traveled: SV = CSA x VTI. CO can then be easily derived by multiplying the calculated SV by the heart rate (HR): CO (cm^3/min) = SV x HR.

This approach to SV and CO calculations has shown very good correlation with thermodilution-derived CO measurements. There are, however, several potential sources of error. First, CSA determination often leads to the greatest source of error. When using any diameter for CSA determination, any error in measurement will be squared, as CSA = aD/2. This translates to a 20% error in calculation of CO for each 2 mm error when measuring a 2 cm-diameter LVOT. Studies have shown that while the Doppler velocity curves can be recorded consistently with little inter-observer measurement variability (2–5%), the variability in 2D LVOT diameter measurements for CSA is significantly greater (8–12%). Second, the Doppler signal is assumed to have been recorded at a parallel or near-parallel intercept angle, called β, to blood flow. The Doppler equation has a cos β term in its denominator. With an intercept angle of 0°, the cos β term equals 1. Deviations of up to 20° in intercept angle are acceptable since only a 6% error in measurement is introduced. Third, velocity and diameter measurements should be made at the same anatomical site. When the two are measured at different places, the accuracy of SV and CO calculations is reduced. Finally, while the pattern of flow is assumed to be laminar, in reality the flow profile is parabolic. This has some impact on velocity-based calculations. However, in routine clinical practice this factor is of little significance and can essentially be ignored.

Assessment of Fluid Responsiveness

One of the most challenging and crucial tasks in the management of a hemodynamically unstable patient is to accurately predict whether the patient would benefit from fluid therapy. Overhydration may lead to pulmonary edema, hypoxia, and worst outcome, and therefore should be avoided. Fluid responsiveness is best assessed by following the dynamic parameter approach (SV variation [SVV] or pulse pressure variation), and can be easily performed with echocardiography. The increase in intrathoracic pressure during the inspiratory phase of positive-pressure ventilation (PPV) leads to simultaneous but different physiological effects on the left and right sides of the heart. In the left side, SV increases as blood is pushed forward out of the pulmonary veins into the LV. In the right side, RV inflow decreases secondary to compression of the inferior vena cava. At the beginning of the exhalation phase, SV decreases since both the pulmonary veins and the RV are relatively ‘empty.’ This is a normal phenomenon in all patients ventilated with PPV, and is known as reversed pulsus paradoxus. In hypovolemic patients this phenomenon is further
respectively). This index can be calculated rapidly by either transthoracic and non-responders with high sensitivity and specificity (100 and 89%, respectively). An index of >12% has been shown to discriminate between fluid responders and non-responders with high sensitivity and specificity (100 and 89%, respectively). This index can be calculated rapidly by either transthoracic echocardiogram (TTE) or transesophageal echocardiogram (TEE).

**Aortic Flow Index**

The increase in SV during PPV as described above leads to increased peak flow across the aortic valve. Similarly, the decrease in SV during exhalation leads to a decrease in peak flow across the aortic valve. The aortic flow index can efficiently predict fluid responsiveness in patients ventilated with PPV. To calculate the aortic flow index, PW Doppler is used to sample flow at the ascending aorta. This will generate a series of peak-flow spectral displays that are increased during inspiration and decreased during exhalation (see Figure 3). The formula to calculate the aortic flow index is:

\[
\text{Aortic flow index} = \frac{(\text{PEAK}_{\text{max,ins}} - \text{PEAK}_{\text{min,exp}}) \times 100}{\text{mean}}
\]

An index of >12% has been shown to discriminate between fluid responders and non-responders with high sensitivity and specificity (100 and 89%, respectively). This index can be calculated rapidly by either transthoracic echocardiogram (TTE) or transesophageal echocardiogram (TEE).

**Superior Vena Cava Collapsibility Index**

This concept is similar to other dynamic parameters. During the inspiratory phase of PPV, the superior vena cava (SVC) collapses due to an increase in the intrathoracic pressure. The SVC relaxes back to its baseline during exhalation. The degree of collapsibility depends on the degree of hypovolemia: as less volume circulates in the intravascular compartment, the SVC will be susceptible to an increase in intrathoracic pressure, and thus this phenomenon is exacerbated in a state of hypovolemia. The SVC index can be calculated with TEE only by using either 2D or M-mode to measure the SVC diameter during PPV (see Figure 3). The formula to calculate this index is:

\[
\text{SVC collapsibility index} = \frac{(\text{Dmax}_{\text{exp}} - \text{Dmin}_{\text{ins}}) \times 100}{\text{Dmax}_{\text{exp}}}
\]

An index of >36% has been shown to predict fluid responsiveness with high sensitivity and specificity (90 and 100%, respectively), and can be useful in predicting the need for fluid therapy in hemodynamically unstable patients.

**Extracardiac Causes of Hemodynamic Instability**

**Pericardial Tamponade**

Pericardial tamponade is a clinical diagnosis; echocardiography may, however, be of assistance in equivocal cases. The echocardiographic diagnosis of tamponade first requires demonstration of an effusion. From there, the examination should focus on identifying cardiac chamber collapse. As the pericardial pressure increases, the cardiac chambers will show collapse in sequence from lowest to highest pressure: the atria will collapse first, followed by the RV and then the LV. Furthermore, the collapse of each chamber will be most pronounced during the portion of the cardiac cycle during which the pressure is the lowest in that chamber: ventricular systole for the atria and ventricular diastole for the ventricles. This collapse can be evaluated with M-mode interrogation of the chamber walls. PW Doppler echocardiographic interrogation of ventricular inflow across both the mitral and the tricuspid valves can also be used to assess for the effects of respiratory variation on ventricular filling—the echocardiographic equivalent of pulsus paradoxus (see Figure 4). In the setting of tamponade, the peak LV inflow velocities will decrease by more than 25% with spontaneous inspiration, while peak RV velocities will decrease by more than 25% during expiration.

**Pulmonary Embolism**

Diagnosis of pulmonary embolism (PE) in hemodynamically unstable ICU patients can be extremely challenging. TTE has been described as a routine screening test in patients with suspected PE. When TTE is non-diagnostic and the clinician has a high level of suspicion, or there is evidence of RV overload or hemodynamic instability, TEE examination is indicated. In these circumstances, TEE has a sensitivity of 80% and a specificity of 100%. 2D echo visualization of the main and proximal right and left pulmonary arteries may allow visualization of an embolus lodged in those locations. The left pulmonary artery may be difficult to visualize as the left bronchus is frequently interposed between the TEE probe and the artery. Echocardiographic findings of acute PE with hemodynamic instability include signs of RV pressure overload. These include a dilated, hypokinetic
RV, increased RV/LV ratio (secondary to bulging of the interventricular septum into the LV), and tricuspid regurgitation (TR). In addition, the inferior vena cava is usually dilated and does not collapse on inspiration in spontaneously breathing patients. Pulmonary hypertension can be seen. Classically, pulmonary pressure is only mildly elevated in patients without chronic pulmonary hypertension. This is because the RV is unable to generate high pressures in the absence of hypertrophy. More than 80% of patients with documented PE have 2D imaging or Doppler abnormalities of RV dimension or dysfunction that may suggest acute PE (see Figure 5).24-26 Unfortunately, these findings are non-specific and can be found in other clinical situations such as acute chronic obstructive pulmonary disease (COPD) exacerbation and chronic pulmonary hypertension. Nevertheless, a more specific sign for acute PE has been described. McConnell sign—akinesia of the mid-free RV wall with apical sparing—has been shown to have 77% sensitivity and 94% specificity for the diagnosis of acute PE.27 In addition, a new tissue Doppler index demonstrated a sensitivity and specificity of 92% in detecting acute PE,28 and can differentiate chronic from acute pulmonary hypertension. Despite these improved sensitivities and specificities, a definite echocardiographic diagnosis of acute PE can be made only with visualization of emboli in the main or branched pulmonary arteries, or in the right atrium or ventricle.

Echocardiography as a Hemodynamic Monitor in the Intensive Care Unit

Intensivists can utilize echocardiography in the management of the hemodynamically unstable patient in the ICU—an often challenging and time-consuming exercise. In this setting, a lower level of echocardiographic expertise is needed compared with a full cardiac assessment performed by cardiologists. The Focused Assessed Transthoracic Echo (FATE) protocol (see Figure 6) is a relatively easy-to-use and efficient protocol for monitoring patients in the ICU.29 It is a rapid echocardiographic assessment performed to screen for significant pathology (e.g. cardiac tamponade, pulmonary embolism, pneumothorax, etc.) and obtain information about the contractility and volume of the heart. The steps of the protocol include: excluding obvious pathology; assessing wall thickness and chamber dimensions; assessing contractility; visualizing the pleura on both sides; and relating the information to the clinical context. The exam can be performed by physicians with only limited training in echocardiography. It requires imaging the heart and pleura in the most favorable sequence from one or more tomographic planes. The protocol has been shown to be a practical and useful hemodynamic monitoring tool in ICU patients. A study of the FATE protocol demonstrated that it added new information in 37.3% of patients and contributed decisive information in 24.5% of patients. The information was too limited to aid in patient management in only 2.6% of the exams performed.24 These findings and other reports support the benefit of an echocardiographic exam when performed by a non-cardiologist in the ICU.27-30

Conclusion

Echocardiography is extremely useful in the management of hemodynamically unstable patients. It allows for rapid assessment of ventricular systolic function, calculation of CO, and assessment of fluid responsiveness and need for fluid therapy. In addition, it allows for the rapid diagnosis of major causes of cardiovascular collapse, such as cardiac tamponade and pulmonary embolism at the patient’s bedside. Intensivists should expand the use of echocardiography in their management of ICU patients, as it is a safe modality that can be performed and interpreted by physicians with only limited training in echocardiography.