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critical care review

Bedside Ultrasonography in the ICU*

Part 1

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Ultrasonography has become an invaluable tool in the management of critically ill patients. Its safety and portability allow for use at the bedside to provide rapid, detailed information regarding the cardiovascular system and the function and anatomy of certain internal organs. Echocardiography can noninvasively elucidate cardiac function and structure. This information is vital in the management hemodynamically unstable patients in the ICU. In addition, ultrasonography has particular value for the assessment and safe drainage of pleural and intra-abdominal fluid and the placement of central venous catheters. A new generation of portable, battery-powered, inexpensive, hand-carried ultrasound devices have recently become available; these devices can provide immediate diagnostic information not assessable by physical examination alone and allow for ultrasound-guided thoracocentesis, paracentesis, and central venous cannulation. This two-part article reviews the application of bedside ultrasonography in the ICU.

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Key words: bedside ultrasonography; cardiac function; central-line cannulation; critically ill; hand-carried ultrasound; ICU; paracentesis; thoracocentesis; transesophageal echocardiography; transthoracic echocardiography

Abbreviations: ACP = acute cor pulmonale; CO = cardiac output; CSA = cross-sectional area; DLVOTO = dynamic left ventricular outflow tract obstruction; 2D = two dimensional; E/A ratio = ratio of early to late transmitral diastolic filling; EF = ejection fraction; FS = fractional shortening; IVS = interventricular septum; LA = left atrial/atrium; LV = left ventricular/ventricle; LVEDA = left ventricular end-diastolic area; LVEDV = left ventricular end-diastolic volume; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; MR = mitral regurgitation; PAC = pulmonary artery catheter; PAP = pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; PE = pulmonary embolism; PR = pulmonary regurgitation; RA = right atrial/atrium; RV = right ventricular/ventricle; SV = stroke volume; TEE = transesophageal echocardiography; TR = tricuspid regurgitation; TTE = transthoracic echocardiography

Bedside ultrasonography has become an indispensable tool in the management of critically ill patients. Ultrasonography in the ICU allows for the rapid assessment of cardiac function and provides information that may be more valuable than that derived from invasive hemodynamic monitoring. In addition, ultrasonography has particular value for the assessment and safe drainage of pleural and intraabdominal fluid collections and the placement of central venous catheters. Although transesophageal echocardiography (TEE) was once the principal diagnostic approach to study the ICU patient, advances in ultrasound imaging, including harmonic imaging, digital acquisition, and contrast for endocardial enhancement, have improved the diagnostic yield of the simpler and safer approach of transthoracic echocardiography (TTE). Furthermore, a new generation of portable, battery-powered, inexpensive, hand-carried ultrasound devices have recently become available. The true portability, ease of use, and low cost make these devices ideally suited for use by the intensivist. The safety and utility of bedside ultrasonography performed by adequately trained intensivists has now been

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well demonstrated. This two-part review will be devoted to the application of bedside ultrasonography in the ICU. The emphasis will be placed mostly on echocardiography and cardiovascular diagnostics that could potentially be performed at the bedside by the intensivist with proper training. The use of bedside ultrasound to facilitate central line placement and to aid in the care of patients with pleural effusions and intra-abdominal fluid collections will also be explored.

Acoustic Window in the Critically Ill Patient

The practical value of bedside ultrasonography in the management of critically ill patients is now widely accepted, despite its inherent limitations.[1] These limitations are related mostly to the suboptimal imaging conditions that are often associated with the critically ill patient and as a consequence of the constrained physical environment of the ICU. For an ultrasound study to be deemed adequate, a good acoustic "window" is required to allow accurate analysis. Ultrasonography uses the physical principle that sound is reflected from tissue interfaces, allowing a two-dimensional (2D) image of the anatomic structure studied to be constructed.[2] Anything hindering the reflection of this acoustic signal, be it air, bone, calcium, a foreign body, or an another interposed structure, will interfere with ultrasound transmission and will diminish the overall quality of the examination. In the critical care unit, up to one third of patients may be receiving mechanical ventilation, and adequate imaging is frequently limited due to interposition of the inflated lung between the heart and chest wall. Other important factors limiting data acquisition in critically ill patients are related to surgical wounds and dressings, tapes, tubing, surgical emphysema, obesity, and COPD. In addition, lack of patient cooperation and the difficulty of moving patients into the optimal position contribute to a high prevalence of technically inadequate studies.[1]

Although ultrasonography provides a window for the evaluation of structure and function of the heart and other important organs and structures, acquisition of data and interpretation of results are fraught with potential traps.[3] Performing an ultrasound examination requires a thorough knowledge of anatomy and instrumentation, including attention to gain control, gray scale, Doppler velocity settings, and transducer placement. Obtaining adequate images and knowledge of the limitations and pitfalls is key to the performance of ultrasonography in the ICU.

Table 1—*General Indications for Performance of an Echocardiographic Examination in the ICU*

Hemodynamic instability
Ventricular failure
Hypovolemia
Pulmonary embolism
Acute valvular dysfunction
Cardiac tamponade
Complications after cardiothoracic surgery
Infective endocarditis
Aortic dissection and rupture
Unexplained hypoxemia
Source of embolus

Indications for Bedside Ultrasonography

Performance of an ultrasound examination in the ICU allows for procedures at the bedside that previously required transport to the radiology suite. This is an important advantage in the critically ill patient, as the portability of the examination prevents many of the potential complications that are known to occur during patient transport out of the ICU.[4–6] Ultrasonography has become an invaluable tool in the management of critically ill patients; its safety and portability allow for use at the bedside to provide rapid, detailed information regarding the cardiovascular system and the anatomy and function of certain internal organs.[7] It can also be used by the clinician for assessment of the pleural and intraabdominal spaces and for the safe performance of a number of invasive procedures. General indications for performance of an echocardiographic examination in the ICU are listed in Table 1. Major indications for performance of a primary TEE study in the ICU are listed in Table 2. Other indications for the use of bedside ultrasonography by the intensivist are listed in Table 3.

Table 2—*Major Indications for Performance of a Primary TEE Study in the ICU*

Diagnosis of conditions in which high image quality is vital
Aortic dissection
Assessment of endocarditis
Intracardiac thrombus
Imaging of structures that may be inadequately seen by TTE
Thoracic aorta
LA appendage
Prosthetic valves
Echocardiographic examinations of patients with conditions that prevent image clarity with TTE
Severe obesity
Emphysema
Mechanical ventilation with high level of positive end-expiratory pressure
Presence of surgical drains, surgical incisions, dressings
Acute perioperative hemodynamic derangements

Table 3—*Other Indications for Use of Bedside Ultrasonography by the Intensivist*

Central line placement
Assessment of pleural effusions and intra-abdominal fluid collections
Urinary bladder scan
Focused assessment of the trauma patient
Intra-aortic balloon counterpulsation

Performance of bedside TTE and other noninvasive ultrasonographic examinations is safe and not associated with significant risk to the patient. Performance of bedside TEE has a low incidence of serious complications, with a rate of $< 0.5\%$.[8] Daniel et al[9] reported significant complications related to TEE in 18 of 10,218 examinations (0.18%). The reported mortality rate associated with TEE is 0.01 to 0.03%.[10] The sedated and endotracheally intubated critically ill patient is at increased risk for traumatic injury to the GI tract compared with an awake patient, as the sedated patient cannot assist with probe insertion by swallowing and will not resist when insertion is difficult.[11] Increased difficulty in directing the TEE probe can also be met in the presence of a nasogastric tube. Colreavy and coworkers[11] reported a complication rate of 1.6% in 255 critically ill patients who underwent TEE performed by intensive care physicians. TEE is thus associated with surprisingly few complications, given the high severity of illness in ICU patients; however, close monitoring of the hemodynamic and oxygenation parameters are essential.[8] Specific contraindications to the insertion of a TEE probe are listed in Table 4.

Preparation of the Patient

Adequate positioning of the patient is an important step in obtaining adequate image quality. For performance of TTE and TEE, optimal imaging is usually obtained by having patients in the left lateral decubitus position. For other types of ultrasonographic studies, adequate positioning of the patient will vary depending on the structures being assessed (pleural space, abdominal space, vascular structures, bladder). Care must be taken when positioning the critically ill patient who is usually attached to multiple vascular catheters, an endotracheal tube, drains, and other ICU-related accessories. When the ultrasound examination is done to localize and mark pleural or abdominal fluid collections for subsequent drainage, it is essential that the patient remains in the same position in which the marking was done until the actual drainage of the collection is performed. Risks of perforating surrounding organs (heart, spleen, liver, lungs, bowel) and inducing significant morbidity are increased if the drainage is performed in a position different than the one in which the marking was done.

Table 4—*Contraindications to TEE Examination*

Absolute contraindications
Esophageal pathology
Stricture
Mass or tumor
Diverticulum
Mallory-Weiss tear
Dysphagia or odynophagia not previously evaluated
Cervical spine instability
Relative contraindications
Esophageal varices
Recent esophageal or gastric surgery
Oropharyngeal carcinoma
Upper GI bleeding
Severe cervical arthritis
Atlantoaxial disease

To optimize the ultrasonographic examination, the patient must be cooperative, calm, and not agitated. Noninvasive procedures such as TTE and abdominal ultrasound are usually well tolerated by the patient, and additional sedation is rarely needed to perform these procedures. The patient should be kept from eating or drinking for at least 4 h prior to a TEE. Topical anesthesia of the oropharynx is recommended prior to TEE probe insertion.[2] Even when adequate topical anesthesia is provided, insertion of the TEE probe can still cause a significant amount of discomfort and anxiety to the patient, so providing adequate sedation is very important. Frequently used sedative agents include IV midazolam, fentanyl, and propofol.

Bedside Echocardiography in the Critically Ill Patient

Echocardiography can noninvasively elucidate cardiac structure and mechanical function. The supplementary information provided by this technique can help determine the cause of hypotension refractory to volume expansion and inotropic or vasopressor infusions.[2] It can help in the diagnosis of a wide spectrum of cardiovascular abnormalities and guide therapeutic management. An adequate understanding of echocardiography is thus essential for the intensivist.

TTE vs TEE

Accurate and prompt diagnosis is crucial in the ICU. The easiest and least invasive way to image cardiac structures is echocardiography by the transthoracic approach.[2] This noninvasive imaging modal-

ity is of great value in the critical care setting because of its portability, widespread availability, and rapid diagnostic capability. In the ICU, TTE may in certain cases fail to provide adequate image quality because of factors that can potentially hinder the quality of the ultrasound signal, as described previously. The failure rate (partial or complete) of the transthoracic approach has been reported to be from 30 to 40% in the ICU setting.[12,13] However, significant improvements in transthoracic imaging with the advent of harmonics and contrast and digital technologies have resulted in a lower failure rate (10 to 15% in our institutions). As a result of the significantly improved technical quality of TTE imaging, the majority of ICU patients can be satisfactorily studied with this modality. However, immediate TEE will be preferable in certain specific clinical situations where TTE is likely to fail or be suboptimal (Table 2).[13] Even in cases where TEE imaging is necessary, data from the transthoracic examination will often be essential and integrated into the final clinical interpretation

TEE is particularly useful for evaluation of suspected aortic dissection, prosthetic heart valves (especially mitral), source of cardiac emboli, valvular vegetation, detection of intracardiac shunt, and unexplained hypotension. This modality allows better visualization of the heart in general (especially the posterior structures), owing to the proximity of the probe and favorable acoustic transmission.[7] However, TEE also has its limitations. For several areas of the heart and great vessels, TEE may provide limited images. For example, the view of the left ventricular (LV) apex will often be foreshortened with TEE, and an apical LV clot could potentially be missed. Because of interposition of the left mainstem bronchus, the superior portion of the ascending aorta is another important area that may not be well visualized with TEE. With TEE, transducer position and angulation are constrained by the relative positions of the esophagus and heart. This "fixed" relationship between the probe position and the heart will often lead to inability to align the Doppler beam parallel to flow of interest (for example to evaluate the jet of aortic stenosis or tricuspid regurgitation [TR] velocity). In addition, standard anatomic measurements are often more difficult to obtain with TEE due to the 2D image planes.

Assessment of LV Systolic Function

The evaluation of LV performance by echocardiography is often of paramount importance in the ICU. Accurate and timely assessment of systolic function should be an integral part of the medical management of the hemodynamically unstable critically ill patient. Global assessment of LV contractility includes the determination of ejection fraction (EF), circumferential fiber shortening, and cardiac output (CO). The simplest quantitative approach is to measure the LV end-diastolic dimension and LV end-systolic dimension for determination of the fractional shortening (FS) percentage. FS is related directly to EF, and a normal FS is 30 to 42%.[7]

$$\text{FS} = \frac{\text{end-diastolic dimension} - \text{end-systolic dimension}}{\text{end-diastolic dimension}}$$

It is important to note that in the setting of regional wall motion abnormalities, FS may underestimate or overestimate global ventricular function and must be interpreted in light of what is seen in all of the 2D imaging planes of the ventricle.[14] Global systolic ventricular function can also be quantitatively assessed by fractional area change (normal value is from 36 to 64%) and EF calculation (normal value is 55 to 75%).[15]

$$\text{Fractional area change} = \frac{\textit{end-diastolic area} - \textit{end-systolic area}}{\textit{end-diastolic area}}$$

$$\text{EF} = \frac{\text{end-diastolic volume} - \text{end-systolic volume}}{\text{end-diastolic volume}}$$

These measurements require a good image quality, as endocardial border contours need to be traced. Machine-integrated software will compute the data and provide volumes, areas, and the resultant EF. In patients with regional wall motion abnormalities, more precise measures of stroke volume (SV) can be made by approximating ventricular volumes as a stack of elliptical discs on biplane imaging (modified Simpson method).[7,15] In the critical care setting, endocardial border definition may potentially be suboptimal because of technically limited TTE and poor imaging quality.[12,16,17] In these cases, the global ventricular function will often be qualitatively assessed by visual inspection alone. This method has been found to be very reliable when used by experienced clinicians.[18] Real-time visualization of the kinetics and size of the cardiac cavities by an experienced critical care intensivist with sufficient echocardiographic background will allow an immediate functional diagnosis.

Analysis of regional wall motion includes a numerical scoring system in order to describe the movement of the different regions of the LV and right ventricle (RV): 1 = normokinesia, 2 = hypokinesia, 3 = akinesia, 4 = dyskinesia, 5 = aneurysmal.[15] Visualized from the short-axis view of the LV, a

complete overview of myocardial areas perfused by the three major coronary arteries can be obtained (Fig 1). In technically difficult transthoracic examinations with poorly visualized endocardium, the use of harmonic imaging and contrast, if needed, can dramatically improve endocardial border visualizations and subsequent evaluation of global systolic function (as discussed later in this review). For the remaining minority of technically challenging cases with suboptimal transthoracic imaging, performance of TEE will allow for a more precise evaluation of ventricular function.

LV Failure in the ICU

In the critically ill patient with unexplained hemodynamic instability, determination of cardiac function is an integral part of the medical management. Echocardiography is of the utmost importance to evaluate cardiac function in this setting, as clinical examination and invasive hemodynamic monitoring are unreliable and often misleading. In a study by Fontes et al,[19] who compared the pulmonary artery catheter (PAC) with TEE, the overall predictive probability for conventional clinical and hemodynamic assessment of normal ventricular function was 98%, whereas for abnormal ventricular function (EF $< 40\%$) it was 0%. In this study, TEE was used as the "gold standard" against which the PAC data were compared. It should be kept in mind that despite the fact that TEE is an excellent tool for measurement of function and volume, it does have some limitations for assessment of ventricular output and filling pressures. Several other studies[20–22] have demonstrated the superiority of TEE when compared with pulmonary artery catheterization for assessment of LV function in the ICU.

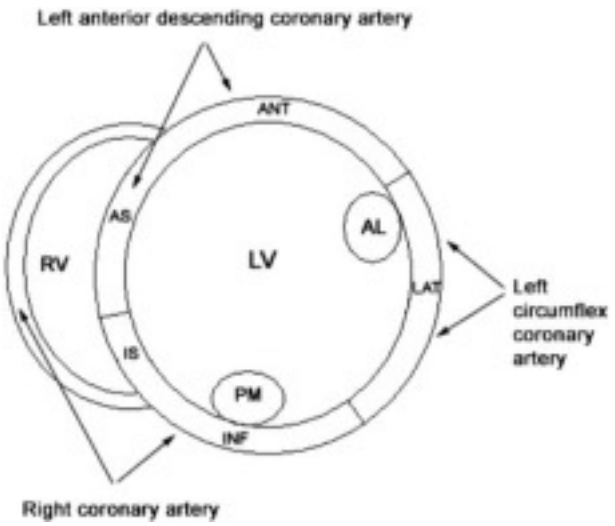

FIGURE 1. Transthoracic short-axis echocardiographic view of the LV and RV at the mid-papillary muscle level. In this tomographic view of the heart, areas of myocardium and papillary muscles (PM = posteromedial; AL = anterolateral) supplied by all three major coronary arteries are represented. ANT = anterior; AS = anteroseptal; IS = inferoseptal; INF = inferior; LAT = lateral.

Significant LV dysfunction is common in critically ill patients. Patients with unexplained hemodynamic instability should have ventricular function determined by echocardiography, as this information is particularly important for adequate therapeutic and resuscitative measures. TTE will provide adequate information of LV function in the majority of ICU patients ($> 80\%$). In a study by McLean[23] on the use of TEE in the ICU, the most common reason to request a TEE was for assessment of LV function (42% of patients). In the majority of patients, LV function was adequately assessed by TTE prior to the TEE. In a study by Vignon et al,[17] TTE allowed adequate evaluation of global LV function in 77% of patients receiving mechanical ventilation in the ICU. In a study by Bruch et al,[24] the most common indication for an echocardiographic study was hemodynamic instability (67% of patients). Of these hemodynamically unstable patients, 26% were found to have significant LV dysfunction (EF $< 30\%$). Despite the fact that TEE may often be needed for other indications, TTE continues to be an excellent diagnostic tool for assessment of LV function in the ICU (Fig 2).

Sepsis-Related Cardiomyopathy

Classically, septic shock has been considered a "hyperdynamic" state in which the CO is either normal or increased. However, echocardiographic studies done in septic patients have suggested that the term *hyperdynamic* is not appropriate to describe a setting in which the heart is in fact very often "hypodynamic."[25,26] Parker et al[27] were the first to describe LV hypokinesis in septic shock, in which patients with severely depressed LV EF (LVEF) in whom an adequate LV stroke output could be maintained through acute LV dilatation.[28] Jardin et al[25] studied 90 patients with septic shock with daily bedside assessment of LV volume and LVEF using TTE, and observed that LVEF was significantly depressed in all patients, resulting in severe reduction in LV SV. Thirty-four patients (38%) were weaned from hemodynamic support; these patients demonstrated a significant improvement in LVEF and ultimately recovered. In the remaining 56 nonsurviving patients (62%), the degree of LV dysfunction was somewhat less than in survivors but did not improve over time. Thus, the severity of the LVEF reduction did not predict worse outcome in individ-

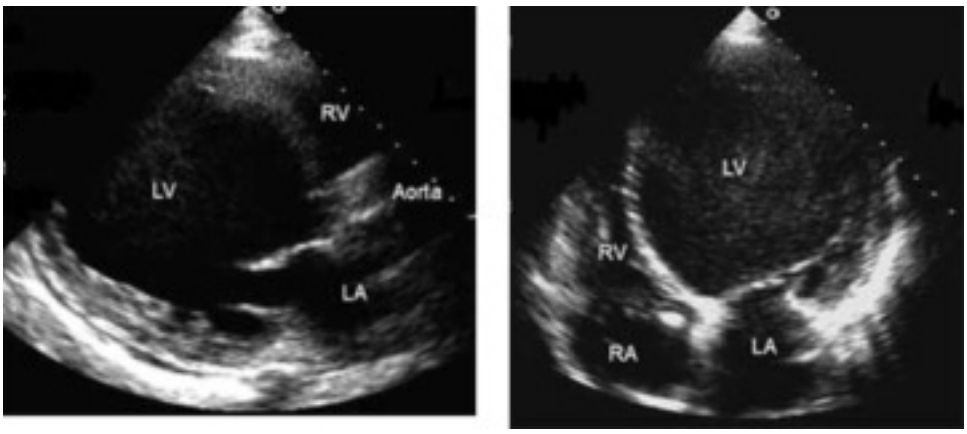

FIGURE 2. Dilated cardiomyopathy. Transthoracic examination of a severely dilated LV in the parasternal long-axis (*left*) and apical four-chamber (*right*) views in a 65-year-old patient who presented with flash pulmonary edema and who was later found to have severe diffuse coronary artery disease.

ual patients; in fact, patients who survived the episode of septic shock had a lower EF than those who did not. This paradoxical relation between the degree of LV dysfunction and the likelihood of recovery has been described by others.[25,27,29,30] However, in patients who survive, LV dilatation and systolic dysfunction are reversible. LVEF fraction might not be a reliable index of LV systolic function in patients with early septic shock, since this is a state of low systemic vascular resistance that unloads the LV.[25] Therefore, the finding of what appears to be a normal or supranormal EF in early sepsis might be misleading, and these patients are potentially at risk of having a marked LVEF reduction if their afterload is increased by the use of vasopressor agents.

In the septic patient, bedside echocardiography is valuable for identification of the cause of hemodynamic instability (which may be of hypovolemic, cardiogenic, or distributive origin) and for the subsequent optimization of therapy (*ie*, fluid administration, inotropic or vasoconstrictor agent infusion, or various combinations of the above).[31] The ability to perform repeat bedside examination is vital in assessing the adequacy and efficacy of therapy.[31]

LV Diastolic Function

In the ICU, diastolic dysfunction may be suspected in the setting of elevated ventricular filling pressure (pulmonary capillary wedge pressure [PCWP]) with a normal or supranormal EF.[7] Evaluation of the diastolic properties of the ventricle have been done mostly using Doppler echocardiographic mitral inflow and pulmonary venous flow patterns. These filling parameters are related to intrinsic diastolic myocardial properties and are influenced by many different factors, particularly left atrial (LA) pressure, heart rate, ischemia, ventricular hypertrophy, and valvular pathologies. Only modest correlation has been found between Doppler indexes of diastolic function and invasive parameters.[32,33] Although mitral and pulmonary vein flow patterns may be useful in the diagnosis of abnormalities of myocardial relaxation, their use as a primary end point for therapy or for specific diagnosis must be done with caution in the critically ill patients

RV Function and Ventricular Interaction

RV dysfunction is common, and its role is underestimated in critically ill patients.[34–36] Based on an echocardiographic definition,[37] massive pulmonary embolism (PE) and ARDS are the two main causes of acute cor pulmonale (ACP) in adults.[38] In the critical care setting, RV function is also altered by other conditions increasing RV afterload, including high levels of positive end-expiratory pressure or increased pulmonary vascular resistance (from vascular, cardiac, metabolic, or pulmonary causes). Depressed RV systolic function is also associated with RV infarction, most commonly in the setting of inferior myocardial infarction. Acute sickle-cell crisis, air or fat embolism, myocardial contusion, and sepsis are other causes of acute RV dysfunction.

Adequate assessment of RV function is very important in hemodynamically unstable critically ill patients, specifically in patients with massive PE and ARDS, as the presence of significant RV dysfunction with these two pathologies may alter therapy (fluid loading, vasopressors, thrombolytics) and is of prognostic value.[38,39] Echocardiographic examination of the RV requires primarily an assessment of the size and kinetics of the cavity and septum.[37,40] The RV appears normally relatively flat. As it dilates, the

apical region of the RV becomes more rounded (Fig 3). In the short axis, the RV (usually having a crescentic shape) becomes oval because of septal displacement and bulging of the RV free wall[7] (Fig 3). RV size and function are generally evaluated by visual comparison with the LV. RV diastolic dimensions can be obtained by measuring RV end-diastolic area in the long axis, from an apical four-chamber view, by either TTE or TEE. Because pericardial constraint necessarily results in LV restriction when the RV acutely dilates (*ie*, ventricular interaction), one of the best way to quantify RV dilatation is to measure the ratio between the RV end-diastolic area and LV end-diastolic area (LVEDA), which circumvents individual variations in cardiac size.[37,40] Moderate RV dilatation usually corresponds to a diastolic ventricular ratio > 0.6, and severe RV dilatation to a ratio ≥ 1.[37,40] RV diastolic enlargement is usually associated with right atrial (RA) dilatation, inferior vena cava dilatation, and TR. When pressure in the RA exceeds that in the LA, the foramen ovale may open. Pressure and volume overload of the RV can lead to distortion of LV geometry and abnormal motion of the interventricular septum (IVS). With

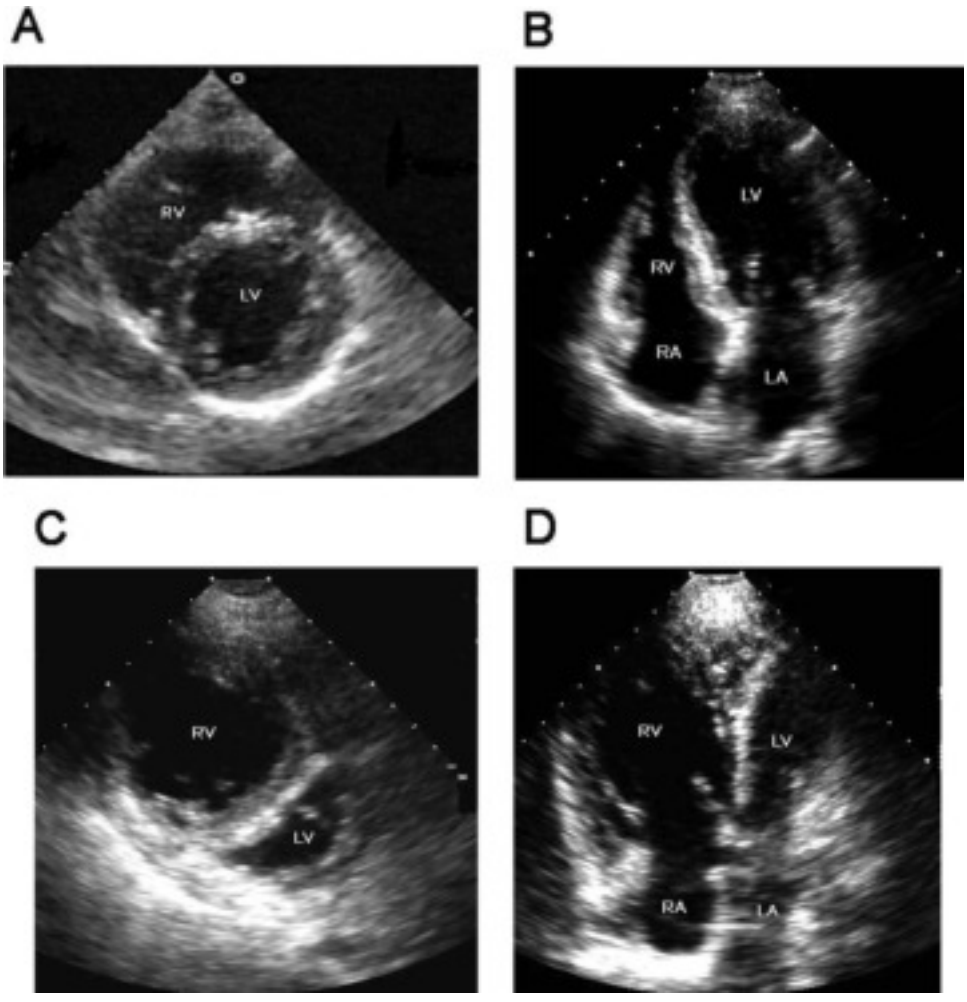

FIGURE 3. Severe RV failure and dilatation. *Top left*, *A*: Normal transthoracic, parasternal, short-axis view of the LV and RV at the midpapillary muscle level. *Top right*, *B*: Normal, transthoracic, apical, four-chamber view of the LV and RV in a normal heart depicting the relationship between the LV and RV, with the LV being normally larger than the RV, and the IVS bulging slightly toward the RV. *Bottom left*, *C*: Transthoracic, parasternal, short-axis view of the LV and RV in a patient with severe RV failure and dilatation. The RV cavity is seen to be much larger than the LV cavity. Because of the high volume and pressure in the RV, the IVS is bulging toward the left. This gives the LV a characteristic D-shaped appearance. *Bottom right*, *D*: Transthoracic, apical, four-chamber view of the same patient showing again the inverse relationship between the LV and RV sizes. The RV dilatation can take place only if associated with a proportional reduction in LV diastolic dimension ("ventricular interaction"). This reduction in LV diastolic dimension will significantly impair LV relaxation and change the pressure-volume relationship of the left heart chambers.

conditions of high strain imposed on the RV (volume and/or pressure overload), the IVS flattens and the LV appears "D shaped"[3,37] (Fig 3). This "paradoxical" septum motion will also be seen at the interatrial level.

Because the two ventricles are enclosed within the relatively stiff pericardium, the sum of the diastolic ventricular dimensions has to remain constant.[41] Thus, any acute RV or LV dilatation can take place only if it is associated with an acute and proportional reduction in LV or RV diastolic dimension, respectively (*ie*, ventricular interaction). With acute RV dilatation, septal displacement will impair LV relaxation, and the opposite will occur with acute LV dilatation. In these situations, the pressure-volume relationship of left and right heart chambers are altered and information obtained from a PAC could be misleading (*ie*, high filling pressures reading despite a state of normovolemia or hypovolemia).

PE

Hemodynamic instability from ACP as a consequence of massive PE is not an uncommon occurrence in the ICU patient. Contrast pulmonary angiography has long been the "gold standard" for the diagnosis of PE. However, it is an invasive procedure and may carry a risk of major complications in patient with circulatory failure.[42] Enhanced-contrast helical CT is an accurate and noninvasive measure and is now commonly used as an alternative to angiography for the diagnosis of PE. However, both of these diagnostic tests require transportation of the potentially unstable patient outside of the ICU, and this may carry significant risks on its own. Echocardiography is well suited in this population of patients, as it can be done within minutes directly at the bedside. The diagnosis of ACP at the bedside with TTE has been shown to have a good positive predictive value for massive PE.[43,44] TTE can detect acute RV dilatation and dysfunction following large PE. The finding of RV dilatation and dysfunction is, however, not specific for PE, as it may be observed with a variety of other conditions increasing RV strain as described above. In a study by McConnell et al,[45] patients with acute PE were found to have a distinct regional pattern of RV dysfunction with akinesia of the mid-free wall but normal motion at the apex on TTE. This was in contrast to patients with primary pulmonary hypertension who had abnormal wall motion in all regions. 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, with a positive predictive value of 71% and a negative predictive value of 96%. The presence of regional RV dysfunction in which the apex is spared should thus raise the level of clinical suspicion for the diagnosis of acute PE (vs the other possible causes of RV dysfunction described above).

Central pulmonary emboli are present in half of patients with PE and ACP on TTE.[8] TTE can, however, not usually visualize emboli lodged in the proximal pulmonary arteries.[8] As other clinical conditions may produce ACP in the ICU, a better visualization of the pulmonary arteries is needed to achieve a higher accuracy for the diagnosis of PE. That can be achieved with TEE. TEE has a good sensitivity for detecting emboli that are lodged in the main and right pulmonary arteries but is limited for the detection of more distal or left pulmonary emboli.[8,46,47] If an embolus is visualized, the diagnosis is substantiated. However, if the study result is negative in a patient with a high suspicion of PE, the TEE must be followed by a more definitive test such as angiography or helical CT to confirm the diagnosis. In those patients with a high clinical suspicion of PE in whom no emboli are visualized on TEE, the potential "nonthrombotic" causes of PE such as air and fat emboli should be kept in mind.

The demonstration of ACP by echocardiography has important prognostic and therapeutic implications.[48,49] The presence of ACP with PE is associated with an increased mortality rate.[39] It has been suggested that the presence of ACP in the setting of PE is an indication for acute thrombolysis; however, there is no clear consensus on this issue.[50,51]

Assessment of CO

Measurement of CO remains a cornerstone in the hemodynamic assessment of the critically ill patient. Its determination can classify patients in high- or low-CO states, thus indicating categories of circulatory failure and subsequently help in providing the appropriate treatment. Thermodilution, utilizing a method based on the Fick principle, is considered the "gold standard" to determine CO at the bedside. This technique requires placement of a PAC, and, although a useful technique, carries considerable risks and a potential for inaccuracies. Unreliable values are particularly common in the ICU, where extremes of hemodynamic and respiratory conditions are often found with either very low or very high CO and frequent TR related to high pulmonary artery pressure (PAP). Several methods for determining CO have been described using both 2D and Doppler echocardiography. With this technique, SV and CO can be determined directly by combining Doppler-derived instantaneous blood flow velocity through a conduit with the cross-sectional area (CSA) of the conduit. Blood flow can be calculated through vari-

ous cardiac structures, including the pulmonary valve,[52] the mitral valve,[53,54] and the aortic valve.[55–58] In the absence of intracardiac shunts, blood flow through these structures should be the same (continuity equation).[59] Of these methods, the one using the LV outflow tract (LVOT) and aortic valve as the conduit is probably the most reliable and most commonly used, with an excellent agreement with thermodilution in most situations.[55–58] The LV SV is obtained by measuring the CSA of the LVOT (area [centimeters squared] = (LVOT diameter [centimeters]^2) × [π/4], assuming that just below the aortic annulus, the LVOT is circular) multiplied by the transaortic flow velocity time integral derived from a spectral Doppler tracing. The SV thus obtained is then multiplied by the heart rate to give the CO: CO = CSA × velocity time integral × heart rate. With the transthoracic approach, the LVOT diameter is usually obtained from the parasternal long-axis view, just below the insertion of the aortic valve leaflets. The Doppler interrogation is then performed through the aortic valve from the apical view. With the transesophageal approach, the LVOT diameter is usually obtained from the five-chamber view of the LV. The transgastric view is usually used to obtained an apical long axis-view of the aortic valve through which Doppler interrogation is then performed.[60] Using TTE, McLean et al[61] demonstrated an excellent correlation (r = 0.94) between CO determined by the LVOT Doppler method and the thermodilution method in a population of critically ill patient. Other studies[55] have shown similar results. In a study by Feinberg et al,[58] CO, as determined by TEE Doppler imaging, was obtainable in 88% of a population of 33 critically ill patients with good correlation (r = 0.91) with the thermodilution method. Descorps-Declère et al[60] also showed that transgastric pulsed Doppler measurement across the LVOT with TEE to be a clinically acceptable method for CO measurement in the critically ill (r = 0.975, when compared to the thermodilution method).

Assessment of Filling Pressures and Volume Status

Assessment of LV preload and intravascular volume status is an integral component in the management of critically ill patients. Invasive pressure measurements to assess LV filling are commonly used at the bedside to make inferences regarding LV preload. These pressure measurements, however, correlate weakly with ventricular volume.[62] This is largely due to the fact that in the critically ill patient, a number of factors alter ventricular compliance, affecting the relationship between pressure and volume.[14,63–67] The identification of preload as LV end-diastolic volume (LVEDV) is a very important issue. In 1895, Frank[68] suggested that ventricular preload is stretch (or filling volume) and not pressure. Echocardiography can be of great value in assessing the preload status of the ICU patient by analyzing a number of parameters with both 2D (LVEDV and LVEDA) and with Doppler interrogation (transmitral diastolic filling patterns, pulmonary venous flow, and respiratory variation in aortic blood velocity).

2-D Imaging: Echocardiography has been validated for LV volume measurements.[15] Subjective assessment of LV volume by looking at the size of the LV cavity in the short- and long-axis views is often adequate to guide fluid volume therapy at the extreme ends of cardiac filling and function. But more precise, quantitative values are desirable and can be obtained by using endocardial border tracing (as described earlier). Normal LVEDVs as determined by echocardiography are from 80 to 130 mL,[15] with normal LVEDV index from 55 to 65 mL/m^2.[15] LVEDA measured in the left parasternal, short-axis view at the level of the midpapillary muscle is commonly used to estimate volume status. Range for values of normal LVEDA in the short axis are from 9.5 to 22 cm^2.[15] 2D TTE evaluation of ventricular dimensions has been found to be useful in assessing preload and optimizing therapy in the critically ill patient.[25,69–71] However, image quality may be suboptimal precluding adequate visualization of the endocardial border. This potential limitation of TTE has partly been circumvented in recent years with the advent of harmonic imaging and contrast echocardiography (as discussed later). In cases in which endocardial border visualization remains suboptimal, TEE is the modality of choice. With TEE, LV volume can be rapidly estimated by subjective assessment of the LV size. Quantitatively, it is most often estimated by determining the LV CSA at the end of diastole, most commonly using the transgastric short-axis view at the level of the midpapillary muscle. End-diastolic area measured with TEE correlates with LV volume determined by radionuclide studies.[69]

Systolic obliteration of LV CSA accompanies decreased end-diastolic area and is considered to be a sign of severe hypovolemia (Fig 4). Although a small end-diastolic area generally indicates hypovolemia, a large end-diastolic area may or may not indicate adequate preload in the patient with LV dysfunction. This is analogous to LV end-diastolic pressure measurements in which an LV end-diastolic pressure of 15 to 20 mm Hg, which usually represents normal to hypervolemia in a patient with normal LV function,

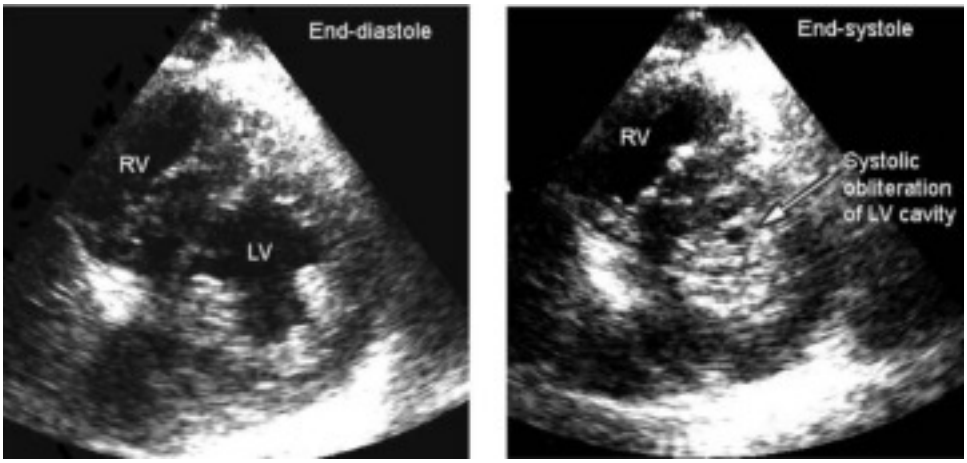

FIGURE 4. Systolic obliteration of the LV in a patient with severe LV hypertrophy and dehydration. This transthoracic, parasternal, short-axis view shows the LV at end-diastole (*left*) and at end-systole (*right*). Nearly complete obliteration of the LV cavity is seen at end-systole. Systolic obliteration of the CSA accompanies decreased end-diastolic area and is considered to be a sign of severe hypovolemia. In this case, the patient presented with hypotension and was found to be severely dehydrated because of a viral gastroenteritis.

can represent inadequate preload for the patient with LV dysfunction.[72] Also, in conditions presenting with a low systemic vascular resistance, such as early sepsis, LV emptying will be improved because of the lowered afterload. In these situations, it may be difficult to differentiate hypovolemia from low systemic vascular resistance by echocardiography alone, as both conditions will lead to a decreased end-diastolic area. Knowledge of the LVEDV or absolute preload does not necessarily allow for accurate prediction of the hemodynamic response to alterations in preload.[73,74] Tousignant et al[75] investigated the relation between LV SV and LVEDA in a population of ICU patients and found only a modest correlation ($r = 0.60$) between single-point estimates of LVEDA and response to fluid loading. Based on the assumption that changes in end-diastolic area occur because of changes in LV volume, the determination of this area and of its subsequent degree of variation after a fluid challenge could help better assess the degree of preload responsiveness (*ie*, fluid challenge leads to an increase in CO) or preload in a particular patient. Studies[76] have demonstrated that changes in EDA measured by TEE using endocardial border tracing are closely related to changes in CO and are superior to PCWP in predicting the ventricular preload associated with maximum CO.

In certain circumstances, patients may have hemodynamic instability with a low cardiac index and despite being severely hypovolemic, have a high PCWP. This hemodynamic profile is seen when dynamic LV obstruction is present. It is very important that this entity be recognized early and that the pathophysiologic process be well understood, as inadequate management of this condition can rapidly lead to worsening of the hemodynamic status and death. Dynamic obstruction of the LV can presents itself in different forms. One of these is dynamic LVOT obstruction (DLVOTO). Although DLVOTO is often seen in association with asymmetric septal hypertrophy, it can also occur in other situations.[77,78] DLVOTO is thought to be caused by the "Venturi effect," which results when an excessive acceleration of blood through a conduit produces a fall in pressure. In the LVOT, such a drop in pressure leads to a suction phenomenon that draws the anterior mitral leaflet and chordae inwards toward the IVS.[79] Patients with a small, hypertrophied LV (typically seen in elderly patient with chronic hypertension) can acquire midventricular obstruction, because of hyperdynamic systolic obliteration of the LV cavity, if they are submitted to a reduced afterload, dehydration, or significant cathecholaminergic stimulation.[80] Dynamic LV obstruction has also been very well described in patients with acute myocardial infarction, mostly in association with apical infarction.[78,81,82] In a study by Chenzbraun[82] in ICU patients, four patients with hemodynamic instability were found to have a small hyperdynamic ventricle on TEE. Of these four patients, three had PCWPs > 20 mm Hg. A study by Poelaert et al,[20] evaluating the diagnostic value of TEE vs the PAC, showed that PAC failed to diagnose the presence of hypovolemia in 44% of patients in which TEE showed systolic obliteration of the LV cavity, supporting a diagnosis of hypovolemia.

Circulating volume status can also be assessed on 2D echocardiography by indirectly estimating RA pressure. This is often done by assessing the diameter and change in caliber with inspiration of the

inferior vena cava (Fig 5). This method has been shown to discriminate reliably between RA pressures < 10 mm Hg or > 10 mm Hg.[83] A dilated vena cava (> 20 mm) without the normal inspiratory decrease in caliber ($> 50\%$ on gentle sniffing) usually indicates elevated RA pressure. However, it is important to note that in patients receiving mechanical ventilation, this measure is less specific because of a high prevalence of inferior vena cava dilatation.[84,85] A small vena cava will reliably exclude elevated RA pressure in these patients.[84,85] It is important to emphasize that RA pressure should not be taken as a direct correlate of LV preload as, in some clinical situations (*eg*, PE, RV infarction), a high RA pressure can be seen in the presence of low LVEDV.

Doppler Flow Patterns: Information obtained by analysis of the Doppler signal at the level of the mitral valve and pulmonary vein offers additional information about the preload of a critically ill patient.[86,87] These Doppler profiles can be obtained by either TTE or TEE. Transmitral parameters that have been studied include the ratio of early to late transmitral diastolic filling (E/A ratio), isovolumetric relaxation time, and the rate of deceleration of early diastolic inflow (deceleration time).[7]

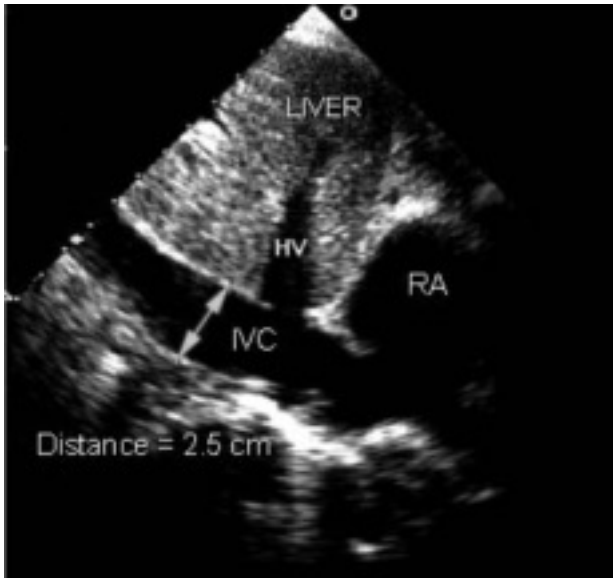

FIGURE 5. Indirect assessment of circulating volume status on 2D echocardiography by assessing the diameter and change in caliber with inspiration of the inferior vena cava (IVC). This method has been shown to discriminate reliably between RA pressures < 10 mm Hg or > 10 mm Hg. A dilated vena cava (> 20 mm) without the normal inspiratory decrease in caliber ($> 50\%$ on gentle sniffing) usually indicates elevated RA pressure. A small vena cava will reliably exclude elevated RA pressure in these patients. In the above case, the inferior vena cava was dilated at 2.5 cm with minimal respiratory variation in a spontaneously breathing patient. The RA pressure was thus estimated to be approximately 10 to 15 mm Hg. Images were obtained in the subcostal view. HV = hepatic veins.

A decrease in preload causes a significant reduction in the E wave (early filling flow wave) velocity at the mitral level in conjunction with a decrease of the S wave (systolic flow wave) in the pulmonary vein. In clinical practice, the E/A ratio is easy to assess; the normal value of this ratio being approximately 1.[2,7] In conjunction with normal contractility of the LV, a low E/A ratio is usually a characteristic sign of inadequate preload.[88] Pulmonary venous flow can also be used to assess LA pressure. A normal pulmonary venous flow pattern, demonstrating a predominance of flow during systole (S phase) compared to early diastole (D phase), will usually indicate a LA pressure < 8 mm Hg, whereas the opposite predominance of flow will usually indicate an elevation in LA pressure (in the absence of significant mitral regurgitation [MR]).[7] It is important to keep in mind that both transmitral and pulmonary vein Doppler patterns are strongly dependent on intrinsic and external factors and are not purely affected by the loading conditions of the LV. The interpretation of Doppler parameters should therefore be done in conjunction with a global analysis of cardiac function and other available hemodynamic or anatomic variables.

Positive-pressure ventilation alters SV by transiently increasing intrathoracic pressure and thereby decreasing preload. This phasic variation in SV results in a cyclic fluctuation in arterial pressure.[73,89] The magnitude of respiratory variation in aortic blood velocity (as recorded echocardiographically by pulsed-wave Doppler at the level of the aortic annulus) is a dynamic parameter that is superior to static measurement of LVEDA (or LVEDV) to predict fluid responsiveness in critically ill patients.[71,90] Feissel et al[91] demonstrated that when patients in septic shock experienced a magnitude of respiratory variation of peak aortic velocity $> 12\%$, infusion of 500 mL of fluid increased SV and CO by $> 15\%$, while decreasing proportionately the magnitude of the respiratory variation of peak aortic velocities. Although practical and reliable, use of this echocardiographic dynamic parameter to assess volemic status can be applied only to patients who are receiving mechanical ventilation and who are perfectly adapted ("synchronous") to the ventilator and have no cardiac arrhythmia.

Assessment of PAP

Pulmonary hypertension is common in the critically ill patient and is a manifestation of various pulmonary, cardiac, and systemic processes. It is characterized hemodynamically as a systolic pulmonary pressure > 35 mm Hg, diastolic pressure > 15 mm Hg, and mean pulmonary pressure > 25 mm Hg.[59] A number of echocardiographic methods, both

with TTE and TEE, have been validated for the noninvasive estimation of PAP.[59,92] These can be of great help in the ICU setting. Systolic and diastolic PAPs are determined from TR and pulmonary regurgitation (PR) velocities, respectively (some degree of regurgitation is essential to be able to obtain a Doppler signal and subsequently determine PAP). TR is present in > 75% of the normal adult population[59] and in approximately 90% of critically ill patients.[93] Peak TR velocity, usually obtained by continuous-wave Doppler from the RV inflow or the apical four-chamber view position, reflects the pressure difference during systole between the RV and the RA.[94,95] Peak systolic PAP is thus determined from the peak TR Doppler velocity using the modified Bernoulli equation[96]: $p = 4 \times (\text{peak TR velocity})^2$. To the peak systolic pressure gradient between RV and RA is added the estimated RA pressure to obtain the peak RV systolic pressure. In the absence of pulmonic stenosis or RV outflow obstruction, peak RV systolic pressure is equal to systolic PAP. Echocardiography can also determine diastolic PAP by applying the modified Bernoulli equation using the regurgitant Doppler velocity of the pulmonary valve to obtain the gradient between the pulmonary artery and the RV at end diastole. The estimated RA pressure is added to this pressure gradient to obtain end-diastolic PAP: $4 \times (\text{peak PR velocity})^2 + \text{estimated RA pressure}$. TR and PR are present at the same time in > 85% of subjects[97] and approximately 70% of critically ill patients have an adequate Doppler signal for calculation of PAPs.[98]

Assessment of Valvular Function and Integrity

Attention has been drawn to the limitations of the physical examination for the detection of cardiovascular abnormalities.[99,100] This problem is enhanced in acutely ill patients in the ICU, and many cardiovascular abnormalities may be concurrent with noncardiac illness without being clinically suspected.[101] Significant valvular abnormalities can be present in the critically ill patient without being clinically recognized.[101] Even in the presence of invasive monitoring, significant valvular pathologies may be missed. Precise evaluation of the valvular apparatus may thus often be warranted in the ICU. The most common indications for bedside echocardiography for evaluation of valvular apparatus in this population are for suspected endocarditis,[11,23] for evaluation of acute aortic or mitral valve regurgitation,[102,103] and for evaluation of prosthetic valve dysfunction.[16] Echocardiography is uniquely suited to the evaluation of valvular heart disease because of its ability to provide information regarding the etiology and severity of valvular lesions. In the intensive care setting, TTE can provide valuable information concerning valvular integrity and function,[16] but it may be suboptimal and not sensitive enough in cases of suspected endocarditis, dysfunctional mitral valve, and for the evaluation of most prosthetic valves. Thus, a TEE may often be warranted for optimal results.

Valvular Regurgitation and Prosthetic Valve Dysfunction

In a patient with unexplained hemodynamic instability and a grossly normal TTE examination, performance of a subsequent TEE is important to rule out the presence of significant undetected valvular pathologies. Common valvular pathologies that can be missed are MR and prosthetic valve dysfunction. In some situations, TTE may provide better imaging than TEE for evaluation of anterior structure such as the aortic valve (native or prosthetic) and for Doppler measurements. However, TEE has clearly been shown to be superior to TTE for evaluation of mitral valve pathologies (both native and prosthetic). In a study by Alam[16] in ICU patients, TTE was shown to either miss or severely underestimate the severity of regurgitation of St. Jude and bioprosthetic valves in the mitral but not in aortic position compared to TEE. With acute severe MR, the diagnosis may clinically be difficult, as the murmur is often of short duration and of low intensity (because of rapid pressure equalization between the LV and the relatively noncompliant LA). By TTE, the size of the regurgitant jet in acute MR may appear small and lead to underestimation of the severity.[104] Because of its close anatomic proximity, TEE will provide a much more precise evaluation of the degree of MR and will also most of the time yield a cause for the MR. The diagnosis of acute MR represents a medical emergency that may necessitate urgent surgery, and so the threshold to perform a TEE when this entity is suspected should be low.[11,12,102] Also, several investigators[105–108] have confirmed the superior accuracy, sensitivity, and reliability of TEE over TTE for dysfunction of mitral prosthesis, where ultrasonic shadowing of the LA often occurs with the standard transthoracic studies.

Traumatic Valvular Injuries

Traumatic valvular injuries associated with myocardial injury may present as acute regurgitation. Bedside exclusion of major trauma to the aorta, valves, and myocardium is important in patients who have sustained both blunt and penetrating chest trauma.[109,110] The aortic valve is most frequently injured, followed by the mitral and tricuspid valves.[111] Valvular dysfunction is most commonly due to a leaflet tear or papillary muscle or chordal rupture.[111] In trauma patients, TEE is the bedside imaging modality of choice to detect these pathologies.[109,110] In a study by Chirillo et al,[112] which

assessed the usefulness of TTE and TEE in recognition and management of cardiovascular injuries after blunt chest trauma, TTE provided suboptimal imaging in 62% of patients, and the poor quality of images obtained was the main cause for the low sensitivity of TTE compared with TEE.

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