Advanced Point-of-Care Cardiac Ultrasound Examination

Doppler Applications, Valvular Assessment, and Advanced Right Heart Examination

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ABSTRACT

Basic point-of-care cardiac ultrasound involves assessment of left ventricular systolic function, right ventricular size and systolic function, intravascular volume status, and the pericardium. These simple tools are sufficient to aid in the hemodynamic management of most acutely ill patients; more complex patients may benefit from the use of advanced echocardiographic techniques. This paper describes the use of ultrasound in the advanced evaluation of the right heart, in the assessment of valvular function, and touches on several advanced Doppler applications.

The basic applications of point-of-care ultrasound (US) from a cardiac perspective include assessment of left ventricular (LV) systolic function, right ventricular (RV) systolic function, intravascular volume status, and the pericardium [1]. In the management of complex patients, more hemodynamic data is often required; fortunately, there is a wide assortment of more advanced US techniques that can provide this information quickly and relatively easily.

Use of inferior vena cava (IVC) diameter and respiratory variation to assess intravascular volume status is well described and clinically useful [2]. Many patients, however, fall into a "gray zone" where results are difficult to interpret; mechanically ventilated patients making spontaneous respiratory efforts are a good example. Techniques using Doppler US may help in these situations and can be used to estimate stroke volume (SV) and cardiac output (CO) in the clinical scenarios where such information is useful.

Two potential causes of unexplained shock that are notoriously difficult to diagnose at the bedside are acute valvular dysfunction and right heart failure. Point-of-care echocardiography is the ideal tool to diagnose or exclude these important clinical entities, and the approach to addressing these conditions is discussed herein.

DOPPLER

Overview

In 1842, the Austrian physicist Christian Doppler described a change in the frequency of a wave for an observer moving relative to its source. This principle has been greatly expanded, and its primary contemporary medical application involves gathering information related to the function of organs [3]. Three specific Doppler techniques will be reviewed here: pulsed wave (PW); color flow (CF); and continuous wave (CW).

In PW Doppler, a small area along the length of the US beam path is selected for interrogation and a pulse of US waves are sent to this "sample volume" and reflected back (Fig. 1B). The US transducer alternates periods of transmission and reception of US waves, analogous to speaking then listening in turn. Knowing the distance to the sample volume and the frequency of the created and reflected sound waves, the US machine can calculate the velocity of the red blood cells within the sample volume [4] and thus record blood flow velocity within discrete regions of the heart and great vessels. The main disadvantage is its inability to accurately measure blood moving at high velocity, as PW is limited by the pulse-repetition frequency. Because the transducer must alternate between periods of transmitting and receiving, it must receive the last echo from a pulse of sound waves before it proceeds to emit the next pulse; thus the frequency of sequential tone bursts is limited, as are the maximal velocities that can be measured [5].

CF Doppler is based on the same principles as PW: An area of interest is selected for interrogation and the same process for determining the velocity of blood flow is applied. In contrast to PW, the blood velocity for every small region within the area of interest is measured, and these velocities are color-coded and displayed on a familiar color scale with red typically indicating flow toward the transducer and blue flow away. Like PW, CF Doppler is limited in terms of the maximal velocity it can process; velocities above this "Nyquist limit" (one-half the pulse-repetition frequency) will result in aliasing, the scrambling of the color scale to shades of green [6].

CW Doppler has the ability to both "speak" and "listen" simultaneously and continuously, recording the velocity of all red blood cells along the path of interrogation rather than within a discrete sampling volume as is the case for PW and CF. As such, the outer contour of the Doppler velocity profile will correspond to the fastest moving blood cell along the line of interrogation. CW is not limited in terms of the maximum velocity it can measure, but it is not able to discern exactly where

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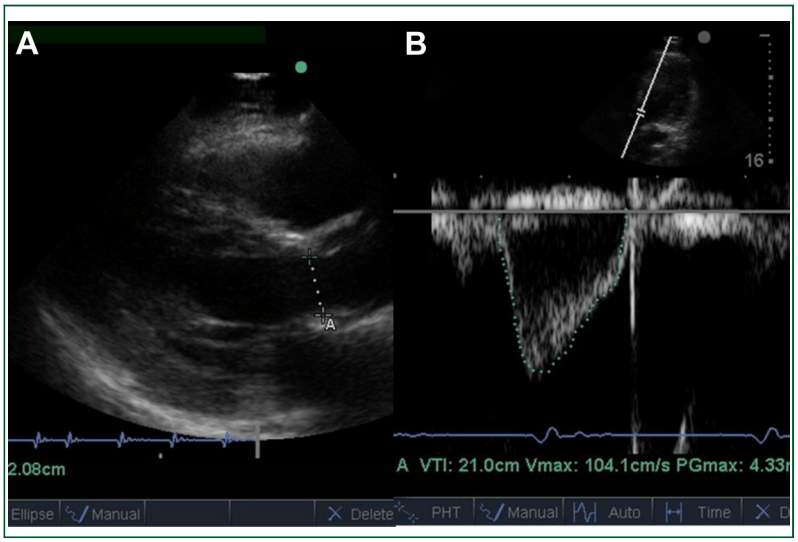

FIGURE 1. Estimating cardiac output using transthoracic echocardiography. (A) Measuring the LVOT diameter from the parasternal long-axis view. (B) Using pulsed wave Doppler to measure the velocity-time integral at the level of the LVOT. LVOT, left ventricular outflow tract.

along the line of interrogation that maximum velocity occurred [4].

Use in estimating SV and CO

There are several techniques described to measure CO by US [4], but all rely on the estimation of SV by PW Doppler. The preferred site for this calculation is the left ventricular outflow tract (LVOT) [7], where SV is the product of the cross-sectional area (CSA) of the LVOT multiplied by the velocity-time integral (VTI) at that same location.

$$SV = CSA \times VTI$$

VTI is the sum of the instantaneous velocities measured at a specific location by PW and is equal to the area under the curve of the Doppler velocity profile. It can be thought of as the distance the average blood cell travels during systole [6].

To calculate CO, the following steps are required (Fig. 1) [8]:

1. Measure the diameter (D) of the LVOT. This is most accurate if measured via the transesophageal route, but it can be done from a surface parasternal long-axis view if the image quality is reasonable. Measurements should be taken in early systole from the junction of the atrioventricular leaflet with the septal endocardium to the junction of the other leaflet with the mitral valve posteriorly, using inner edge to inner edge [7]. The largest of 3 to 5 repeated measurements should be used given the tendency to underestimate the true value.
2. Convert the LVOT diameter into a CSA using the following formula:

$$CSA = D^2 \times 0.785$$

3. Record the velocity at the LVOT using an apical 5-chamber or 3-chamber view. Place the PW sample volume 5 mm above the aortic valve and record a Doppler waveform with a well-defined envelop.
4. Record the VTI by tracing 3 consecutive waveforms and taking the average of the 3 measurements.
5. Calculate the SV using the following formula:

$$SV = CSA \times VTI$$

6. Calculate the CO using the following formula:

$$CO = SV \times \text{heart rate}$$

Whereas calculating CO can be intellectually satisfying, it is important to keep in mind the significant limitations of this technique. There is a large margin of error associated with LVOT diameter measurement, especially when using a transthoracic approach. Variability in VTI occurs from breath-to-breath (especially in hypovolemic patients) and from beat-to-beat in the setting of cardiac arrhythmias.

Use in estimating volume responsiveness

It is possible to glean useful information from the LVOT VTI value alone, a process that eliminates the inaccuracy associated with LVOT diameter measurement. Because the LVOT CSA is constant, variations in LVOT VTI can be assumed to represent changes to the patient's SV [9]; although the absolute value will remain unknown, measuring a change with therapy is often clinically useful.

To evaluate volume responsiveness, the LVOT VTI at baseline can be compared with its value after administration of a fluid bolus; if the VTI increases, it can be assumed that the SV has increased. If the heart rate has remained constant, it can be further assumed that the CO has increased, and further fluid administration should be considered depending on the clinical context. Care should be taken to administer fluid quickly and in a sufficient quantity as to exert an effect that will be measurable—boluses of approximately 500 cc over 15 to 30 min are common in clinical practice and in trials [10]. The same technique can be performed using a passive leg raise in lieu of a fluid bolus [11,12].

A related concept can be used to predict volume responsiveness without actually administering a fluid bolus (or performing a leg raise). Respiratory variation of the LVOT VTI will be exaggerated in the presence of hypovolemia, a phenomenon that is closely related to using the pulse-pressure variation of an arterial line tracing to predict

fluid responsiveness [13]. This technique is performed by generating a PW Doppler tracing of the LVOT then altering the sweep speed such that enough waveforms are visible on screen to span a full respiratory cycle (Fig. 2). At this point, the difference between minimum and maximum LVOT VTI is measured, with a variation of >9% suggestive of a volume-responsive state [14]. The same process can be repeated using LVOT peak velocities in lieu of VTI, with variation of >12% as a threshold [10]. It should be noted that whereas these techniques have been studied in animals [15], in patients after myocardial infarction [16], and in children [17], their applicability to undifferentiated acutely ill patients remains unclear. Additionally, there are several potentially confounding factors including cardiac arrhythmias, the influence of spontaneous respiratory efforts, and the effect of varying tidal volumes for patients who are mechanically ventilated.

Use in assessing for pericardial tamponade physiology

The term "pericardial tamponade" is traditionally used to describe the presence of severe clinical manifestations caused by cardiac compression related to pericardial fluid. Echocardiography is the gold-standard modality for detecting a pericardial effusion, but the diagnosis of tamponade remains primarily a clinical one. Because physical examination findings are nonspecific, US features that can aid in understanding the hemodynamic impact of pericardial fluid are very useful. Tamponade is not an all-or-none phenomenon [18], and thus it is crucial to combine clinical evaluation with echocardiographic findings to arrive at an accurate diagnosis.

Diameter of the inferior vena cava (IVC) If tamponade physiology exists, the IVC should be dilated due to elevated right-sided filling pressures and resistance to venous inflow. Whereas a dilated IVC is very nonspecific, a nondilated IVC effectively rules out a hemodynamically significant effusion [19].

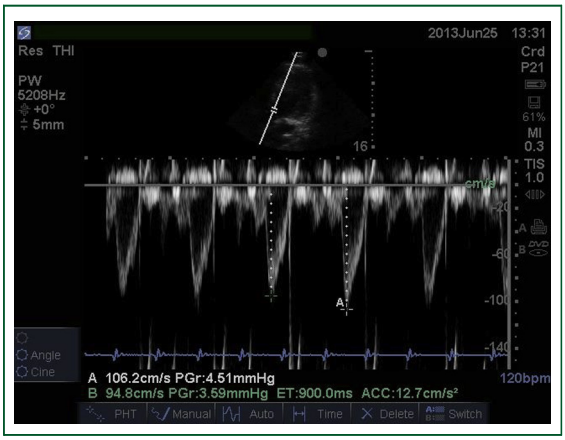

FIGURE 2. The respiratory variation of blood flow at the level of the left ventricular outflow tract using pulsed wave Doppler. Maximal velocities at different phases of the respiratory cycle are compared.

Diastolic collapse of the right atrium or RV As the quantity of pericardial fluid increases, so will the intrapericardial pressure. When this pressure exceeds intracardiac pressure, cardiac collapse will ensue. Because intracardiac pressure is higher in systole, this collapse will typically occur in diastole, affecting the right atrium (RA) first (in early diastole) and then the RV (in late diastole) if intrapericardial pressures continue to rise. This phenomenon can be appreciated if image quality permits, with the subcostal 4-chamber view typically the best window. While the exact sensitivity and specificity of this sign is not known, it appears to be reliable [20,21].

Transvalvular respiratory variation In the presence of tamponade physiology, intracardiac and intrathoracic pressures become dissociated and the phenomenon of ventricular interdependence is exaggerated [22]. This phenomenon causes the respiratory variation of blood flow across any of the 4 cardiac valves to be increased, a phenomenon that is explained in detail elsewhere [23,24]. Exact sensitivities and specificities for this technique are also unknown, as are the exact threshold values for each valve that signify the onset of tamponade physiology.

VALVULAR ASSESSMENT

Overview

Point-of-care sonographers evaluating valvular heart disease must be as skilled as they are humble. A rich understanding of hemodynamics and US physics, a wide scope of Doppler techniques, and a deep respect for pitfalls and artifacts must all be in play when assessing valvular heart disease.

Techniques for valvular assessment

A multitude of echocardiographic techniques (Table 1) are supported and often required for the characterization of valvular lesions and their severity. A description of the Doppler and hemodynamic principles of each technique is beyond the scope of this paper, and the motivated reader should seek out a reputable textbook and refer to the American Society of Echocardiography's many guideline documents [25—27] on the topic.

Scope of examination

Whereas many aspects of comprehensive echocardiographic exams can be distilled into a more focused approach, the complexity of assessing valves makes the notion of limited valvular assessment somewhat controversial [28].

In a point-of-care setting, valvular assessment is typically performed in the context of acute circulatory or respiratory failure. As such, the scope of the assessment is typically centered on identifying suitably important

TABLE 1. Techniques for valvular assessment and characterization

2D or M-mode	Color Doppler	Spectral Doppler
2D inspection of function and appearance	Regurgitant jet area and width	Pressure half-time
Planimetry	Presence of turbulence/aliasing	Transvalvular peak gradient
2D inspection of chamber size and function	Width of vena contracta	Transvalvular mean gradient
	Proximal isovolemic surface area	Presence of flow reversal
		Calculation of valve area by continuity equation
		Calculation of regurgitant volume
		Calculation of regurgitant fraction

left-sided lesions. In practice, this often means assessment for severe mitral regurgitation (MR) or aortic regurgitation that may be associated with endocarditis, trauma, ischemia, or aortic dissection. It is important to note that the pursuit of valvulopathy is not necessarily the same as the pursuit of potentially causative disease states (such as endocarditis or aortic dissection) which, though related to the question at hand, typically relies on advanced US skills or different diagnostic modalities.

Chronic valvular lesions, especially chronic severe MR, mitral stenosis, or aortic stenosis, may significantly complicate the management of critically ill patients but are typically not isolated causes of cardiorespiratory failure.

Regurgitant lesions

Identification of important regurgitant lesions is considered a basic skill for those using echocardiography to care for the critically ill [1]. CF Doppler, widely available on most machines and intuitive to interpret, is suggested as a tool to screen for these lesions. Assigning severity is guided, at this basic level, by color jet size. Clinicians must be aware of the many pitfalls, including variation in color width created by eccentric jets (underestimated size due to energy loss) versus central jets (relatively overestimated size due to entrainment) [7]. As with other areas of US, understanding the underlying PW physics that govern color Doppler mapping, including the Nyquist limit, pulse-repetition frequency, and aliasing phenomena, is critical to the safe use of this modality.

If a suspicious regurgitant lesion is identified, further clarification of severity may require the use of the additional methods listed in Table 1. The vena contracta is favored by the authors for its simplicity, as it is an extension of the CF techniques already in use, and it remains accurate in the setting of eccentric jets (Fig. 3). If uncertainty remains, methods such as proximal isovolemic surface area, pressure half-time, regurgitant fraction, or regurgitant volume may prove useful but require advanced skills and extensive experience to perform accurately.

Of note, the diagnostic severity of a regurgitant lesion and its hemodynamic and clinical significance may be different. This is particularly true in acutely acquired lesions where a moderate or even mild lesion by strict diagnostic standards may produce severe clinical consequences as the receiving chamber (the LV in aortic regurgitation or the left atrium in MR) is naïve to increased volume and may be easily overwhelmed.

Stenotic lesions

Suspicion of comorbid aortic stenosis or mitral stenosis in a critically ill patient starts with a 2-dimensional examination of each valve. Findings such as a calcific annulus, thickened leaflets, or restricted leaflet mobility may be suggestive of stenosis. When using color, aliasing or turbulent blood flow near the valve may also frequently be present. It is important to assess the size and function of the cardiac chambers associated with the valve in question, as this can contribute significant additional information. Significant mitral stenosis, for example, is associated with a dilated left atrium, and the absence of such a finding should call into question the initial suspected valvular pathology.

Though many data points may help in grading the severity of a stenotic lesion, the most efficient and realistic point-of-care technique is the use of a well-aligned CW

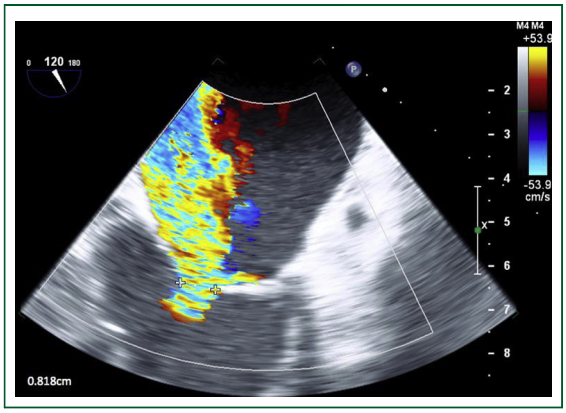

FIGURE 3. A point-of-care transesophageal echocardiography exam in a patient with shock in the context of an acute coronary syndrome demonstrates an eccentric jet along the left wall of the left atrium. Vena contract is measured at 8 mm, which is indicative of severe mitral regurgitation.

Doppler line of interrogation through the valve in question. Using the modified Bernoulli equation, the spectral Doppler information may be readily converted to a mean or peak pressure gradient. Standardized reference values are easily available and typically guide decision making for definitive therapeutic options [27]. The point-of-care clinician must be mindful of how hemodynamic alterations, arrhythmias, and varying loading conditions (all common during an acute illness) may significantly alter the measured gradient. As such, previous baseline echocardiographic information for comparison is invaluable.

Special circumstances

Prosthetic valves confer significant complexity and, in general, if concern exists, the involvement of an advanced echocardiographer who also has transesophageal echo skills is advisable.

Systolic anterior motion of the mitral valve and other forms of dynamic obstruction of the LVOT may be detected readily in those with structural heart disease or those with profound hypovolemia who are receiving excessive inotropic therapy. A point-of-care provider may detect this condition on 2-dimensional examination of the mitral valve or may note the late peaking CW Doppler profile through the LVOT, which is characteristic of this lesion (Fig. 4).

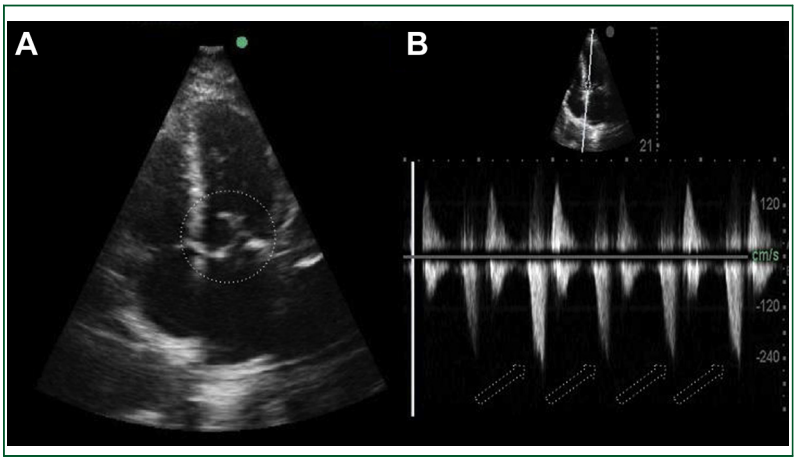

FIGURE 4. A point-of-care echo demonstrating features of systolic anterior motion due to profound hypovolemia in a burn patient. (A) The anterior mitral valve leaflet is seen to be nearly abutting the septum during systole. (B) The late peaking CW Doppler envelopes during systole (arrows) as obtained from the left ventricular outflow tract are pathognomonic for dynamic obstruction.

RIGHT HEART

Overview

Despite the crucial role it plays in the circulatory system, the RV has historically received relatively little attention compared with its neighbor to the left. When treating acutely ill patients, be it in the intensive care unit, the emergency department, the operating room or elsewhere, understanding the RV is often crucial; a number of common disease states affect it directly, and its examination using US is often critical to diagnosing and treating them.

Traditional RV assessment

RV size is traditionally measured using an RV-focused apical 4-chamber view, with measurements of maximum RV diameter taken at the base, mid-level, and in the longitudinal dimension [29,30]. In the acute care setting, acquiring these measurements is often not feasible and thus a simplified, semiquantitative method is recommended: comparing the ratio of maximum RV/LV width [31]. A normal RV/LV ratio should be $\leq$0.6; a ratio of 0.6-1.0 represents mild RV dilation and a ratio of $>$1.0 represents severe LV dilation. A secondary check can be made using the apex as a guide [32]. Normally the cardiac apex should be made up almost entirely of the LV apex; equal "sharing" of the cardiac apex by the LV and the RV suggests at least mild RV dilation, whereas a dominant RV apical presence implies severe RV dilation.

Assessment of RV systolic function is often done using a subjective technique, although the reliability of this "eyeball" method has been shown to be limited when compared with the gold standard, MRI [33]. Many operators evaluate the motion and thickening of the RV free wall (usually best appreciated in the subcostal 4-chamber view) and the displacement of the lateral aspect of the tricuspid valve (TV) annulus (usually best appreciated in the apical 4-chamber view) and then proceed to make their subjective determination, usually taking into account the size of the RV as well.

Advanced assessment of RV systolic function

Five main techniques have been developed in an attempt to objectify the evaluation of RV systolic function, but only 2 are simple enough to routinely apply to the care of acutely ill patients. Right ventricular index of myocardial performance [34], 2-dimensional fractional area change [35], and real-time 3-dimensional echocardiography [36] have all been correlated to assessment of RV function by MRI, but are generally too unwieldy to apply at the point of care. The 2 remaining techniques—tricuspid annular plane systolic excursion (TAPSE) and assessment of tricuspid annulus velocity by pulsed Doppler (S^1)—are more straightforward to perform and may help to improve the accuracy and reliability of RV assessment.

TAPSE is a technique to measure the systolic excursion of the lateral aspect of the tricuspid valve annulus. Beginning from an apical 4-chamber view, an M-mode cursor is placed through the lateral TV annulus and the distance traveled from end diastole to peak systole is measured (Fig. 5). This is a regional technique, and it assumes that annular motion is correlated to global RV function, an assumption that is untrue in many pathological states. Despite this, TAPSE appears to be useful, with displacement values of $<$17 mm correlating with poor global RV function [37].

A related technique, S^1, relies on the use of PW Doppler to quantify the speed of movement of the RV annulus. To

FIGURE 5. Using TAPSE to measure systolic excursion of the lateral aspect of the tricuspid valve annulus. In this case, the excursion is normal, measured at 27 mm. TAPSE, tricuspid annular plane systolic excursion.

perform this technique, an apical 4-chamber view is again used, to which a tissue Doppler imaging filter is applied. Tissue Doppler imaging alters the gating of the Doppler software to focus on the higher-amplitude, lower-velocity signals that emanate from movement of myocardial tissue. A pulsed Doppler sample volume is placed at the level of the lateral TV annulus (Fig. 6) and the velocity is measured. In theory, higher maximum Doppler velocities correlate with better global RV systolic function, although the same caveats apply as for TAPSE. A maximum velocity of <10 cm/s suggests abnormal RV global systolic function [38].

Summary of assessment of size and function

In acute care settings, and under reasonable imaging conditions, the following stepwise approach is recommended by the authors:

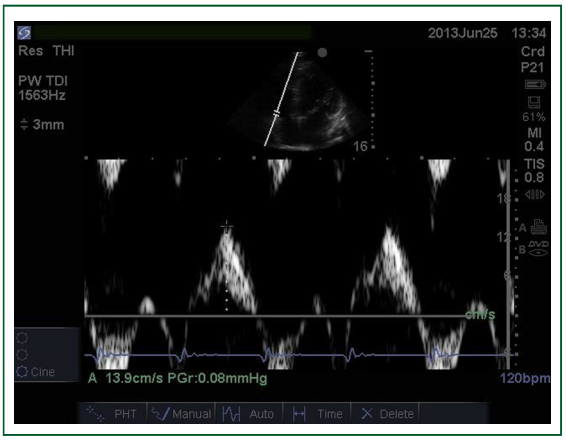

FIGURE 6. Using S^1 to quantify the speed of movement of the lateral aspect of the tricuspid valve annulus. In this case the velocity is normal, measured at 13.9 cm/s. S^1, assessment of tricuspid annulus velocity by pulsed Doppler.

1. From the apical 4-chamber view, assess the following elements:
   a. RV size using the RV/LV ratio and by examining the cardiac apex;
   b. RV systolic function by subjective assessment of RV free wall movement and thickening, and by assessment of lateral TV annular displacement; and
   c. RV systolic function by TAPSE and S^1.
2. From the subcostal 4-chamber view, assess the following elements:
   a. RV size using the RV/LV ratio; and
   b. RV systolic function by subjective assessment of RV free wall movement and thickening, and by assessment of lateral TV annular displacement.

Under conditions where image quality is suboptimal, it may be necessary to rely solely on the subcostal view. Sonographers should be aware that this could introduce a number of potential errors. Mistakes due to foreshortening are more common, and the assessment of RV systolic function will be completely subjective, as the more objective techniques cannot be performed from the subcostal position.

Estimation of PA pressures

In most patients a reliable estimate of pulmonary artery (PA) systolic pressure can be made relatively easily using the velocity of the tricuspid regurgitation (TR) jet, assuming there is no obstruction of the right ventricular outflow tract [30]. From the apical 4-chamber view, a color Doppler window is placed over the TV to localize the TR color jet, assuming it exists (as it does in a majority of patients). The maximum TR jet velocity is then measured using CW Doppler. Care must be taken to align the Doppler interrogation with the direction of TR jet; otherwise, the systolic PA pressure will be underestimated. This jet velocity is then converted to a pressure gradient using a modified Bernoulli equation, to which an estimate of RA pressure is added. The final equation becomes:

$$\text{Systolic PA pressure} = 4(\text{TR jet velocity})^2 + \text{RA pressure}$$

The estimation of RA pressure is done using the size and respiratory variation of the IVC when the central venous pressure cannot be directly measured. There is an inherent tendency to underestimate TR jet velocities, thus PA pressure should be considered a rough estimate rather than a precise measurement, and these measurements should be interpreted with care.

SUMMARY

Physicians are commonly asked to assess patients in shock where the etiology is initially unclear. Although simple point-of-care ultrasound techniques are often very helpful, it is not uncommon to encounter scenarios where basic level information is difficult to interpret and where more hemodynamic information would be useful. This chapter

discussed, in 3 parts, advanced techniques that can be applied in certain clinical scenarios, such as:

- when basic assessment of volume status is confounded;
- when estimating SV and CO is useful;
- when pericardial tamponade is suspected as a cause of shock;
- when RV failure is suspected as a cause of shock; and
- when valvular dysfunction is suspected as a cause of shock.

The scope of cardiac US applications for the care of acutely ill patients is nearly limitless, and whereas this range of possibility can seem overwhelming, it is important to realize that many advanced techniques have not been validated and some require high-quality images that are often challenging to obtain. As such, caution is required in interpreting results, which must always be integrated into the clinical context as a whole. Despite these limitations, there is great potential to improve patient care; the realization of this potential, however, will depend on the enthusiastic deployment and scientific study of these and other advanced point-of-care US tools.

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