

Cardiac Ultrasonography in the critical care setting: a practical approach to assess cardiac function and preload for the “non-cardiologist”

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Abstract

Cardiac ultrasonography has become an indispensable tool in the management of hemodynamically unstable critically ill patients. Some consider it as the modern stethoscope. Echocardiography is non-invasive and safe while the modern portable devices allow to be used at the bedside in order to provide fast, specific and vital information regarding the hemodynamic status, as well as the function, structure and anatomy of the heart. In this review, we will give an overview of cardiac function in general followed by an assessment of left ventricular function using echocardiography with calculation of cardiac output, left ventricular ejection fraction (EF), fractional shortening, fractional area contraction, M mode EF, 2D planimetry and 3D volumetry. We will briefly discuss mitral annulus post systolic excursion (MAPSE), calculation of dP/dt , speckle tracking or eyeballing to estimate EF for the experienced user. In a following section, we will discuss how to assess cardiac preload and diastolic function in 4 simple steps. The first step is the assessment of systolic function. The next step assesses the left atrium. The third step evaluates the diastolic flow patterns and E/e' ratio. The final step integrates the information of the previous steps. Echocardiography is also the perfect tool to evaluate right ventricular function with tricuspid annular plane systolic excursion (TAPSE), tissue Doppler imaging, together with inferior vena cava dimensions and systolic pulmonary artery pressure and right ventricular systolic pressure measurement. Finally, methods to assess fluid responsiveness with echocardiography are discussed with the inferior vena cava collapsibility index and the variation on left ventricle outflow tract peak velocity and velocity time integral. Cardiac ultrasonography is an indispensable tool for the critical care physician to assess cardiac preload, afterload and contractile function in hemodynamically unstable patients in order to fine-tune treatment with fluids, inotropes and/or vasopressors.

Key words: cardiac ultrasound, critical care, preload, cardiac function, filling status, fluid responsiveness

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The past decade has witnessed the introduction of bedside ultrasonography in the critical care setting (emergency room (ER), operating room (OR) and intensive care units). All over Europe intensive care unit (ICU) physicians are participating in ultrasound courses to further improve their knowledge and skills; in order to rapidly establish a diagnosis and provide optimal treatment [1].

The use of cardiac ultrasound has proven to be invaluable in order to assess hemodynamic function and preload. Pulmonary artery catheters (PAC) have been replaced by

less invasive continuous cardiac output (CO) measurements, either calibrated, such as transpulmonary thermodilution or uncalibrated, such as pulse contour analysis, or a combination of both. In most cases, however, these CO measurements need to be completed by a cardiac ultrasound. It is by far the most complete, comprehensive and vital investigation technique and is crucial for correct clinical decision making.

Some ultrasound techniques require more expertise and advanced skills than others. It is our aim to describe

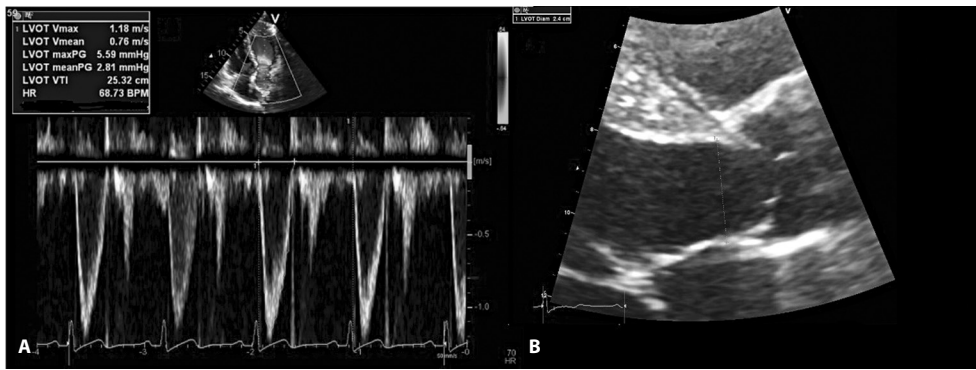


Figure 1. Calculation of stroke volume. **Panel A** shows the velocity time integral (VTI) of the left ventricular outflow tract (LVOT) obtained from an apical 5 chamber window. **Panel B** demonstrates the measurement of the diameter of the LVOT. CSA (Circumferential Surface Area) = $(\text{Diameter LVOT}/2)^2 \times \pi = (2.4/2)^2 \times 3.14 = 4.5 \text{ cm}^2$. Stroke volume = $VTI \times LVOT \times CSA = 25.3 \text{ cm} \times 4.5 \text{ cm}^2 = 113.83 \text{ cm}^3$. Cardiac output = stroke volume \times heart rate = $113.83 \text{ cm}^3 \times 68.7 \text{ beats per minute} = 7820 \text{ cm}^3/\text{minute}$.

the most practical measurements, their validation and their usefulness in the ICU ward and the broader critical care setting (ER and OR). We will refrain from describing complex measurements that may require profound acquisition skills or significant post-processing time. Guidelines for cardiac ultrasound in emergency settings have already been issued and updated [2, 3]. In order to facilitate speedy diagnosis in emergency care, pocket-held devices have been developed and are in widespread use today [4].

CARDIAC FUNCTION IN GENERAL

Cardiac function can be measured by several parameters. In the ICU the most implemented parameter is CO. This can readily be measured with a PAC catheter and its later derivatives, e.g. transpulmonary thermo- or dye dilution techniques as with the PiCCO (Pulsion Medical Systems, Feldkirchen, Germany), the EV1000 (Edwards Lifesciences, Irvine, USA) or the LiDCO (LiDCO Group plc, London, UK). These devices use a surrogate gold standard (calibrated) technique based on the Stewart Hamilton method. The newer less invasive devices using uncalibrated pulse contour analysis cannot be recommended in unstable patients, with frequently changing preload, afterload or contractility.

ICU clinicians focus on cardiac function in general. Cardiac output is a real-time measurement, regardless of regional hypokinesia and/or valvular dysfunction. Cardiologists and ultrasound technicians prefer ejection fraction (EF) and rather prefer to describe regional wall motion. The cardiologists' main task is to diagnose the aetiology of cardiac dysfunction. In their area of expertise, CO measurement *per se* is too limited and vague.

ASSESSMENT OF LV SYSTOLIC FUNCTION CARDIAC OUTPUT (CO)

Cardiac output is measured, by convention, in the left ventricular outflow tract (LVOT) using pulsed wave (PW)

Doppler velocity (Fig. 1). Since we can measure the LVOT diameter, we can calculate its cross sectional surface area (CSA) and derived stroke volume (SV).

- $CSA = 3.14 \times (D/2)^2 = 0.785 \times D^2$
- $SV = VTI \times CSA$, with VTI the velocity time integral

The first description of CO measurement with ultrasound at the LVOT was described and validated by Otto in 1988 [5]. Although CO can also be measured at other locations (mitral valve annulus [6], ascending aorta [7, 8], the right ventricular outflow tract (RVOT) [9] and pulmonary artery [10]), this has been less validated. While cross section of the LVOT at diastole ($LVOT_d$) can also be measured [11], large inter-observer variability exists up to 0.2 cm [12]. There also exists a difference between LVOT measured by transthoracic (TTE) and transesophageal echocardiography (TEE). It has been revealed that TTE tends to underestimate the LVOT by 0.1 cm [12]. Variation of LVOT in the general population ranges between 18 and 22 mm [13, 14] and is related to body surface area. Therefore, it can be estimated by a given formula, which is time-effective and reduces error [15]:

$$LVOT_d = 5.7 \times BSA + 12.1$$

Some ultrasonography labs use fixed values like 1.8 for female and 2.0 for male patients.

The calculated CSA, using these values, varies between 2.6–3.1 cm². The velocity time integral (VTI) can be derived with *pulsed wave Doppler* measured at the LVOT. A normal VTI varies between 20–25 cm [13]. This implies that a VTI > 20 cm refers to a normal CO, without the need for further calculation.

$$CO (\text{cm}^3/\text{min}) = SV \times HR = HR (\text{bpm}) \times CSA (\text{cm}^2) \times VTI (\text{cm})$$

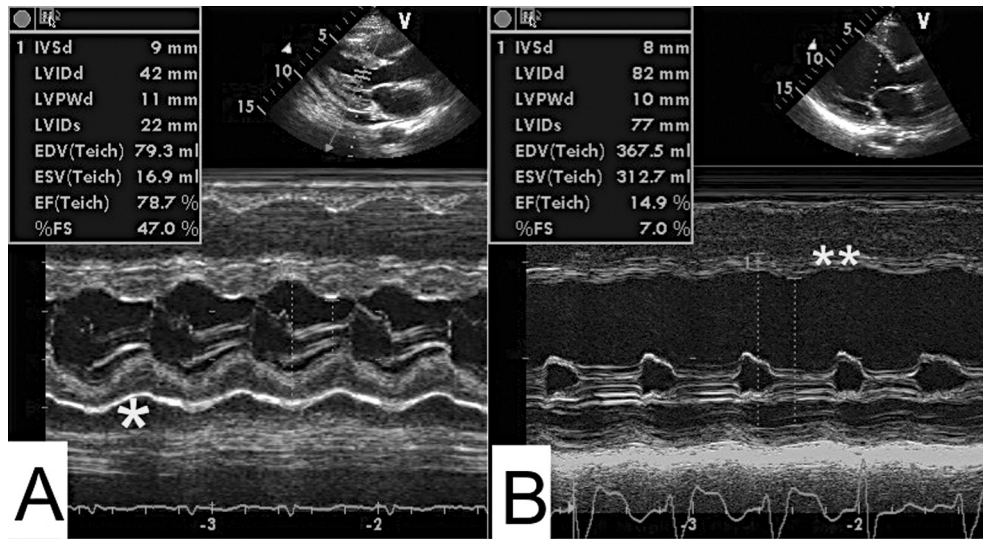


Figure 2. M Mode Left Ventricle and Fractional Shortening. **Panel A** shows an M Mode obtained from the parasternal long axis of a patient with preserved left ventricular function. This patient suffered from cardiac amyloidosis. Note also the small amount of pericardial fluid indicated by the asterisk (*). **Panel B** shows a patient with an anteroseptal myocardial infarction with severely diminished LV contractility. The septum is thin scar tissue. Note the absence of systolic thickening of the interventricular septum.

In order to facilitate quick bedside calculation in the ICU, CSA can be assumed to be around 3 cm², which simplifies the equation to:

$$CO (mL\ min^{-1}) = 3 \times HR \times VTI_{LVOT}$$

This equation is easy to memorize, the heart rate is readily available on the ICU monitor and the VTI measurement can be mastered without extensive time-consuming training.

EJECTION FRACTION (EF)

Ejection fraction is very common in the cardiology literature. The measurement is, although not very complicated, quite user-dependent and prone to many errors. The physiological basis of left ventricular ejection fraction (LVEF) is simple: the ejected volume is related to the left ventricular end-diastolic volume (LVEDV). Normal LVEF is above 55%.

$$LVEF = (LVEDV - LVESV)/LVEDV$$

FRACTIONAL SHORTENING

Fractional shortening (FS) can be derived by calculating the linear shortening of the following measurements:

FS = (LVEDD – LVESD/LVEDD) × 100, with LVEDD as the left ventricular end-diastolic diameter and LVESD as the left ventricular end-systolic diameter.

This measurement in itself is correlated with LVEF, without further calculation. Normal FS is between 25–40%. Unless one is familiar with this measurement, the number never gives an intuitive “correlation” with other known variables in the ICU.

FRACTIONAL AREA CONTRACTION (FAC)

Fractional area contraction (FAC) can be derived by calculating the linear shortening of the following measurements:

FAC = (LVEDA – LVESA / LVEDA) × 100, with LVEDA as the left ventricular end-diastolic area and LVESA as the left ventricular end-systolic area.

As for FS, this measurement is also correlated with LVEF, without further calculation. Normal FAC is between 35–45%.

M MODE LVEF

Fractional shortening can subsequently be used to calculate an actual LVEF. All these calculations require linear acquisition of ventricular diameters. After determination of the end-diastolic and end-systolic left ventricular diameters, several methods can be used to estimate LVEF. Cubed formulas, such as the Teichholz formula [16] and the modified Quinones formula equation [17] have been described previously, and are usually programmed on most available ultrasonography equipment. Several pitfalls make these methods less desired in the critical care setting (Fig. 2):

- M Mode acquisition requires a lot of expertise, and is not always easy to perform in dorsal decubitus;
- M Mode border identification is not easy in non-expert hands;
- Only 2 segments out of a total of 17 cardiac segments are used to calculate LVEF.

2D PLANIMETRY

This is, according to the current guidelines, the method of choice to estimate CO [18, 19]. This method needs area

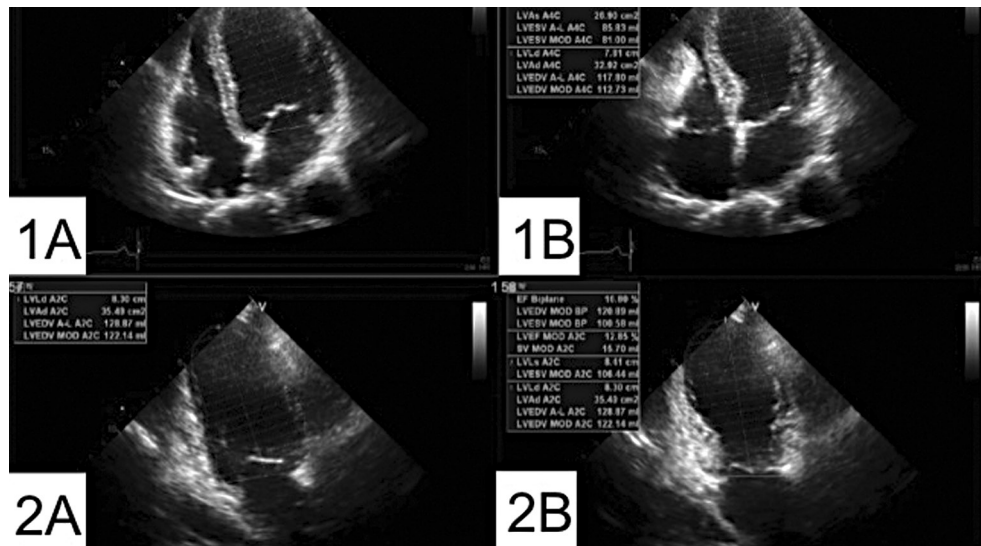


Figure 3. LV EF by Simpson method. This figure shows a calculation of the LV EF by the Simpson method. The 4 chamber and 2 chamber frames at end systole and end diastole are used. The software applications of modern ultrasound machines automatically calculate the LV EF by using the Simpson method, once the ultrasonographer traces the endocardial border. This example concerned a patient suffering from apical ballooning. Only the basal segments were contractile. **Panels 1A and 1B** are 4 chamber views at end-diastole and end-systole respectively. **Panels 2A and 2B** show the analogous 2 chamber views.

tracings of the left ventricle in two perpendicular views. In this way it makes no geometrical assumptions of the ventricle. Since the ventricular endocardium is traced from base to apex, there is less possibility to under- or overestimate the ventricular function due to regional hypokinesia. The method is generally described as Simpson’s method, which is based on the summation of the smaller volumes in order to obtain the overall left ventricular volume. The length of the LV is divided into 20 parallel discs, from base to apex, with a diameter of each disc determined in two apical views (two-chamber and four-chamber) (Fig. 3).

The left ventricular endocardium is traced in end-diastole and end-systole in both views. These parameters are used to calculate both left ventricular end-diastolic and end-systolic volumes, as well as the LVEF. The crucial element in the echocardiographic evaluation of LVEF, using Simpson’s method, is accurate identification of the ventricular endocardium. Poor image quality and failure to identify papillary muscles will produce significant errors.

It is worth noting that in cases of suboptimal image quality, the use of an echocardiographic contrast may improve the endocardial definition.

Some of the more recent ultrasound devices may also have an automatic endocardial border detection.

3D VOLUMETRY

This method renders a 3D image and estimates EF, based on the use of a dedicated 3D probe. The imaging is in itself not difficult, since a single volume acquisition suffices to calculate EF. Automated border detection of the endocardium

in Real Time 3D echo seems promising, but remains yet to be validated [20]. The question remains if this high-end ultrasonography equipment will be within the practical scope of an emergency/ICU department. For the time being, this method requires tedious post-processing techniques and cannot be recommended; hence it falls outside the scope of this article.

EYEBALLING

If one is familiar with cardiac ultrasound, one can estimate the left ventricular function by just viewing movie-loops of the ventricular motion. Some articles state that focused training in the ‘eyeballing technique’ can result in acceptable accuracy in estimating LVEF [21].

MITRAL ANNULUS POST SYSTOLIC EXCURSION (MAPSE)

A quick method to estimate LVEF is to measure systolic movement of the mitral annulus ring. Using M Mode in an apical four-chamber view, one can visualise this movement with ease. The technical considerations and pitfalls involved are, however, obvious. One has to align the M Mode perpendicular to the annulus movement, in order not to underestimate the systolic excursions. Since one only measures MAPSE in one or two positions of the atrio-ventricular plane, it extrapolates LVEF function based on sparse data. However, MAPSE can be easily obtained in patients with poor imaging quality and in dorsal decubitus. Intra- and inter-observer variability is around 5%, which is acceptable [22]. Furthermore, it is an independent predictor of 28-day mortality [23]. Some key-points to remember are as follows (Fig. 4):

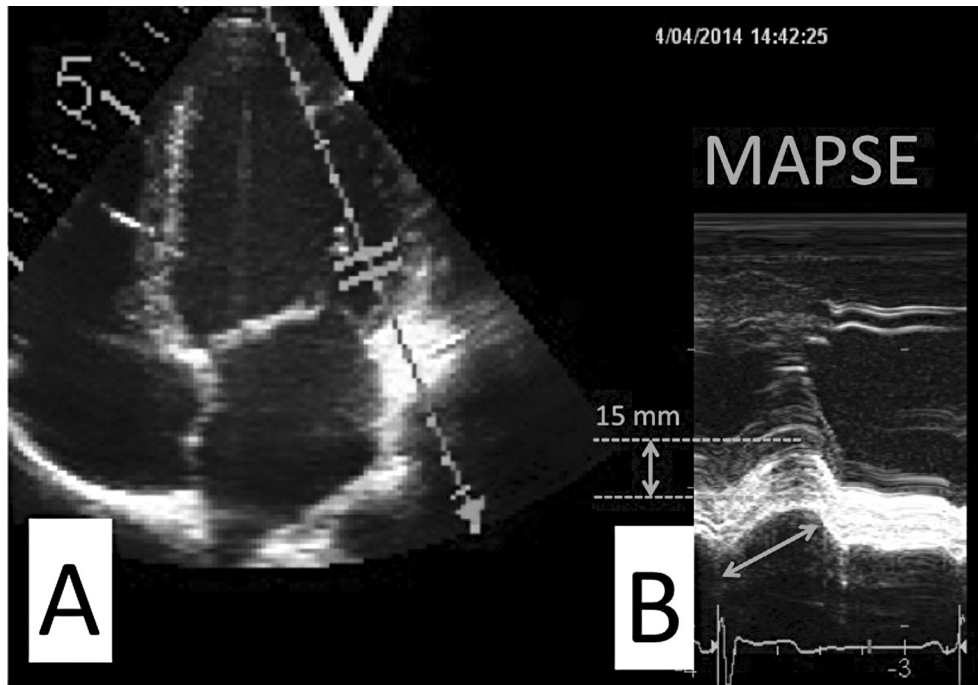


Figure 4. Mitral Annulus Post Systolic Excursion (MAPSE). **Panel A** shows the acquisition of MAPSE from the mitral valve insertion on the left ventricular free wall. **Panel B** shows M Mode registration with clear systolic movement of the mitral annulus plane. MAPSE is estimated at 15 mm.

- MAPSE of > 10 mm correlates with an EF > 55%
- MAPSE of < 8 mm relates to a reduced ejection fraction
- This leaves an “indeterminate” grey zone between 8–10 mm, where no statement regarding LV function can be made

CONTRACTILITY OR DP/DT

This is an underutilized indicator of LV function. Although mitral regurgitation dP/dt is afterload independent, it is influenced by preload. It is a measurement of contractility of the LV in the isovolumetric contraction phase [24, 25]. This technique requires a measurable mitral regurgitation on the Continuous Wave signal, obtained from a four-chamber view. It necessitates alignment of the regurgitated jet with the ultrasound beam. Most echocardiographic ultrasound machines will provide reference lines at 1 and 3 m sec⁻¹ and will calculate and display dP/dt automatically (Fig. 5).

- The normal dP/dt is > 1200 mm Hg sec⁻¹
- dP/dt between 800 to 1200 mm Hg sec⁻¹ suggests mild LV dysfunction
- dP/dt < 800 mm Hg sec⁻¹ suggests severe LV contractile dysfunction

SPECKLE TRACKING ECHOCARDIOGRAPHY (STE)

Two-dimensional speckle tracking echocardiography (STE) is a relatively novel and sensitive method for assessing ventricular function by measuring the myocardial deformation (strain). STE has been demonstrated to be

able to unmask myocardial dysfunction before it can be detected with conventional echocardiography. Contrary to Doppler Strain imaging, STE is an angle independent technique. This independence of alignment is a potential advantage when echocardiographic imaging has to be performed in suboptimal conditions, as is often the case in patients in the ICU.

A recent study demonstrated that STE is a feasible technology for assessing left ventricular deformation in septic patients in the ICU. Furthermore, a greater portion of the patients in this study were identified as having systolic dysfunction of both the RV and LV when assessed by STE as compared with conventional echocardiography [26].

Another study has shown that the combination of global longitudinal strain (GLS) and the APACHE II score have additional value in the prediction of ICU and hospital mortality in septic shock patients admitted to the ICU [27]. STE may help in the early identification of high-risk patients in the ICU. STE is a promising field in cardiac ultrasound that will be developed further over the next years and standardization of this technique is currently under review [28] (Fig. 6).

CARDIAC PRELOAD AND DIASTOLIC FUNCTION

Even in the optimal setting of an echo laboratory, assessment of the loading conditions and diastolic function is often challenging. Bedside echo Doppler evaluation in the ICU is often more difficult due to suboptimal image

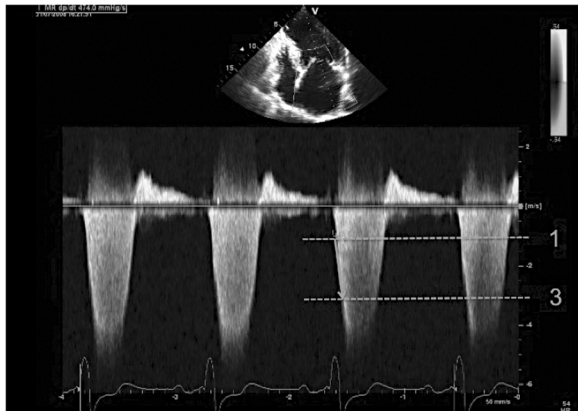


Figure 5. Contractility dP/dT. The panel shows a mitral valve regurgitation signal by continuous wave (CW) Doppler. Change in velocity is measured, by definition, between 1 and 3 m/sec. The dP/dT is 474 msec, and implies severely diminished LV function.

quality: patients lie in a supine position in the ICU, whereas the patients in the ultrasound laboratory lie on their left side, while echocardiography studies are being performed.

As a consequence, these measurements in the ICU often have to be obtained from suboptimal acoustic windows. Imperfect alignment with the ultrasound beam is a major limitation for the use of an echo Doppler, especially for quantitative assessment. Despite these limitations, adequate evaluation of the diastolic function is an essential part of the echocardiographic assessment. Moreover, the evaluation of the loading condition is often the main reason for ordering a cardiac ultrasound study in patients, hospitalized in the ICU.

Over recent decades, several parameters have been proposed to assess the diastolic function. The goal of this paper is not to review all these methods, but to describe a comprehensive approach for the evaluation of the diastolic function, and which is feasible for use in the ICU. Assessment of the diastolic function is not based on a single measurement. The diastolic “mitral inflow” patterns, but also quantification of the left atrium and the systolic function, have to be taken into account. In most echocardiography training programs the following stepwise approach is proposed:

STEP 1: ASSESSMENT OF THE SYSTOLIC FUNCTION

Paradoxically, assessment of the systolic function, as described in the previous sections, is the first step in the evaluation of the diastolic function. In heart disease, the diastolic function is affected before a decrease in LV EF appears. Thus, an impaired systolic function excludes a normal diastolic function. Interpreting systolic function is the first step in the ESC/EACVI (European Society of Cardiology/ European Association of Cardiovascular Imaging) algorithm for diastolic assessment [29].

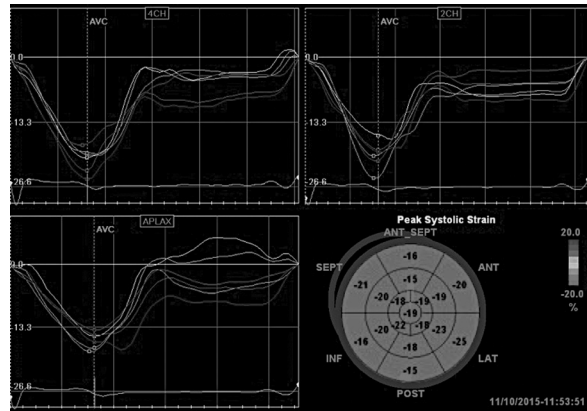


Figure 6. Speckle Tracking Longitudinal Strain (STE). This figure shows STE of a patient with preserved LV EF and normal global longitudinal strain.

STEP 2: ASSESSMENT OF THE LEFT ATRIUM

Quantification of the left atrium (LA) is the next step in the procedure. LA dilation is the consequence of longstanding LA pressure and/or volume overload. Thus, LA enlargement does not necessarily mean LA pressure is definitely increased, but it indicates a likelihood of elevated LA pressure (LAP). Due to this property, LA volume is sometimes called the HbA_{1c} of diastology. LA dilation is *considered incompatible* with preserved diastolic function. Quantification of LA volume according body surface area (LA_{vol}/BSA) (30) is as follows:

- LA_{vol}/BSA < 29 mL m⁻²: normal
- LA_{vol}/BSA: 29–33 mL m⁻²: mild
- LA_{vol}/BSA: 33–39 mL m⁻²: moderate
- LA_{vol}/BSA > 39 mL m⁻²: severe

The European Association of Echocardiography and the American Society of Echocardiography guidelines use the threshold value of LA_{vol}/BSA of 34 mL m⁻² in combination with Doppler measurements in algorithms to estimate the LAP [31] (Fig. 7).

STEP 3: EVALUATION OF THE DIASTOLIC FLOW PATTERNS

THE MITRAL INFLOW DOPPLER VELOCITY PATTERN

This measurement is the keystone of the diastolic flow patterns. At the end of the systole, the aortic valve closes. The left ventricle starts to relax and left ventricular pressure decreases. Once the left ventricular pressure falls below the LAP, the mitral valve opens and the diastolic filling of the LV begins. The interval between the closing of aortic valve and the opening of the mitral valve is called the isovolumetric relaxation time (IVRT). The mitral inflow pattern consists of an early rapid filling E (early) wave, with a peak and decel-

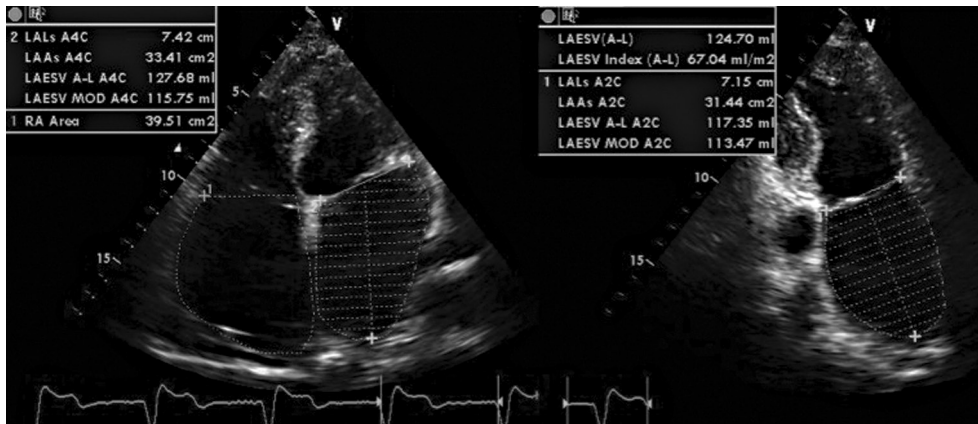


Figure 7. Quantification of the LA volume. This figure demonstrates the measurement of the LA volume. A frame at start of the diastole in the 2 chamber and 4 chamber view is used. The automated software of the ultrasound machine calculates the LA volume indexed to BSA.

eration (down) slope (DT). Due to the rapid filling of the LV cavity, the left ventricular pressure increases and filling velocity decreases. At the end of the diastole, atrial contraction occurs, resulting in the A (atrial) peak [32].

ABNORMAL DIASTOLIC FLOW PATTERNS

1. Impaired relaxation

With aging and heart disease, there is a decrease in diastolic relaxation, as well as elastic recoil. This results in a slower LV pressure decline. Subsequently, it takes more time before the LV pressure becomes equal to the LAP. Thus, the IVRT is prolonged. Due to the decreased relaxation of the LV, the filling occurs at lower velocities (E peak decreases) and the filling-time prolongs, with a subsequent increase in DT. The mitral E velocity is decreased while the A velocity is increased. This results in an E/A ratio < 1.

An impaired diastolic filling pattern typically occurs in case of hypertrophic cardiomyopathy and can be summarized as follows [32]:

- Decreased E velocity
- Increased A velocity
- E/A ratio < 1
- Prolonged DT (> 160 msec)
- Prolonged IVRT (> 90 msec)

2. Restrictive filling pattern (decreased compliance)

The increase in LAP results in a faster opening of the mitral valve, with subsequent shortening of the IVRT. The high LAP results in an increased initial trans-mitral gradient (higher E peak). Due to noncompliance of the LV, the early diastolic filling results in a fast rise in pressure in the LV with early equalization of LV and LA pressure, which results in a shortened DT. The atrial contraction results in a small A wave with a shortened duration, as the increase in LV pressure increases more rapidly as consequence of the noncom-

pliant state of the LV. Thus, in a restrictive filling pattern, the mitral inflow velocities can be summarized as follows [32]:

- Increased E velocity
- Decreased A velocity
- E/A ratio > 2
- Shortened DT (< 160 msec)
- Shortened IVRT (< 70 msec)

3. Pseudonormalized pattern

This pattern is a transition from an impaired to a restrictive filling pattern. As the name indicates, it resembles a normal filling pattern, with normal E/A ratio and DT. This filling pattern is the result of increased LA pressure superimposed on a relaxation abnormality.

E/e' RATIO

Tissue Doppler Imaging (TDI) is able to measure the longitudinal mitral annular velocity. The measurement e' is the early diastolic peak velocity obtained by TDI. It has been demonstrated that e' is relatively load-independent. Meanwhile, the measurement E is dependent of loading conditions as well as ventricular relaxation [33].

Thus, dividing E/ e' reflects better the loading conditions of the LV (Fig. 8).

E/ e' is considered as the single best parameter to estimate the LAP. As mentioned earlier, in order to evaluate the filling pattern, it is wise never to rely on one single measurement. Nonetheless E/ e' is very useful in order to discriminate the normal diastolic filling pattern from a pseudonormalized filling. In the latter e' is decreased. Keypoints to remember are [31]:

$$E/e' \approx \frac{\cancel{LAP \times relaxation}}{\cancel{relaxation}} \approx LAP$$

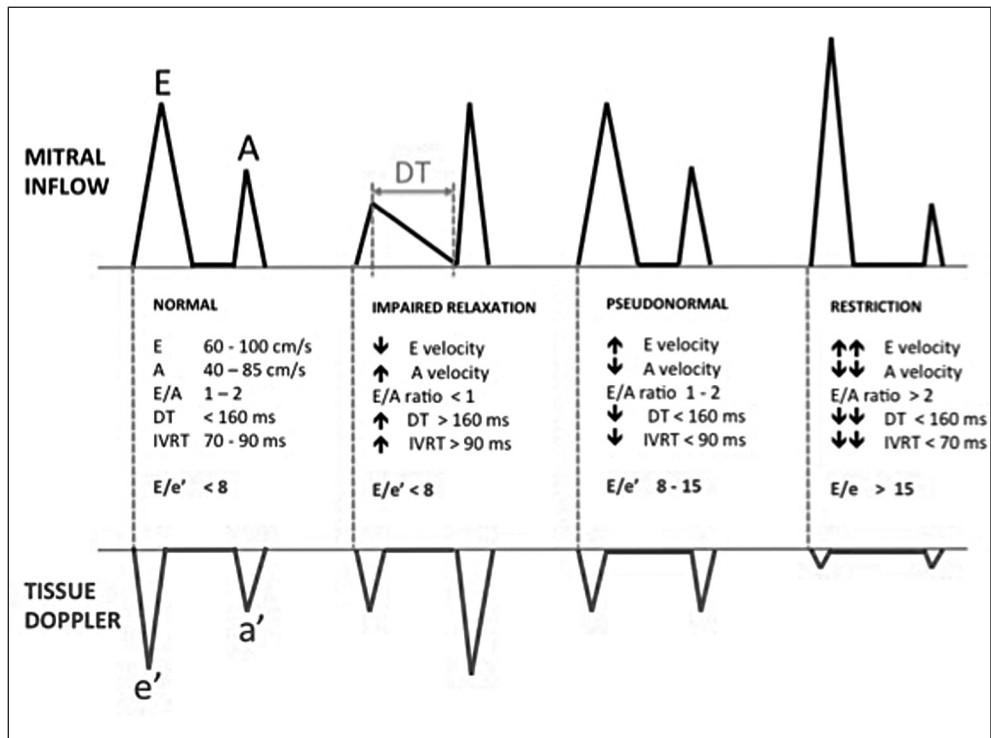


Figure 9. Diastolic Filling Patterns. Theoretical appearance of Mitral Inflow signal and Tissue Doppler Imaging in the 4 different states of diastolic function: normal, impaired relaxation, pseudonormalized flow pattern and restriction.

- $E/e' < 8$ reflects normal LA pressure
- $E/e' > 15$ implies increased LA pressure

Unfortunately, there is a large grey area in between these two values. For ICU purposes it is interesting to note that E/e' correlates well with pulmonary artery occlusion pressure (PAOP \approx LAP). The following formula has been suggested by Nagueh *et al.* [34]:

$$PAOP = 1.24 * (E/e') + 1.9 \text{ mm Hg}$$

The M Mode acquisition of the mitral annulus post systolic excursion, or MAPSE, is, in essence, a less accurate measurement of tissue displacement. Studies have shown that BNP levels correlate inversely to MAPSE and even better than E/e' [35].

STEP 4: INTEGRATION OF THE INFORMATION OF THE PREVIOUS STEP

By putting the information, obtained in the previous steps together, the diastolic dysfunction can be graded into 4 classes:

- Diastolic dysfunction grade I: impaired relaxation
- Diastolic dysfunction grade II: pseudonormalized pattern
- Diastolic dysfunction grade III: reversible restrictive pattern

- Diastolic dysfunction grade IV: irreversible restrictive pattern

The differentiation between diastolic dysfunction grade III and IV is difficult to make in ICU patients, as this requires cooperation of the patient while performing echo Doppler measurements with or without the Valsalva Manoeuvre (Figs 9, 10).

The EAE algorithm to estimate the filling pressure in patients with preserved LV EF starts with E/e' [31] (Fig. 11):

- $E/e' < 8$: normal LAP
- $E/e' 9-14$: LA_{vol}/BSA is used to further discriminate:
 - $LA_{vol}/BSA < 34 \text{ mL m}^{-2}$: normal LAP
 - $LA_{vol}/BSA > 34 \text{ mL m}^{-2}$: elevated LAP
- $E/e' > 15$: elevated LAP

In patients with decreased LV EF the algorithm, the mitral inflow pattern is the first step in the evaluation (Fig. 12):

- $E/A < 1$ and $E < 50 \text{ cm s}^{-1}$: normal LAP
- $E/A > 2$ and $DT < 150 \text{ cm}$: elevated LAP
- $E/A 1-2$ or $E/A < 1$ and $E > 50 \text{ cm/s}$ use E/e' to make further discrimination:
 - $E/e' < 8$: normal LAP
 - $E/e' > 15$: elevated LAP

Thus, LV systolic function, LA volume, mitral inflow velocities and E/e' are the most important factors in order to

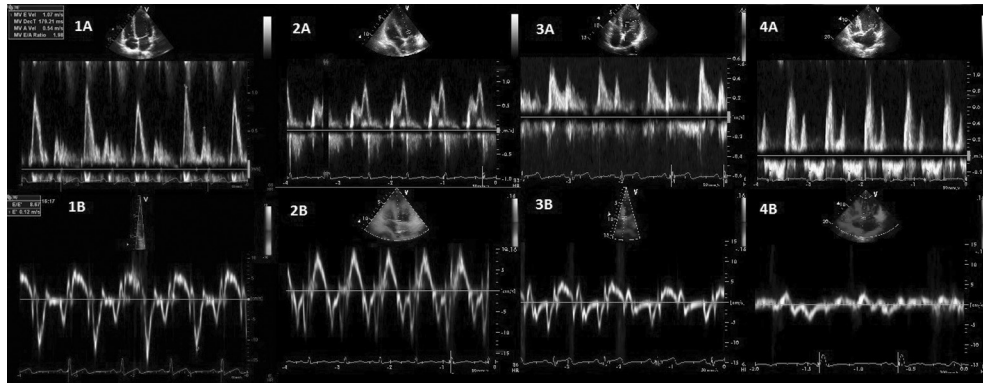


Figure 10. Diastolic function in reality. **Panels 1A and 1B** show respectively the mitral inflow velocity and e' in a 23-year-old man with normal left atrial pressure (LAP). $E/A = 1.5$, $e' = 12$ cm/s and $E/e' = 8.67$. **Panels 2A and 2B** show respectively the mitral inflow velocity and e' in a patient with delayed relaxation (diastolic dysfunction grade 1) in a 68-year-old female suffering from hypertensive left ventricular hypertrophy. $E/A = 0.53$, $e' = 9$ cm/s and $E/e' = 7.8$. **Panels 3A and 3B** show respectively the mitral inflow velocity and e' in a patient with pseudo normalized mitral inflow pattern (diastolic dysfunction grade 2). $E/A = 1.34$, $e' = 4.5$ cm/s and $E/e' = 13.3$. Note the contribution of the e' to allow discrimination between normal and pseudo normal inflow velocities. **Panels 4A and 4B** show respectively the mitral inflow velocity and e' in a patient with restrictive inflow pattern due to cardiac amyloidosis. $E = 110$ cm/s, $E/A = 2.2$, $e' = 3.3$ cm/s and $E/e' = 33$.

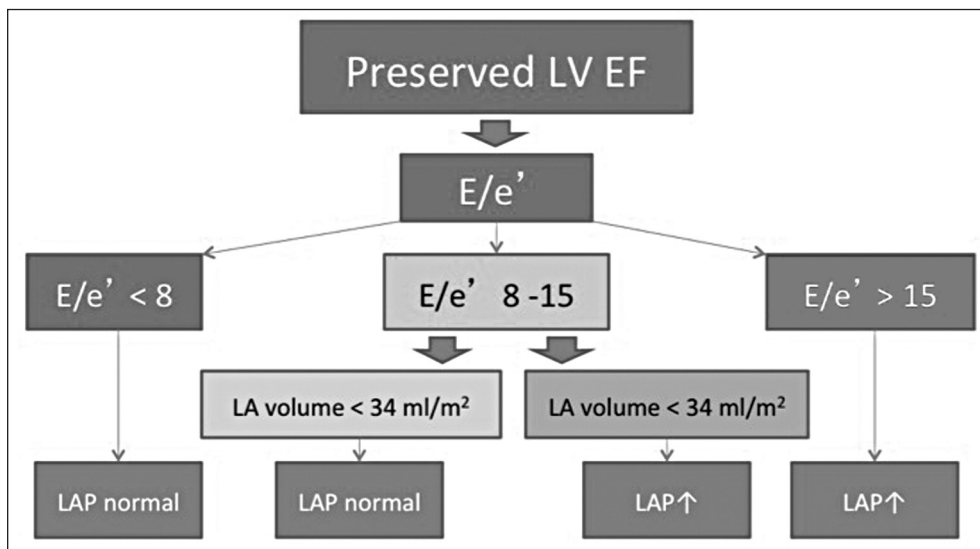


Figure 11. Preserved LV EF. Proposed simplified flowchart for assessment of loading conditions and/or filling pressures

determine the grade LV diastolic function. The Pulmonary Vein Velocity Patterns and colour flow mapping (CFM) trans-mitral flow propagation also provide additional information. Their acquisition and interpretation are outside the scope of this article, since they require greater expertise.

RIGHT VENTRICULAR ASSESSMENT

RIGHT VENTRICLE

Assessment of right heart structure and function is a basic part of cardiac ultrasound evaluation. It is an essential addition to left heart parameters and has long been neglected. Due to its geometry, the right ventricular (RV) function is more difficult to quantify. Since its cavity is not

circular, 2D Simpson's method is not feasible. Moreover, short axis linear measurements in M Mode do not correlate well with RV function.

3D echocardiography has been demonstrated to be able to accurately measure the RV EF. As mentioned afore, this requires sophisticated modern cardiac ultrasound machines.

The goal of this paper is to provide a comprehensive and relatively easy method to perform bedside evaluation of the RV function. The combination of 4 measurements, as described below, allows a basic assessment of the RV function and RV pressures. In patients with severely increased RV pressure or volume overload, examination of the global RV shape may be helpful (Fig. 13).

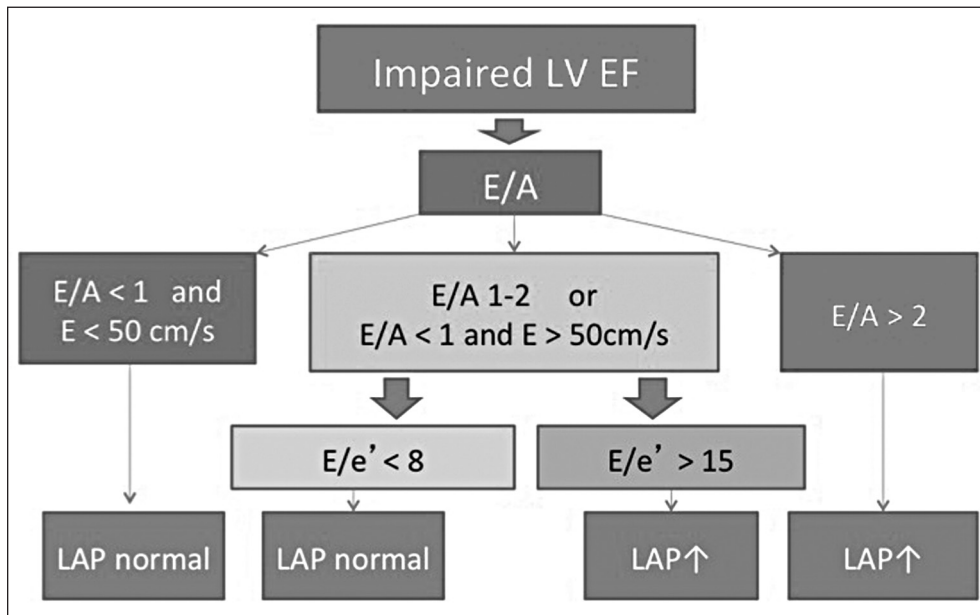


Figure 12. Impaired LV EF. Proposed simplified flowchart for assessment of loading conditions and/or filling pressures.

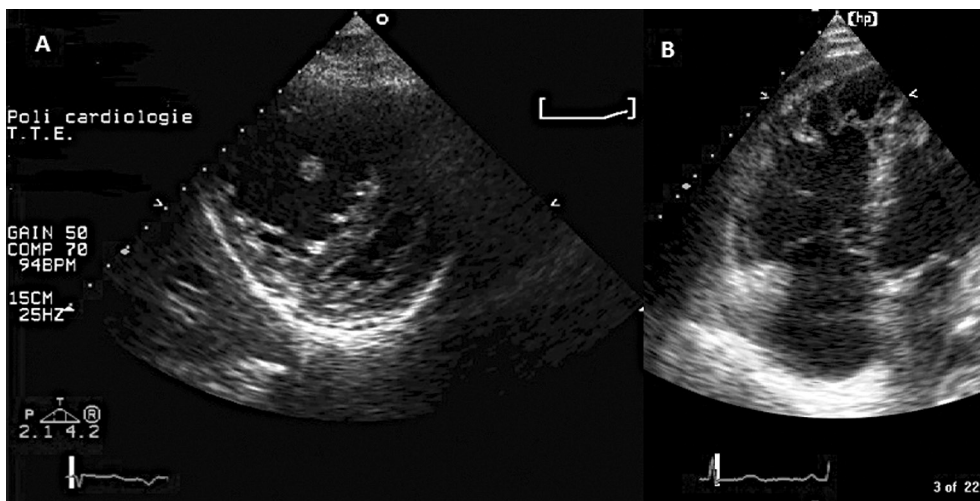


Figure 13. RV overload. These images were made in a 55-year-old woman suffering from severe pulmonary hypertension, due to chronic pulmonary embolism. **Panel A** shows an image obtained from the parasternal short axis view and **panel B** an image from the 4 chamber window. Note the dilated RV, that exceeds the LV dimensions, with rightward ventricular septal shift (*D Shape* of LV).

- In normal conditions, the LV dimensions are larger than the RV dimensions with a rightward ventricular septal shift
- If the RV “oversizes” the LV with leftward ventricular septal shift, there is major volume or pressure overload (*D-shaping* of left ventricle)
 - In cases of pressure overload, the leftward ventricular shift is most prominent at end-systole
 - In case of volume overload, this shift is most pronounced at end-diastole

TAPSE: TRICUSPID ANNULAR PLANE SYSTOLIC EXCURSION

TAPSE measures the distance of the systolic excursion of the RV annular segment along its longitudinal plane [36]. This measurement is obtained from an apical 4-chamber view. Thus, TAPSE represents longitudinal function of the right ventricle. The limitation of this method is the assumption that the displacement of the basal segment is representative for the entire RV.

Nonetheless, the European and American Society of cardiology recommend TAPSE in the routine use for an as-

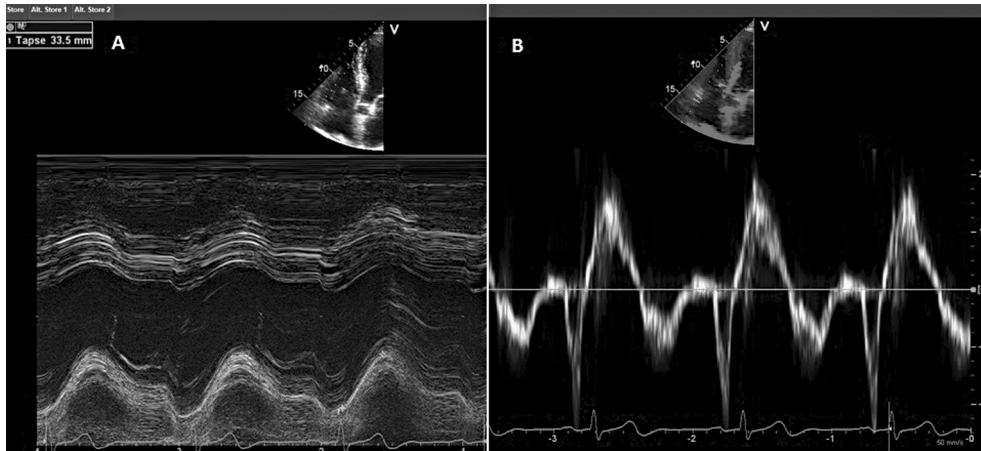


Figure 14. Tricuspid annular plane systolic excursion (TAPSE) and systolic excursion velocity. **Panel A** and **B** were measured in a patient with normal right ventricular function. **Panel A** shows TAPSE (Tricuspid annular plane systolic excursion). This measurement is performed from the 4 chamber window. It measures the distance of systolic excursion of the RV annular segment along its longitudinal plane. **Panel B** shows a Doppler Tissue Imaging measurement of the RV S' or systolic excursion velocity.

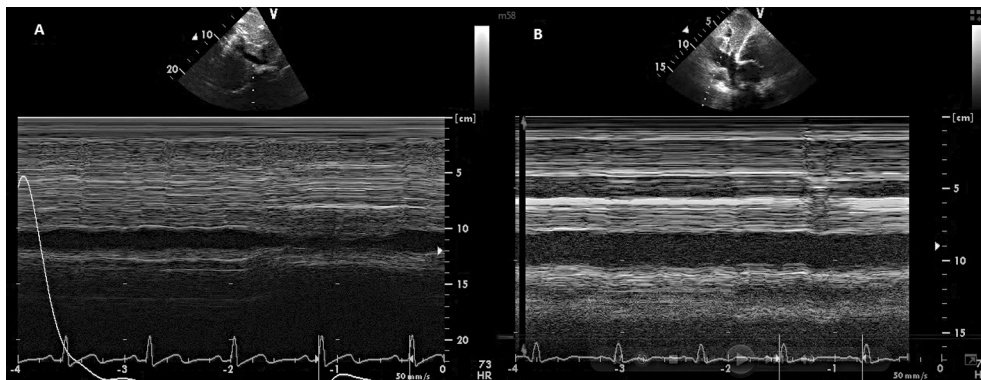


Figure 15. Collapse of inferior vena cava (IVC). **Panel A:** IVC diameter < 2.1 cm that collapses $> 50\%$ with a sniff: normal right atrial (RA) pressure of 3 mm Hg (range, 0–5 mm Hg). **Panel B:** IVC diameter > 2.1 cm that collapses $< 50\%$ with a sniff suggests high RA pressure of 15 mm Hg (range, 10–20 mm Hg). (See text for explanation)

assessment of the RV function. In their consensus document, the advantages and limitations were summarized as follows [29]: “Advantages: TAPSE is simple, less dependent on optimal image quality, and reproducible, and it does not require sophisticated equipment or prolonged image analysis.

Disadvantages: TAPSE assumes that the displacement of a single segment represents the function of a complex 3D structure. Furthermore, it is angle dependent, and there are no large-scale validation studies. Finally, TAPSE may be load dependent.”

- TAPSE > 16 mm is considered indicative for normal RV function
- TAPSE < 16 mm implies impaired RV function

TISSUE DOPPLER IMAGING: RV S' OR SYSTOLIC EXCURSION VELOCITY

According to the above-mentioned consensus document [29], Pulsed TDI can be used to measure the longitudinal velocity of excursion RV S' . It is easy to measure, reliable

and reproducible. This Doppler measurement is, however, prone to errors due to suboptimal alignment of the annulus with the Doppler cursor. Lindqvist *et al.* [37] validated this method in a population based study. S' velocity has been demonstrated to correlate well with other measures of global RV systolic function (Fig. 14).

- RV S' velocity > 15 cm sec^{-1} at the annulus (RV free wall) is considered normal, with lower velocities at the mid- and apical segments
- RV S' velocity < 10 cm sec^{-1} indicates RV systolic dysfunction

INFERIOR (VENA CAVA) IVC DIMENSIONS

The IVC and its inspiratory collapse can be measured in the subcostal window. IVC diameter should be measured just proximal to the entrance of hepatic veins (Fig. 15). The European Association of Echocardiography recommends quantification as follows [29]:

- IVC diameter < 2.1 cm that collapses $> 50\%$ with a sniff, suggests normal RA pressure of 3 mm Hg (range, 0–5 mm Hg)

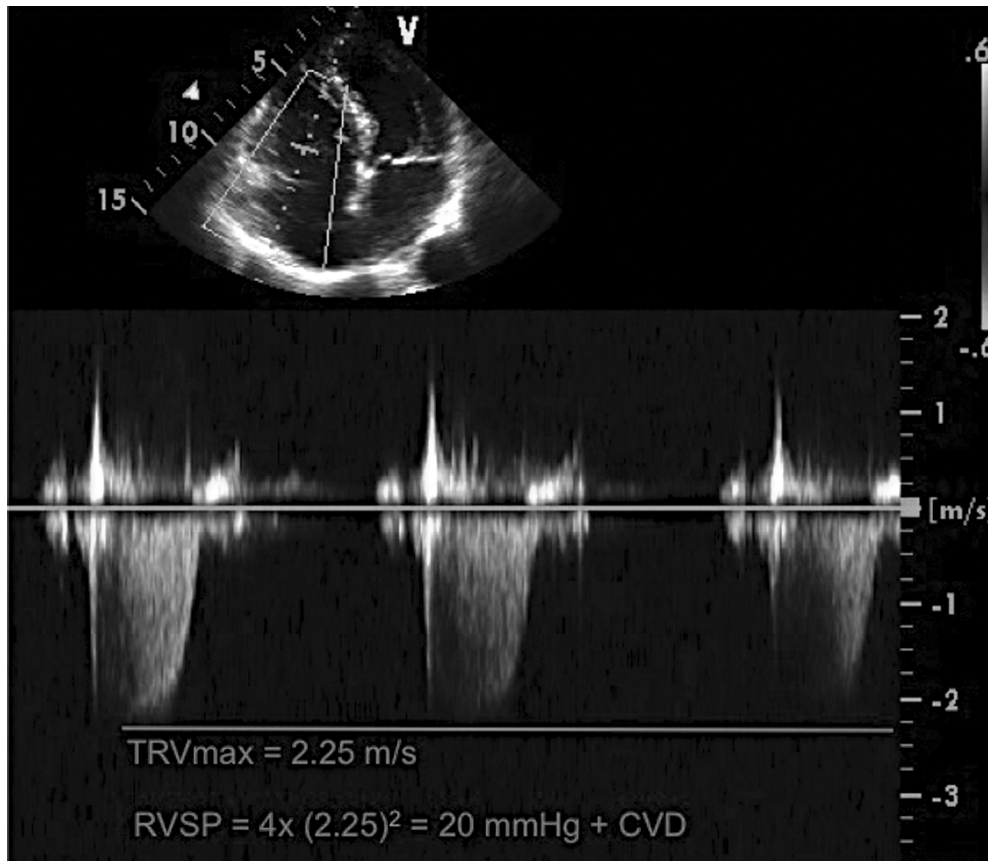


Figure 16. Tricuspid regurgitation signal. This figure shows a Tricuspid Regurgitation velocity measurement from the 4 chamber view. The peak velocity equals 2.3 m s^{-1} . According to the simplified Bernoulli equation, the systolic pressure gradient between RV and RA = $4 \Delta V^2 = 4 \times (2.6)^2 = 21.2 \text{ mm Hg}$. **Panel A** from figure 15 was obtained the same patient. Right atrial pressure (RAP) was determined to be 3 mm Hg. Right ventricle systolic pressure (RVSP) is calculated using the simplified Bernoulli equation: pressure = $4 \times$ velocity (in meters/second) squared. Thus, RVSP = RAP + TR gradient = $3 + 21.2 \text{ mm Hg} = 24.2 \text{ mm Hg}$.

- IVC diameter > 2.1 cm that collapses < 50% with a sniff, suggests high RA pressure of 15 mm Hg (range, 10–20 mm Hg)
- In scenarios in which IVC diameter and collapse do not fit this paradigm, an intermediate value of 8 mm Hg (range, 5–10 mm Hg) may be used
However, there are constraints to the use of IVC and its collapse in patients on the ICU:
 - The IVC is commonly dilated and may not collapse in patients on ventilators
 - IVC may be dilated in the presence of normal pressure in normal young athletes

SYSTOLIC PULMONARY ARTERY PRESSURE (SPAP) AND RIGHT VENTRICULAR SYSTOLIC PRESSURE (RVSP)

Assuming the absence of a relevant right ventricular obstruction, Tricuspid Regurgitation (TR) velocity reliably permits estimation of RVSP with the addition of Right Atrial (RA) pressure, using an RA pressure estimated from IVC dimension and its collapsibility. In order to estimate pres-

sure gradients out of maximal velocity, one needs to use a simplified Bernoulli equation [13]:

$$\Delta P = 4V^2$$

An estimated TR velocity of 3 m sec^{-1} will thus correspond to a gradient of 36 mmHg. In order to estimate RVSP, we need to add RA pressure. This can be estimated using the IVC protocol from the previous section. As SPAP is also stroke volume dependant and may increase with age, SPAP may not always indicate increased pulmonary vascular resistance (PVR) (Fig. 16).

FLUID RESPONSIVENESS

The primary question that fluid responsiveness monitoring seeks to answer whether the patients’ CO will increase after volume expansion [38]. The cardiac ultrasound quantification methods to measure stroke volume, described in an earlier section, may contribute to answer this difficult question. Most of these methods evaluate

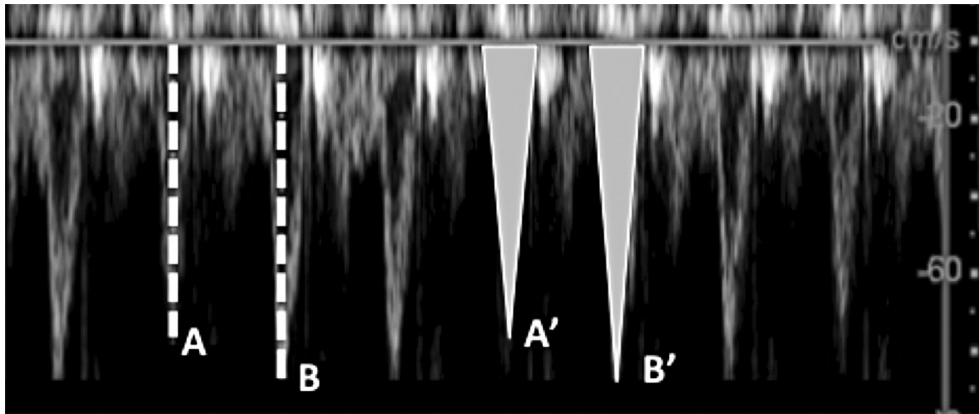


Figure 17. Flow variation on left ventricular outflow tract. Pulsed wave analysis at the level of the left ventricle outflow tract (LVOT). Fluid responsiveness is indicated by large variations (> 10–15%) between expiratory (A) and inspiratory (B) values of peak velocity (cm/sec) or velocity time integral (A' and B' respectively).

the changes in stroke volume after fluid challenging by the administration of a bolus of fluid. Alternatively, they use the passive leg raise method to increase venous return to the right atrium. In the medical community, measurement of changes in IVC dimensions after administration of an i.v. bolus of fluid is of particular interest, because of its technical simplicity.

It should be noted that although the echocardiographic methods to predict fluid responsiveness are promising, nowadays there is lack of robust validation of these methods among the different subpopulations of patients in the ICU. Further research is needed before practical guidelines for their daily use can be made.

LEFT VENTRICULAR OUTFLOW TRACT (LVOT) VELOCITY TIME INTEGRAL (VTI) VARIATION WITH VOLUME LOADING

In critically ill patients, the variation of CO and VTI after the administration of 50 mL crystalloid solution over 10 seconds can accurately predict fluid responsiveness [39]. The utility of this method of fluid challenging to determine fluid responsiveness has also been demonstrated in mechanically ventilated children in the postoperative period [40, 41] (Fig. 17).

An increase in VTI > 15% after administration of 50 mL crystalloid solution over 10 seconds predicts fluid responsiveness [39].

LEFT VENTRICULAR OUTFLOW TRACT (LVOT) VELOCITY TIME INTEGRAL (VTI) INCREASE WITH PASSIVE LEG RAISE (PLR)

Passive leg elevation (PLR to 45°) results in increased venous return to the right atrium. Using the NICOM (Non-Invasive Cardiac Output Monitor; Cheetah Medical, Tel Aviv, Israel), recent studies found PLR to be a promising tool for

the evaluation of fluid responsiveness [42, 43]. Echocardiographic assessment of changes in stroke volume due to PLR have also been demonstrated to be useful predictors for fluid responsiveness [44]. However, it is questionable if this method has been sufficiently validated among the different subgroups of ICU patients, in order to make general recommendations for its use. We were also unable to find a consensus threshold for the change in stroke volume, allowing discrimination of the responders from the non-responders (in general a 5 to 10% increase is used).

LEFT VENTRICULAR END DIASTOLIC AREA (LVEDA)

This a controversial method to determine fluid responsiveness. At the level of the papillary muscles in the parasternal short axis window, the area of the left ventricle at end-diastole is measured by tracing the endocardial border [45] (Fig. 18). It has been advocated that:

- An LVEDA of less than 10 cm² or a LVEDA index (LVEDA/BSA) of less than 5.5 cm m⁻² indicates significant hypovolaemia (normal range of LVEDAI is between 8 to 12 cm m⁻²)
- An LVEDA of more than 20 cm² suggests volume overload

It should be mentioned that severe concentric hypertrophy can reduce LVEDA even without any hypovolaemia. Furthermore, there is lack of consensus on the use this method for the assessment of fluid responsiveness. Cannesson *et al.* stated that LVEDA should not be used to predict fluid responsiveness, as it is inaccurate and requires too much technical skill and training [38]. Given these major criticisms, its use cannot be promoted [46].

IVC COLLAPSIBILITY INDEX

The IVC collapsibility index is expressed as the difference between the value of the maximum and minimum

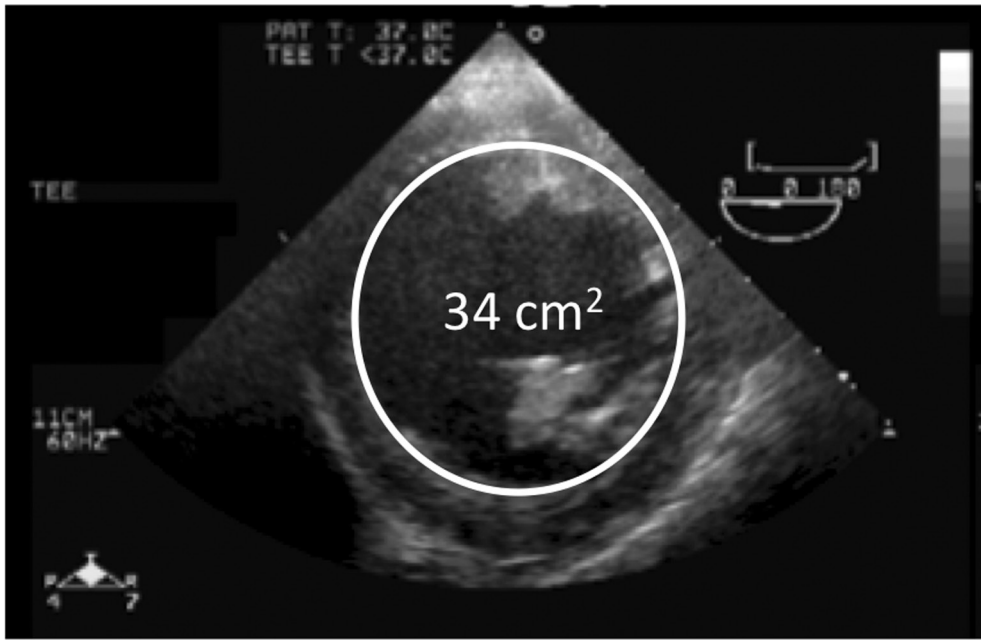


Figure 18. Left ventricular end diastolic area. Large left ventricular end diastolic area obtained with transesophageal echocardiography in a patient with dilated cardiomyopathy. Note that the papillary muscles are included within the surface area

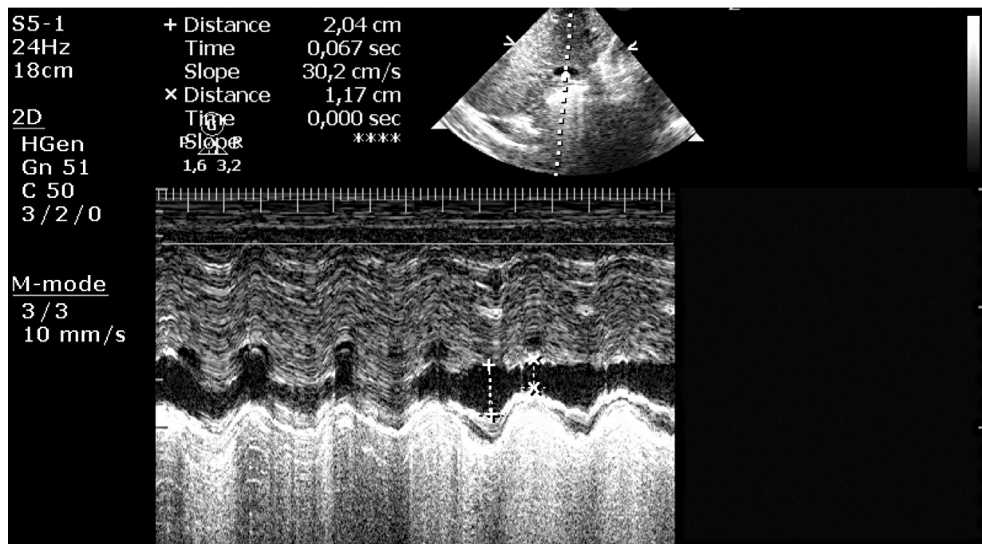


Figure 19. Fluid responsiveness. Inferior vena cava collapsibility index (IVCCI) of 50% in a patient with shock who was severely underfilled and fluid responsive

diameters, divided by the maximum of the two values. This is an index right atrial pressure (see previous section) and volume status (Fig. 19).

Given the relative simplicity of the measurement technique and its noninvasive nature, the use of these parameters to predict fluid responsiveness seems very attractive. Moreover, it is known that changes in both IVC and CVP are apparent during an infusion of a standardized fluid bolus.

Stawicki *et al.* demonstrated that the dynamic change in IVC as a measurement of responsiveness to a fluid bolus

is inversely related to changes seen in CVP in patients in the surgical ICU. They also found that an IV bolus tends to produce an early response in IVC, while the CVP response is more gradual [47]. However, other studies have shown that bedside ultrasonographic measurement of the inferior vena cava fails to predict fluid responsiveness in the first 6 hours after cardiac surgery [48] and hemodynamic response to early hemorrhage [49]. Despite its promising potential, IVC collapsibility to bolus fluid challenging cannot be recommended as a predictor of fluid responsiveness. Thus, further research is needed.

CONCLUSION

Hemodynamic assessment by echocardiography is an important if not vital tool in unstable critically ill patients admitted to the ICU. The technique enables fast and accurate bedside diagnosis, allowing focussed treatment. Cardiac ultrasound is feasible in almost all ICU patients, while even suboptimal image quality will not impede the measurement of Doppler signals that can provide important clues for ICU physicians. Cardiac output, MAPSE, and TAPSE can be used in order to obtain an insight into systolic cardiac function within minutes. After a focused training ICU physicians can learn to “eyeball” ventricular function rapidly. Basic training allows one to evaluate diastolic filling patterns and to guide fluid management. Thus, we suggest adding emergency and critical care cardiac ultrasound to the core curriculum of ICU physicians. The consensus document of the European Society of Cardiology on emergency echocardiography can serve as a guide in order to identify the necessary standards one should attain to become a skilled critical care cardiac sonographer.

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