medicina *intensiva*

UPDATE IN INTENSIVE CARE MEDICINE: HEMODYNAMIC MONITORIZATION IN THE CRITICAL PATIENT

Role of echocardiography in the hemodynamic monitorization of critical patients[☆]

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Abstract The use of echocardiography in intensive care units in shock patients allows us to measure various hemodynamic variables in an accurate and non-invasive manner.

By using echocardiography not only as a diagnostic technique but also as a tool for continuous hemodynamic monitorization, the intensivist can evaluate various aspects of shock states, such as cardiac output and fluid responsiveness, myocardial contractility, intracavitary pressures, heart–lung interaction and biventricular interdependence.

However, to date there has been little guidance orienting echocardiographic hemodynamic parameters in the intensive care unit, and intensivists are usually not familiar with this tool.

In this review, we describe some of the most important hemodynamic parameters that can be obtained at the patient bedside with transthoracic echocardiography in critically ill patients.
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Papel de la ecocardiografía en la monitorización hemodinámica de los pacientes críticos

Resumen El uso de la ecocardiografía en las unidades de cuidados intensivos para los pacientes en estado de shock permite la medición precisa de varias variables hemodinámicas de una forma no invasiva.

Mediante el uso de la ecocardiografía, no como un instrumento diagnostico sino como herramienta de monitorización hemodinámica continua, el intensivista puede evaluar varios aspectos de los estados de shock, como el gasto cardíaco y la respuesta de fluidos, contractilidad miocárdica, las presiones intracavitarias, la interacción corazón-pulmón y las interdependencia biventricular.

Sin embargo, hasta la fecha, ha habido pocas guías que orienten respecto a los parámetros hemodinámicos ecocardiográficos en la unidad de cuidados intensivos, y la mayoría de los intensivistas por lo general no están familiarizados con esta herramienta.

En esta revisión, se describen algunos de los parámetros hemodinámicos más importantes que pueden obtenerse con la ecocardiografía transtorácica en los pacientes críticos.

Introduction

Hemodynamic instability is common in critical patients, and the usefulness of monitorization systems in situations of shock is based on the capacity to obtain quantifiable and reliable hemodynamic variables able to assess preload (central venous pressure or pulmonary capillary pressure), afterload (vascular resistances), and contractility (ventricle function and cardiac output, CO).[1] Once these variables have been obtained, they can be grouped to establish hemodynamic profiles in specific clinical situations producing shock, such as hypovolemia, left or right ventricle dysfunction or diminished peripheral resistances, and to assess different anatomical structures (pericardium, major vessels and heart valves).

The need to obtain continuous hemodynamic information in these patients, and the ongoing controversy over use of the Swan-Ganz catheter, have focused attention on the usefulness of ultrasound in determining systolic and diastolic function indexes that can be used for monitoring the cardiovascular system.[2]

The role of echocardiography as a useful tool for the evaluation and monitorization of cardiovascular function in these patients has been clearly established, with class A indication as recently defined by the recommendations for the appropriate use of echocardiography.[3] This is because in addition to its applicability at the patient bedside, its non-invasiveness in the case of transthoracic echocardiography (TTE) or semi-invasive nature in the case of transesophageal echocardiography (TEE), the technique offers further advantages of great importance in evaluating patients with hemodynamic instability, such as immediate (real-time) image analysis and the obtainment of reliable etiological as well as functional parameters.[4–6]

Usefulness of echocardiography in situations of hemodynamic instability in the critical patient

The current technological advances applied to echocardiography make it possible to study most patients via the transthoracic route, using the standard windows and planes (Fig. 1) from which clinically applicable conclusions can be drawn.

Two-dimensional (2D) echocardiography[7] allows us to visualize most of the heart structures, with an assessment of the morphology and size of the right and left cavities, cardiac walls and of the presence of masses within the heart chambers. This information is basically qualitative, and Doppler ultrasound[8] in its different modes (pulsed, continuous and color) is required to obtain information on the direction and velocity of blood flow in the different chambers and major vessels. Knowing the flow velocity, and simplifying the Bernoulli equation, we can calculate the pressure gradient between the cavities, establish the valve areas and determine the intracavitary pressures of the different heart chambers.

TEE is reserved for situations where the existing window is inadequate or suboptimal for TTE; when we need to evaluate structures that are difficult to examine with TTE (e.g., the appendages, thoracic aorta or valve prostheses); or where a diagnosis must be established in which high imaging quality is crucial—as in acute aortic syndrome, endocarditis and its complications, and the detection of thrombi or masses, or prosthesis dysfunction.[9,10]

As with all imaging techniques, its main limitation is dependency on operator experience and on the apparatus being used. Adequate training and capacitation are therefore needed. Recent articles[11–14] show the usefulness of different echocardiographic methods in obtaining hemodynamic measurements that can be used for both diagnostic purposes and for analyzing the variability and changes induced by applied treatments.

Likewise, focused ultrasound and echocardiography (FUSE)[15,16] has been shown to be useful in critical, emergency and out-hospital patient care.[17,18] In this context, a basic two-dimensional echocardiographic study allows us to establish or discard clinical situations based on binary (yes/no) responses to ensure the provision of urgent treatment.

A series of basic questions must be considered on performing echocardiography in the critical patient under conditions of shock.

How is patient left ventricle function (LVF)?

The analysis of LVF is one of the basic elements and the first to be considered before carrying out volume replacement and/or providing inotropic support. There are multiple causes of reversible left ventricle myocardial dysfunction in non-cardiac critical disease in the Intensive Care Unit (ICU): pancreatitis, sepsis, neurogenic causes, intoxications, brain death and anaphylaxis. A left ventricle dysfunction rate of up to 40% has also been described in septic shock.

The quantitative methods used for estimating left ventricle systolic function with echocardiography include linear determinations in M-mode, two-dimensional measurements in 2D-mode, and calculations derived from intracardiac flows using Doppler echocardiography (Table 1).

At basic level, and following the criteria of the Focused Echocardiography Entry Level (FEEL) protocol,[19,20] the evaluation of LVF should be made visually with the purpose

Table 1 Echocardiographic parameters in evaluating left ventricle function.

Evaluation of left ventricle function
Echocardiography, two-dimensional and M-mode:
Size and configuration of cavities.
Global systolic function and ejective phase indexes:
Ejection fraction (EF)
Shortening fraction (SF)
Global and regional systolic function of LV and RV.
Detection and assessment of pericardial effusion.
Assessment of valve anatomy.
Echo-Doppler:
Calculation of cardiac output.
Evaluation of regurgitations and valve stenoses.
Evaluation of diastolic function.
Estimation of pulmonary capillary pressure (PCP).

of establishing whether the left ventricle (LV) is dilated, and whether function is normal or moderately/severely depressed, in order to integrate the response in the clinical context, with a view to defining a correct management strategy.

Subjective visual estimation of the ejection fraction (EF) is widely used in daily clinical practice, though experience is required on the part of the explorer in order to establish the different degrees of dysfunction (<30% severely depressed, >30% to <40% moderately depressed, >40% to <55% slightly depressed and >55% normal) with acceptable correlation to the quantitative determinations.[21]

The extreme values are easily identified. In this context, a group of intensivists with 2 h of theoretical training and only 4 h of practice, using a portable echocardiograph, were able to correctly identify normal LVF in 92% of the cases and depressed LVF in 80% of the cases—the most common error being the overestimation of ventricle function.[22]

Other forms of quantitative evaluation require greater experience. With the transducer in the parasternal longitudinal plane and combining two-dimensional imaging
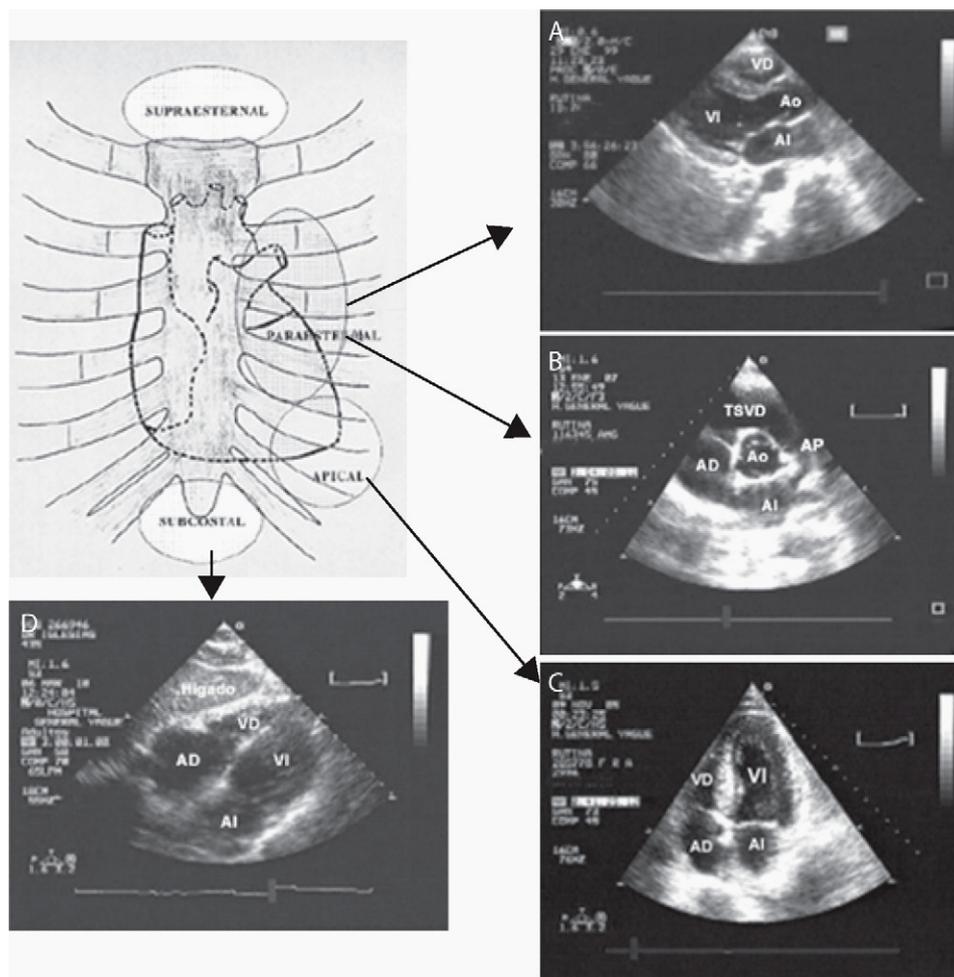

Figure 1 Schematic representation of windows and images obtained by transthoracic echocardiography. Parasternal longitudinal plane of the LV (1A) showing the left atrium (LA), left ventricle (LV), aorta (Ao) and right ventricle (RV). Parasternal transverse plane at major vessel level (1B), with the right ventricle outlet tract (RVOT), pulmonary artery (PA) and right atrium. Apical four-chambers plane (1C), and subcostal plane (1D) showing the right and left cavities.

with the M-mode, the ultrasound beam should be made to section the LV as perpendicular as possible (never tangentially), in order to register the smallest diameters, the end-diastolic diameter (DEd coincides with the peak of the R-wave of the ECG tracing) and the end-systolic diameter (DEs with the maximum excursion point of the endocardial margin). From these values we calculate the shortening fraction $(SF = (DEd - DEs)/DEd \times 100)$, which expresses the percentage relationship between the diastolic and systolic diameters standardized with respect to the diastolic diameter (normal value > 30%), and the ejection fraction, which relates the end-diastolic ventricular volume (VEd) and the end-systolic ventricular volume (VEs), standardized with respect to the end-diastolic volume $(EF = (VEd - VEs)/VEd \times 100)$.

The technique has important limitations in the case of non-global structural and functional alterations (ischemic heart disease) or when the section plane does not meet the required perpendicularity between the cardiac walls. It is therefore very dependent upon the operator. The methodology is also dependent upon preload and contractility, and only assesses the function of the segments sectioned by the ultrasound beam. Accordingly, function of the left ventricle is estimated assuming contraction to be symmetrical. Nevertheless, the parameters are easy to obtain, reproducible and reliable for evaluating function, since no mathematical model needs to be assumed.

In relation to the different methods available for measuring the ejection fraction with two-dimensional echocardiography,[23–25] the so-called modified Simpson method has been established by consensus as the option of choice. This method assumes the volume of the left ventricle to conform a series of ellipses stacked over the length of the cavity, using measurements in orthogonal planes to determine the systolic and diastolic volumes. The standard planes are the apical four-chamber (4C) and two-chambers (2C) planes, taking special care not to include the papillary muscles in the planimetric tracing of the cavities, using a complex formula included in the measuring software of the system. In this way, calculation is made of the end-systolic volume (ESV) and the end-diastolic volume (EDV), with the ejection fraction being derived from the formula: $EF = (EDV - ESV)/EDV$. The main limitations of the method are the difficulty in many cases of delimiting the endocardial margin, and the establishment of an apical sagittal plane at true apical level (of lesser thickness than the surrounding level), where it is very common to obtain a two-chambers apical acquisition tangential and not orthogonal to the four-chamber apical acquisition.

These methods analyze LVF globally, but can also be used to assess segmental systolic function. In this context, in the presence of myocardial ischemia we observe a decrease in contractility of the affected ventricular segments that can be evidenced by echocardiography as a decrease in segment motility and systolic thickening (hypokinesia). In turn, if ischemia is prolonged, motility and systolic thickening are abolished (akinesis).

The left ventricle model currently accepted for the analysis of segmental contractility[26] includes 17 segments obtained from apical four-, two- and five-chamber planes, and parasternal transverse planes in the three sections.

Table 2 Echocardiographic parameters in evaluating right ventricle function and central venous pressure.

Evaluation of right ventricle function

Basic level. Discard acute cor pulmonale:
RV dilatation and LV relationship.
Paradoxical septal motion
TAPSE
Dilatation of inferior vena cava, non-collapsible

Advanced level. Evaluate RV function:
Dimensions of RV and RA
Systolic function and BSA
Pulmonary artery flow: type and acceleration time
Systolic pulmonary artery pressure due to TI
Doppler tissue imaging (DTI)
Inferior vena cava and right atrial pressure

How is patient right ventricle function? Is there acute cor pulmonale?

Until recently, right ventricle function (RVF) has been ignored, partly due to a lack of awareness of its importance, and partly because of technical difficulties in assessing function in a reliable and reproducible manner. Under normal conditions, the right ventricle (RV) functions as a low-pressure chamber, with great capacity to adapt to volume overload, but with a poor response to pressure increments in the pulmonary territory. However, knowledge of RVF is of great diagnostic interest in pulmonary embolism, acute respiratory distress syndrome or cardiac tamponade, and for assessing the interactions with function in patients subjected to mechanical ventilation.

With the use of different planes and despite the anatomical and technical difficulties involved (complex geometry of the RV, with an irregular, half-moon shape and thin walls, an irregular endocardial surface with the presence of a moderator band and trabeculae that make it difficult to recognize the endocardial margins), echocardiography[27,28] allows us to study the global right-side cavities, particularly with the two-dimensional mode, which serves as a guide for measurements in M-mode and orientation of the ultrasound beam in the Doppler technique (Table 2).

According to the echocardiographic protocol, the parasternal longitudinal plane (LP) is generally the first plane obtained, and reveals the size of the RV and its relationship with the LV. It therefore offers a first impression of the presence or absence of dilatation: an end-diastolic diameter of the RV in LP is >30 mm is considered pathological (normal = 9–26 mm in left lateral decubitus).

The apical four-chambers plane is the projection offering the best information and with which it is easier to obtain measurements of both the right atrium and right ventricle. In general, different studies have demonstrated the possibility of measuring[29] the diameters, establishing the areas via planimetry, and of calculating the ejection fraction, though the procedure is difficult due to the geometry of the RV. In this context, different methods have been developed, involving the measurements of different diameters, that are scantly reproducible in routine emergency practice.
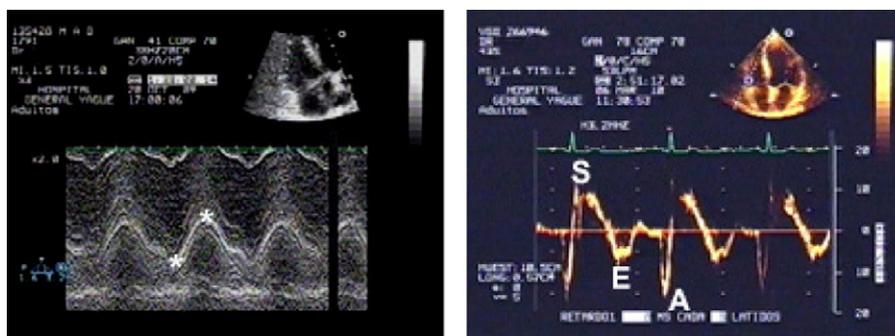
Figure 2   Tricuspid annular plane systolic excursion (TAPSE) represented between the two asterisks (1) and registry of systolic wave velocity using Doppler tissue imaging (DTI) (2), showing a systolic wave (S) and two retrograde E- and A-waves. Normal registries.

For these reasons, analysis is made of the relative size relationship between RV and LV at the end of diastole (normal ratio < 0.6 with RV < LV; moderate dilatation RV = LV; severe dilatation RV > LV, with ratio > 1).

The interventricular septum (IVS) is a muscle wall separating the right ventricle (RV) from the left ventricle (LV), though it is anatomically and functionally part of the latter. The diameter in diastole is 7–11 mm, which is three-fold greater than the RV free wall, with a systolic thickening of 35%.

Ultrasound is the only technique allowing us to study interventricular dynamics and inter-dependency. Mobility of the IVS can be analyzed using two-dimensional ultrasound, particularly in the parasternal transverse plane, at mitral valve level. The LV appears as a circle and the septum as an arc encompassing 2/5 of the circumference. Under normal conditions the IVS is convex towards the RV and concave towards the LV, and maintains this morphology over the entire cardiac cycle. Movement alterations in the form of systolic fluttering, or the configuration of the RV in both systole and diastole, can be used to identify the existence of diastolic or systolic overload. In situations of RV diastolic overload, the IVS undergoes flattening during diastole, since the RV diastolic pressure equals or exceeds that of the LV. Systolic overload, in the presence of greater pressure in the RV than in the LF, is recognized by the presence of a flattened septum (septum in D) in both systole and diastole, with diminished dynamics.

The analysis of segmental contractility alterations acquires greater importance in right ventricle infarction, which is typically characterized by akinesis or dyskinesia of some of the explored surfaces in the different projections. In pulmonary embolism, in the presence of pressure overload, the McConnell sign has been described as contractility alteration in the form of akinesis of the free wall but without affecting the apical zone—though its usefulness is questionable.[30]

Tricuspid annular plane systolic excursion (TAPSE) is measured in M-mode and represents the excursion or distance displaced by the tricuspid ring from the end of diastole to the end of systole.[31] To this effect, and in the apical four-chambers plane, the M-mode cursor is positioned at the free margin of the tricuspid ring, with measurement of its systolic displacement. It is not influenced by heart rate (HR) but is affected by pre- and afterload. This is an easy method for assessing right ventricular contractility. Values of under 15 mm are considered pathological and of prognostic value (Fig. 2).

Registry of the maximum velocity of the systolic wave using Doppler tissue imaging (DTI)[32] is carried out at RV lateral wall level in four-chambers apical acquisition. A positive wave is recorded after QRS, and is preceded by another wave of shorter duration, corresponding to isovolumetric contraction. Normality is taken to be 15 ± 2 cm/s, while RV dysfunction is considered to be present when <10 cm/s (Fig. 2).

By using ultrasound in its M and 2D modes, we can obtain direct and indirect data relating to pulmonary hypertension, suspect its presence even though its evaluation is not quantitative, and study its repercussions upon the heart valves and chambers. However, Doppler echocardiography[33] is the technique that informs of and quantifies pulmonary hypertension.

Analysis of the morphology of pulmonary flow is carried out in the parasternal transverse plane at major vessel level, positioning the pulsed Doppler sample volume at pulmonary valve level. Under normal conditions a triangular pattern is observed, gradually accelerating with a peak in the mid-portion of systole, followed by a slow decrease ending just before valve closure (Type I). The presence of a mid-systolic notch is indicative of severe pulmonary hypertension (Type III), with a sensitivity of 56% and a specificity of 100% for PAPs > 50 mmHg.[34,35] Another very useful and accessible option from the same plane is the measurement of the acceleration time (AT) of pulmonary flow, which is measured from the start of the wave to the maximum velocity (if >90 ms, pulmonary hypertension is discarded).[36–38]

The presence of tricuspid valve insufficiency (TI) is detected as systolic flow in the right atrium, with a maximum velocity that can be quantified by continuous Doppler with the help of color Doppler for correct assessment and positioning of the sample, allowing us to calculate the transvalvular pressure gradient between the right atrium (RA) and right ventricle (RV) from the modified Bernoulli equation: $PRV - PRA = 4V_{max} TI^2$.

Right ventricle systolic pressure equals the sum of the systolic gradient between RA and RV plus the right atrial pressure (RAP), and in the absence of obstruction of the right atrium outlet tract, it equals the systolic pressure of the pulmonary artery, whereby: $PAPs = 4V_{max} IT^2 + RAP$.

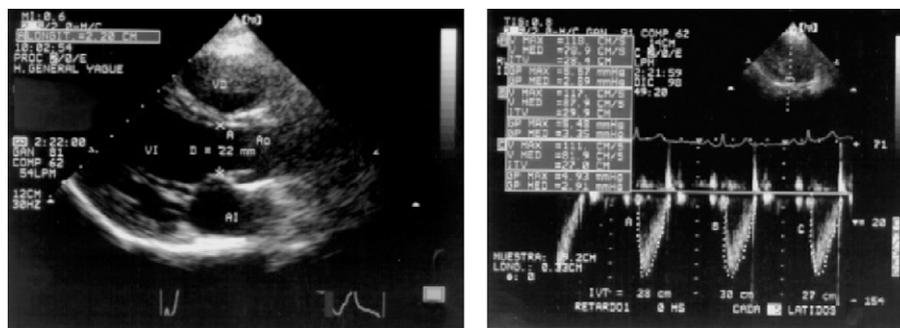Different alternatives are available for determining the mean right atrial pressure to be summed to the gradient:

Figure 3 Obtainment of the diameter of the left ventricle outlet tract (LVOT) in the parasternal longitudinal plane (1) and estimation of the area ($0.785 \times 2.2^2 = 3.79\,cm^2$. We then (2) register the LVOT flow with pulsed Doppler, in the apical 5-chambers plane, and obtain VTI (velocity time integral) (the figure registers three measurements with a mean of 24 cm). The stroke volume (SV) = $3.79 \times 24 = 91$ ml.

- Use of a central venous catheter.
- Use of the diameter of the inferior vena cava (IVC) (normal diameter 16 ± 2 mm) and its inspiratory collapse (IC) index, due to the existence of good correlation between these parameters and right atrial pressure. An IVC ≤ 21 mm with IC > 50% estimates a RA pressure of between 0 and 5 mmHg, while IVC > 21 mm and a non-collapsible vein indicates a pressure of ≥ 15 mmHg.

The reliability of the estimation is well established in the echocardiography laboratory; however, there are technical limitations in patients admitted to the ICU. Mechanical ventilation in particular complicates the technique due to the absence of an adequate echocardiographic window. In non-ventilated patients, the presence of dyspnea and the impossibility of tolerating left lateral decubitus are the main impediments. The presence of arrhythmias, particularly atrial fibrillation, makes it necessary to average the measurements over 5–10 beats. Despite these limitations, the measurement of pulmonary artery systolic pressure using Doppler ultrasound is reliable, with good correlation to the invasive methods.

Is cardiac output normal or reduced?

Cardiac output is an estimator of global cardiovascular system function, and is calculated as the product of heart rate and stroke volume (SV). According to classical hydrodynamics, the volume passing through a certain section can be calculated by multiplying the area of the section or zone (A, cm^2) by the integral of velocity versus time of the flow passing through it (IVT, cm)—this representing the systolic distance traveled by the blood during the measured time period: SV = $A \times$ IVT (Fig. 3).

The area most often used for this purpose in clinical practice is the area of the aortic valve ring. The annular or ring diameter (D) is measured at the level of insertion of the valve cusps or leaflets, which in most cases is correctly visualized in the parasternal longitudinal plane of the left ventricle in TTE and/or in the two-chambers mid-esophageal plane in TEE, assuming a circular geometry. To this effect zoom is applied to the zone at the start of systole, measuring from the insertion or junction of the anterior cusp on the endocardium to the same point of the posterior cusp: area = $\Pi \times (D/2)^2 = 0.785 \times D^2$.

Stroke volume is determined by echocardiography usually by calculating the volume of blood crossing the aortic valve in each beat. Pulsed Doppler yields the flow velocity spectrum at that level, using the apical 5-chambers plane, which allows more parallel alignment between the direction of flow and the Doppler interrogation line. Measurement of the flow (IVT of the left ventricular outlet tract, LVOT) is made placing the sample volume close to the valve area. The normal values are between 18 and 23 cm—a value of <12 cm indicating low output. At present, all echocardiography systems provide IVT (in cm) when the Doppler signal curve is delineated with the incorporated measurement software.

$$CO(cm^3/min) = 0.785 \times D^2 \quad (cm^2) \times IVT(cm/beat) \times$$
$$HR(beats/min)$$

This method,[39] despite all the assumptions upon which it rests and the need for careful and good quality exploration, has revealed acceptable global correlations with the invasive techniques—though with great individual variability and the need for a normal left ventricular outlet tract (LVOT) and the absence of significant aortic insufficiency. The main limitation is represented by errors in measurement of the aortic ring diameter, particularly when considering that such error is moreover magnified by having to square the measure (i.e., raise it to the second power) (Fig. 3).

Precisely with the idea of avoiding this error, Evangelista et al.[40] have shown that we can obviate measurement of the aortic ring, since there is a closer correlation between the cardiac index (CI) estimated by thermodilution and the mean velocity in the LVOT obtained by pulsed Doppler ($r = 0.97$) than between the cardiac index and the estimation made with the usual method described in this section ($r = 0.90$). The resulting regression equation is: CI (ml/min/m^2) = $172 \times$ mean velocity $- 172$.

The standard deviation of the estimation is 0.24 l/min/m^2. The mean flow velocity in the LVOT is an extraordinarily useful parameter for estimation of the cardiac index, and especially for monitoring the changes occurring in the face of new hemodynamic situations or as a result of treatment.

Table 3 Echocardiographic indexes in evaluation of left ventricle filling pressures.

Indexes suggesting elevated PCP
E/A ratio ≥ 2
Mitral filling E-wave deceleration time < 150 ms
DT of the diastolic wave of PVF ≤ 160 ms
Relationship duration reverse PVF wave and mitral A-wave ($AP_{dur} > A_{dur}$)
E/VP > 2.5 → PCP: 15 mmHg
$100/[(2 \times IVRT) + Vp] > 5.5 \rightarrow$ PCP: 15 mmHg
$E/e' > 15 \rightarrow$ PCP: 15 mmHg

Can we estimate the filling pressures?

The filling pressure values classically have been the values provided by the pulmonary artery catheter for hemodynamic assessment of a patient. Posteriorly, with the incorporation of the hemodilution techniques, it became possible to calculate SV, CO and peripheral vascular resistance (PVR). The usefulness of the filling pressures has been greatly questioned as a result of scientific confirmation that they are not valid either for assessing heart function or for predicting response to volume.[41,42]

Nevertheless, in a patient with hemodynamic instability, the knowledge of pulmonary wedge pressure is basic when complemented by important data such as the anatomical characteristics of the left ventricle (hypertrophic/dilated) and its global function. Wedge pressure is an indicator of left ventricle preload, and has diagnostic applications by allowing us to distinguish among the different etiologies of lung edema. Although theoretical preload is correlated to left ventricle end-diastolic volume, in practical terms it is assimilated to wedge pressure. At present, pulmonary capillary pressure (PCP), as a reflection of left atrial pressure, can be reliably estimated from diastolic function parameters obtained by Doppler echocardiography[43–46] (Table 3).

Mitral valve filling flow

During diastole, left ventricle filling is explored by pulsed Doppler (Pw), placing the sample volume at the free margin of the mitral valve leaflets in the apical four-chambers plane. At each timepoint during diastolic filling, the velocity represented by the wave reflects the instantaneous pressure gradient between the left atrium and ventricle, in accordance with the Bernoulli equation.

A recording composed of two waves is obtained in individuals under sinus node rhythm: the E-wave, corresponding to rapid protodiastolic filling, and the A-wave, dependent upon atrial contraction. Likewise, on placing the sample volume of the pulsed Doppler between the mitral valve and the left ventricle outlet tract, in the apical 5-chambers plane and using color Doppler as a guide, we obtain the systolic flow curve at that level and can thus measure the isovolumetric relaxation time (IVRT). Its duration is measured between the aortic flow closing artifact and the start of the mitral flow (E-wave).

Typically, the normal mitral filling flow curve in a middle-aged individual presents an E-wave (velocity 80 ± 16 cm/s) that is slightly larger than the A-wave (56 ± 13 cm/s), with

an E/A ratio > 1, an E-wave deceleration time (DT) of about 180 ms (199 ± 32), and an IVRT in the order of 90 ms (69 ± 12 ms).

The diastolic ventricular filling patterns are the result of the interaction between filling flow and the diastolic properties of the LV and the loading conditions.[47,48] Thus, the mitral flow pattern affords global and nonspecific information on diastolic function, resulting from interaction among the ventricular relaxation state, ventricular compliance and the left atrial pressure value. Accordingly, the morphology of the curve is modified by different factors, such as the existing hemodynamic conditions, heart rate and age. Its characteristics and the evolution of the filling patterns in relation to the degree of diastolic dysfunction are summarized in Figs. 4 and 5.

The mitral filling flow velocity curve is technically very simple to obtain, reproducible and easy to perform at the patient bedside. The Mayo Clinic[49] investigated the relationship between these parameters and filling pressure in two groups of patients: 42 subjects with left ventricular systolic dysfunction (EF < 40%) and 55 patients with hypertrophic myocardiopathy. In this clinical context, left atrial pressure (LAP) was inversely correlated to the DT of the E-wave ($r = 0.73$, $p < 0.001$) and directly correlated to the E/A ratio ($r = 0.49$, $p = 0.004$). An E/A ratio of ≥ 2 had low sensitivity (52%) but high specificity (100%) in detecting LAP ≥ 20 mmHg. In the case of DT < 180 ms there was an associated PCP of ≥ 20 mmHg, with a sensitivity and specificity of 100%. Due to their dependence upon the ventricular relaxation state, these parameters were only useful in the advanced systolic dysfunction group (EF < 40%) and under conditions of sinus rhythm.

This study coincides with earlier findings[50–52] in patients with normal systolic function, where none of these measures showed sensitivity and specificity levels acceptable for clinical use, except in patients with depressed EF.

E-wave velocity by color Doppler in M-mode (Vp)

For recording in the apical four-chambers plane, we analyze the color Doppler signal corresponding to mitral filling, adjusting the depth to include the entire left ventricle from the mitral valve to the apex (approximately 45 mm). After zooming in on the zone, we align the cursor of the M-mode at the center of the color signal. We thus obtain a wave corresponding to propagation of the color Doppler in M-mode, which almost instantaneously reaches the apex of the LV in individuals with normal relaxation. After freezing the image, we measure Vp as the gradient of the line separating first aliasing of the early diastolic flow (blue/red transition) from the mitral valve ring to the apex (normal > 60 cm/s). Theoretically, it could also be measured as the gradient of any of the isovelocity lines; to this effect it is useful to modify the color Doppler baseline.

It has been shown that Vp is independent of mean atrial pressure and is closely correlated to *tau*; as a result, it can be used as an estimator of ventricular relaxation.

Two combined parameters have been found to be of practical use:

- Usefulness of the ratio of the peak E-wave velocity to propagation velocity (E/Vp) in estimating PCP. An E/Vp
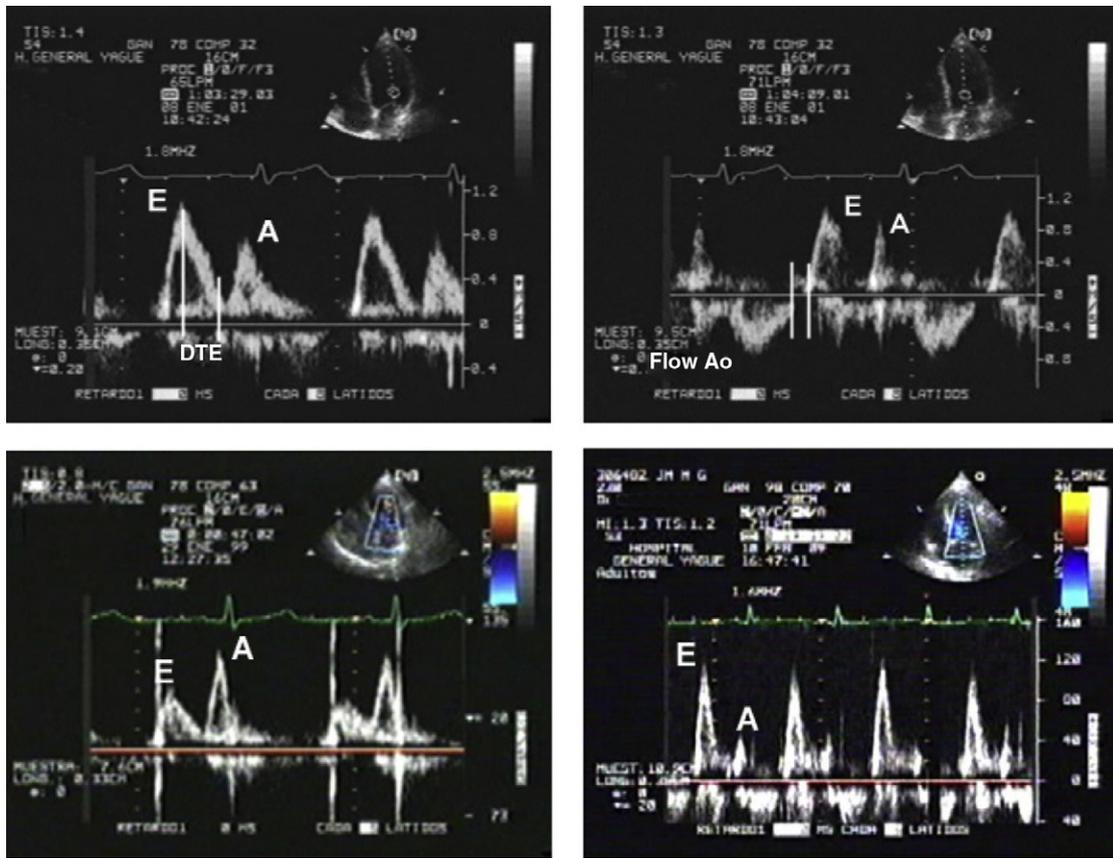
Figure 4 Pulsed Doppler recording showing the isovolumetric relaxation time (IVRT) and the ventricular filling patterns in normal filling (1 and 2) and inadequate relaxation (3), the restrictive pattern (4) and its relation to the ECG tracing. An inadequate relaxation pattern (2) is characterized by a decrease in E-wave velocity, an increase in A-wave velocity, E/A ratio < 1, and a prolongation of the deceleration time (DT) of the E-wave and of IVRT. Diminished ventricular elasticity in turn produces an increase in left atrial pressure that implies a filling pattern inverse to the previously described pattern, known as the restrictive pattern (4), with an increase in E-wave velocity, a decrease in A-wave velocity, and shortening of DT and IVRT. In the progressive transition between the inadequate or delayed relaxation pattern and the restrictive pattern, mitral flow may present a ''pseudonormal'' morphology, that can shift to an inadequate relaxation pattern if the patient performs the Valsalva maneuver.

of >2.5 offers acceptable predictive value in predicting PCP > 15 mmHg ($r = 0.80$, $p < 0.001$)—its value being estimated from PCP = $5.27 \times$ [E/Vp] + 4.6.[53] However, its usefulness decreases in the presence of atrial fibrillation, dilated myocardiopathy with an eccentric filling jet, hypertrophic myocardiopathy, mitral valve stenosis or prosthesis, and in the presence of preserved LV systolic function.

- The combined parameter 1.000/([2 × IVRT] + Vp) is closely correlated to PCP, according to the regression equation
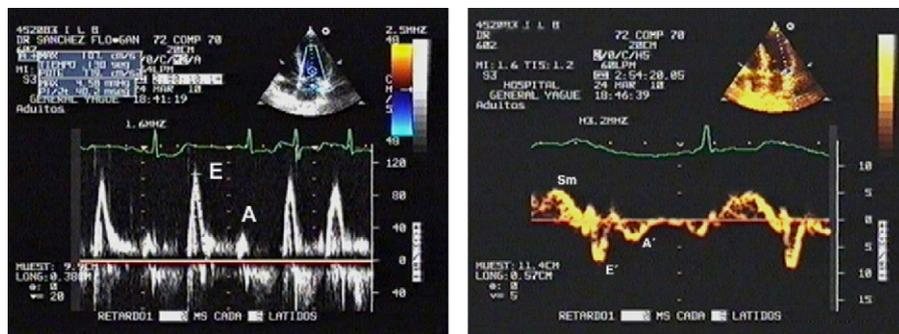

Figure 5 Restrictive mitral filling pattern (1) in a patient presenting Killip class III after anteroseptal infarction, with DT = 130 ms and an E/A ratio of >2. E-wave velocity = 1.07 m/s. DTI (2) with e'-wave velocity = 0.80 cm/s, E/e' ratio > 12, suggesting increased filling pressures.
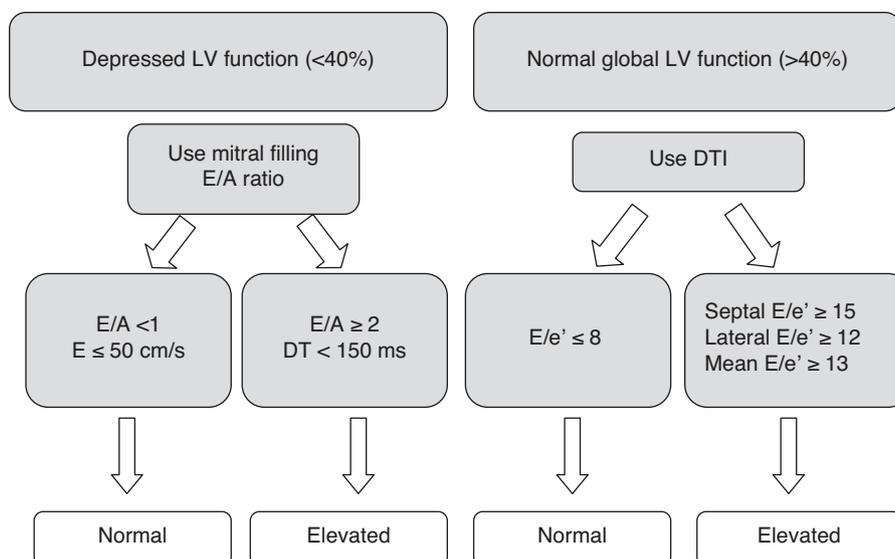
Figure 6   Doppler evaluation of filling pressures. The extreme values have been represented, use being required of the E/Vp ratio, pulmonary vein flow, IVRT/T E/e' < 2, 100/([2 × IVRT] + PV) or combined parameters in the intermediate values.
Source: Adapted from: Nagueh SF, Appleton CP, Gillebert TC, Marino PN, Oh JK, Smiseth OA *et al*. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. Eur J Echocardiogr 2009;10:165–93.

PCP = 4.5 × 1.000/[(2 × IVRT) + Vp] − 9, independently of ventricular systolic function.[54] A value of >5.5 for this parameter discriminates between PCP over or under 15 mmHg in 96% of all cases. In practical terms, IVRT < 80 ms in the presence of a low EF is indicative of decompensation.

Doppler tissue imaging (DTI)
When the ultrasound beam is directed towards the heart, the waves are reflected from the cardiac structures. In the same way as with the red blood cells, mobile tissues such as the myocardium reflect low-velocity Doppler signals. The longitudinal muscle fibers in the heart are located in the basal segments. Positioning of the pulsed Doppler sample, generally in the lateral portion of the mitral valve ring or in the basal portion of the septum, in the apical four-chambers plane, allows us to quantify the maximum velocities of this myocardial zone in the different phases of the cardiac cycle. By means of a series of Doppler signal modifications, we can record a velocity/time curve, obtaining a type of signal referred to as Doppler tissue imaging (DTI). In clinical practice, DTI measurements have been standardized in the mitral and tricuspid ring to determine variables of systolic and diastolic function. A normal registry comprises systolic and diastolic waves:

- Sm, systolic wave, showing two components in some patients, and representing isovolumetric contraction and the ejective systolic phase. A peak systolic velocity Sm > 5.4 cm/s predicts EF >50% with a sensitivity of 88% and a specificity of 97%.

- e', early diastolic wave, representing the rapid filling phase.
- a', late diastolic wave, representing the late filling phase and atrial contraction.

The peak velocity of the E-wave obtained by DTI (e') is correlated to ventricular relaxation and is relatively independent of the preload. The ratio between the peak velocity of the mitral E-wave and the E velocity of the lateral mitral ring (E/e') shows close correlations to PCP (PCP = 1.24 [E/e'] + 1.9)—values above 15 being predictive of PCP > 15 mmHg.[55] Values of <8 in turn are associated with normal PCP values). However, E/e' ratios between 8 and 15 have low predictive value. At present, septal $E/e' \geq 15$, lateral $E/e' \geq 12$ and mean $E/e' \geq 13$ is considered indicative of increased filling pressures.[56]

The method has been validated in the presence of sinus node tachycardia, atrial fibrillation and hypertrophic myocardiopathy. Its limitations are the presence of mitral valve stenosis or prosthesis, moderate to severe mitral valve insufficiency, severe mitral ring calcification and the existence of posterolateral wall akinesis. Recently[57] the recommendations for evaluating diastolic function via echocardiography have been published. In this context, Fig. 6 shows the recommendations for estimating the filling pressures in patients with normal and depressed systolic function.

The clinical usefulness of the described parameters depends on two factors: (a) the experience and technical knowledge of the explorer and the available technology; and (b) the physiological mechanisms that inter-relate the Doppler data with the diastolic properties of the LV, the loading conditions and their variations in different etiological situations, or the response to applied treatment.
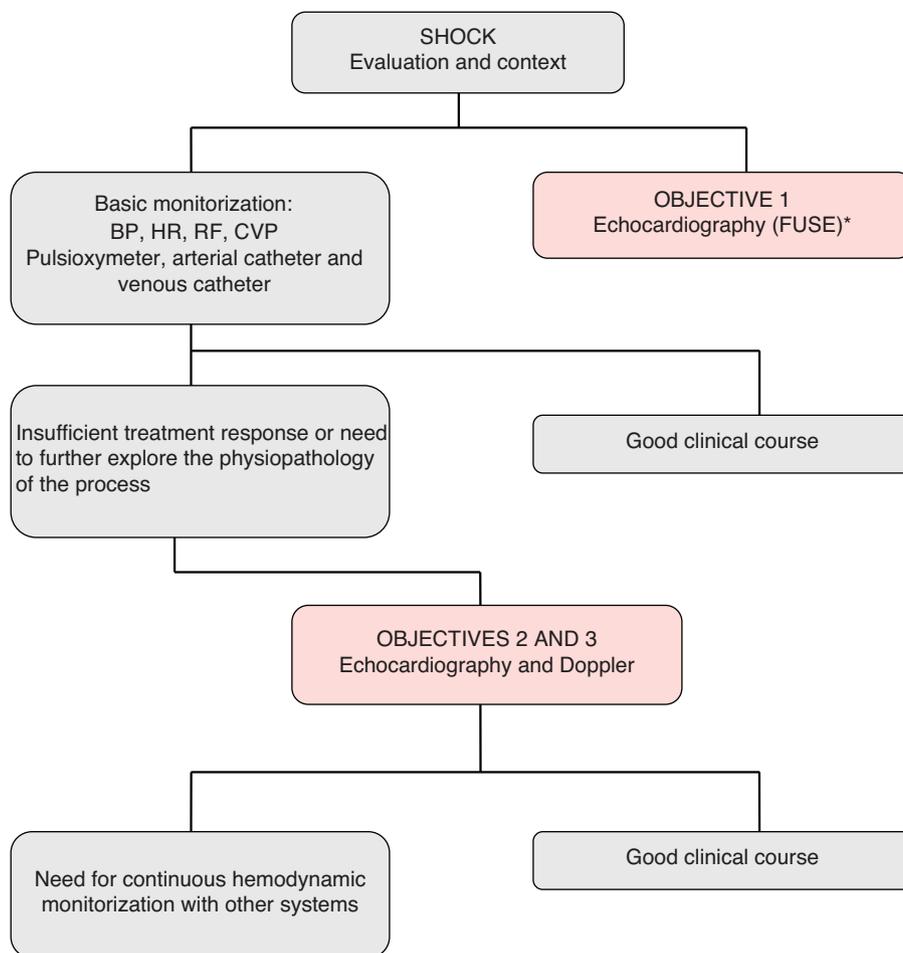
Figure 7 Evaluation and monitorization algorithm based on echocardiography among patients in shock. FUSE: focused ultrasound exam.

The main limitations of these studies are the number of patients involved, their very different characteristics, and the by now classical controversy regarding the relationship between pulmonary wedge pressure and left atrial pressure, and its usefulness as an indicator of left ventricular preload. Thus, no single parameter is correlated in a universal and statistically significant manner to PCP, and we must use and integrate all the information that can be obtained from the different echocardiographic methods, either isolately or in combination.

Nevertheless, data such as the E/A ratio, IVRT, DT of the E-wave in patients with depressed EF, and the E/e' ratio via DTI (at present all systems are equipped with DTI) in patients with normal EF not only can be easily obtained but moreover form part of the routine protocol used in basic echocardiographic studies such as measurement of the size of the different heart chambers or the evaluation of systolic or valve function. Other data, including pulmonary vein flow (PVF), or combined parameters, can be used in those cases in which the mitral filling pattern presents some of the limitations commented above. It should be remembered that all of them are quickly obtainable by transthoracic echocardiography at the patient bedside, and can be repeated as often as necessary.

Is the heart dependent upon preload?

Measurement of the size of the heart cavities (left ventricle end-diastolic surface and volume) has been proposed as reliable preload indexes. However, there are no sufficiently sensitive concrete values allowing us to predict the response to volume expansion.

Recently, Doppler ultrasound and 2D imaging have led to the development of new hypovolemia indicators and to the prediction of response to volume expansion. These parameters should serve to identify those patients who will benefit from volume expansion, increasing their systolic volume (responders) and at the same time avoiding useless and potentially harmful therapy (non-responders). The dynamic parameters, which analyze cardiovascular response to the respiratory changes in pleural pressure produced by a mechanical respiratory cycle, are the variables which have been most extensively studied to date.

Analysis of the diameter of the inferior vena cava (IVC)
The subcostal plane often allows us to evaluate the inferior vena cava and the suprahepatic veins and to indirectly estimate right atrial pressure. Changes in intraabdominal pressure and breathing rapidly modify their volume. During

inspiration, venous return increases and the IVC decreases in diameter. The size and decrease in diameter during inspiration (collapse index: maximum diameter in expiration − minimum diameter in inspiration/maximum diameter in expiration) are related to mean right atrial pressure.

The respiratory variability index of the IVC ($\triangle$IVC) has been defined as $D_{max} - D_{min}/(D_{max} + D_{min})/2 \times 100$, where D_{max} and D_{min} are respectively the maximum and minimum values of the IVC in a mechanical respiratory cycle. The observation of $\triangle VCd \geq 12\%$ allows us to distinguish between volume expansion responders and non-responders, with high sensitivity and specificity.[58]

Analysis of the variability of the diameter of the superior vena cava (SVC)

In patients subjected to mechanical ventilation, transesophageal echocardiography can be used to explore the inspiratory increase in pleural pressure during lung insufflation, which produces a total or partial collapse of the SVC. The observation of collapse suggests that at this point the external pressure exerted by the chest cavity upon the superior vena cava is greater than the venous pressure. In this context, variability in the diameter of the SVC of over 36% has been shown to predict a positive response to volume expansion, with a sensitivity of 90% and a specificity of 100%.[59,60]

Analysis of the variation in aortic peak flow velocity (PFV)

This parameter is obtained by recording aortic flow in the left ventricle outlet tract (see cardiac output). A value $\triangle$PFV ($PV_{max} - PV_{min}/PV_{max} + PV_{min}/2 \times 100$) of 12% can also be used in the evaluation of volume expansion.[61–64]

Conclusions

Echocardiography, by generating a large body of information on the anatomy of the heart, ventricle function and the hemodynamic condition of the critical patient, is becoming increasingly common in the ICU as a diagnostic tool and for evaluating cardiovascular function. Only adequate training can allow intensivists to perform reliable echocardiographic explorations of help in the diagnostic and therapeutic management of situations characterized by hemodynamic instability.

Hemodynamic monitorization using echocardiography has the following objectives:

- **Objective 1**: To exclude serious structural heart disease as the cause of hemodynamic instability, particularly cardiac tamponade, infectious endocarditis, structural valve disease, acute aortic syndrome and acute myocardial infarction and its mechanical complications, based on two-dimensional (2D) imaging.
- **Objective 2**: To monitor right and left ventricle function.
- **Objective 3**: To monitor dynamic parameters of preload, and contractility. Table 4 summarizes the basic echocardiographic parameters in hemodynamic monitorization.

Fig. 7 offers a monitorization proposal based on the echocardiographic evaluation of patients in shock. We

Table 4 Echocardiographic parameters in hemodynamic monitorization.

Preload	Contractility
Diameter of inferior vena cava and RA pressure	Visual estimation visual of LV and RV function
Diameter, area and end-diastolic volume of LV and RV	Systolic collapse of LV cavity
Estimation of filling pressures: E/A and E/e' ratios	Ejection fraction and shortening
Variation infusion of fluids	Cardiac output and VTI Pulmonary flow and TI TAPSE TDI-s of RV free wall

consider that the use of this protocol does not discard other monitorization methods; rather, the protocol can be used as a complement to other techniques depending on the concrete parameters to be examined, the invasiveness allowed by the patient condition, and the continuity considered opportune. In aspects such as the evaluation of extrapulmonary water in patients with respiratory distress syndrome, close monitorization of right-side cardiac function in patients subjected to mechanical ventilation, pulmonary hypertension of any origin, etc., or the definition of tissue perfusion and O_2 transport and consumption (DO_2/VO_2), use must be made of the existing monitorization systems along with echocardiography.

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Conflict of interest

The authors declare no conflicts of interest.

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