The Perioperative Use of Echocardiography for Fluid Management

6

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Abstract

This chapter will discuss some common "static" echocardiographic measurements that can guide fluid management including echocardiographic quantification of ventricle dimensions, areas, and volumes. The chapter will mainly focus on "dynamic" echocardiographic measurements of fluid responsiveness that can guide the perioperative physician in the fluid management of patients in the operating room, the postanesthesia care unit (PACU), and in the critical care unit. These include echocardiographic quantification of inferior vena cava and superior vena cava diameters and collapsibility index to guide fluid therapy. In addition, Doppler ultrasound guided approaches to quantification of stroke volume changes both in mechanically ventilated patients (using respiratory-induced changes) and in spontaneously breathing patients (using the passive leg raising test) will be described.

Key Points

1. Echocardiography can be used as a monitoring tool for fluid management if after a diagnostic assessment, repetitive hemodynamic, or anatomic assessments are being made over a period of minutes, hours, or days in the same patient to guide management.
2. Echocardiographic "static" parameters such as left ventricle end systolic and end-diastolic areas and volume are helpful in differentiating the different mechanisms of shock but are not helpful in predicting fluid responsiveness.
3. Echocardiographic "dynamic" measures of fluid responsiveness including IVC and SVC collapsibility index and respiratory variations in left and right ventricle

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stroke volume can be used to predict the response to fluid loading in mechanically ventilated patients before an actual fluid bolus is given and is therefore an essential component of goal-directed fluid therapy. Prediction of fluid responsiveness reduces perioperative morbidity associated with overhydration and fluid overload, including pulmonary complications, postoperative ileus, and increased length of stay.

4. Passive leg raising test coupled with echocardiographic measurement of stroke volume variations is the only validated measure for prediction of fluid responsiveness in spontaneously breathing patients.
5. Echocardiographic dynamic measures of fluid responsiveness have several limitations including the different cutoff values for identification of fluid responders as well as their inability to accurately predict fluid responsiveness in patients with heart rhythms other than sinus, in those with right or left ventricle dysfunction, in patients with pulmonary hypertension, as well as in patients with "low" tidal volume mechanical ventilation that reduces the respiratory variations in echocardiographic dynamic parameters. In addition, these echocardiographic measurements require expertise in performance and interpretation of perioperative echocardiography (transthoracic and transesophageal)

Introduction

In the last decade, there has been an increased understanding of the limitations of "static measures" of volume responsiveness in predicting the response to fluid administration.

While perioperative fluid management requires the integration of several clinical data points including, but not limited to, perioperative fluid balance (fluid deficit, estimated blood loss, urine output), hemodynamic data (blood pressure and heart rate), as well as laboratory data such as lactate level, acid base status, and mixed venous oxygen saturation, these data points are insufficient in predicting the response to fluid loading [1].

Most nonechocardiographic-derived static markers of cardiac preload, especially central venous pressure or pulmonary artery occlusion pressure, but even some echocardiographic-derived parameters such as left ventricular end-diastolic dimension and early/late diastolic wave ratio, do not identify fluid responders from nonresponders [2]. While these static markers can identify whether a cardiac chamber is full or empty and may help identify different mechanisms of shock states (which is certainly important), they do not reliably predict the hemodynamic response to a subsequent fluid bolus administration [3–5].

Identifying that the end goal for optimum fluid management is the optimization of stroke volume, cardiac output for optimum oxygen delivery to tissues and vital organs has resulted in the use of more "dynamic" measures of fluid responsiveness that can inform clinicians whether a subsequent fluid bolus will result in an increase in stroke volume [6].

The physiologic benefit of a fluid bolus is based on the Frank-Starling relationship whereby an increase in cardiac preload results in an increased stroke volume and subsequently an increased cardiac output. This concept assumes that a patient's preload is on the steep portion of the Frank-Starling curve. However, there are several curves that rely on stroke volume and cardiac preload, depending on the ventricular function. A given value of cardiac preload can be associated with an increase in stroke volume and the presence of preload reserve in patients with good ventricular function, whereas the same value of preload will not be associated with an increase in stroke volume (no preload reserve) in patients with poor ventricular function. Thus, it is the actual interaction among the three parameters—preload, stroke volume, and cardiac contractility—that determines fluid responsiveness [7].

Whether administration of a fluid bolus will result in an improvement in stroke volume or whether it will precipitate the occurrence of acute pulmonary edema and result in "overhydration" with its associated complications of gut edema, delayed bowel function, cardiorespiratory complications, and worsening morbidity are at the heart of this dilemma. The last decade has therefore witnessed a steady increase in the use of perioperative echocardiography as a means of obtaining real-time "dynamic" measures of fluid responsiveness in a noninvasive fashion [8–10].

This chapter will discuss some common "static" echocardiographic measurements that can guide fluid management, but will mainly focus on "dynamic" echocardiographic measurements of fluid responsiveness that can guide the perioperative physician in the fluid management of patients in the operating room, the postanesthesia care unit (PACU), and in the critical care unit.

Indications for Echocardiography in Assessment of Volume Status

In a recent report from the American Society of Echocardiography titled "Guidelines for the Use of Echocardiography as a Monitor for Therapeutic Intervention in Adults" [10], the authors proposed that echocardiography be used as a monitoring tool if after a diagnostic assessment, repetitive hemodynamic or anatomic assessments are being made over a period of minutes, hours, or days in the same patient to guide management, including fluid management. This recommendation is in response to the increasing use of echocardiography to guide therapeutic interventions by anesthesiologists, intensivists, cardiologists, and trauma physician and several observational trials and review articles [9, 11] that demonstrate the potential role of echocardiography in decision-making for patients undergoing noncardiac surgery.

This report also extends the indications for the use of echoacardiography beyond the 2010 multidisciplinary guidelines published by the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists [12] that recommended the use of transesophageal echocardiography (TEE) in patients who are undergoing noncardiac surgery and exhibit persistent hypotension or hypoxia despite therapeutic intervention.

Table 6.1 Differentiation of hypovolemia from other disease states by changes in LVID

	Hypovolemia	Low SVR and or high C.O.
LVIDS or LVESA	Decreased	Decreased
LVIDD or LVEDA	Decreased	Normal

Reference ranges for LVIDD are 3.9–5.3 cm in women and 4.2–5.9 cm in men [13]
SVR systemic vascular resistance, *C.O.* cardiac output, *LVEDA* left ventricle end-diastolic area, *LVESA* left ventricle endsystolic area, *LVIDD* left ventricle internal diameter diastole, *LVIDS* left ventricle internal diameter systole

Two-Dimensional Echocardiographic Assessment of Left Ventricle Chamber Dimensions

Serial measurements of cardiac chamber internal diameter and/or area can be helpful in assessment of volume status. While left ventricle chamber measurements are more common, both right ventricle and left ventricle serial measurements have been described. Small left ventricle internal diameter can be indicative of hypovolemia if measured at end-diastole (Table 6.1) [13].

The timing of measurements is of utmost importance, since a small left ventricle internal diameter at end systole can also occur as a result of increased contractile states (high cardiac output causing a hyperdynamic state) or due to reduction in systemic vascular resistance such as in cases of sepsis and anaphylaxis with resultant vasoplegia.

Several views can be used to measure left ventricle internal dimensions: If transthoracic echocardiography is used, the parasternal short axis or long axis are typically utilized, while transesophageal measurements are typically done using the midesophageal 2-chamber view at the mitral valve leaflet tips (Fig. 6.1). Alternatively, the transgastric long axis view can be used. While the transgastric mid short axis view at the level of the papillary muscles can also be utilized, improper alignment can result in erroneous measurements.

M-mode imaging of the LV minor axis utilizing the aforementioned parasternal TTE views (1 cm distal to the mitral valve annulus at the MV valve leaflet tips) or the TEE transgastric midpapillary SAX view (Fig. 6.2) can also be utilized to measure the LV chamber dimensions in systole and diastole [10].

Regardless of the view used, serial measurement of LV dimensions is recommended to monitor the response to fluids.

Two-Dimensional Echocardiography for Assessment of Ventricle End-Diastolic and End Systolic Areas

It is worthwhile noting that while left ventricle cavity obliteration in the transgastric midshort axis view can provide a rapid diagnosis of inadequate LV preload, 20% of cases with systolic cavity obliteration occur due to an increase in ejection fraction (hyperdynamic circulation with high cardiac output states) state or due to a reduction in afterload (sepsis, anaphylaxis with resultant vasoplegia), highlighting the

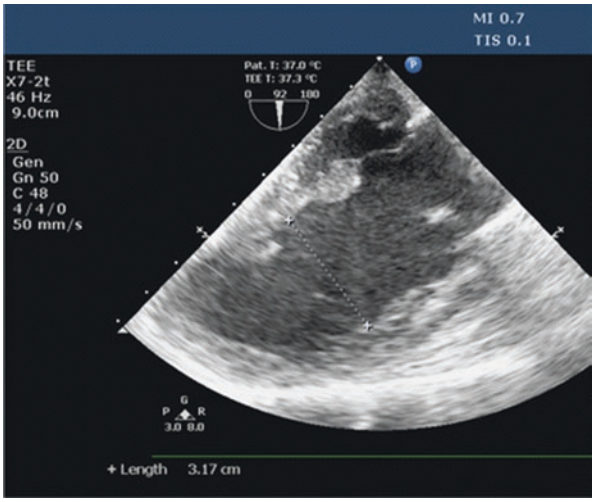

Fig. 6.1 Transesophageal echocardiographic measurements of left ventricular (LV) minor-axis diameter (LVD) from transgastric 2-chamber view of LV, usually best imaged at an angle of approximately 90–110°

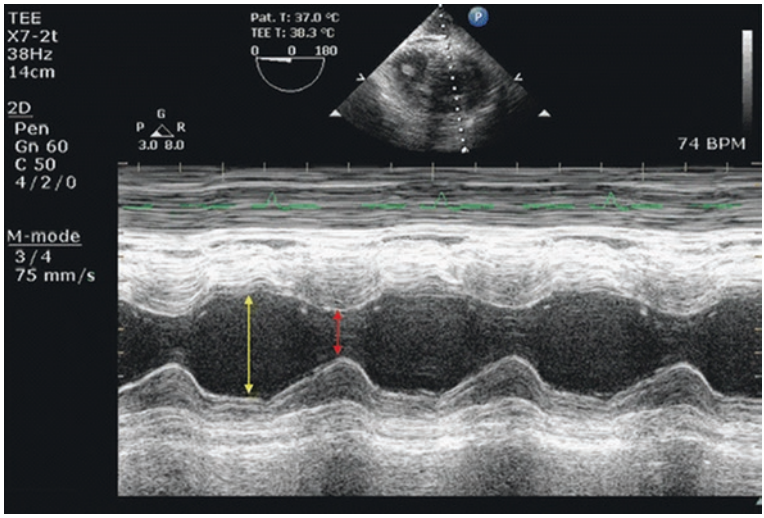

Fig. 6.2 Transesophageal M-mode imaging through the transgastric mid short axis view identifying end-diastolic (*yellow arrow*) and endsystolic (*red arrow*) left ventricle internal diameters

importance of measuring both end-diastolic and endsystolic left ventricle dimensions to differentiate hypovolemia from other conditions (Table 6.1).

Two-Dimensional Echocardiography for Assessment of Left Ventricle Volume

The recommended method for 2-dimensional (2D) echocardiographic volume calculations is the biplane method of disks summation (modified Simpson's rule) [13].

Table 6.2 Normal value ranges for left ventricle volumes in systole and diastole [13]

	Normal LVEDV (LVEDV/BSA)	Normal LVESV (LVESV/BSA)
Women ml (ml/m^2)	56–104 (35–75)	19–49 (12–30)
Men ml (ml/m^2)	67–155 (35–75)	22–58 (12–30)

LVEDV left ventricle end-diastolic volume, *LVESV* left ventricle endsystolic volume, *BSA* body surface area

In transthoracic echocardiography, this is accomplished in the apical 2- and 4-chamber views. In transesophageal echocardiography, the midesophageal 4 chamber (0° on omniplane) and 2 chamber (90° on omniplane) views are used.

Volumetric measurements are usually based on tracings of the interface between the compacted myocardium and the LV cavity in end systole and end-diastole. Table 6.2 identifies normal value ranges for left ventricle volumes in systole and diastole [13]. At the mitral valve level, the contour is closed by connecting the two opposite sections of the mitral ring with a straight line. LV length is defined as the distance between the middle of this line and the most distant point of the LV contour. The advantages of the disk summation method is that it corrects for shape distortion and has less geometric assumptions compared to linear dimension. However, foreshortening of the left ventricle is a frequent problem and can result in volume underestimation. Foreshortening can be reduced by acquiring the views at a reduced depth to focus on the left ventricle cavity. For better delineation and tracing of the left ventricle endocardial border, contrast agents can be injected intravenously [14].

Three-Dimensional Echocardiography for Assessment of Left Ventricle Volume

In patients with good image quality, three-dimensional (3D) echocardiographic measurements are accurate and reproducible [15]. In addition, they do not rely on geometric assumptions and are therefore less prone to foreshortening. 3D image acquisition should therefore focus on including the entire left ventricle within the pyramidal data set [16].

2D and 3D volumetric assessment of left ventricle volume during systole (ESV) and diastole (EDV), in addition to monitoring of fluid status, is most commonly used to calculate ejection fraction using the formula:

$$\mathrm{EF} = \left(\mathrm{EDV} - \mathrm{ESV}\right) / \mathrm{EDV}$$

Inferior Vena Cava Size and Collapsibility

The inferior vena cava (IVC) ends at the floor of the right atrium, just after crossing the diaphragm, and carries about 80% of the venous return to the right atrium. Its route is purely abdominal and is therefore only subject to intra-abdominal pressure [8].

In spontaneously breathing patients, inspiration causes a negative intrathoracic pressure and a subsequent reduction in IVC diameter. This normal inspiratory reduction in IVC diameter is exaggerated in the hypovolemic state. Periodic measurement of IVC diameter and its collapsibility with inspiration has been used to guide fluid management in patients with shock states.

It is important to obtain an appropriate imaging window that maintains the inferior vena cava in view throughout the respiratory cycle, since this facilitates measurement of the IVC size both in inspiration (minimum diameter) and expiration (maximum diameter).

Transthoracic Echocardiography in the Spontaneously Breathing Patient

From subcostal 4-chamber view, the transducer is rotated 90° counterclockwise, always keeping the right atrium on the screen (transducer orientation marker is at 12 o'clock). A depth of 16–24 cm is used, with imaging adjusted to ensure that the merging of the IVC into the right atrium is visualized, thereby confirming that the descending aorta is not erroneously imaged instead (Fig. 6.3).

Both 2D imaging and M-mode imaging can be used to measure IVC diameter and collapsibility. M-mode imaging allows high frame rate measurements of diameter changes that occur throughout the respiratory cycle. The diameter of the IVC should be measured 2–3 cm before it merges with the right atrium. IVC collapsibility index is measured as follows:

$$\text{IVC collapsibility index} = \text{Maximum Diameter IVC}\left(\text{DIVC max}\right) - \text{Minimum Diameter IVC}\left(\text{DIVC min}\right) / \text{Maximum Diameter IVC}\left(\text{DIVC max}\right) \times 100$$

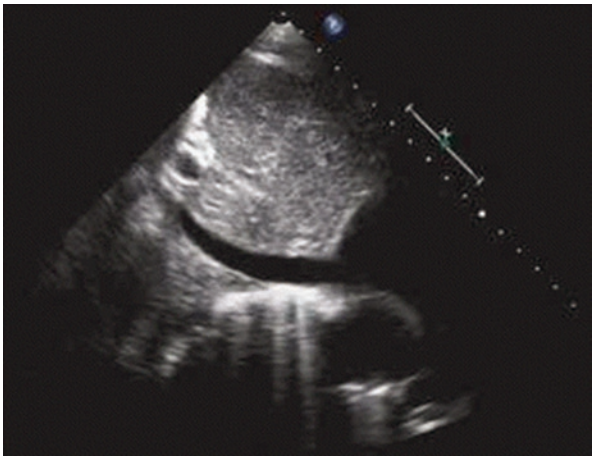

Fig. 6.3 Transthoracic subcostal view of the IVC

Table 6.3 Estimation of right atrial pressure based on IVC diameter and collapsibility with sniff [17]

IVC size (cm)	Collapsibility with sniff (%)	Right atrial pressure (mmHg)
≤2.1	>50	0–5
≤2.1	<50	5–10
>2.1	>50	5–10
>2.1	<50	10–20

Uses of IVC collapsibility index in spontaneously breathing patients:

1. IVC diameter and collapsibility can be used to estimate right atrial pressures (Table 6.3) [17].
2. Assessment of volume status (hypovolemia, hypervolemia).
3. In spontaneously breathing patients, IVC collapsibility index has *not* been validated for assessment of fluid responsiveness.

Transesophageal Echocardiography in the Mechanically Ventilated Patient

Measurement of IVC collapsibility index has also been used in mechanically ventilated septic patients using either transthoracic (TTE as described above) or transesophageal echocardiography (TEE) with 2D and/or M-mode imaging of the IVC.

In TEE, from the midesophageal bicaval view (90–110°), the probe is advanced deeper into the esophagus to bring the IVC to the center of the display, which is followed by multiplane rotation back to 40–70°. On this view, the posterior and anterior walls of the IVC are observed on the top and the bottom of the display, respectively.

The second option to view the IVC starts at the level of the aortic valve at 0°. From this point, the probe is advanced and turned to the right until the tricuspid valve and the coronary sinus come into view. Further advancing and turning to the right will show the IVC and bring it to the center of the display [17].

Uses of IVC collapsibility index in mechanically ventilated patients:

1. Prediction of fluid responsiveness: IVC collapsibility index of 15% and above typically predicts fluid responsiveness [18, 19]
2. Due to its intra-abdominal location, the IVC is *not* suited for estimation of right atrial pressure during mechanical ventilation, especially because positive pressure ventilation causes a dilation of IVC diameter [20, 21]. However, a small IVC diameter (<1.2 cm) has a 100% specificity (with a low sensitivity) for a RA pressure of less than 10 mmHg [22].

Superior Vena Cava Size and Collapsibility

The superior vena cava (SVC) ends at the top of the right atrium. Unlike the IVC, its route is purely intrathoracic. It carries about 20% of the venous return to the right atrium [8].

Transesophageal Echoacardiography in the Mechanically Ventilated Patient

In mechanically ventilated patients, superior vena cava (SVC) collapsibility index has been proposed as a gauge of volume status [23]. Measurements of the SVC will be taken in the midesophageal bicaval view (90–110°) using 2D and/or M-mode echocardiography, 1–2 cm away from the entry point into the right atrium. This technique is analogous to that recommended when measuring IVC diameter and collapsibility [20] and was previously described by Cowie et al. [24] Collapsibility index is defined as maximal SVC diameter during expiration minus minimal diameter during inspiration divided by maximal diameter:

$$\text{SVC collapsibility index} = \text{Maximum Diameter SVC}\left(\text{DSVC max}\right) - \text{Minimum Diameter SVC}\left(\text{DSVC min}\right) / \text{Maximum Diameter SVC}\left(\text{DSVC max}\right) \times 100 \quad \left(\text{Fig. 6.4}\right)$$

A number of 36% allows discrimination between fluid responsive and fluid-unresponsive patients in mechanically ventilated septic patients [23]. Alternatively, a quick qualitative visual approach has been suggested by the same authors [25] to gauge fluid responsiveness based on the presence and degree of SVC collapse. Patients with complete or partial collapse would be considered fluid responsive, while patients with no collapse would be considered fluid nonresponsive (Fig. 6.4):

Major respiratory variation → complete SVC collapse fluid responsive
Moderate respiratory variation → partial SVC collapse fluid responsive
No respiratory variation → no SVC collapse fluid unresponsive

Limitations of the use of respiratory changes in vena caval diameter:

1. In spontaneously breathing patients, respiratory variations of the vena cava cannot be used to predict fluid responsiveness. In these situations, passive leg raising to mimic a fluid bolus (see below) with measurement of left ventricle stroke volume before and after the maneuver is the only described method for assessment of fluid responsiveness in the spontaneously breathing patient.

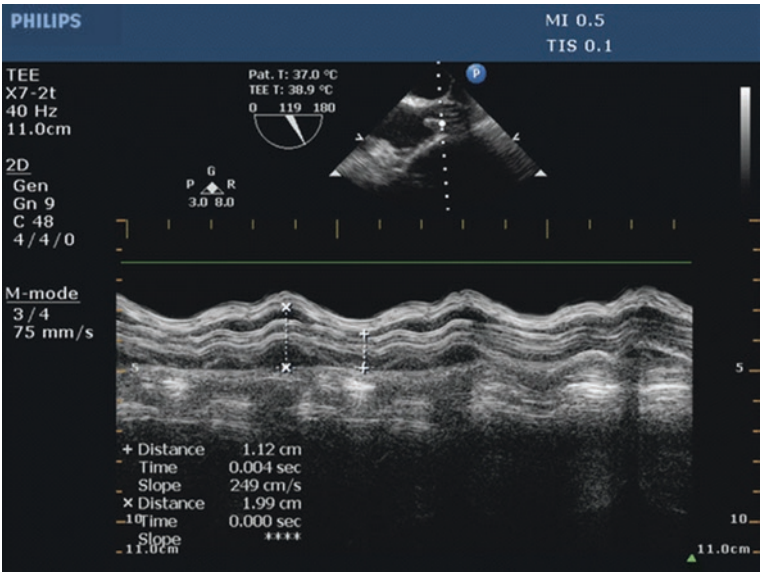

Fig. 6.4 M-mode Assessment of SVC collapsibility index utilizing the TEE midesophageal bicaval view at ~120°. SVC collapsibility index = 1.99 cm − 1.12 cm/1.99 cm × 100 = 43% indicating fluid responsiveness. Note the echogenic density in the SVC representing an indwelling central venous catheter that should not be confused with the wall of the SVC

2. In mechanically ventilated patients, the vena cava diameters and collapsibility cannot be used to estimate right atrial pressure.
3. Fluid responsiveness based on SVC and IVC collapsibility indices has not been validated in patients with rhythms other than sinus rhythms, in those with small tidal volume ventilation (<6 ml/kg) in patients with right or left ventricle dysfunction, or with those with pulmonary hypertension.
4. The cutoff for fluid responsiveness for SVC and IVC collapsibility index vary markedly (around 15% for IVC and 35% for SVC). In addition, the fluid responsiveness cutoff for each one of the vena cava varies across studies, introducing the "gray zone" concept. The gray zone concept of fluid responsiveness refers to patients where the value of the collapsibility index does not definitely determine whether they will or will not be fluid responsive (e.g., a value for IVC collapsibility index between 10 and 15%) [26].

Respiratory Variations in Left Ventricle Stroke Volume

Using transesophageal echocardiography, mechanical ventilation-induced changes in left ventricle stroke volume can be assessed in the deep transgastric 5-chamber view at a transducer angle of 0–20° to align the pulsed wave Doppler signal with the left ventricle outflow tract [8].

The area under the curve of left ventricle flow, also called stroke distance or velocity time integral (VTI) is multiplied by the cross-sectional area of the aortic valve to measure left ventricle stroke volume. (Stroke volume multiplied by the patient's heart rate can then be used to measure the cardiac output.)

Since the cross-sectional area is constant throughout the respiratory cycle, changes in velocity time integral reflect changes in left ventricle stroke volume:

$$\text{Delta}\left(\text{Velocity Time Integral}\right)\ \text{VTI}\ \% = \text{VTI max} - \text{VTI min} \times 100 / \text{VTI mean}$$

where the mean VTI equals the VTI max + VTI min/2 [27].

In hypovolemic patients, the magnitude of respiratory changes that occur with mechanical ventilation exaggerate the difference between the inspiratory and the expiratory left ventricle stroke volume and can be used to assess biventricular preload dependence and fluid responsiveness.

In an attempt to simplify the measurements even further, maximum and minimum peak velocities throughout the respiratory cycle have been used instead of velocity time integrals to measure left ventricle stroke volume changes [22].

While velocity time integral measurements require tracing of the area under the curve of the maximum and minimum VTI area, measuring velocities only requires identification of the maximum and minimum peak velocities throughout the respiratory cycle. Changes in peak velocity can then be calculated as follows:

$$\text{Delta Vpeak}\ (\%) = 100 \times \left(\text{Vpeakmax} - \text{Vpeak min}\right) / \text{Vpeak mean}$$

Where the mean peak velocity equals (Vpeakmax + Vpeakmin)/2.

A Delta Vpeak threshold value of 12% allowed discrimination between responders and nonresponders with a sensitivity of 100% and a specificity of 89% [19] (Fig. 6.5).

The inspiratory phase of positive pressure ventilation causes a reduction in right ventricle stroke volume (through an increase in pleural pressure and transpulmonary pressure) and a concomitant increase in left ventricle stroke volume. In the expiratory phase of positive pressure ventilation, these changes are reversed, with a decrease in left ventricle stroke volume during expiratory phase. These mechanical ventilation-induced changes have also been termed "reverse pulsus paradoxus," since their direction is opposite to those occurring during spontaneous ventilation (whereby the right ventricle stroke volume increases during inspiration and the left ventricle stroke volume decreases during inspiration, with these changes being reversed during spontaneous expiration).

Mechanical ventilation-induced changes of right ventricle stroke volume can also be assessed with pulsed wave Doppler in the right ventricle outflow tract using the midesophageal ascending aorta short axis view at 0–20° on the transducer angle or using the upper esophageal aorta short axis view at 90° with the pulmonary artery outflow in view (Fig. 6.6). This is especially important when transgastric views cannot be obtained for assessment of left ventricle stroke volume changes.

Esophageal Doppler devices introduced through the mouth and adjusted to obtain the highest Doppler velocity signal from the descending aorta have also been

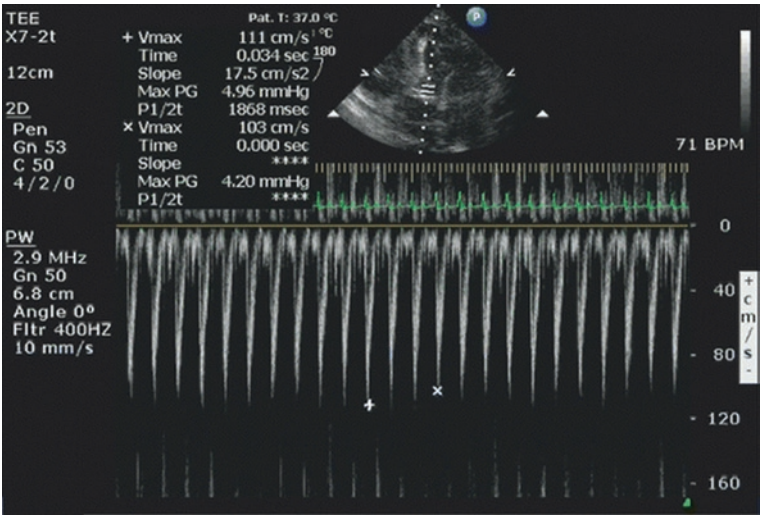

Fig. 6.5 TEE Assessment of Delta V peak in the deep transgastric 5 chamber view. Delta V peak = $111_{cm/s} - 103_{cm/s}/(111_{cm/s} + 103_{cm/s}/2) \times 100 = 7.5\%$ indicating lack of fluid responsiveness

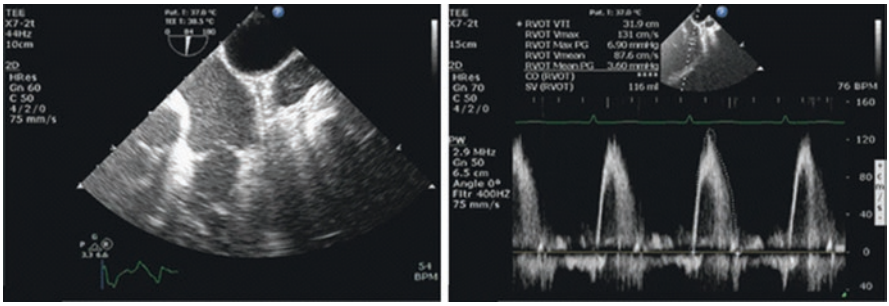

Fig. 6.6 TEE assessment of delta VTI % or delta Vpeak % can also be done using the upper esophageal aortic short axis view so that the pulsed wave Doppler signal is parallel to the right ventricle outflow tract

used successfully to assess fluid responsiveness by measuring variations in aortic blood flow (ABF) using the following formula:

$$\text{Delta ABF\%} = \left(\text{ABFmax} - \text{ABF min}\right) \; / \; \text{ABFmean} \times 100$$

where ABFmax and ABFmin are the maximal and minimal peak ABF values over 1 respiratory cycle, respectively and ABFmean equaling (ABFmax + ABFmin)/2. The delta ABF value is typically averaged over five respiratory cycles [22, 28, 29].

Passive Leg Raising Test for the Prediction of Volume Responsiveness in the Spontaneously Breathing Patient (Combined with Changes in Stroke Volume)

Mechanical ventilation-induced changes in hemodynamic signals cannot be used in predicting fluid responsiveness in spontaneously breathing patient. Passive leg raising (PLR), by lifting the legs passively from the horizontal position, induces a gravitational transfer of blood from the lower extremities toward the intrathoracic compartment [1]. In order to induce sufficient venous blood shift that can create a significant increase in cardiac preload, the lower limbs are elevated to 45° (automatic bed elevation) while simultaneously placing the patient in the supine from a 45° semirecumbent position. TTE measurement of stroke volume before (at baseline) and after passive leg raising can predict fluid responsiveness. An increase in stroke volume by 12% or more during passive leg raising is highly predictive of positive hemodynamic response and stroke volume increase with subsequent fluid bolus administration [30, 31].

On a parasternal 2D view, aortic diameter is measured just below the level of the aortic annulus at the left ventricle outflow tract (LVOT). Aortic valve area (AVA) is calculated as follows:

$$\mathrm{AVA} = \left(\pi\left[\mathrm{pi}\right] \times \mathrm{LVOT\ \ diameter}^2\right) / 4 = 0.785 \times \mathrm{LVOT\ \ diameter}^2$$

On an apical 5-chamber view, aortic blood flow is recorded using pulsed Doppler, with the sample volume placed just below the aortic annulus. The velocity–time integral of aortic blood flow (VTIa) is calculated. Stroke volume is then calculated as SV = VTIa × AVA and CO is calculated as SV X HR. The aortic valve area is only measured once at baseline since it is considered to remain unchanged. To reduce error in VTI measurement, 3–5 consecutive measurements averaged over one respiratory cycle are reported for each VTI measurement [30].

Echocardiographic measurements to detect changes in VTIa require experienced echocardiographers to perform the measurements, especially because the changes in VTIa may not persist beyond a couple of minutes. In addition, any malalignment of the pulsed Doppler beam can introduce errors in measurements mainly due to underestimation of the VTI caused by angulation of the Doppler beam if not strictly parallel to the aortic blood flow (a 15° angle inducing a 5% error in measurement) [32].

In mechanically ventilated patients, esophageal Doppler measurements of changes in descending aortic blood flow in response to passive leg raising have been used in several studies [29, 33, 34]. Since these probes are uncomfortable in conscious spontaneously breathing patients, they are not used in non-intubated patients.

Conclusion

Several echocardiographic methods for the assessment of volume status and the prediction of fluid responsiveness have been described. Echocardiographic "dynamic" measures for the assessment of fluid responsiveness in mechanically ventilated patients include SVC and IVC collapsibility index, left (and right) ventricle delta velocity time integral percentage, left (and right) ventricle delta peak velocity percentage, and delta aortic blood flow percentage.

In the spontaneously breathing patient, the only validated test for assessment of fluid responsiveness is the passive leg raising test and requires the simultaneous transthoracic echocardiographic assessment of changes in aortic velocity time integrals or peak velocities as a dynamic measure of fluid responsiveness.

A detailed understanding of the various limitations of these echocardiographic measurements is essential in avoiding the wrong decision-making regarding fluid loading.

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