



Notable Grand Rounds
of the

**Michael & Marian Ilitch
Department of Surgery**

Wayne State University
School of Medicine

Detroit, Michigan, USA

Jason Kurle, DO

**VOLUME STATUS AND FLUID
RESPONSIVENESS IN SURGICAL
AND CRITICAL CARE PATIENTS**

March 11, 2026

About Notable Grand Rounds

These assembled papers are edited transcripts of didactic lectures given by mainly senior residents, but also some distinguished attending and guests, at the Grand Rounds of the Michael and Marian Ilitch Department of Surgery at the Wayne State University School of Medicine.

Every week, approximately 50 faculty attending surgeons and surgical residents meet to conduct postmortems on cases that did not go well. That "Mortality and Morbidity" conference is followed immediately by Grand Rounds.

This collection is not intended as a scholarly journal, but in a significant way it is a peer reviewed publication by virtue of the fact that every presentation is examined in great detail by those 50 or so surgeons.

It serves to honor the presenters for their effort, to potentially serve as first draft for an article for submission to a medical journal, to let residents and potential residents see the high standard achieved by their peers and expected of them, and by no means least, to contribute to better patient care.

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Volume Status and Fluid Responsiveness in Surgical and Critical Care Patients

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Grand Rounds Presentation

March 11, 2026

Editor's Note: This is an edited summary of a Grand Rounds talk given by Dr. Kurle on March 11, 2026 at the Ilitch Department of Surgery, Wayne State University School of Medicine.

Abstract

Determining when to administer intravenous fluid remains a central challenge in perioperative and critical care medicine. Modern management focuses on **fluid responsiveness**, defined as an increase in cardiac output following a preload challenge, rather than relying solely on static indicators of volume status. Importantly, fluid responsiveness must be distinguished from **fluid tolerance**, as some patients cannot safely accommodate additional intravascular volume.

This review summarizes physiologic principles and bedside methods used to assess fluid responsiveness, including stroke volume variation, pulse pressure variation, echocardiographic LVOT VTI measurement, vena cava variation, ventilator occlusion testing, and the passive leg raise maneuver. Integrating these tools with clinical judgment helps guide safer, individualized fluid therapy.

Introduction

Optimal fluid management remains a central challenge in perioperative and critical care medicine. Both inadequate and excessive fluid administration can contribute to organ dysfunction, hemodynamic instability, and adverse clinical outcomes. Surgical patients frequently present with complex physiologic conditions in which the clinician must rapidly determine not only whether additional fluid will increase cardiac output, but also whether the patient can physiologically tolerate such expansion of intravascular volume.

The concept of **fluid responsiveness** has therefore become central to modern hemodynamic management. Rather than focusing solely on static measurements of intravascular volume, clinicians increasingly attempt to determine whether a patient’s cardiac output will increase in response to a preload challenge. Importantly, an increase in cardiac output does not necessarily translate into an increase in blood pressure, because mean arterial pressure reflects the interplay among preload, myocardial contractility, and afterload.

Equally critical is the distinction between **fluid responsiveness** and **fluid tolerance**. A patient may demonstrate increased cardiac output after fluid administration but still be unable to tolerate additional intravascular volume without developing pulmonary edema or other organ dysfunction. Thus, the evaluation of a patient’s hemodynamic status requires consideration of three related but distinct questions:

1. Is the patient fluid responsive?
2. Can the patient physiologically tolerate additional volume?
3. Is fluid administration appropriate for the current clinical scenario?

These principles frame the modern approach to fluid management in surgical and intensive care environments.

Physiologic Foundations of Fluid Responsiveness

The physiologic basis for fluid responsiveness derives from the **Frank–Starling relationship (Fig. 1)**, which describes the relationship between ventricular preload and cardiac output. As ventricular filling increases, myocardial fiber stretch leads to increased stroke volume up to a physiologic plateau. Patients on the steep portion of the Frank–Starling curve demonstrate significant increases in cardiac output with relatively small increases in preload. In contrast, patients on the plateau portion exhibit minimal increases in output despite additional fluid administration.

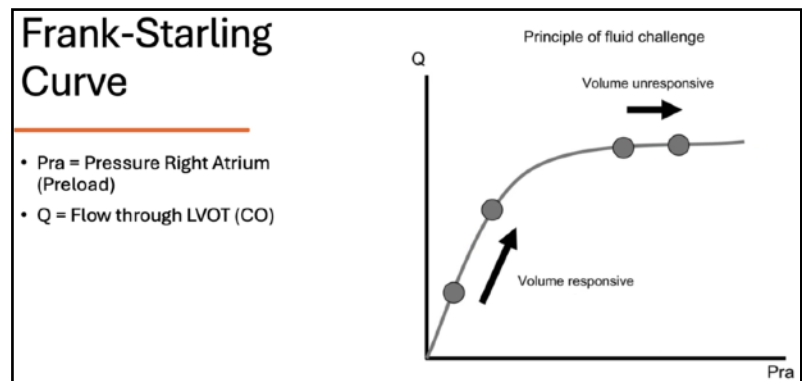


Fig. 1. Frank–Starling relationship illustrating fluid responsive versus volume-unresponsive regions of the curve.

In clinical practice, the objective of fluid assessment is therefore to determine where a patient lies on this curve. If the patient occupies the steep portion, fluid administration may significantly augment cardiac output. Conversely, if the patient lies on the plateau, additional fluid is unlikely to improve hemodynamics and may increase the risk of complications such as pulmonary edema or right ventricular failure.

It is important to recognize that most patients fall within an intermediate zone in which prediction of fluid responsiveness is uncertain. Static measurements of preload alone—such as central venous pressure—often fail to accurately predict the hemodynamic response to fluid administration. Consequently, modern approaches increasingly rely on **dynamic assessments of cardiopulmonary interaction** to guide fluid management.

Assessing Fluid Tolerance

Although determining fluid responsiveness is essential, clinicians must also assess whether the patient can physiologically tolerate additional intravascular volume. Fluid tolerance reflects the capacity of both the heart and lungs to accommodate increased preload without causing pathologic congestion or organ dysfunction.

Evaluation of fluid tolerance requires assessment of multiple physiologic domains including:

- The central venous reservoir
- Right ventricular size and morphology
- Left ventricular size and function
- Valvular abnormalities
- Pulmonary interstitial fluid status

These parameters collectively provide insight into whether fluid administration is likely to worsen cardiac congestion or pulmonary edema.

Right Ventricular Assessment

Right ventricular performance plays a critical role in determining fluid tolerance. A failing right ventricle may be unable to effectively transmit increased preload through the pulmonary circulation, resulting in systemic venous congestion rather than improved cardiac output.

Key echocardiographic features used to evaluate right ventricular tolerance include:

- Right ventricular dilation
- Ventricular wall thickness
- Severity of tricuspid regurgitation
- Overall right ventricular contractile function

- Evidence of systemic venous congestion

One commonly used quantitative parameter is **tricuspid annular plane systolic excursion (TAPSE)**. TAPSE measures longitudinal motion of the tricuspid annulus and serves as a surrogate marker of right ventricular systolic function. Values greater than approximately 17 mm are generally considered consistent with preserved right ventricular contractility.

Right ventricular dysfunction can substantially limit the benefits of fluid administration, because inadequate right ventricular output prevents effective filling of the left ventricle. Consequently, assessment of right ventricular performance is essential before initiating aggressive fluid resuscitation.

Left Ventricular Function and Risk of Pulmonary Edema

Left ventricular performance determines the degree to which the pulmonary circulation can accommodate increased preload without developing pulmonary edema. Both **systolic and diastolic ventricular function** must be considered in this evaluation.

While clinicians frequently focus on the left ventricular ejection fraction (LVEF), diastolic function is often equally or more important in determining fluid tolerance. A ventricle with impaired relaxation may exhibit elevated filling pressures even when systolic function is preserved.

Important echocardiographic parameters for assessing left ventricular tolerance include:

- Left ventricular dilation or hypertrophy
- Left ventricular systolic function (LVEF)
- Diastolic function parameters
- Mitral regurgitation
- Pulmonary interstitial fluid

Assessment of diastolic function commonly includes evaluation of:

- Left atrial size

- Mitral inflow velocities (E/A ratio)
- Mitral annular tissue velocities (e')
- The E/ e' ratio as an estimate of filling pressures
- Pulmonary venous flow patterns

Among these measurements, the **E/ e' ratio** serves as a useful indicator of elevated left ventricular filling pressures and risk of pulmonary edema.

Pulmonary Interstitial Fluid and Lung Ultrasound

Assessment of pulmonary interstitial fluid is an essential component of evaluating fluid tolerance. Lung ultrasound has emerged as a rapid bedside tool for detecting pulmonary edema through identification of characteristic artifacts known as **B-lines**.

In normal lung parenchyma, ultrasound demonstrates horizontal reverberation artifacts known as **A-lines**, reflecting the air-filled nature of lung tissue. In contrast, the presence of pulmonary interstitial fluid generates vertical artifacts called **B-lines**, which extend from the pleural surface to the bottom of the ultrasound image. (Fig. 2, top right.)

As pulmonary edema increases, lung ultrasound findings progress along a continuum from normal aerated lung to diffuse B-lines and ultimately to consolidation. (Fig. 3.)

The presence of diffuse B-lines across multiple lung zones suggests

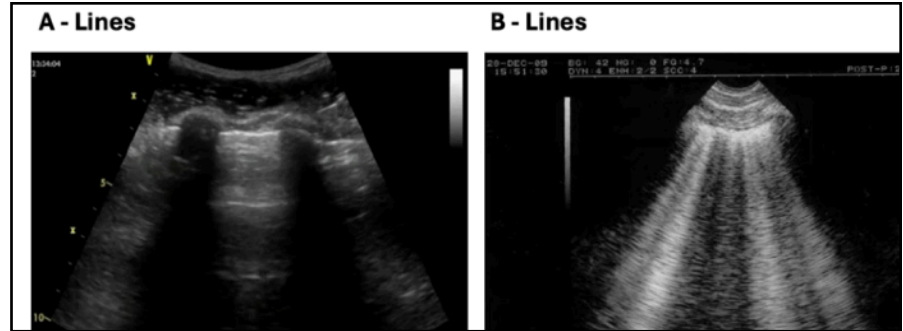


Fig. 2. Lung ultrasound demonstrating A-lines in normal lung and B-lines representing pulmonary interstitial edema.

significant pulmonary interstitial edema and may indicate that additional fluid administration would worsen respiratory function.

Static Measures of Volume Status

Historically, clinicians relied heavily on **static measures of preload** to estimate intravascular volume status. These measurements represent single-point estimates of cardiac filling pressures or ventricular volumes and are often used as indirect surrogates of circulating blood volume. While such measurements can be informative in extreme circumstances, they correlate poorly with the physiologic response to fluid administration.

Examples of commonly used static measures include:

- Central venous pressure (CVP)

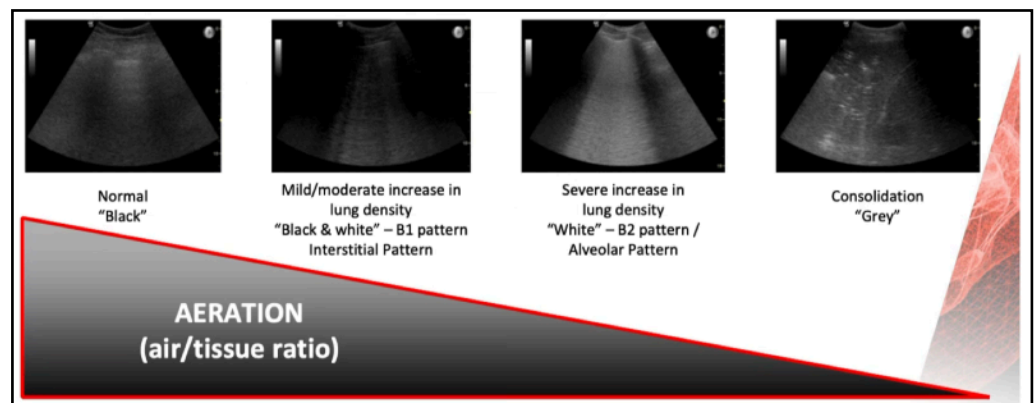


Fig. 3. Spectrum of lung ultrasound findings illustrating progressive interstitial fluid accumulation from normal lung to consolidation. Adapted from Gargani L. *Cardiovascular Ultrasound*, 2011.

- Right atrial pressure
- Pulmonary artery wedge pressure (from pulmonary artery catheterization)
- Ventricular end-diastolic volume or end-diastolic area
- Inferior vena cava diameter

Static measurements are most useful at physiologic extremes. For example, a severely collapsed inferior vena cava or very low filling pressure strongly suggests hypovolemia, while markedly elevated filling pressures suggest a volume-replete or overloaded state. However, most patients fall within an intermediate range in which static measurements cannot reliably predict the effect of additional fluid administration.

Central Venous Pressure

Central venous pressure remains one of the most widely available measurements of preload. CVP is commonly measured through a central venous catheter and serves as a surrogate for **right ventricular end-diastolic pressure (RVEDP)**. Normal values generally range between **8 and 12 mmHg**. (Fig. 4.)

Although CVP can provide useful information regarding the overall venous reservoir, several limitations restrict its usefulness as a predictor of fluid responsiveness. First, CVP represents only a static measure of preload and therefore cannot determine whether additional volume will increase cardiac output. Second, CVP is influenced by numerous physiologic factors including mechanical ventilation, intrathoracic pressure, and intra-abdominal pressure.

Despite these limitations, CVP remains clinically valuable when interpreted in context. Changes in CVP reliably reflect changes in preload, but **preload itself does not reliably predict fluid responsiveness**. Thus, while a rising CVP indicates increasing filling pressure, it does not necessarily indicate that the heart will generate additional cardiac output in response to that preload.

Dynamic Measures of Fluid Responsiveness

Because static measures have limited predictive value, contemporary hemodynamic assessment emphasizes **dynamic measurements of cardiopulmonary interaction**. Dynamic measurements evaluate how cardiac output or stroke volume changes in response to physiologic perturbations such as mechanical ventilation or transient preload challenges.

Dynamic methods are generally more accurate because they simulate the effect of fluid administration without actually delivering large volumes of fluid. By observing the cardiovascular system's response to these perturbations, clinicians can determine whether the patient remains on the responsive portion of the Frank–Starling curve.

Common dynamic measures include:

- Stroke volume variation (SVV)
- Pulse pressure variation (PPV)
- Systolic pressure variation (SPV)
- Dynamic arterial elastance
- Passive leg raise testing
- End-expiratory occlusion testing

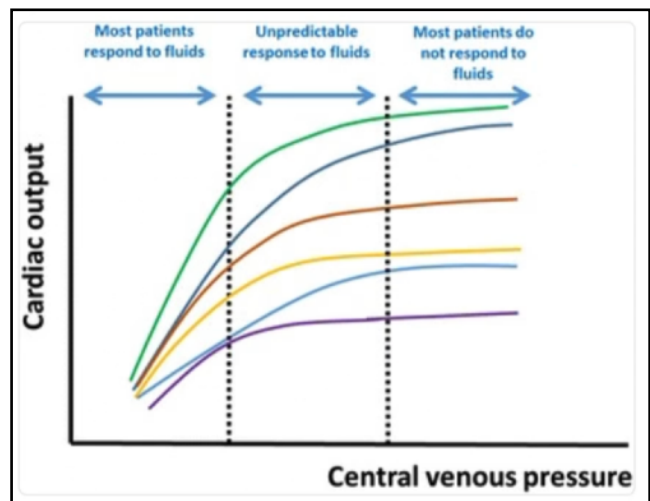


Fig. 4. Relationship between central venous pressure and cardiac output demonstrating wide variability in patient response to fluid administration.

Stroke Volume Variation

Stroke volume variation is one of the most widely studied dynamic predictors of fluid responsiveness. The principle underlying SVV is the interaction between **positive pressure ventilation and venous return**.

During mechanical inspiration, intrathoracic pressure rises, which transiently decreases venous return and reduces right ventricular preload. In patients who are volume depleted and therefore operating on the steep portion of the Frank–Starling curve, these respiratory changes produce substantial variations in stroke volume. Conversely, patients who are already volume replete demonstrate relatively little variation in stroke volume throughout the respiratory cycle.

Stroke volume variation is typically calculated using the following relationship:

$$SVV = \frac{SV_{max} - SV_{min}}{SV_{mean}} \times 100$$

Values **greater than approximately 12–13%** are generally considered predictive of fluid responsiveness in mechanically ventilated patients.

Stroke volume variation can be measured through several methods, including advanced hemodynamic monitors such as the **FloTrac/Hemosphere system** or through echocardiographic assessment of left ventricular outflow tract velocity.

Systolic Pressure Variation and Pulse Pressure Variation

In situations where direct stroke volume measurements are unavailable, arterial waveform analysis can provide alternative surrogates.

Systolic pressure variation (SPV) represents the difference between the maximum and minimum systolic blood pressure during a respiratory cycle. This measurement can often be estimated visually from the arterial waveform and has

historically been described as the “poor man’s stroke volume variation.” (Fig. 5.)

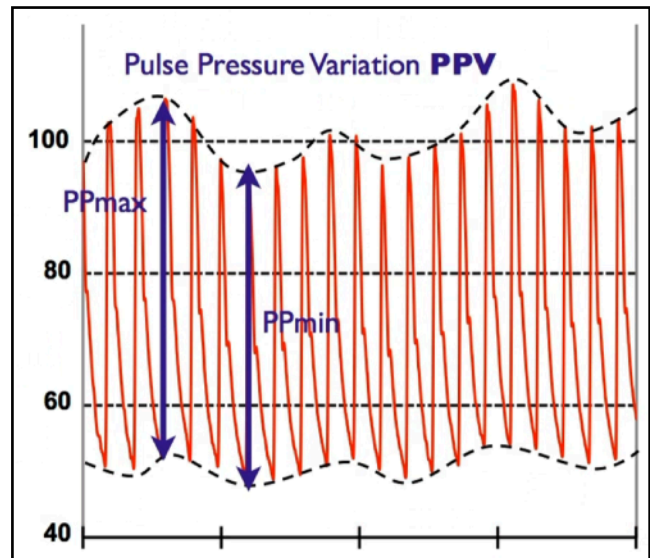


Fig. 5. Arterial waveform demonstrating systolic pressure variation and pulse pressure variation during mechanical ventilation.

A related parameter, **pulse pressure variation (PPV)**, evaluates the variation in pulse pressure throughout the respiratory cycle and is calculated as:

$$PPV = \frac{PP_{max} - PP_{min}}{PP_{mean}} \times 100$$

Unlike stroke volume variation, which reflects the amount of blood ejected from the left ventricle, pulse pressure variation is influenced by **arterial compliance and vascular stiffness**. Nevertheless, PPV and SVV demonstrate similar predictive accuracy for identifying fluid responsive patients in appropriately selected clinical scenarios. (Fig. 6, next page.)

Dynamic Arterial Elastance

Advanced hemodynamic monitoring platforms provide additional physiologic parameters beyond stroke volume variation. One particularly useful measurement is **dynamic arterial elastance (EaDyn)**.

Systolic Pressure Variation		$SP_{max} - SP_{min}$
Pulse Pressure Variation		$\frac{(PP_{max} - PP_{min})}{[(PP_{max} + PP_{min})/2]}$
Stroke Volume Variation		$\frac{(SV_{max} - SV_{min})}{SV_{mean}}$

Fig. 6. Comparison of systolic pressure variation, pulse pressure variation, and stroke volume variation calculations.

Dynamic arterial elastance represents the ratio of pulse pressure variation to stroke volume variation:

$$Ea_{Dyn} = \frac{PPV}{SVV}$$

This parameter reflects the relationship between ventricular output and arterial system compliance. Clinically, EaDyn can help determine whether fluid administration in a volume-responsive patient will also result in an increase in arterial blood pressure.

Values **greater than approximately 1.0–1.2** suggest that fluid administration will likely increase mean arterial pressure, whereas values **below approximately 0.8–1.0** suggest that stroke volume may increase without a corresponding rise in blood pressure.

This distinction can be clinically important in hypotensive patients, as it may guide the decision between administering fluid versus initiating vasopressor therapy.

Echocardiographic Assessment of Stroke Volume

Bedside echocardiography provides a powerful method for evaluating cardiac output and fluid responsiveness. One of the most widely used echocardiographic techniques involves measurement of **left ventricular outflow tract velocity–time integral (LVOT VTI)**.

The standard approach begins with acquisition of an **apical four-chamber view**, (Fig. 7) followed by slight angulation of the probe to obtain the **apical five-chamber view**, (Fig. 8, next page) which allows visualization of the left ventricular outflow tract (LVOT) and aortic valve.

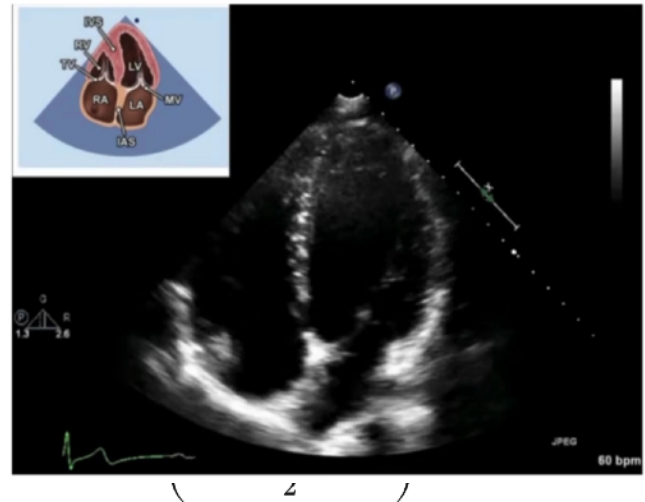


Fig. 7. Apical four-chamber echocardiographic view demonstrating the four cardiac chambers.

Stroke volume can be calculated from LVOT flow using the formula for the volume of a cylinder. In this model, the LVOT cross-sectional area represents the base of the cylinder and the velocity–time integral represents the height (Fig. 9, next page):

$$SV = \pi \times \left(\frac{LVOT \text{ diameter}}{2} \right)^2 \times LVOT \text{ VTI}$$

However, in clinical practice the LVOT diameter remains relatively constant for a given patient. As a result, clinicians often monitor **changes in LVOT VTI alone** as a surrogate for changes in stroke volume.

The VTI represents the **distance traveled by a column of blood during a single cardiac cycle**. When stroke volume increases, the area under the Doppler velocity curve correspondingly increases. Thus, observing changes in VTI before and after a preload challenge can provide a rapid estimate of changes in cardiac output. (Fig. 10.)

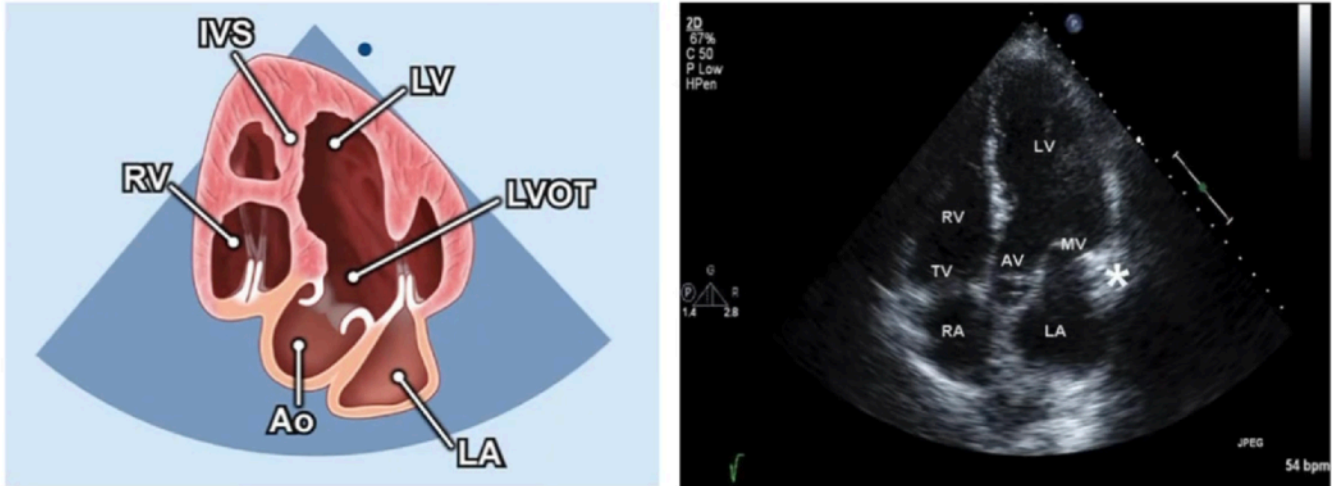


Fig. 8. Apical five-chamber echocardiographic demonstrating the LVOT and aortic valve used for Doppler assessment of stroke volume.

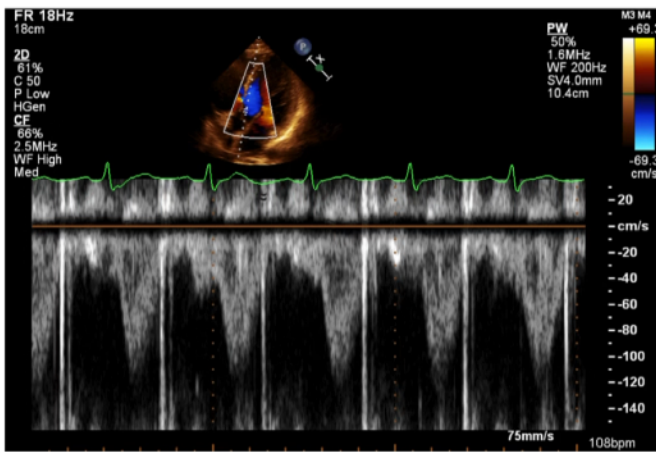


Fig. 9. Pulsed-wave Doppler measurement of LVOT velocity-time integral demonstrating stroke volume calculation.

Limitations of Stroke Volume Variation

Despite their usefulness, dynamic measurements such as stroke volume variation have important limitations that must be recognized.

First, SVV measurements were originally validated in patients undergoing **controlled mechanical ventilation with relatively large tidal volumes (8–10 mL/kg)**. Modern lung-protective ventilation strategies frequently employ lower tidal volumes, which may reduce the accuracy of these measurements.

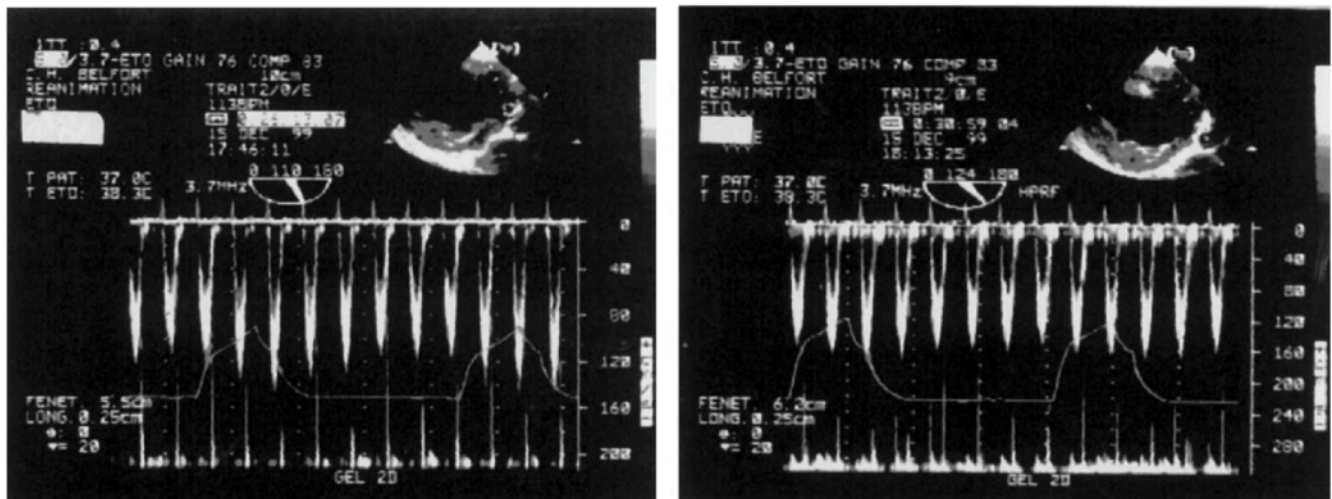


Fig. 10. LVOT Doppler waveforms demonstrating variation in stroke volume before and after fluid administration.

Second, accurate SVV measurement generally requires:

- Controlled mechanical ventilation
- Minimal spontaneous respiratory effort
- Adequate sedation
- Regular cardiac rhythm

Several clinical conditions can produce misleading values. Factors that may **falsely increase stroke volume variation** include:

- Spontaneous breathing or assisted ventilation
- Cardiac arrhythmias
- Elevated intra-abdominal pressure
- Right ventricular failure

Conversely, factors that may **falsely decrease stroke volume variation** include:

- Low tidal volume ventilation
- Reduced lung compliance such as ARDS
- High ventilator respiratory rates relative to heart rate
- Open chest or open abdomen

Because these limitations are common in critically ill patients, alternative dynamic tests are frequently required.

End-Inspiratory and End-Expiratory Occlusion Tests

Ventilator occlusion tests provide an alternative method of assessing fluid responsiveness by temporarily altering intrathoracic pressure.

During an **end-expiratory occlusion test**, the ventilator holds the patient at end expiration for approximately 10–30 seconds. This maneuver decreases intrathoracic pressure and transiently increases venous return, effectively creating a brief auto-transfusion.

In contrast, the **end-inspiratory occlusion test** holds the ventilator at end inspiration, increasing intrathoracic pressure and transiently decreasing venous return. (Fig. 11.)

Fluid responsiveness can be inferred by evaluating changes in cardiac output or VTI during these maneuvers. Typical thresholds include:

- Approximately **5% increase in cardiac output during end-expiratory occlusion**
- Approximately **13% difference between combined inspiratory and expiratory occlusion tests**

These tests offer several advantages compared with stroke volume variation. They remain valid in patients with:

- Low tidal volume ventilation
- Poor lung compliance
- Irregular cardiac rhythms
- Assisted mechanical ventilation

Vena Cava Variation

Ultrasound evaluation of the vena cava provides another rapid bedside method for estimating fluid responsiveness.

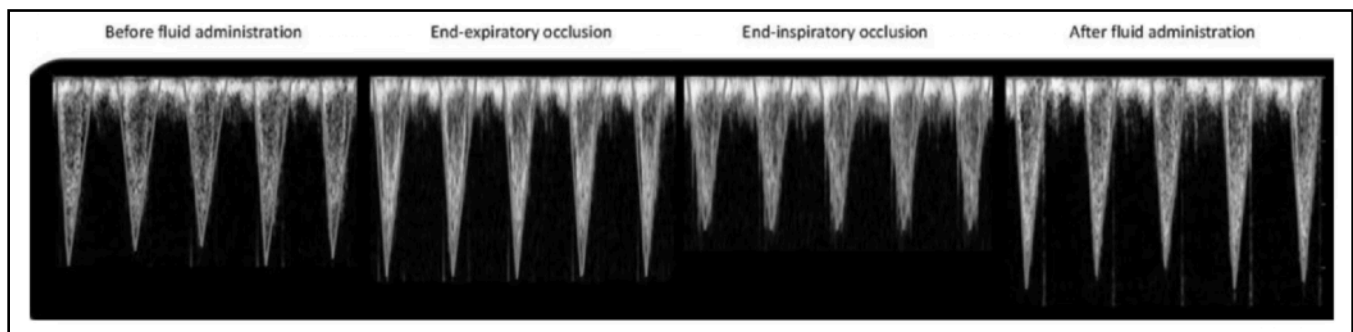


Fig. 11. Doppler waveform demonstrating changes in LVOT VTI during end-expiratory and end-inspiratory occlusion tests.

The **inferior vena cava (IVC)** is typically assessed in the subcostal view approximately 1–2 cm proximal to its junction with the right atrium (Fig. 12, below).

IVC measurements can be interpreted in two different ways depending on the patient’s respiratory status.

(1) Spontaneously Breathing Patients

During spontaneous inspiration, negative intrathoracic pressure increases venous return and causes the IVC to collapse. The **IVC collapsibility index** is calculated as:

$$\text{Collapsibility Index} = \frac{IVC_{max} - IVC_{min}}{IVC_{max}}$$

Values **greater than approximately 50%** are generally associated with fluid responsiveness.

(2) Mechanically Ventilated Patients

In mechanically ventilated patients the physiology is reversed. Positive pressure inspiration reduces venous return and causes the IVC to distend. In this setting clinicians evaluate the **IVC distensibility index**:

$$\text{Distensibility Index} = \frac{IVC_{max} - IVC_{min}}{IVC_{min}}$$

Values **greater than approximately 18%** suggest fluid responsiveness.

When visualization of the IVC is limited, similar measurements can be performed using the **internal jugular vein** (Fig. 13, next page). However, accurate measurement requires careful probe positioning because even minimal external pressure can compress the vein and produce misleading results.

Limitations of Vena Cava Measurements

Although widely used, vena cava measurements also have important limitations.

Technical factors such as poor probe alignment or excessive probe pressure can create erroneous measurements. Additionally, physiologic conditions may alter vena cava dynamics independently of intravascular volume.

Examples include:

- Cardiac tamponade
- Pulmonary embolism
- Obstructive shock
- Abdominal compartment syndrome

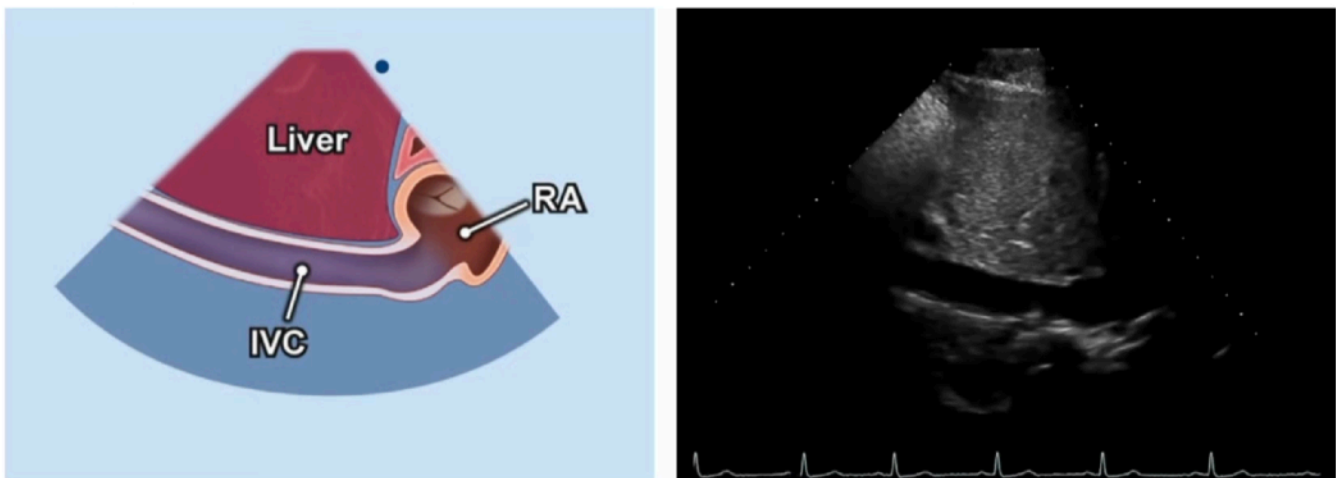


Fig. 12. Subcostal ultrasound view demonstrating the inferior vena cava entering the right atrium.

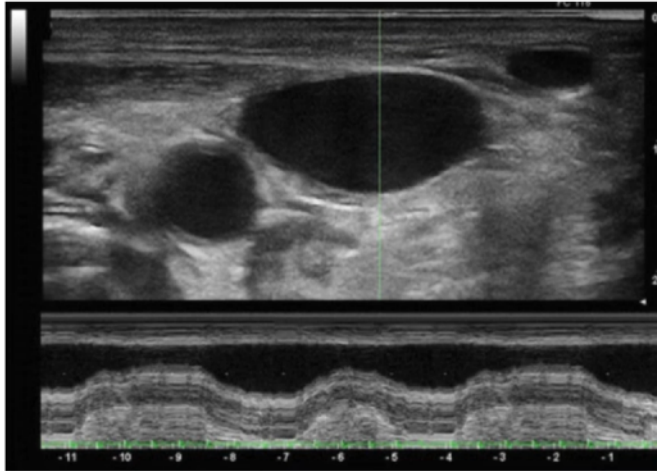


Fig. 13. Ultrasound measurement of internal jugular vein variation as an alternative to IVC assessment.

- Pregnancy
- Severe COPD or lung hyperinflation

In obstructive shock states such as cardiac tamponade, the IVC may appear plethoric despite the patient being fluid responsive, because impaired right ventricular filling causes venous congestion.

Passive Leg Raise Test

Among bedside techniques for assessing fluid responsiveness, the **passive leg raise (PLR) test** is widely considered the most reliable and versatile.

The maneuver involves raising the patient’s legs to approximately 45 degrees while the upper body is lowered into a supine position (Fig. 14). This transfers venous blood from the lower

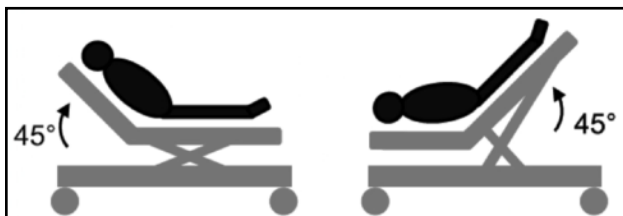


Fig. 14. Passive leg raise maneuver demonstrating transfer of venous blood toward the central circulation.

extremities into the central circulation, producing a transient **auto-transfusion of approximately 300 mL of blood**.

The hemodynamic response is measured using any available cardiac output surrogate, including:

- Stroke volume variation
- LVOT VTI
- Arterial waveform analysis
- Direct cardiac output monitoring

Measurements should be obtained:

1. At baseline
2. Approximately one minute after the maneuver
3. After returning the patient to the original position

Because the transferred blood rapidly redistributes, the effect is transient. Observing both the rise and subsequent return toward baseline confirms that the response is attributable to the preload challenge rather than other hemodynamic fluctuations.

The passive leg raise test offers several advantages. It is reliable in:

- Spontaneously breathing patients
- Mechanically ventilated patients
- Patients with arrhythmias
- Patients with varying lung compliance or tidal volumes

Limitations of Passive Leg Raise

Despite its versatility, the passive leg raise test is not universally applicable.

Situations that may limit its use include:

- Open abdomen or severe abdominal hypertension
- Pelvic or lower extremity fractures
- Pregnancy
- Bilateral lower limb amputation
- Extreme hemodynamic instability

In such cases, a **mini fluid challenge** (for example 50–250 mL) may serve as an alternative preload test.

Integrating Fluid Assessment Into Clinical Practice

Effective fluid management requires integration of physiologic assessment with clinical judgment. No single measurement can determine the correct management strategy in every patient. Instead, clinicians must synthesize multiple sources of information.

A practical framework includes four key questions:

1. Is the patient in shock?
2. What is the patient's current volume status and responsiveness?
3. Can the patient tolerate additional volume?
4. Is fluid administration appropriate in this clinical context?

By systematically addressing these questions, clinicians can avoid both under-resuscitation and fluid overload while tailoring therapy to the patient's physiologic state.

Summary

Fluid management remains one of the most important and complex aspects of perioperative and critical care practice. While no single test reliably predicts fluid responsiveness in every patient, combining physiologic understanding with dynamic bedside assessment allows clinicians to tailor fluid therapy to the individual patient. Thoughtful integration of these tools can help avoid both under-resuscitation and fluid overload while improving hemodynamic management in surgical and critically ill populations.

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