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PERIOPERATIVE CARDIOVASCULAR ROUNDS

Hemodynamic instability and fluid responsiveness

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Scenario

The rapid response team was called to evaluate a 64-yr-old hypotensive and hypoxic female patient hospitalized for deep venous thrombosis. Low-molecular-weight heparin had been initiated two days previously. The rapid response team observed her vital signs: heart rate, 110 beats·min^{-1}; blood pressure, 90/65 mmHg; and oxygen saturation, 90% while receiving 100% oxygen via a non-rebreather face mask. Using bedside focused ultrasonography, the patient's inferior vena cava was found to be < 2 cm and to collapse during inspiration. Lung ultrasonography revealed a spontaneous left-sided hemothorax. She was transferred to the intensive care unit, and a chest tube was inserted while coagulation was corrected using fresh frozen plasma. A chest radiograph confirmed the left-sided hemothorax. An inferior vena cava filter was inserted and the patient's condition rapidly stabilized. She was discharged from hospital three days later.

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Interpretation

This case illustrates the use of focused echocardiography in the presence of hemodynamic instability and hypoxemia.[1,2] A diagnosis of pulmonary embolism was considered in this patient because of the history of deep venous thrombosis; however, within a minute, bedside ultrasonography revealed the presence of hypovolemia from a hemorrhagic shock secondary to a left-sided hemothorax. The diagnosis of hypovolemia is challenging and is discussed in more detail.

For decades, intravenous fluid administration has played a key role in the management of patients with hemodynamic instability such as septic and hypovolemic shock. A landmark study from Rivers *et al*. showed that a protocol of early goal-directed therapy partly based on volume optimization reduces organ failure and improves outcomes in patients with severe sepsis or septic shock.[3] Uncorrected hypovolemia leading to inappropriate infusion of vasopressors may increase organ hypoperfusion and ischemia.[4] On the other hand, hypervolemia and excessive fluid administration have been associated with increased complications and mortality.[5,6] Based on these evidences, there is a compelling need for rapid reliable noninvasive methods to help clinicians assess the fluid responsiveness of their patients in situations of hemodynamic instability.

Clinical implications of hypovolemia

The first step in the management of hemodynamic instability is to recognize the clinical manifestations associated with this condition (Table 1). These variables are suggestive of tissue hypoperfusion and need to be corrected; however, these signs are non-specific and could be, for example, from a cardiogenic or non-cardiogenic

Table 1 Clinical manifestations of hypoperfusion

Loss of mentation
Mottled skin/cold extremities
Diminished capillary refill
Low urine output
Low mean arterial pressure
Tachycardia
Low cerebral saturation (ScO_2)
Low mixed venous oxygen saturation ($ScvO_2$)
Elevated difference in venous-arterial carbon dioxide pressure ($PvCO_2$ - $PaCO_2$)
Metabolic acidosis
High blood lactate

etiology. In a clinical setting, fluid administration is then often considered. Nevertheless, clinical studies have shown that only 50% of hemodynamically unstable critically ill patients are volume responsive,[7] i.e., they have increased their cardiac output following a fluid challenge. How can we show fluid responsiveness?

Based on the work by Guyton in a previous article on perioperative hemodynamic rounds,[8] venous return is physiologically determined by the difference between mean systemic pressure and right atrial pressure divided by the resistance to venous return. During steady state, venous return is equal to cardiac output. Hypovolemia could be defined as a reduction in mean systemic venous pressure without any change in venous compliance. When volume is given, mean systemic pressure increases, leading to an increase in venous return. This is associated with an increase in right and left ventricular (LV) preload.[8,9] According to the Frank-Starling principle, as the LV preload increases, stroke volume increases until the optimal preload is reached, at which point, the stroke volume will remain constant having reached the flat part of the curve.[10] This relationship with preload (*x* axis) is related to cardiac output (*y* axis), as illustrated in Fig. 1. Curve *A* represents a patient with a normal cardiac function. A1 and A2 are on the ascending part of the curve, i.e., cardiac output can be increased by an increase in preload. We can see that A3 is on the flat part of the curve. An increase in preload will not have an effect on cardiac output even if cardiac function is normal. On the other hand, curve *B* represents a case of heart failure. On this curve, B1 will be responsive to a preload increment, B2 and B3 will not, being on the flat part of the curve. It is therefore important to determine the patient's individual position on the Frank-Starling curve, since the only reason to give a fluid challenge would be to increase the patient's stroke volume.

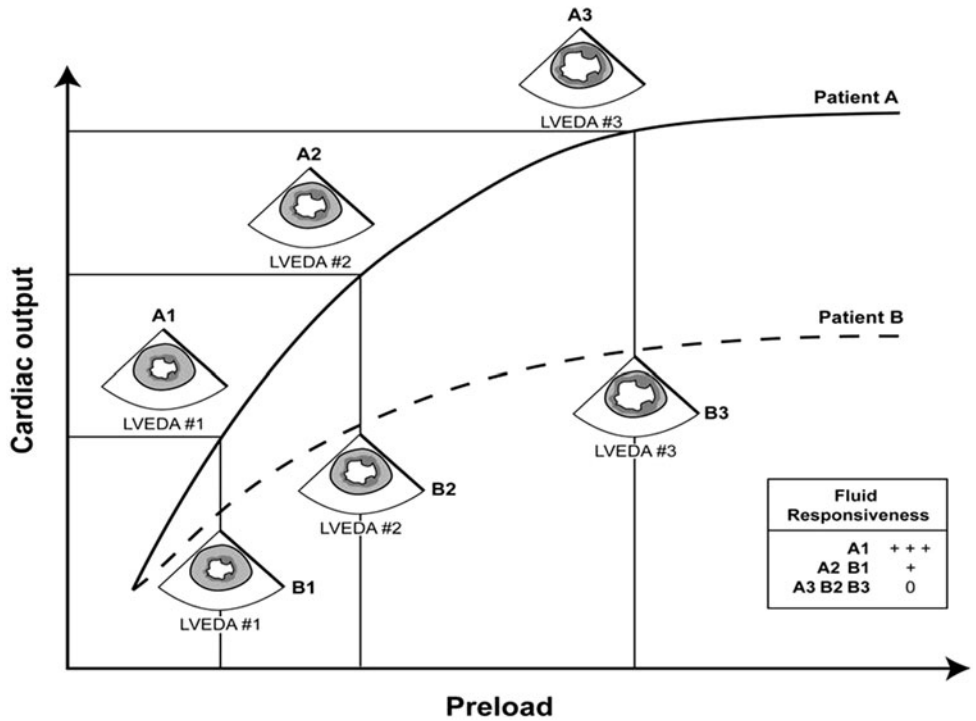


Fig. 1 Fluid responsiveness, preload, and cardiac function. The relation between cardiac output and preload estimated using LVEDA in two different patients (A and B) with different ventricular function is shown. The response to fluid will be different for the same LVEDA depending on the position where the actual LVEDA lies in the ventricular function curve. Therefore, the response of patient A1 to volume will be more than that of patient B1. Patients A3, B2, and B3 will respond poorly to volume. This shows why absolute LVEDA does not necessarily correlate with volume responsiveness. LVEDA = left ventricular end-diastolic area. (With permission of Denault *et al.* © Informa Healthcare, Transesophageal Echocardiography Multimedia Manual: A Perioperative Transdisciplinary Approach, 2011)[41]

Static variables

For years, physicians have studied new ways of predicting fluid responsiveness. Traditionally, central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) have been used to guide fluid management in critically ill patients. These static variables reflect right and left atrial pressure, respectively. It has been shown that these two variables are inaccurate in predicting fluid responsiveness and should not be used to guide fluid management.[11,12] Figure 2 summarizes common static variables, suggestive or not of fluid responsiveness. Both hemodynamic and two-dimensional Doppler echocardiographic variables can be used.

In patients monitored with a pulmonary artery catheter, CVP, PCWP, and right ventricular (RV) waveform analysis can be performed to estimate right and LV filling pressure. Figures 2A and 2B illustrate two markers currently used in our institution to help predict fluid responsiveness.[13] Figure 2A represents a CVP or PCWP waveform with the *a* and *v* waves. A higher *a* wave in relation to the *v* wave (normal *a/v* ratio) is typically associated with normal ventricular filling pressures and cardiac function. Therefore, normal waveforms are more likely to predict fluid responsiveness. As shown in Fig. 2B, RV pressure waveform analysis can help identify normal or abnormal RV function. A horizontal diastolic slope represents normal RV compliance, normal RV diastolic function, normal filling pressures, and most likely fluid responsiveness. On the other hand, fluid responsiveness would be less likely if an oblique diastolic curve were observed, which corresponds to reduced RV compliance and high filling pressures.[14]

The two-dimensional echocardiographic measurements of the right and left end-diastolic areas have also been studied. The results of these studies show that these variables cannot always reliably predict fluid responsiveness except when they are continuously monitored and when the left ventricle is small and hyperkinetic, as shown in Fig. 2.[15,16]

In terms of Doppler variables, both LV filling pressures and PCWP can be estimated by the E/A ratio of the transmitral flow (TMF) Doppler signal. High E/A ratios represent high filling pressures. Preload reduction is associated with a reduction in filling pressure and a low E/A ratio.[17,18] Lattik *et al*. observed that patients with a low to normal E/A ratio may be volume responders as opposed to patients with an elevated E/A ratio.[19] Nevertheless, another study shows conflicting data regarding this observation.[15] The combination of tissue Doppler imaging with TMF has been shown to reflect filling pressure more accurately than their use as single variables.[20] Tissue Doppler imaging measures velocity at the level of the mitral valve annulus in contrast with TMF where the blood flow velocity is measured at the tip of the mitral valve in the LV cavity. In the operating room where hemodynamic changes are frequent, the combination of these modalities as predictors of fluid responsiveness has not been extensively studied and validated.

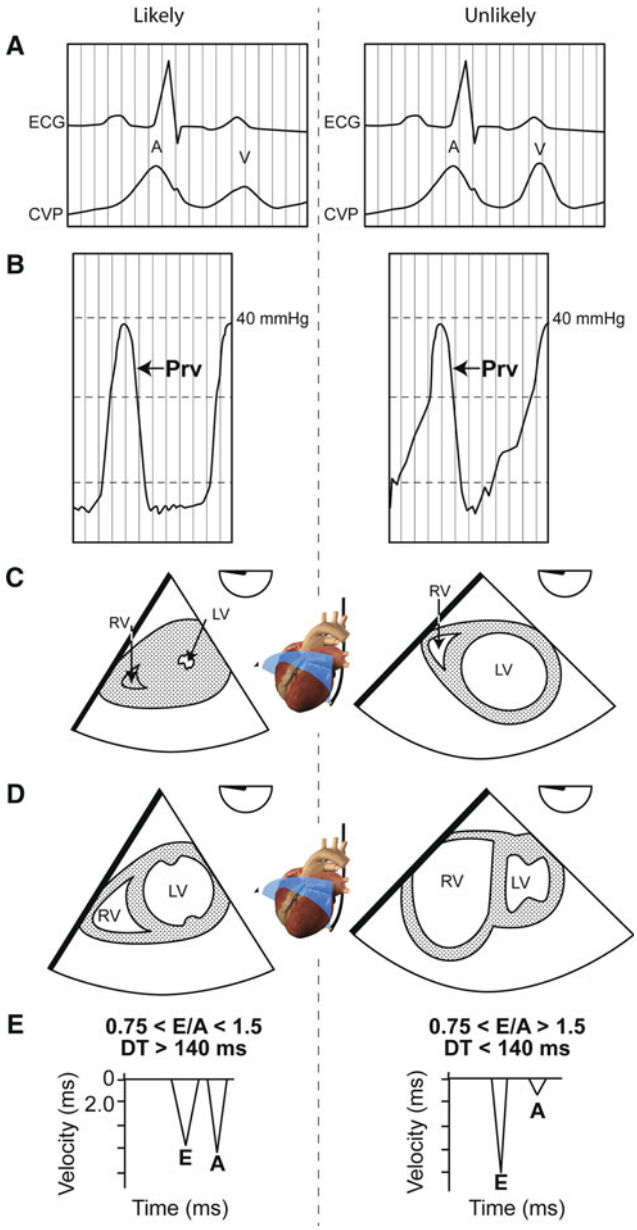


Fig. 2 Static variables of fluid responsiveness defining response as likely or unlikely. (A) CVP or pulmonary capillary wedge pressure curve waveform analysis[13] (B) Prv waveform analysis[13] (C) LV end-diastolic area[15,16] (D) RV area[42] (E) Transmitral flow velocity using pulsed Doppler.[19] A = atrial transmitral filling; CVP = central venous pressure; E = early transmitral filling; EKG = electrocardiogram; DT = deceleration time; LV = left ventricular; Prv = right ventricular pressure; RV = right ventricular. (With permission of Denault *et al*. © Informa Healthcare, Transesophageal Echocardiography Multimedia Manual: A Perioperative Transdisciplinary Approach, 2011)[41]

Table 2 Predictive value of techniques used to determine fluid responsiveness

Method	Technology	AUC	Reference
Superior vena cava respiratory variation	Transesophageal echocardiography	0.99 (0.98-1.00)	30
Passive leg raising (PLR)	Cardiac output measurement	0.95 (0.92-0.97)	36
Passive leg raising (PLR)	End-tidal carbon dioxide ($ETCO_2$)	0.94 (0.82-0.99)	38
Pulse pressure variation (PPV)	Arterial waveform	0.94 (0.93-0.95)	10
Inferior vena cava respiratory variation	Echography	0.91 (0.84-0.98)	28
Pulmonary capillary wedge pressure (PCWP) waveform analysis	Pulmonary artery catheter	0.89 (0.79-0.99)	13
Systolic pressure variation (SSV)	Arterial waveform	0.86 (0.82-0.90)	10
Stroke volume variation (SVV)	Pulse contour analysis	0.84 (0.78-0.88)	10
Left ventricular end-diastolic area (LVEDA)	Echocardiography	0.64 (0.53-0.74)	10
Global end-diastolic volume (GEDV)	Transpulmonary thermodilution	0.56 (0.37-0.67)	10
Central venous pressure (CVP)	Central venous catheter	0.55 (0.48-0.62)	10

AUC = area under the curve with 95% intervals

Dynamic variables

Using variations in venous return (preload) and cardiac output, dynamic variables have been proven to be much more reliable than static variables to assess fluid responsiveness, as shown in Table 2.[10,16]

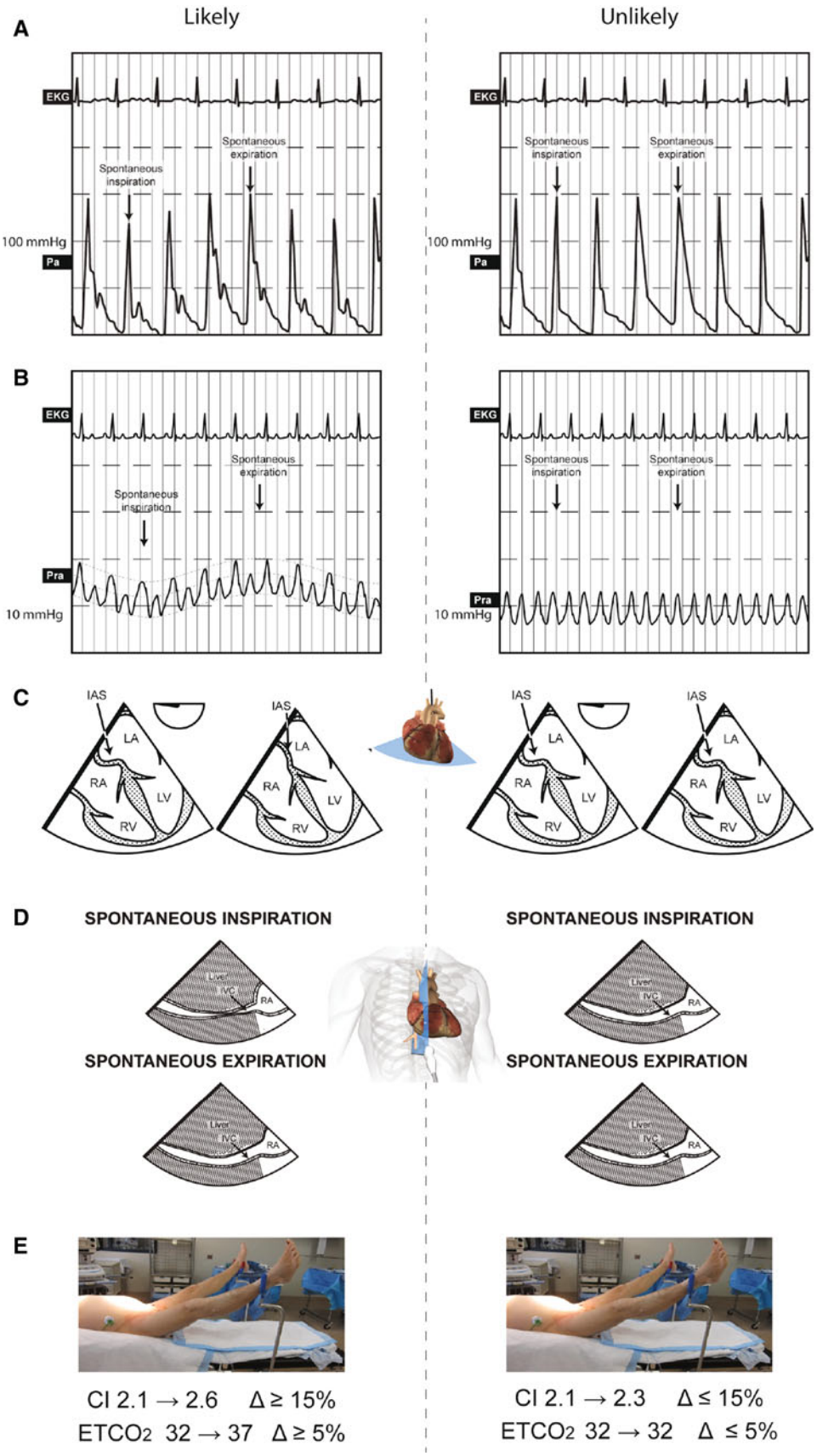
Pulse pressure variation (PPV) and systolic pressure variation (SPV)—both derived from analysis of the arterial waveform—, stroke volume variation (SVV), and the variation of the amplitude of the pulse oximeter plethysmographic waveform are predictive of fluid responsiveness.[7,21,22] These techniques use heart-lung interactions during mechanical ventilation. During inspiration, pleural pressure increases, causing a reduction in RV preload and an increase in RV afterload. The RV stroke volume is then reduced, which creates a decrease in LV filling and LV stroke volume two to three heart beats later.[23] These changes are greater when the ventricles operate on the steep part of the Frank-Starling curve, i.e., fluid responders should experience greater change in these variables compared with non-responders who operate on the flat part of the curve. A variation $> 12\%$ of these values has been reported to be highly predictive for fluid responsiveness.[7,10] The SPV in a passive patient with mechanical ventilation is illustrated in Fig. 3A. Right atrial pressure and CVP, its surrogate, respiratory variations are also predictive of fluid responsiveness as shown in Fig. 3B.[24]

These approaches have several limitations. Arrhythmias and spontaneous respiration will influence the measured responses. These respiratory variations cannot be used in an open chest condition such as cardiac surgery. Also, De Backer *et al.* have shown that PPV is unreliable when tidal volume is < 8 mL·kg^{-1}, i.e., larger tidal volumes are needed to induce these respiratory variations.[25] Pulse

Fig. 3 Dynamic variables of fluid responsiveness defining response as ▶ likely or unlikely. (A) Pulse pressure and systolic pressure variation[10] (B) Pra spontaneous respiratory variation[24] (C) IAS systolic movement, reflecting left atrial pressure[43,44] (D) IVC or superior vena cava respiratory variation.[28,30] This figure shows a case of an IVC respiratory variation in a spontaneously breathing patient. (E) Passive leg raising and change in CI and $ETCO_2$.[34,38] CI = cardiac index; EKG = electrocardiogram; $ETCO_2$ = end-tidal carbon dioxide; IAS = interatrial septum; IVC = inferior vena cave; LA = left atrium; LV = left ventricle; Pra = right atrial pressure; RA = right atrium; RV = right ventricle. (With permission of Denault *et al.* © Informa Healthcare, Transesophageal Echocardiography Multimedia Manual: A Perioperative Transdisciplinary Approach, 2011)[41]

contour-based hemodynamic monitoring devices, such as the FloTrac-Vigileo™ (Edwards Lifesciences, Irvine, CA, USA), the most recent and most widely studied device, have inherent inaccuracies. As shown in a recent review, these devices may provide variable SVV measurements in situations of low systemic vascular resistance, including septic shock, and problems may be encountered when tracking changes in stroke volume after a volume challenge or after the use of vasopressors.[26] Nevertheless, these devices are much more reliable and accurate for measuring PPV, which does not use pulse contour analysis.[7] This suggests that PPV should then be the variable used with these devices.

Echocardiographic evaluation of the respiratory changes in the diameter of the two venae cavae can also help predict fluid responsiveness. Using a subxiphoid view in completely passive patients with mechanical ventilation, inferior vena cava respiratory variation has been associated with a positive fluid responsiveness,[27,28] as shown in the current case and in Fig. 4. Either M-mode, as shown in Figs. 4 and 5, or direct visualization could then be used. This variable is not as sensitive in mechanically ventilated


A
Likely
Unlikely
EKG
Spontaneous inspiration
Spontaneous expiration
100 mmHg
Pa
B
Pra
10 mmHg
C
IAS
LA
RA
LV
RV
D
SPONTANEOUS INSPIRATION
SPONTANEOUS EXPIRATION
Liver
IVC
RA
E
CI 2.1 → 2.6 Δ ≥ 15%
ETCO2 32 → 37 Δ ≥ 5%
CI 2.1 → 2.3 Δ ≤ 15%
ETCO2 32 → 32 Δ ≤ 5%

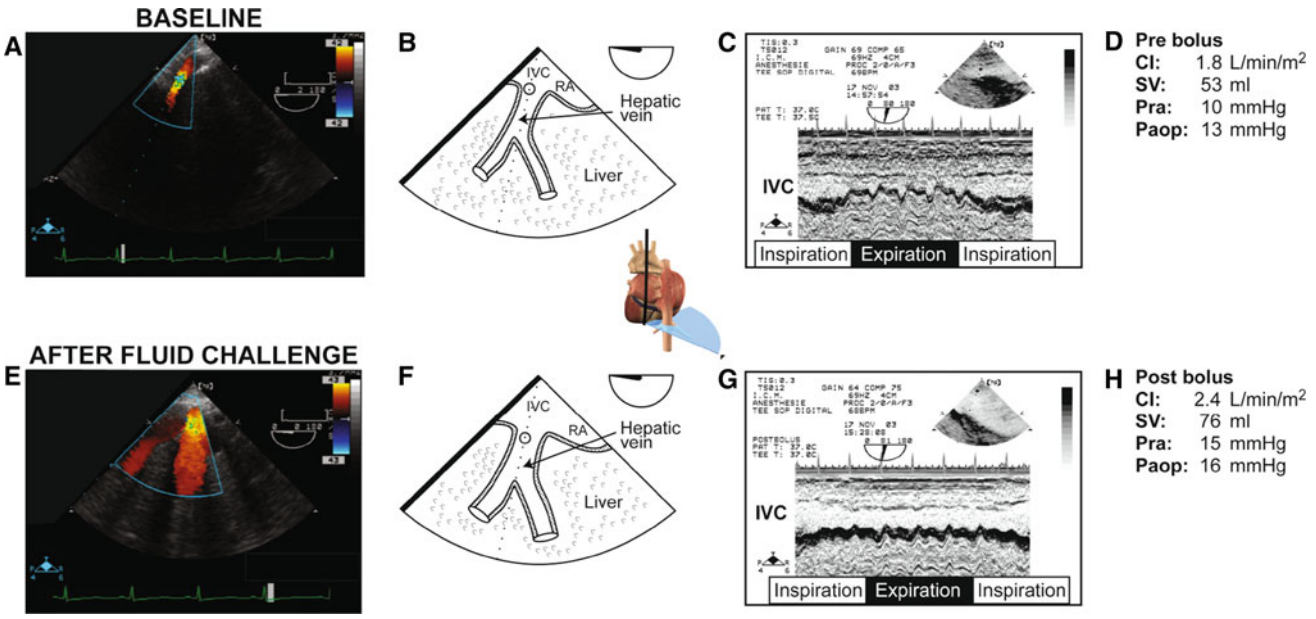


Fig. 4 The effect of fluid loading on hemodynamic and echocardiographic variables in a 62-yr-old man on mechanical ventilation is shown. (A, B) At baseline, the diameter of the IVC and hepatic veins is small. (C) Using M-mode, the IVC collapses during the lowest period of intrathoracic pressure, the expiration phase of positive-pressure ventilation. (D) The cardiac index (CI) is 1.8 $L \cdot min^{-1} \cdot m^{-2}$. (E–G) After a fluid challenge, the diameter of the IVC and hepatic veins has enlarged without significant IVC collapse. (H) CI and filling pressures increased. CI = cardiac index; IVC = inferior vena cava; Paop = pulmonary artery occlusion pressure; Pra = right atrial pressure; RA = right atrium; SV = stroke volume. (With permission of Denault *et al.* © Informa Healthcare, Transesophageal Echocardiography Multimedia Manual: A Perioperative Transdisciplinary Approach, 2011)[41]

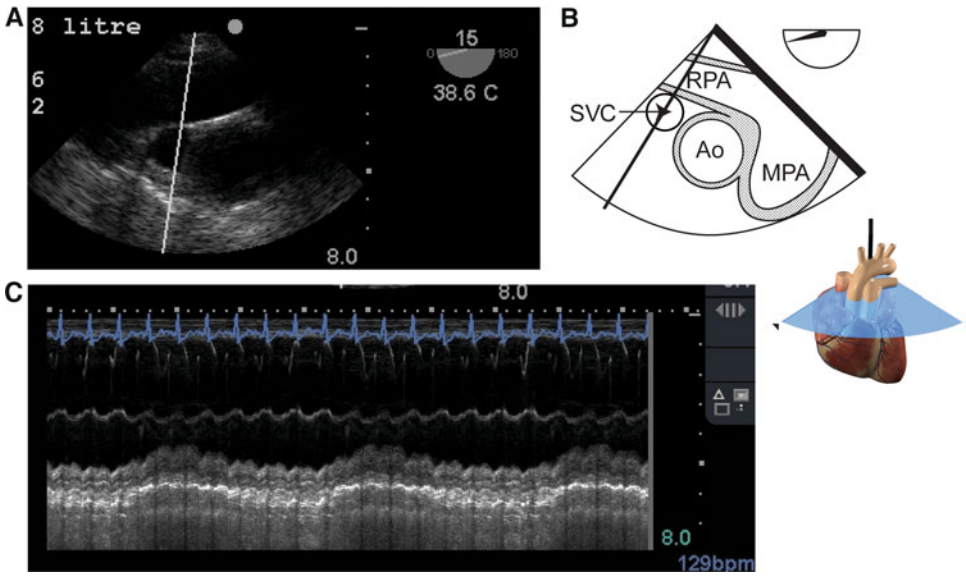


Fig. 5 Respiratory variation of the SVC. (A, B) Mid-esophageal ascending Ao short-axis view in a 76-yr-old man in the intensive care unit after removal of 1.8 L with dialysis is shown. (C) Using M-mode, significant respiratory variation of the diameter of the SVC was present. *Abbreviations*: Ao = aorta; MPA = main pulmonary artery; RPA = right pulmonary artery; SVC = superior vena cava. (With permission of Denault *et al.* © Informa Healthcare, Transesophageal Echocardiography Multimedia Manual: A Perioperative Transdisciplinary Approach, 2011)[41]

patients.[29] The superior vena cava (SVC) can be assessed using transesophageal echocardiography. Its cyclic collapsibility is also related to fluid responsiveness (Fig. 5).[30]

Echocardiographic assessment of static and dynamic parameters of fluid responsiveness needs to be performed by an experienced operator. The clinician's expertise is thus the major limiting factor. Figure 3 summarizes

common dynamic variables, suggestive or not of fluid responsiveness.

Passive leg raising as a marker of fluid responsiveness

Since dynamic variables cannot be used in spontaneously breathing patients, new variables have been studied to predict fluid responsiveness. Passive leg raising (PLR), as shown in Fig. 3E, induces a transfer of blood from the lower limbs to the intrathoracic compartment, creating an intrinsic volume challenge of 350 mL.[31] This action has the benefit of being reversible, applicable to the bedside, maximal in 60 sec, and will not create fluid overload.[32] Passive leg raising has been validated to predict fluid responsiveness in both spontaneous breathing patients and passive patients with mechanical ventilation,[33,34] but it is not reliable in the presence of abdominal hypertension.[35,36] To assess the maximal hemodynamic effects of PLR, cardiac output and stroke volume variations must be continuously monitored. Transthoracic echocardiography, esophageal Doppler, pulse contour analysis, and femoral Doppler have all been validated in this situation, as shown in a recent meta-analysis.[34] This same meta-analysis confirms the excellent value of PLR to predict fluid responsiveness with an area under the receiver operating curve of 0.95, which is best among the different dynamic variables, as shown in Table 2. Recently, end-tidal carbon dioxide ($ETCO_2$) has also proved its reliability as a surrogate to cardiac output measurement to predict fluid responsiveness with PLR.[37,38]

Finally, once it has been established whether or not there is fluid responsiveness in the presence of hemodynamic instability, the next step is to determine why the patients became fluid responsive. In the current case, two possibilities were contemplated: loss of volume (hypovolemic or hemorrhagic shock) or increased venous compliance such as septic shock. Point-of-care echocardiography can be used for rapidly identifying or ruling out the presence of blood in the thoracic and abdominal cavity, but it can also be used to identify conditions associated with septic shock, such as pneumonia, empyema, peritonitis, liver abscess, cholecystitis, and pyelonephritis.

In conclusion, assessing fluid responsiveness is an important feature in the management of patients with hemodynamic instability. In our experience, we do not rely on one variable alone. Combinations of multiple signs are more useful. In some patients, fluid responsiveness can be diagnosed easily using PLR, but in others, it is more complex. Occult hypoperfusion can still occur despite what seems to be adequate fluid resuscitation. The use of such modalities as the renal or splenic Doppler resistance index in trauma is promising for identifying those patients at the bedside, but these modalities will need further confirmation in critically ill patients.[39,40] Dynamic variables are most useful in finding the patient's individual point on the Frank-Starling curve.

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Conflicts of interests None declared.

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