EDITORIAL

Using echocardiography to predict fluid-responsiveness and manage the need for fluids

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Management of fluids has progressively moved toward a more dynamic and functional approach based on the prediction of fluid responsiveness (FR), to improve efficacy and benefits and limit detrimental effects of fluids [1]. Application of an optimal threshold for validated parameters above which the patient could be fluid-responsive generates a "gray zone" [2]. Therefore, the objective of intensivists should be to adapt the threshold to the respective sensitivity and specificity they need to predict FR, according to the patient's condition. Intensivists should consider being very specific in severely hypoxemic patients where fluids can be significantly harmful and mainly sensitive in patients without blood gas abnormalities, as maintaining non-optimal volume could be detrimental here. Applying a continuous approach to the prediction of FR, i.e., how much the cardiac output is expected to increase after fluid bolus, rather than the classic binary one, i.e., will the patient be a "responder", could be more efficient [3].

Echocardiography is crucial for management of patients with respiratory or circulatory failure. Many echo parameters have been proposed to predict FR. Measurement of aortic velocity time integral (VTI) is cardiac beat-related and precise enough to detect small changes in stroke volume, i.e., 6–4% precision when measured in one or averaged in 3 cardiac beats, respectively, in patients in sinus rhythm with no impact of the type of ventilation [4]. This measurement requires a good alignment with the ejection flow and a nice envelope with the aortic closing sound [5].

We propose here how to use echocardiography to predict FR and differentiate 3 situations (Table 1). In the first, filling should not be considered, and sometimes contra-indicated; echo parameters indicate that the patient is already overfilled and congestive. In the second, filling should be highly considered; echo parameters suggest it is very likely the patient is hypovolemic and then fluid-responsive. In the last, filling could be optional; hypovolemia is already corrected, but echo parameters indicate that cardiac output could still increase to a certain level after fluid bolus.

However, a few key points should be first discussed: (i) Echocardiography must be interpreted as a whole, while most parameters are unfortunately validated in isolation; (ii) intensivists do not treat an echo image but a patient and echocardiography is no more than a help; (iii) in many cases, intensivists have just to fill the patient, as during bleeding or at the very early phase of septic shock; and (iv) echocardiography is limited to give direct information on end-organ perfusion [6].
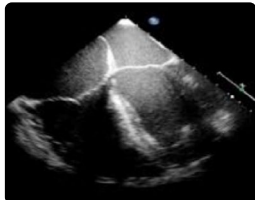
Do not fill when

1   The right ventricle (RV) is severely dilated (the right ventricle is bigger than the left ventricle (LV)), with or without a paradoxical septal motion [7]. Giving more fluids could be deleterious even in the case of non-severe right ventricular dilatation, while the optimal threshold of RV/LV end-diastolic area is unknown.

2   End-expiratory inferior vena cava (IVC) diameter is higher than 25–27 mm. This reflects significant

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Table 1 Main echo parameters (route and views, ventilation, clinical context, indication, limitations).

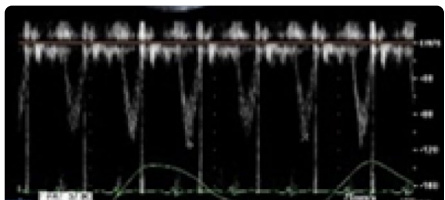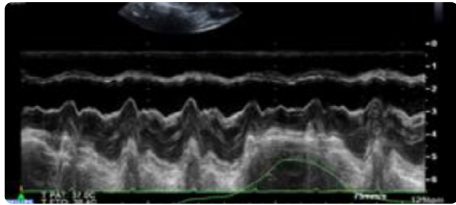| RV size: RV/LV EDA	
Route and views	TEE transverse mid-esophageal view. TTE apical 4-chamber view.
Type of ventilation	SB and IMV.
Clinical context	Described in ARDS and in septic shock.
Fluids	Do not fill when RV severely/markedly dilated.
Limitations	Optimal threshold of RV/LV EDA is unknown; could be lower than 1. A non- severely dilated right ventricle do not always exclude to fill the patient according to the other parameters.

Respiratory variations of aortic maximal velocity: Vmax - Vmin/Vmean	
Route and views	TEE transgastric oblique view at 110°. TTE apical 5-chamber view.
Type of ventilation	IMV without spontaneous effort.
Clinical context	Vt 6-8 mL/kg. Shock.
Fluids	Optional when > 10% during tidal ventilation.
Limitations	Sinus rhythm is mandatory. May be present in unresponsive patient with severe RV dilatation. False negative may occur in the case of very low tidal volume.

Table 1 (continued)

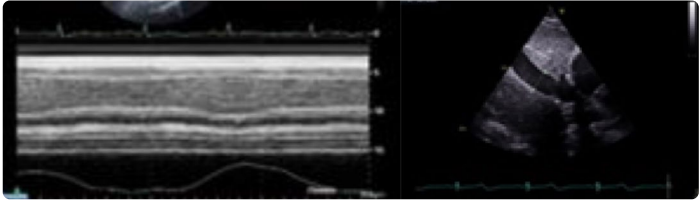| SVC respiratory variations (SVC collapsibility index): Dmax - Dmin/Dmax	
Route and views	TEE longitudinal (90°) upper esophageal view. 2D may be combined with time-motion.
Type of ventilation	IMV without spontaneous effort.
Clinical context	Vt 6-8 mL/kg. Shock. Still usable in arrhythmia.
Fluids	Fill when collapsibility >31% or partial/complete SVC collapse. Optional when mild SVC respiratory variations. It is likely that in the absence of any respiratory variation the patient is non- responder, i.e. do not fill.
Limitations	TEE is required. False negative in case of very low Vt?
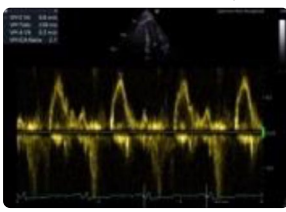
End-expiratory IVC diameter	
Route and views	TTE sub-costal view. 2D may be combined with time motion.
Type of ventilation	SB or IMV.
Clinical context	Vt 6- 8 mL/kg. Shock. Still usable in arrhythmia.
Fluids	Fill when diameter < 10 mm. Do not fill when diameter > 25-27 mm.
Limitations	2/3 of patients are between 10 and 25-27mm. Place of measurement can be questionable.

Table 1 (continued)

LV filling pressure	
Route and views	TEE transverse mid-esophageal view. TTE apical 4-chamber view. Pulsed wave Doppler and tissue Doppler imaging.
Type of ventilation	SB or IMV.
Clinical context	Most studies done in the cardiological field.
Fluids	Do not fill when elevated (restrictive mitral inflow with E/A>1.8 and/or E/E'>15).
Limitations	A restrictive mitral inflow may be observed in young patients (low LV compliance). E/E' was mainly validated in cardiac patients.

Valvulopathy	
Fluids	Do not fill when severe MR, AR or AS
Limitations	Skills of intensivists. Discussion with an echo expert can be useful. Patients with severe AS poorly tolerates hypovolemia.
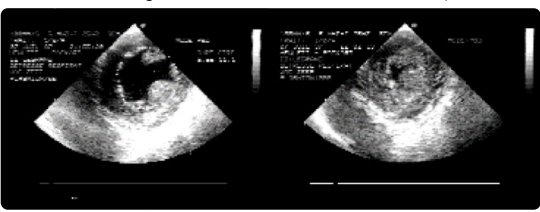
LV size and function	
Route and views	TEE transverse transgastric view. TTE parasternal short axis view. (At papillary muscle level).
Type of ventilation	SB or IMV.
Clinical context	Septic shock. Dynamic obstruction with SAM may be associated.
Fluids	Fill when "kissing" LV with low end-diastolic area (<5 cm²/m²).
Limitations	A "kissing" LV is observed in profound vasoplegia but LVEDA is preserved.

Table 1 (continued)

Passive Leg Raising	
Type of ventilation	SB or IMV.
Clinical context	Validated regardless type of ventilation, cardiac rhythm, Vt, lung compliance.
Fluids	Optional when increase in aortic VTI ≥10% within 1 minute.
Limitations	False negative if venous compression stockings or intra-abdominal pressure ≥ 16 mmHg.

ACP Acute cor pulmonale, *AR* aortic regurgitation, *ARDS* acute respiratory distress syndrome, *AS* aortic stenosis, *Dmax* maximal diameter at end-expiration, *Dmin* minimal diameter at end-inspiration, *EDA* end-diastolic area, *IMV* invasive mechanical ventilation, *IVC* inferior vena cava, *LV* left ventricle, *MR* mitral regurgitation, *RV* right ventricle, *SAM* systolic anterior motion of mitral leaflet, *SB* spontaneous breathing, *SVC* superior vena cava, *TEE* transesophageal echocardiography, *TTE* transthoracic echocardiography, *Vt* tidal volume, *VTI* velocity time integral, *Vmax* maximal velocity at end-inspiration, *Vmean* mean velocity, *Vmin* minimal velocity at end-expiration

congestion, with effects on the liver and kidney. It is associated with 80–90% of fluid non-responders [8].

3. An echo pattern of cardiogenic shock is observed, defined by a decreased LV ejection fraction (LVEF) with elevated LV filling pressure (restrictive mitral inflow with E/A > 1.8 and/or E/E′ > 15 [9]). In contrast, patients with septic cardiomyopathy could be fluid-responders despite decreased LVEF, but LV filling pressure is not increased here (no restrictive mitral inflow and even E/A < 1).

4. Grade 3 or 4 mitral or aortic regurgitation is observed. If there is difficulty in evaluating severity, the intensivist should confer with the cardiologist. This is also probably true when severe right-side valve lesion is observed. If so, the right ventricle is usually dilated.

Fill when

1. A "kissing" left ventricle is observed. This is associated with LV hyperkinesia and a low end-diastolic area (< 5 cm^2/m^2); in profound vasoplegia, the left ventricle is still hyperkinetic but its end-diastolic area is normal; this does not require more fluids. Dynamic obstruction may also be observed and corrected by fluids [10]. "A kissing left ventricle" may be observed in case of RV failure; the left ventricle is then also underfilled but filling is not recommended.

2. End-expiratory IVC diameter is lower than 10 mm. This is usually associated with a low central venous pressure and 90% of patients are fluid-responders [8].

3. The decrease in superior vena cava (SVC) diameter is greater than 31% during tidal ventilation, or more obvious when a partial or complete SVC collapse is observed. Its specificity in predicting a response to fluids is 90% [11]. This is only validated in mechanically ventilated patients without any spontaneous

effort and requires transesophageal echocardiography.

Optional when

Echo parameters are usually in the gray zone. As discussed above, prediction of how much cardiac output/aortic VTI is expected to increase after fluid bolus could be an adequate approach to decide whether to perform fluid expansion. The patient's clinical situation is more crucial here, as severely hypoxemic patients will probably not be filled.

1. Mild SVC respiratory variations are observed. Here, the sensitivity in predicting a significant increase in aortic VTI is close to 90%, but the specificity is very low (25%) [11]. The greater the respiratory variations, the greater is the expected increase in aortic VTI [3].

2. An increase in aortic maximal velocity is observed during tidal ventilation. This is only validated in mechanically ventilated patients without spontaneous effort. This is the most sensitive parameter [11]; it reflects LV preload dependency, like pulse pressure variations, and may occur in unresponsive patients with severe RV dilatation [12]. Therefore, it cannot be interpreted without information on RV size. The greater the respiratory variations, the more the aortic VTI is expected to increase in patients without RV impairment [3].

3. In case of doubt, or in association, a passive leg raising (PLR) may be performed. It is very likely that the patient is responder when PLR induces an increase in aortic VTI greater than 10% within 1 min after starting the maneuver [13]. PLR is considered a very good way to assess FR [14], especially when other parameters are not available, but has its own limitation (high intra-abdominal pressure). It mimics fluid expansion

(300 mL is mobilized from the legs and abdomen to the chest) and allows intensivists to include severely hypoxemic patients in clinical research on fluid management. A mini-fluid challenge with 100 mL of saline with assessment of the aortic VTI has been also proposed [15].

Finally, IVC respiratory variations, while popular, cannot accurately predict FR. In spontaneously breathing patients, IVC collapsibility index depends on the level of inspiratory effort and, in mechanically ventilated patients, IVC distensibility index is only obtained in 78% of patients and has a poor predictive performance [11].

In conclusion, our aim was to report our practical vision of how echocardiography could help intensivists to detect FR and to manage fluids. This requires intensivists to be adequately trained in echocardiography with the risk, if not, to misinterpret the exam. This approach is based on our experience at the bedside and on the literature, but this may appear somewhat theoretical and should be formally validated as a whole. One could say that this is simplistic but "simple is always wrong; what is not is unusable" (Paul Valéry, Oeuvres II, 1942).

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Declarations

Conflicts of interest
AV-B declares lecture fees from Baxter and was paid by Air Liquide Healthcare for a research advisory board. MS and FB do not declare any conflict of interest.

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