Intensive Care Med
https://doi.org/10.1007/s00134-024-07708-w

EDITORIAL

Using the ventilator to predict fluid responsiveness

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During acute circulatory failure, infusing fluids as first line therapy is the source of a therapeutic dilemma [increase in cardiac output (CO) and improvement of tissue perfusion vs. inconsistent effectiveness and risk of fluid accumulation]. Predicting effectiveness of a fluid bolus on CO before infusing it avoids administering fluid to patients who do not require it [1]. The principle is simple: if some changes in cardiac preload, induced by external maneuvers or by the ventilator, change CO above a given diagnostic threshold, fluid infusion will likely have a similar effect [2].

The tests and indices using the ventilator (Supplementary Fig. 1) are underpinned by heart–lung interactions. Under mechanical ventilation, increased intrathoracic pressure reduces the pressure gradient of systemic venous return by increasing right atrial pressure [3]. This effect is exaggerated upon insufflation. The resulting drop in CO is larger if both ventricles are preload responsive. Ventilation also increases afterload of the right ventricle (RV) and decreases left-ventricular (LV) afterload [3], but these effects are independent of preload responsiveness and only relevant if the RV is failing.

PPV, SVV

Pulse pressure variation (PPV) results from cyclic decreases in venous return to the RV during inspiration, decreasing right-ventricular output on the next beat, subsequently decreasing LV filling about 2–3 beats later. The resultant cyclic changes in LV filling cause proportional changes in LV stroke volume in volume responsive patients, quantified as stroke volume variation (SVV). PPV in the arterial coupling effect of those LV stroke volume changes. PPV or SVV values ≥ 12% reflect volume responsiveness [4].

Many bedside monitors can measure PPV. However, its diagnostic reliability is reduced by low tidal volume (Vt), low lung compliance, cardiac arrhythmia, spontaneous ventilation, very high respiratory rate, intra-abdominal hypertension, open chest, and likely *acute cor pulmonale* (ACP) [4]. Ultimately, PPV and SVV can only be used in limited number of patients (Supplementary Fig. 1).

Vena cava diameter changes

These compliant vessels change size under mechanical ventilation in the event of preload responsiveness: the extra-thoracic inferior vena cava (IVC) dilates during inspiration as venous return is impeded, while the intrathoracic superior vena cava (SVC) collapses. However, respiratory variations of IVC diameter, and to a lesser extent SVC diameter variations, are less reliable than other tests of preload responsiveness (Supplementary Fig. 1) [5]. Moreover, they share the same limitations of PPV (except arrhythmias), and the IVC variation has limited diagnostic ability in intra-abdominal hypertension [5].

Vt challenge

The Vt challenge consists, in a patient ventilated with a Vt of 6 mL/kg, in transiently increasing Vt to 8 mL/kg for 1 min and noting the change in PPV between the two tidal volume states [6] (Fig. 1). An increase in PPV of ≥ 3.5% reflects preload responsiveness, even though this threshold varies between studies [7]. This test's advantage is that it requires only measuring changes in

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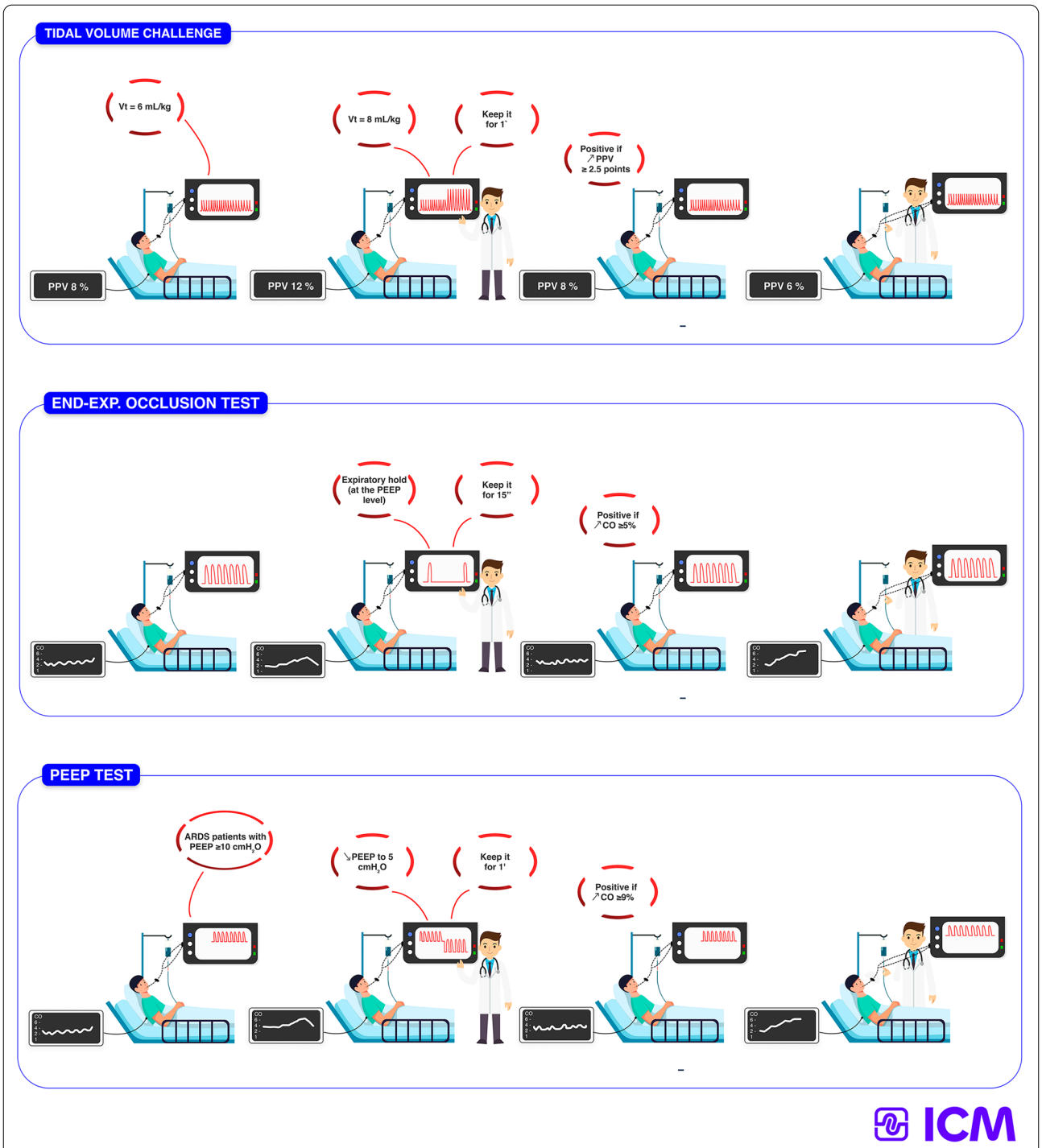

Fig. 1 Practical way of assessing preload responsiveness with three tests using the ventilator. ARDS: acute respiratory distress syndrome; CO: cardiac output; PEEP: positive end-expiratory pressure; PPV: pulse pressure variation; Vt: volume challenge

PPV, so that a simple blood pressure monitor is sufficient (Supplementary Fig. 1). The diagnostic reliability is good, but the diagnostic threshold needs to be better specified [7]. False-positive results may be observed in ACP.

End-expiratory occlusion test

It consists in interruption of ventilation at end-expiration for 15 seconds (Fig. 1). Airway pressure decreases to positive end-expiratory pressure (PEEP), decreasing intrathoracic pressure and increasing cardiac preload [8]. The occlusion must be long enough for the increased right-ventricular preload to be transmitted to the left side. The disadvantage is precisely that patients must tolerate a 15-s expiratory pause without spontaneously triggering inspiration (Supplementary Fig. 1). The test is now well validated, and the diagnostic threshold is an increase in CO ≥ 5% [9]. It may be less reliable in ACP, but this needs confirmation.

This low threshold requires a sufficiently precise CO measurement, which is not provided by ultrasonic techniques. If echocardiography or oesophageal Doppler are used, one should perform not only an end-expiratory pause, which lowers the stroke volume in the event of preload responsiveness, but also an end-inspiratory pause, which has the opposite effect (Supplementary Fig. 2). Adding the changes obtained during the two successive pauses increases the amplitude of the stroke volume changes, which become better detectable by ultrasound [10].

Recruitment maneuvers

By increasing lung volume, recruitment maneuvers increase intrathoracic pressure, which significantly reduces cardiac preload. Some studies showed that the drop in CO observed during recruitment maneuvers detects preload responsiveness [5]. Studies have also quantified preload responsiveness by measuring the slope of blood pressure changes induced by sighs performed at different pressure levels [11, 12]. However, recruitment maneuvers have limited indications and may be dangerous in case of ACP, which lowers the interest of these tests.

PEEP test

In ventilated patients with PEEP ≥ 10 cmH_2O, PEEP is lowered to 5 cmH_2O (Fig. 1). In a recent study, an increase in CO (pulse contour analysis) ≥ 9% reliably detected preload responsiveness [13]. Changes in PPV, but not those in pulse pressure, also allowed a reliable prediction, even if it was less good [13]. The results of the unique study require confirmation, particularly in different populations (patients without ARDS, other CO measurement techniques, etc.) A limitation to this test is the risk of lung de-recruitment.

How to select the right tool?

Supplementary Fig. 1 shows factors to consider for choosing the right tool. Even though PPV and SVV have multiple limitations, these indices received the highest level of evidence (along with the passive leg raising test). If no CO monitor is available, looking at PPV changes during a Vt challenge or a PEEP test (provided the latter is better validated) is adequate. The end-expiratory occlusion test is very reliable but requires that the patient has no strong respiratory activity. All these tests share the same advantage of being easy to perform in ventilated patients.

Conclusions

The effects of positive pressure ventilation on cardiac preload make it possible to easily detect preload responsiveness using different tests and indices. In the future, these tests could be automated by coupling the ventilator to hemodynamic monitoring devices. They enable us to personalize the therapeutic strategy according to the physiological characteristics of the patient [14], and participate in a monitoring-based strategy which may improve the patients' outcome [15].

Supplementary Information
The online version contains supplementary material available at https://doi.org/10.1007/s00134-024-07708-w.

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Funding
None.

Declarations

Conflicts of interest
Daniel DE BACKER received honoraria for consulting or giving lectures for Edwards Lifesciences, Philips, Viatris, and Pharmazz. Xavier MONNET received honoraria for consulting, institutional research grants, and honoraria for giving lectures from Pulsion Medical Systems (Getinge). He received honoraria for giving lectures from Baxter Healthcare and from AOP Health. Michael PINSKY received honoraria for giving lectures from Baxter Medical and Edwards LifeSciences.

Publisher's Note

Received: 15 April 2024 Accepted: 22 October 2024
Published online: 16 December 2024

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