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INVITED REVIEW

Cardiopulmonary interactions and volume status assessment

Alain F. Broccard

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Abstract Assessment of the hemodynamics and volume status is an important daily task for physicians caring for critically ill patients. There is growing consensus in the critical care community that the "traditional" methods—e.g., central venous pressure or pulmonary artery occlusion pressure—used to assess volume status and fluid responsiveness are not well supported by evidence and can be misleading. Our purpose is to provide here an overview of the knowledge needed by ICU physicians to take advantage of mechanical cardiopulmonary interactions to assess volume responsiveness. Although not perfect, such dynamic assessment of fluid responsiveness can be helpful particularly in the passively ventilated patients. We discuss the impact of phasic changes in lung volume and intrathoracic pressure on the pulmonary and systemic circulation and on the heart function. We review how respirophasic changes on the venous side (great veins geometry) and arterial side (e.g., stroke volume/systolic blood pressure and surrogate signals) can be used to detect fluid responsiveness or hemodynamic alterations commonly encountered in the ICU. We review the physiological limitations of this approach.

Keywords Cardiopulmonary interactions · Volume status · Fluid responsiveness · Vena cava ultrasound · Pulse pressure variation · Stroke volume variation · Physiology

A. F. Broccard (✉)
Division of Pulmonary, Allergy, Critical Care and Sleep Medicine, University of Minnesota, Minneapolis, MN, USA
e-mail: brocc001@umn.edu

A. F. Broccard
Critical Care and Respiratory Services, Fairview SouthDale Hospital, Edina, MN 55435, USA

1 Introduction

Understanding cardio-respiratory interactions is extremely important to the practicing intensivist as reviewed elsewhere [1, 2]. The heart and the lungs are anatomically and physiologically coupled. The respiratory and cardiovascular systems are linked functionally in the respiratory chain (oxygen and carbon dioxide exchange). The heart and lungs are in close proximity within the thorax and the lungs serve as conduit between the right and left heart chambers. Together those characteristics account for most of the mechanical interactions between the cardiovascular and respiratory systems. We shall focus here on the key physiological concepts needed, we believe, to recognise, correctly interpret and monitor the hemodynamic changes induced by cardiopulmonary interactions in the critically ill.

2 Venous return, cardiac function and blood volume changes

According to Guyton's representation of the circulation (Chapter 10 of [3]), the flow of venous return is determined by the equation: $Q_{VR} = (MSFP - RAP)/Rv$ where MSFP is the mean systemic filling pressure, RAP corresponds the right atrial pressure and Rv the resistance to venous flow return (Figs. 1, 2). Q_{VR} is thus driven by the pressure gradient between MSFP and RAP and opposed by Rv. The large capacitance veins of the systemic circulation contain approximately 70 percent of the total blood volume. This highly distensible compartment can accommodate a large blood volume while maintaining a quasi constant transmural pressure. The corresponding intravascular volume represents the so-called *unstressed volume*. Magder [4, 5] has proposed to represent the venous capacitance as a

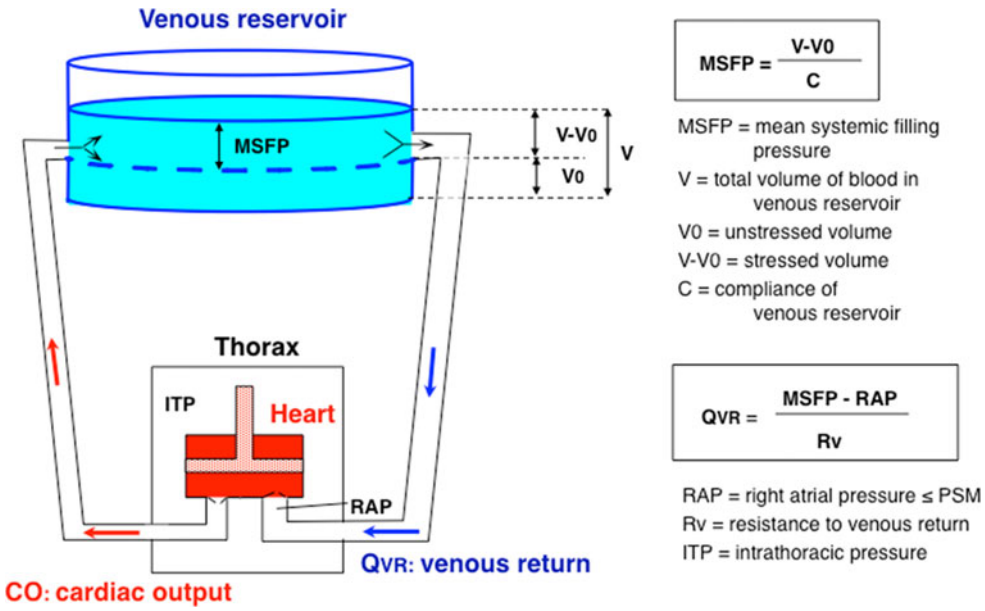

Fig. 1 Simplified representation of the circulation. The relationships between mean systemic filling pressure (MSFP), right atrial pressure, venous resistance, venous return, intrathoracic pressure (ITP) and cardiac output is schematically represented. See text for detailed explanation. Notice that serial and parallel ventricular interdependence and the impact of ITP on the pulmonary circulation and right ventricle are omitted in this representation

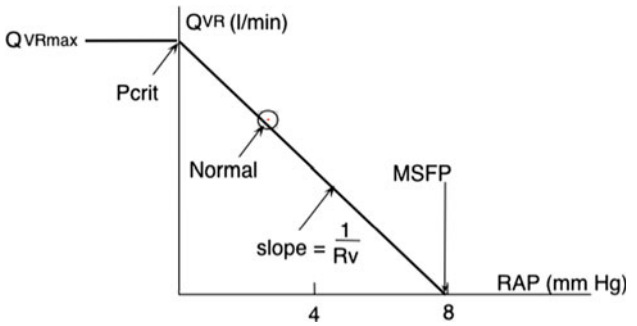

Fig. 2 Venous return curve according to Guyton. Q_{VR} = venous return, Q_{VR} max = maximal venous return. Notice that **a** when RAP decreases below a critical pressure (Pcrit), the transmural pressure of the great veins at the thoracic inlet becomes negative, leading to their collapse which prevents any further increase in Q_{VR} (flow limitation). **b** When VR curve intercepts the *X* axis, $Q_{VR} = 0$ and the right atrial pressure (RAP) equals the mean systemic filling pressure (MSFP). An increase in MSFP (e.g., hypervolemia) shifts the venous return up and to the right (see Fig. 3), while a change in venous resistance (e.g. increased VR) affects the slope of the venous return curve (reduced slope and lower Pcrit) while *X* axis intercept is unchanged as long as MSFP remains constant

reservoir drained through an opening in the side rather than the bottom (Fig. 1). The blood volume below the opening represents the *unstressed volume* that does not directly contribute to venous return. In contrast, the blood volume above the opening directly represents the *stressed volume* that contributes directly to MSFP and thus to Q_{VR} and ultimately cardiac output (CO). Venoconstriction due to adrenergic stimulation or an intravenous (i.v.) fluid administration can cause a "rightward" shift of the venous return curve (Fig. 3, left lower panel, hypervolemia curve). Conversely, absolute or relative hypovolemia (i.e. reduced venous tone and increased venous compliance reduce the effective blood volume as blood is transferred from the stressed to the unstressed volume) shifts the venous return curve "leftwards" (Fig. 3, right lower panel, hypovolemia curve).

In Guyton's representation, the cardiac function and the venous curve are both plotted against the RAP and can thus be superimposed on the same plot (Fig. 3). Over any time frame longer than a few heartbeats, CO must equal Q_{VR}, i.e. the heart can only pump what comes back from the periphery and CO is represented by the intersection of the cardiac function and venous return curves. This intersection represents the operating point of the circulatory system which may vary with any condition that has the potential to alter either or both the venous return or cardiac function curves (Fig. 3). For example, i.v. fluid administration by increasing MSFP shifts the venous return curve "rightwards" and towards the hypervolemia operating point (Fig. 3, right lower panel) and providing that the cardiac curve is normal, cardiac output increases (*fluid responsiveness*). Note that the position of the cardiac function curve integrates all aspects of cardiac performance, including the diastolic function, contractility, and afterload of *both ventricles*, as well as heart rate. An increase in afterload and/or decrease in contractility shifts the operating point downward on the venous return curve (Fig. 3 left lower panel) and cardiac output decreases. Under such conditions, a fluid challenge despite shifting the venous curve to the right will have negligible impact on CO because the operative point is on the flat part of the cardiac function curve (*fluid unresponsiveness*).

A few caveats are in order. In the Guyton's representation, RAP is an intramural pressure but is reported

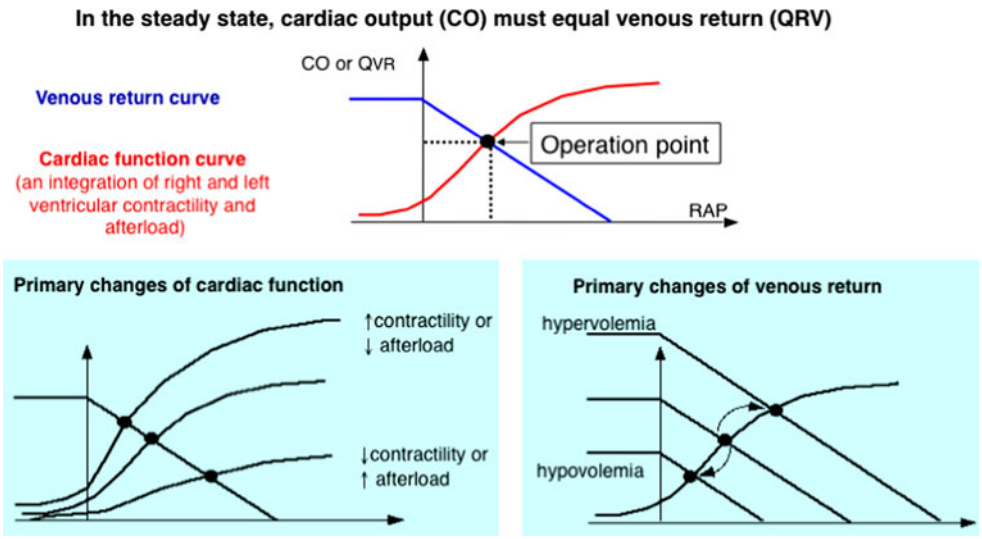

Fig. 3 Interactions of venous return and cardiac function. The *upper panel* represents the Guyton's graphic analysis of cardiac output regulation and the relationship between cardiac output and venous return. The *right lower panel* illustrates how a primary change in contractility and the *left lower one* how a primary change in volume status (or MSFP) affects this relationship. Notice that with the exception of short periods of transition to a new steady state—that cannot last more that a few cardiac beats—the cardiac output must be equal to the venous return regardless of the volume or cardiac function conditions

relative to the atmospheric pressure, a *constant* extramural pressure. RAP in this model is thus equivalent to a transmural pressure, a much better index of ventricular filling than the central venous pressure (CVP), RAP or pulmonary artery occluded pressure (PAOP) which are intramural pressures typically measured in patients without concomitant measure of the *variable* extramural pleuro-pericardial pressure. As a consequence, the same intramural CVP, RAP or PAOP may reflect during spontaneous breathing very different transmural pressures than when measured during positive pressure ventilation. In other words, the CVP, RAP or PAOP may vary due to change in extramural pressure and irrespective of the heart filling conditions, which explains—at least partially—the poor correlation reported between the CVP or PAOP and filling conditions [6, 7]. The Guyton's model assumes that the heart functions as a single unit. In reality, the heart is made of 2 pumps in series sharing myocardial fibers and a deformable separation (septum) all confined in a relatively stiff container (the pericardium). Significant alteration in any element of this complex system has the potential to alter the cardiac curves due to *serial* [e.g., a drop in RV stroke volume (SV) will result a few heart beats later in a LV SV change in similar direction] and/or *parallel ventricular interdependence* (e.g., an increase in RV end-diastolic volume may cause an interventricular septum shift towards the left, which impairs LV filling and SV once the pericardial space constrain prevents any further increase in overall heart volume). Finally, clinicians should keep in mind that a given volume challenge may have no detectable or minimal effects on cardiac output not only because either or both ventricles are unable to increase their SV (true fluid unresponsiveness due to fact that the operative point is on the flat part of an abnormal cardiac function curve) but also because the effects of a given intravenous bolus on venous return depends on other factors than just the heart: e.g., the fluid challenge per se (type of fluid given, volume and rate of administration) and what proportion of the fluid bolus given directly contributes to the stressed volume and to rise the MSFP as oppose to the unstressed volume. In other words, some "apparent" non-responder may just need more and/or faster fluid administration to demonstrate a response while others are truly fluid non responsive (flat part of the cardiac curve). This concept has obvious bedside implication but is also important to understand the limitations of all studies aiming at determining the presence of fluid responsiveness based on the response to a single sized, arbitrary and variably *effective* volume challenge.

3 Mechanical cardiopulmonary interactions

We shall now discussed how respiration and mechanical ventilation have the potential to significantly and mechanically alter the operative point of the cardiovascular system and produce detectable changes that may be taken advantage of to predict fluid responsiveness. We will limit the discussion to the key concepts needed to understand the limits and possible uses of such an approach. From a monitoring and hemodynamic standpoints, the key changes of interest are dynamic and transient as opposed to sustained (e.g., effects

of cyclic change in lung volume during ventilation or transient change in airway pressure as a consequence of a manoeuvre) and primarily mechanically mediated as opposed to caused by neuro-endocrine changes.

3.1 Lung inflation and hemodynamics

Ventilation is defined by cyclic changes in lung volume, the latter increasing during inspiration and decreasing during expiration. Inspiration can be generated by phasic drop in pleural (Ppl) and alveolar pressure (Palv) due to the contraction of inspiratory muscles (spontaneous breathing) or alternatively by phasic increase in Palv and Ppl (passively ventilated patient with positive pressure ventilation) or combination of both in actively breathing mechanically ventilated patients. Expiration is normally passive and associated with a rise in Ppl following a spontaneous breath and a reduction in Ppl following a mechanical one.

Let us first consider the impact of cyclic change in lung volume as this is common to all modes of ventilation. As the lungs inflate, they tend to "hug" and "compress" the heart [8, 9]. This anterior and retro-sternal expansion of the lungs during inspiration generates a local surface pressure on the epicardium, which appears to be at least partially independent of global intrathoracic pressure, since it is not removed by opening the chest [10]. This direct contact effect appears to be particularly important in patients with severe dynamic hyperinflation (or during a recruitment manoeuvre) and could explain the increase in RAP and PAOP, the small heart size observed on chest films [11] and the pulsus paradoxus seen in acute severe asthma [12, 13] and other causes of airway obstruction. The mechanism of pulsus paradoxus in acute severe asthma differs somewhat from that in tamponade. Although both have in common an exaggerated parallel diastolic interdependence due to external compression of the heart, RV SV appears to fall, rather than increase during inspiration in the setting of severe hyperinflation [12]. Lung inflation increases pulmonary vascular resistance (PVR) [14]. Given that the thin-walled right ventricle has limited capacity to generate the high systolic pressure needed to maintain cardiac output across the elevated vascular resistance in the setting of severe hyperinflation, this leads RV SV to drop during inflation followed a few heart beats later by a decrease in LV preload, stroke volume and SBP. In other words, an exaggerated serial ventricular interdependence contributes to pulsus paradoxus in the setting of hyperinflation.

Lung inflation also affects the venous return to the RV and the LV by mechanisms distinct from those related to the opposite change in RAP observed during spontaneous or positive pressure ventilation. In regard to the systemic circulation, the diaphragmatic descent during inspiration elevates abdominal pressure (Pabd) and compresses the splanchnic venous reservoir [15]. This transfer of capacitance blood from the unstressed to the stressed volume [16], increases MSFP. During a spontaneous inspiration the rise in MSFP together with the drop in RAP enhance the MSFP–RAP gradient and thus venous return. The diaphragmatic descent associated with positive airway pressure (e.g., PEEP) causes for similar reasons a rise in MSFP which tends to offset the concomitantly RAP elevation, so that the MSFP–RAP gradient may remain constant [16, 17]. The rise in RAP, thus, does not appear to explain alone the reduction in cardiac output in the setting of positive pressure ventilation. Theoretically, the extent to which the rise in MSFP may offset the increase in RAP may vary depending with the volume status (MSFP may rise less in hypovolemic conditions). Other possible mechanisms for the drop in Q_{VR} include increased resistance to venous return and decrease the maximal value of venous return (increased Pcrit)[16, 18] due to venous geometry distortion at the entrance of the venae cavae into the thorax [19] or further upstream in the portal circulation (i.e. compression of the liver by diaphragmatic descent) [20]. Geometric alteration of the inferior vena cava (ICV) at its point of entry the thorax is not restricted to positive pressure ventilation. Interestingly, the extent of ICV distortion during a spontaneous inspiration is a function of the extent of diaphragmatic descent which vary with inspiratory effort [21] as well as of the breathing pattern (diaphragmatic versus rib cage: femoral venous flow appears to cease completely during pure diaphragmatic breathing while increasing during intercostal breathing [22]). In the actively breathing patient, effort and breathing pattern dependant IVC size variation can thus take place, which renders volume responsiveness assessment based on IVC size problematic.

A final consideration is the impact of lung inflation on pulmonary venous return and LV filling. Experiments in isolated lungs [23, 24] have indicated that, whether actuated by positive airway or negative pleural pressure, an increase in lung volume can "squeeze" blood out of the pulmonary vascular bed, provided that intra-alveolar vessels are filled at end-expiration (West zone 3 conditions, see [24] for a detailed discussion of this issue) and thus increase transiently LF filling and SV.

3.2 Intrathoracic pressure and hemodynamics

Although it is not always possible to tease out completely the effects of lung volume from that of intrathoracic pressure (ITP), we have nevertheless attempted to do so to help understand—in regard to the assessment of volume responsiveness based on cardiopulmonary interactions—how the passive ventilated patient differs from a spontaneously breathing one or an actively breathing ventilated

one. Irrespective of the mode of inflation, the pure effects of a rising lung volume on the hemodynamics tend to occur in the same direction. The key differences between passive mechanical inspiration and spontaneous active one are as follows: A. during spontaneous breathing, one observes greater breath to breath variability of tidal volume and intrathoracic pressures than during passive mechanical breathing, which complicated the analysis of respirophasic change. B. Pressure changes in the alveolar, pleural and juxta-pericardial space occur in opposite direction.

As a result, the ITP changes associated with spontaneous or passive mechanical ventilation tend to change the end-diastolic volumes and compliance of both ventricles cyclically but in opposite directions [25]. Spontaneous inspiration augments venous return, RV filling, which in turn makes the LV stiffer, thus impeding its filling and SV (parallel ventricular interference). As a result, the systolic arterial pressure (SAP) falls slightly (by less than 10 mmHg) in inspiration in a healthy individual. Lung inflation with positive airway pressure tends to act in an inverse fashion in regard to RV [26, 27] and LV filling [23, 24].

The intraluminal pressure in the aortic root decreases less than does ITP due to the connection of this vessel with extrathoracic arteries during a spontaneous breath thus the aortic transmural pressure and LV afterload increase [28–30] while the opposite pressure changes during positive pressure ventilation reduce LV afterload. RV afterload increases mainly due to the alveolar vessels deformation as lung volume rises above FRC during a spontaneous or positive breath but possibly more so due to direct compression of alveolar vessels [31]. RV after load does not necessarily rise under all circumstances during positive pressure ventilation. In the setting of diseased lungs, mean airway pressure and PEEP may have a favourable effect on lung volume, gas exchange, pulmonary vascular resistance and therefore RV afterload if the net effect is lung recruitment. In contrast, acute right ventricular failure in the setting of positive pressure ventilation may be a sign that the lungs are at least regionally overdistended and alveolar pressure thus needs to be reduced (e.g., decrease VT or PEEP, as appropriate). It is also important to keep in mind that not all breaths are equal. The contribution of a positive pressure breath to the juxta-pericardial pressure and resulting degree of heart compression vary in parallel with the size of the tidal volume delivered, the stiffness of the chest wall and theoretically may also depend on the time constant of the lung segments in direct contact with the pericardium [10].

Finally, the timing of respiratory fluctuations in SBP during a passive positive pressure breath in a normal subject differs from that seen during spontaneous breathing. During the former mode of ventilation, LV stroke volume becomes maximal (maximal pulse pressure) near the end of inspiration (Fig. 4) due to combined effect of enhanced LV filling and reduced afterload followed during expiration few heart beats later by a fall in SBP and pulse pressure as the propagation of reduced RV stroke volume during inspiration reaches LV output during expiration (*serial interdependence)*[32].

4 Monitoring and assessing fluid responsiveness based on cardiopulmonary interaction

As mentioned above and with few exceptions (e.g., the lack of an inspiratory drop in RAP suggests an overfilled, noncompliant heart lying on the flat part of its function curve unlikely to respond to i.v. fluid [33]), neither the CVP/RAP [6] nor the pulmonary artery occlusion pressure (PAOP) [34–36] [7] are helpful to predict fluid responsiveness. This has lead to propose alternative methods to establish fluid responsiveness including some based on the cardiopulmonary interaction and the Guyton representation of the circulation discussed above.

Our goal is not to review exhaustively here the different devices, specific methods, or studies published on the topic but to focus on the common physiological background, limitations and clinical implications of the methods that attempt to use cardiopulmonary interactions to assess and monitor fluid responsiveness. Based on Guyton's model, this is best defined as the ability to increase the CO in response to a rise in MSFP, which requires the operative point to be on ascending part of the cardiac function curve. Notice that this condition is fulfilled in hypovolemic as well as in normovolemic conditions for a normal cardiac curve (Fig. 3 left lower panel). The clinical implication is that the presence of fluid responsiveness does not mean that the patient is hypovolemic or needs i.v. fluid. It just facilitate the clinical decision making.

The methods to assess fluid responsiveness based on cardiopulmonary interactions can be divided in 2 broad categories depending on whether the focus is on the venous (venous return based) or arterial side (stroke volume based) of the circulation.

4.1 Arterial side or stroke volume based methods

The "arterial" methods aim overall at identifying the phasic changes in stroke volume associated with respiration using a variety of signals (e.g., pulse oximetry, SBP variation, pulse pressure (PP) variation, analyzed pulse contour, aortic flow by esophageal doppler, or echocardiographic measure of the velocity time integral in the LV outflow track).

In the *spontaneously breathing* patients, there is very limited data to our knowledge to validate the use of

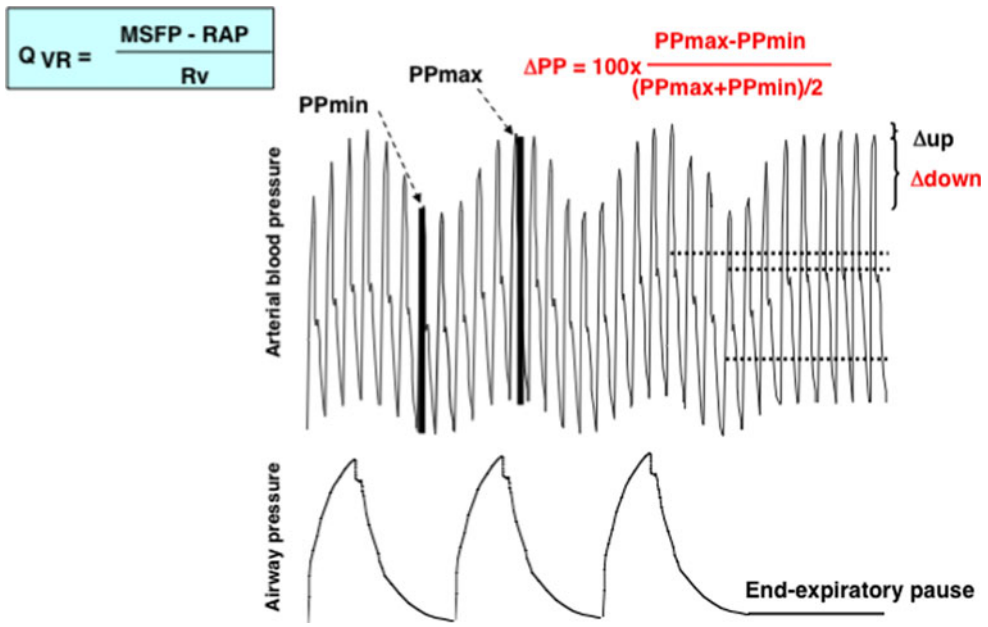

Fig. 4 Respiratory variations of arterial blood pressure in a sedated patient on volume controlled mechanical ventilation. In such conditions, the respiratory fluctuations of either systolic (Δup and Δdown) or pulse pressure (PPmax and PPmin) have been proposed as a method to detect volume responsiveness. The first method requires an end-expiratory pause of sufficient duration for systolic blood pressure to stabilize, to obtain a reference level from which to measure Δup and Δdown as indicated. PPmax and PPmin can be obtained without interrupting ventilation and ΔPP appears to a more robust index of fluid responsiveness [36]. It is important to know the limitation of this measure and to be aware that other conditions than fluid responsiveness can cause pulse pressure variation during ventilation. See text for details

respiratory induced variation in stroke volume or its surrogates as an accurate way to assess fluid responsiveness. Alternatively, one can require the patient to perform a deep inspiration [37] or Valsalva manoeuvre [38]. This is not to say that clinicians should ignore the presence of exaggerated cardiopulmonary interaction in this population. The presence of a Kussmal's sign, pulsus paradoxus or marked PPV always warrant further exploration as those findings provide important clues that patients' condition is changing and/or a catastrophic cardiovascular event is in the making. Overall, alternative method based on passive leg raising are better validated in this population [39].

In the *mechanically ventilated patients*, the most promising data come from heavily sedated patients ventilated in controlled mode with relatively large tidal volumes (≥8 ml/kg) [35, 36, 40] as recently reviewed [41]. However, even for pulse pressure variation—probably the best studied, validated and available bedside measure of stroke volume variation—this approach was reported to be inconclusive in approximately 25 percent despite even when used in an "ideal" population of anesthetized patients [42].

A practical problem with all the methods based on detection of respirophasic change in SV or its surrogates, is the potential confounding influence of the ventilatory conditions and changes in chest wall and/or lung compliance [43]. These methods do not appear to remain as helpful in the presence of active inspiratory or expiratory effort, [44–46], with small tidal volumes [47–49], increased abdominal pressure [50], altered chest wall compliance [44], changes in vascular tone or arrhythmias. Possible solutions to address some of the above shortcomings have been proposed by applying, for instance, a three successive incremental pressure-controlled breath and quantifying the effect on systolic blood pressure [51], by interrupting positive pressure ventilation with a transient end expiratory pause [44] or by using new algorithm for stroke volume variation [52]. Nevertheless, there are still many caveats to keep in mind, which limits the utility of respirophasic change in SV or surrogates to assess fluid responsiveness.

It is important to remember that the phasic signal detected from a cardiopulmonary interaction is a function of the intensity of the tidal volume and ITP change, of the method used to track a respirophasic SV change [53] and of the degree of hypovolemia (relative or absolute). In addition, one cannot assume that exaggerated cardio-pulmonary interaction is synonymous with fluid responsiveness and/or hypovelemia. Any condition predisposing to respirophasic change in ventricular diastolic function, afterload or

contractility has the potential to shift the cardiac curve, alter the operative point and thus SV irrespective of the volume status (Fig. 3, left lower panels: e.g., a positive pressure breath tends in the presence of severe LV dysfunction to shift the cardiac curve up due to reduced LV afterload and down in the presence of cor pulmonale due increased RV afterload).

In summary, although the finding of a marked respirophasic change in SV or its surrogate may indicate fluid responsiveness in the passively ventilated patients, no definitive conclusion should be drawn, we believe, before, considering other possible explanations. This is best done by integrating the findings with the overall clinical picture and by performing a focused echocardiogram whenever fluid administration appears risky or a clear-cut cause cannot be established. Unlike clinical trial patients, real world ones are not carefully selected, commonly present with multiple problems affecting their hemodynamics and they are more complex.

4.2 Venous side approach

The approaches proposed to replace CVP on the venous side to predict fluid responsiveness are based on the assessment of respiratory fluctuations of great veins geometry. The relationship between the volume and transmural pressure of vena cavae is nonlinear and similarly to the more familiar pressure volume curve of the lung, has a steep slope at low distension and a plateau at full repletion [54]. Therefore, the transmural pressure changes associated with respiration are more readily translated into vascular diameter alterations when imposed on a partially empty vessel (hypovolemia), as opposed to a fully repleted one (normo or hypervolemia). Ultrasound based measurement of caval diameters change with respiration (e.g., collapsibility index) have been proposed as non-invasive way to assess intravascular volume status [54–59]. The IVC is typically imaged close to its crossing of the diaphragm. At this point, its extramural and intramural pressure are close to the abdominal (Pabd) and RAP, respectively. During a spontaneous breath, Pabd increases (diaphragmatic descent) while RAP decreases. This tends to reduce the IVC diameter providing the vessel operates on the steep rather than the plateau part of its transmural pressure/diameter relationship. Quantified in various ways with echocardiography, the IVC diameter has been used to characterize volume status in the course of hemodialysis for end-stage renal disease [55, 60]. Prediction of fluid responsiveness in spontaneously breathing ICU subjects using this approach has not been validated to our knowledge. Its limitations include the fact that the degree of intramural and extramural variation during a spontaneous breath is effort dependent and may vary with conditions affecting the operative point irrespective of the volume status. For instance, the breathing pattern and the degree of diaphragmatic descent/contraction may affect the IVC diameter [21]. Along this line, this approach appears to also be problematic in ventilated patient who actively triggered the ventilator [61]. Despite those limitations, many clinicians find helpful to observe an IVC that fully collapses during spontaneous inspiration as opposed to a large, fixed one. This is particularly true in a patient with hypotension or shock as either finding help narrow the differential and first line treatment choice to restore an adequate blood pressure pending a more complete evaluation.

During mechanical ventilation, airway pressure transmits more to the RAP and IVC intramural pressure than to the Pabd or IVC extramural pressure. Therefore, the IVC transmural pressure increases and the IVC dilates if not already completely filled. Although the direction of IVC diameter changes goes in the opposite direction in spontaneous compared to positive pressure breath, the degree of respirophasic change follows the same principle discussed above (position on the transmural pressure/volume curve). The amplitude of phasic changes in IVC geometry, in sedated septic shock patients ventilated in controlled modes was found to be highly predictive of cardiac output response to a fluid challenge [57, 58]. Although not fully evaluated in the ventilated patients, respirophasic IVC diameter changes are likely to depend not only on the intravascular volume, but also on the respiratory pattern, tidal volume, respiratory system mechanics and prevailing level of mean Pabd and right ventricular function, which might explain recent disappointing results [61].

The superior vena cava (SVC) runs mainly intrathoracic so that its extramural pressure is close to pleural pressure and not directly influenced by Pabd unlike the IVC. Positive pressure inflation may transiently create zone 2 conditions (intraluminal pressure $<$ Ppl) in the SVC, which may thus partial collapse [56]. In septic passively ventilated patients, respirophasic SVC diameter changes appear to correlate well with CO responsiveness to iv fluid [59]. The SVC index of hypovolemia has been advocated as superior to that based on IVC diameter [60]. In contrast to the IVC which can be easily seen through a subcostal view, the SVC requires imaging via the transesophageal route.

5 Conclusion

Respirophasic change in stroke volume (and other surrogagte such as PPV) or in the geometry of large vessels may have a role and appears superior to measure of the CVP in assessing fluid responsiness but its utility appears mainly limited to the passive ventilated patient population. Like any other test, fluid responsiveness prediction needs to be

considered in the patients' clinical context. For the problem at hand, the following questions need to be answered: does the patient require an hemodynamic intervention to increase his/her CO? If so, what is the pretest probability that the inadequate CO is mainly the consequence of absolute or relative hypovolemia and not primarily due to another cardiovascular derangement? This is essential to determine if the next best step is to proceed with a fluid challenge, another test or therapeutic intervention. We believe that point of care echocardiography (particularly when coupled with lung ultrasound to assess for the presence of lung edema [62]) constitutes a very promising avenue to answer many of these questions. It allows to assess the venous and cardiac aspects of the Guyton's mode model of the circulation. When used in conjunction with monitoring of respirophasic SVV or PPV, ultrasonograghy is helpful for the characterization of the hemodynamic changes detected by the continuous tracking of those parameters.

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Conflict of Interest The author declares that he has no conflict of interest.

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