Using mechanical ventilation to monitor the circulation

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Abstract. In patients who are on fully controlled mechanical ventilation with tidal volumes of at least 8 ml/kg, the intermittent increase in intrathoracic pressure serves as a repetitive challenge of the circulation, resulting in measurable functional haemodynamic parameters. The degree by which surrogate parameters of the left ventricular stroke volume are influenced by the inspiratory decrease in venous return offers unique information about the fluid responsiveness of the ventilated patient. These parameters are superior to commonly-used static preload parameters in their ability to detect occult hypovolaemia, reflect the response to changes in the effective blood volume, and prevent potentially damaging fluid overload. Within their respective limitations, functional haemodynamic parameters, offer immediate, dynamic, and essential information about cardiovascular function. Following the recognition of its value, functional haemodynamic monitoring is being gradually implemented in new bedside monitors.

Introduction

Although fluid loading is one of the most common therapeutic steps taken in the ICU, it fails to increase the cardiac output (CO) in about 50% of the patients [1]. This sobering reality has been repeatedly shown both during elective surgery and in critically ill patients with circulatory failure (Table 1). The fact that in many of these studies fluid loading was judged by the authors to be ‘clinically indicated’ raises the question about our ability to predict fluid responsiveness. The importance of this problem cannot be overestimated since unnecessary fluid administration may be harmful especially in patients with respiratory, renal and/or cardiac failure. Overzealous fluid administration may indeed be an underestimated occult source of mortality in the ICU, since the excess fluid may increase interstitial oedema in various organs, increase lung water content, postpone weaning and increase the risk of sepsis. Part of this excess fluid administration may also stem from the fact that the end-point of fluid resuscitation is frequently unclear. Hence the importance of an accurate assessment of fluid responsiveness lies not only in the detection of latent hypovolaemia or a meticulous ‘prophylactic optimization’, but also in the withholding of fluids when their administration may not be of benefit.

A major reason for an imperfect fluid management in critically ill patients is the common reliance on the CVP and the PAOP, though these parameters have been clearly shown to be poor predictors of the response of the cardiac output (CO) to fluid loading and to be unable to differentiate between patients that respond to volume loading (responders) and patients that do not (non-responders) [1]. However, even the ‘volumes’ of the cardiac chambers themselves are often mediocre predictors of fluid-responsiveness since their relationship to the stroke volume (SV) depends on the elusive ventricular contractility. This is why even more accurate measures of preload, like the global end-diastolic volume (GEDV) or the LV end-diastolic area, have a limited capability of predicting fluid responsiveness, although they are useful in patients with spontaneous ventilatory activity.

In mechanically ventilated patients, the haemodynamic effects of the increase in intrathoracic pressure offer dynamic information about fluid responsiveness. This direct clinical application of the physiological principles of heart-lung interaction during mechanical ventilation is gaining an ever-growing interest and has been the topic of many reviews and editorials, most of which are quite recent [2-6]. In this chapter we will describe the basic physiological principles of this monitoring approach, review the various parameters that have been developed and delineate the usefulness as well as the limitations of these parameters.

The haemodynamic effects of the mechanical breath

Normally, a positive-pressure breath will cause a decrease of about 20% in right ventricular (RV) filling due to a decreased venous return. In the presence of hypovolaemia the inspiratory decrease in RV outflow may be much higher and reach 70%. The decrease in venous return during the mechanical breath is due not only to the increase of the right atrial pressure, but also due to a “waterfall” effect caused by a significant closure of the cavea especially in hypovolaemic conditions, when the major veins are more compliant [7]. This decrease in venous return decreases, in turn, the right ventricular (RV) stroke output, which, in patients with ARDS, may also occur because of an increase in RV outflow impedance during the mechanical breath.

However, the first and immediate effect of the rise in intrathoracic pressure on the LV is normally an augmentation of the LV stroke volume (SV) [4,5,8,9]. This augmentation, which is more pronounced in the presence of congested (“zone 3”) lungs, is due mainly to the inspiratory squeezing of the pulmonary blood volume and an increase in pulmonary venous flow [9]. Other suggested mechanisms for the early inspiratory increase in LV SV include a decrease in the transmural aortic pressure reflecting an effective decrease in LV afterload, an earlier and longer opening of the aortic valve, external pressure exerted on the LV by the increased lung volume, better LV contractility due to the decreased size of the RV, and lung inflation-induced adrenergic discharge. The second phase of the response of the LV to the mechanical breath is normally a decrease in LV SV, which is the result of the earlier decrease in RV SV. Thus, the mechanical breath induces cyclic changes in the output of
the right and left ventricles, which normally include an early increase in LV SV with a simultaneous decrease in RV SV during inspiration, and an increase in RV SV with a decrease in LV SV during the expiratory phase.

**Basic principles of arterial pressure waveform analysis (SPV, dUp, dDown)**

The previously described respiratory fluctuations in the LV stroke output are reflected in the arterial pressure waveform. The early inspiratory augmentation of the LV stroke fluctuations in the LV stroke output is reflected as an increase in the systolic blood pressure (SBP) termed dUp (delta up, \( \Delta u \)), while the later decrease in LV stroke output is reflected in a decrease in the SBP termed dDown (delta down, \( \Delta d \)) (Figure 1). The dUp is measured as the difference between the maximal value of the SBP and the SBP during a long end-expiratory pause or a short (5 seconds) apnoea, while the dDown is measured as the difference between the end-expiratory SBP and the minimal SBP value. The sum of the dUp and the dDown, which is the difference between the maximal and the minimal SBP values during one mechanical breath, is termed the Systolic Pressure Variation (SPV) (5).

It is important to note again that the dUp and the dDown represent two different haemodynamic events. The dDown is due to the decrease in venous return during the mechanical breath, and its magnitude reflects ‘fluid responsiveness’, namely, the degree by which LV SV decreases in response to the transiently decreased preload. The dUp, on the other hand, reflects the early inspiratory augmentation of the LV SV, and has been originally described as ‘reversed pulsatil paradoxus’.

The SPV and dDown reflect volume status and predict fluid responsiveness.

The SPV and the dDown have been repeatedly shown to be sensitive indicators of changes in blood volume. The dDown gradually increases with each step of controlled haemorrhage, becomes the main component of the SPV during hypovolaemia, and returns to normal values following restitution of intravascular volume (8). The SPV and the dDown have been found to correlate with the intrathoracic blood volume (ITBV), the echocardiographic LV end-diastolic area and even with the PAOP. The SPV and dDown have also been shown to increase simultaneously with the decrease of CO following the application of PEEP (10). In critically ill patients the pulse pressure variation (PPV, see later) values prior to PEEP application were also shown to significantly correlate with the PEEP-induced changes in CO (11). Hence the presence of a significant dDown (or PPV) should prevent the application of PEEP without prior fluid loading or without the application of more advanced haemodynamic monitoring.

However, the main value of the SPV and dDown lies in their accuracy as predictors of fluid responsiveness (12). A number of clinical studies have shown that these parameters have much better correlation to the change in the CO following volume loading than the CVP, the PAOP and even the LV end-diastolic area (13-15). In septic patients, the presence of a dDown > 5 mmHg was found to be highly predictive of a positive response to fluid loading (13). Varying degrees of fluid expansion in humans have always shown the SPV to decrease significantly by anywhere from 2.5-10 mmHg, while experimental data have repeatedly shown that hypervolaemia and/or congestive heart failure were associated with a relatively small SPV value and a practically non-existent dDown segment (16). Rarely, large respiratory fluctuations in BP were observed in cor pulmonale patients who were not responsive to fluids (17).

**The inspiratory increase in arterial pressure - dUp (delta up, \( \Delta u \))**

In experimental studies, the dUp has been repeatedly shown to increase during hypervolaemia and/or congestive heart failure (16,18). In critically ill patients a significant dUp has been shown to be present quite frequently (9), and at times to be the main component of the SPV (13). The fact that the dUp does not normally reflect fluid responsiveness and yet can be significant, has some very important implications. The first one is that simple eyeballing of the arterial pressure fluctuations during mechanical ventilation without relating them to some reference pressure may be misleading (13). Secondly, a patient presenting with a prominent dUp should be considered as being either hypervolaemic or as having compromised LV function. The mechanical breath serves as a repetitive ‘assist device’ to the LV in such conditions. Several studies have explored the potential of using the mechanical breath for this purpose, but to date there is no clinically accepted method that takes advantage of this phenomenon. Weaning patients with a significant dUp from ventilatory support should be done with caution, and prior improvement of the cardiovascular function should be considered. The last but not least implication of the presence of a prominent dUp is that the interpretation of the SPV, SVV and PPV as parameters of fluid responsiveness should be done with caution, since these parameters include the dUp which is not directly related to fluid responsiveness.

**The Pulse Pressure Variation (PPV)**

The Pulse Pressure Variation (PPV) is the difference between the maximal and minimal pulse pressures (PP) during the mechanical breath cycle (Fig. 1) divided by the mean of these two values (11,19). The rationale of using the PPV rather than the SPV as a parameter of fluid responsiveness is that, for a given arterial compliance, the PP is directly related to the LV SV and is not influenced by any transmission of pleural pressure which may affect the systolic BP and hence the SPV. The PPV has indeed been shown to be an excellent predictor of fluid responsiveness during the application of PEEP (11) and in septic patients (20). A PPV value of 13% allowed discrimination between responders (increase in CI≥15%) and non-responders with a sensitivity of 94% and a specificity of 96%, which was somewhat better than that of the SPV, but much better than that of the PAOP and the CVP (19). Other studies have also shown that the PPV performs a little better than the SPV as a predictor of fluid responsiveness (14,20,21). In patients with acute circulatory failure, the changes in PPV and in aortic blood flow during passive leg raising (PLR) were shown to be sensitive indicators of fluid responsiveness (22,23). The PPV has probably become the most popular functional haemodynamic parameter and is continuously measured by a number of commercially available monitors.

**The Stroke Volume Variation (SVV)**

Measuring the respiratory variation of the SV itself, rather than that of surrogate parameters, has become possible with the renewed introduction of pulse contour analysis in a number of commercially available monitors which use different algorithms for this purpose. Basically, the stroke volume variation (SVV) is the difference between the maximal and minimal SV during one mechanical breath (Fig. 1) divided by the mean SV value. The SVV has been shown to be a sensitive
Limitations of functional haemodynamic parameters

The main limitation of functional haemodynamic parameters is that their use is limited to patients who are on fully controlled mechanical ventilation. In patients who are breathing spontaneously or on partial ventilatory support, quantification of the respiratory changes in functional haemodynamic parameters may be inaccurate and difficult to interpret, due to the variability of the inspiratory effort, partial airway obstruction and forced expiration. Although physiologically obvious, this limitation of functional haemodynamic parameters has been recently studied (37). The other main limitation of functional haemodynamic parameters is their dependency on the magnitude of the employed tidal volume, large tidal volumes producing an exaggerated variation and low tidal volumes producing an inadequate response. A tidal volume of at least 8 ml/kg has been found to be necessary to produce a PPV value that can adequately predict fluid responsiveness (38). Exaggerated respiratory variations can also be seen in the presence of air-trapping or reduced chest wall compliance. Decreased lung compliance by itself should not affect the usefulness of the SPV and its derivatives if the tidal volume is unchanged, since the effects of increased airway pressure and its reduced transmission may cancel each other out (39). In fact some of the major clinical studies on functional haemodynamic parameters have been done in patients who were in respirator failure.

Other functional haemodynamic parameters

The respiratory variations of other parameters have also been shown to reflect fluid responsiveness. Due to limitations of space these parameters will be mentioned only briefly.

1. The plethysmographic waveform of the pulse oximeter (31).
2. Superior vena cava collapsibility (echo) (32).
3. Inferior vena cava distensibility (echo) (33).
4. Aortic blood flow velocity (oesophageal Doppler) (34) and velocity-time integral (35) (echo-Doppler).
5. The pre-ejection period (the time interval between the beginning of the R wave on the electrocardiogram and the upstroke of the radial arterial pressure curve) (36).

The respiratory systolic variation test (RSVT)

The change in the arterial pressure following a mechanical breath is dependent not only on the status of fluid responsiveness but also on the magnitude of the tidal volume itself, since larger increases in intrathoracic pressure will reduce venous return to a greater extent. In view of this fact and of other limitations of current functional haemodynamic parameters (see below), we have developed the Respiratory Systolic Variation Test (RSVT) which is based on the production of a ventilatory manoeuvre that consists of 3 consecutive incremental pressure-controlled (10, 20 and 30 cmH₂O) breaths (14,30). Plotting the respective three lowest systolic pressure values (for each breath respectively) against their respective airway pressure, produces a slope that reflects fluid responsiveness. In patients who underwent vascular (30) and cardiac (14) surgery, the RSVT slope decreased significantly following fluid loading, and correlated well with the changes in SV following volume loading. The RSVT slope compared favourably with other functional haemodynamic parameters, which as a group performed much better than traditional ‘preload’ indices (14). The RSVT slope may also provide a numeric approximation of the slope of the LV function curve, which is often mentioned but only rarely measured. At this stage this parameter, which is produced by a true automated linkage of the ventilator and the monitor, is unavailable commercially.
Since functional haemodynamic parameters rely on individually measured beats, any arrhythmias may cause significant inaccuracies. Nodal rhythm, however, may increase the SPV by effectively decreasing preload due to the loss of the ‘atrial kick’. Whenever the PPV or SVV are provided by commercially available devices, it is important to note that the accuracy of their values may depend on the algorithms that are being used, most of which have not yet undergone thorough validation. As mentioned before, the PPV, SVV and PPV include the dUp, a component that is unrelated to fluid responsiveness and that may reduce their ability to accurately reflect fluid responsiveness. This may occur especially when they are in mid-range, since then it is unclear whether they are due to an isolated dUp, an isolated dDown or a combination of both. The dDown has therefore a theoretical advantage in that it directly reflects fluid responsiveness. However, its measurement is complicated by the necessity to introduce a long end-expiratory pause or a short apnoea, following which the effects of the succeeding (or preceding) mechanical breath on the arterial pressure wave form should be carefully analyzed. The apnoea should preferably be achieved without disconnecting the patient from the ventilator, so as not to lose the prevailing PEEP or auto-PEEP.

Last but not least, the presence of large dDown or its equivalent is not an indication, per se, to administer fluids. When all confounding factors have been excluded, a large variation in the arterial pressure following the mechanical breath means only that the patient will probably respond to fluid administration by increasing his cardiac output. The decision whether or not to administer fluids should be individually determined according to the patient status. In addition, at present we have no evidence that fluid loading following the identification of significant fluid responsiveness by a functional haemodynamic parameter affects outcome in any way.

Conclusions

We are often confronted with a variety of static parameters that do not provide a conclusive haemodynamic diagnosis. Challenging the system with a standardized stimulus may provide new insights about the function of the whole system. The normal effects of this stimulus have to be well known, so that interpretation of the response to this stimulus is clear and preferably immediate. Confounding factors may decrease the usability of this approach.

These are general guidelines for the use of any diagnostic or therapeutic functional test, and directly apply to the use of the increase in intrathoracic pressure as a repetitive challenge of the circulation. The resulting functional haemodynamic parameters can be of great value in the monitoring of ventilated patients, in which haemodynamic uncertainty and potential instability are often present. Besides supplying immediate estimation of fluid responsiveness, these parameters are extremely sensitive to changes in preload, and therefore are useful in following the response to fluid loading. Since the normal healthy heart is fluid responsive, the presence of fluid responsiveness is not an indication by itself to administer fluids. By being able to detect occult hypovolaemia, identify the presence of fluid responsiveness or its absence in low-flow states, and reflect the response to changes in effective blood volume, these parameters offer immediate, dynamic, and essential information about cardiovascular function.

References


