Commentary

What is a pressure–volume curve?

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Abstract

The pressure–volume (PV) curve is a physiological tool proposed for diagnostic or monitoring purposes during mechanical ventilation of acute respiratory distress syndrome. The reduction in compliance measured by the PV curve and the different inflection points on the curve are considered interesting markers of the severity of and the levels of opening and closing pressures. Tracing a curve, however, may in itself influence the degree of opening or distension of the lung, and interpretation of the curve has to take this effect into account. In some individuals tracing the curve may even have moderate hemodynamic effects. Fortunately, on average, most of these effects are transient or negligible and do not invalidate the PV curve measurement.

The pressure–volume (PV) curve is a diagnostic or monitoring technique proposed soon after the initial description of acute respiratory distress syndrome (ARDS) [1]. The PV curve was really identified as a potentially important tool, however, by Matamis and colleagues [2], who described the relationship between alterations in respiratory mechanics and the stage of acute lung injury. The PV curve is usually traced from the elastic equilibrium lung volume that corresponds either to the functional residual capacity or to the end-expiratory lung volume. The end-expiratory lung volume can be greater than the residual capacity in the case of air trapping or of ventilation with positive end-expiratory pressure (PEEP).

The classic shape of the PV curve in ARDS patients is more or less sigmoidal, with a general slope (i.e. compliance of the respiratory system) that is markedly reduced compared with normal subjects. The curve is obtained by slowly insufflating the chest, either continuously or in a series of small steps [2,3]. The PV curve is generally viewed as consisting of three segments separated by two inflection points; or, for other authors, it can also be described as a true sigmoid [4]. The first segment, characterized by low compliance, is separated from a more linear part of the curve by the lower inflection point. The intermediate segment can be considered linear and is used to measure the 'linear' compliance between the lower inflection point and the upper inflection point. Beyond the upper inflection point, the PV curve tends to flatten again.

The reduction in linear compliance measured by the PV curve is considered a hallmark of ARDS and is usually explained chiefly by the loss of aerated lung volume. Lung areas with a normal appearance on plain radiograph scans, however, show increases in lung tissue despite preserved aeration on computed tomography images, indicating that lung tissue alterations are diffuse in ARDS [5].

A weak but significant correlation between compliance and markers for collagen turnover was recently described [6], with a logarithmic pattern consistent with a model of collagen-dependent maximal distension. This model reflects the mechanical characteristics of elastin and collagen from freshly excised peripheral pulmonary parenchyma. The model suggests that, until collagen deposition reaches a threshold level, chord compliance is not influenced by or is only slightly influenced by collagen turnover. A reduction of chord compliance until around 30 ml/cmH_2O is therefore essentially a result of a lung volume reduction. Compliance beyond this value may also be limited by collagen deposition, which at the cellular level is called the 'collagen-dependent maximal distension'. One can model the anatomical units of the fibrous skeleton of the lung as being made of extensible elastin and inextensible collagen, which are 'folded' in the lung resting position [7]. The limits of distension are dictated by the inextensible collagen fibers, which work as a 'stop-length' system. The significant correlation linking compliance to biological markers suggests that compliance may be also affected by alveolar remodeling.

The volume recruited by PEEP is usually assessed based on the static PV curve of the respiratory system. Alveolar recruitment leads to an upward shift along the volume axis of the PV curve with PEEP, compared with the curve with zero end-expiratory pressure, and is quantified as the volume increase with PEEP at the same elastic pressure [8,9].

ARDS = acute respiratory distress syndrome; PEEP = positive end-expiratory pressure; PV = pressure–volume.

The technique used to perform the PV curve has been the subject of enormous attention. The three major questions regarding the technique are as follows. To what extent are the findings artifactual? What are the feasibility and the reproducibility of the different techniques? Does the measurement by itself affect the underlying physiology?

The first question (artifactual findings) was related both to the technique (e.g. gas exchange occurring during a prolonged insufflation and deflation with the super-syringe technique [10]) and to the findings in general. Since the measurements are performed during static (or quasi-static) maneuvers, are we measuring phenomena that are relevant during mechanical ventilation (opening and closing of lung units) or is the observation related to the artificial derecruitment imposed by a prolonged expiration to functional residual capacity [11]? There is ample evidence that, during single expirations to lower PEEP values, a derecruitment occurs that becomes more and more pronounced at lower PEEP values, while recruitment continues up to 35–45 cmH_2O during the following reinflation – but the whole picture may be very different during the course of mechanical ventilation from that during a single PV curve maneuver.

The second question (feasibility and reproducibility) has received relatively little attention, but research seems to indicate that the reproducibility is reasonably good [11,12].

The final question is the object of investigation of the study by the group of Papazian in the present issue [13]. Many questions could be addressed regarding the influence of the PV curve measurement on the end-expiratory lung volume, hemodynamics and gas exchange. The authors produced PV curves with two different techniques in patients with acute lung injury and examined arterial blood gas changes in the 2 hours following recordings of each curve. The authors concluded that no significant (or substantial) influence existed on gas exchange in the group as a whole; this was far from true at an individual level, however, and changes occurred in different directions in terms of oxygenation or partial pressure of CO_2. The PV curve is a kind of recruitment maneuver. The influence of this recruitment maneuver in many patients is again limited in terms of time and effect, but these effects may be larger than expected if the pressure used is markedly higher than the plateau pressure during the course of mechanical ventilation.

Among different patients, Papazian's group found sustained increases in PaO_2, decreases in PaO_2 and increases in $PaCO_2$. Although it is difficult to speculate without having more precise data from these patients, several mechanisms can be at work explaining these effects. An increase in oxygenation may easily be explained by the reopening of some areas of the lungs, which were perfused but nonventilated; the increase in oxygenation could have occurred because these areas remained open after the maneuver. A sustained increase in lung volume after determining such PV curves has been observed [14]. A decrease in oxygenation may have resulted from adverse hemodynamic effects resulting in reduced mixed venous oxygen content, especially in patients with limited cardiorespiratory reserve. This may have been facilitated by hypovolemia and impediment to venous return, although this effect should not last long after the end of the maneuver. In patients with ARDS, the problem may be more at the right ventricular level, functioning on the edge of cardiac failure because of severe pulmonary hypertension. A recruitment maneuver can markedly increase the right ventricle afterload, can induce right ventricle dilation and can decrease left ventricular size and function. This was recently illustrated by Nielsen and colleagues [15]. The finding of an increased $PaCO_2$ level may result from the same mechanism. It may also result from the reopening of nonperfused or poorly perfused previously collapsed alveoli. Finally, this finding may be explained by persisting overdistension after the maneuver, especially if some degree of hypovolemia was present, generating high ventilation–perfusion areas or non zone III of the lungs.

The merit of the study by Papazian's group is that it examines, in a systematic manner, the impact of a diagnostic or monitoring technique on the patient's underlying physiology. Studies like this are needed, especially in the intensive care unit setting, to more systematically address the impact of various diagnostic techniques on the underlying physiology or clinical course of patient. The PV curve acts like a recruitment maneuver, and the interpretation of the clinical management incorporating this tool must take this fact into account. Fortunately, as shown in this study, the observed effects of this maneuver are limited or insignificant in many, but not all, patients.

Competing interests

The author declares that they have no competing interests.

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