Pressure-volume curve: methods and meaning

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The pressure-volume curve of the respiratory system is a physiological method used for diagnostic purposes to describe the static mechanical properties of the respiratory system. A renewal of interest in the pressure-volume curve has recently appeared because of experimental evidence regarding the information conveyed by the curve, a better understanding of the pathophysiologic factors influencing its interpretation and the beneficial results of clinical trials based on the use of the pressure-volume curve for ventilatory management of acute respiratory distress syndrome. Thus, adapting ventilatory settings to individual characteristics of the patients in terms of respiratory mechanics may be an extremely important aspect for a better management of the most difficult to ventilate patients with acute lung injury. There is considerable experimental evidence that both the opening-collapse phenomena and the excessive lung stretch may cause damage to the lungs. Therefore tools allowing an individual titration of ventilatory settings taking into account the constraints of the respiratory system seem highly desirable. The pressure-volume curve might be easily achievable at the bedside as a monitoring tool. The low-flow technique using ventilator technology has several potential advantages. It is hopeful to think that in the future the measurement of the P-V curve and the quantification of alveolar recruitment may be easily provided at the bedside and may help for the titration of the ventilatory settings in clinical practice. This review will focus briefly on the physiologic background, technique description, and recent advances concerning the interpretation of the P-V curve in the critically ill patients.

Key words: **Lung mechanics - Pressure-volume curve - Acute respiratory distress syndrome.**

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The pressure-volume (P-V) curve of the respiratory system is a physiological method used for diagnostic purposes to describe the static mechanical properties of the whole respiratory system.[1] It has been easily adapted to mechanically ventilated patients because of the relative easiness to obtain reliable signals in sedated and paralyzed patients, and has given the unique opportunity to apply this pulmonary function test to the most severe situations encountered in pulmonary disease.

This review will focus briefly on the physiologic background, technique description, and recent advances concerning the use and interpretation of the P-V curve in the critically ill patients.

Physiologic background

The P-V relation describes the static behavior of the respiratory system and is used to get information about the structures (lung, chest-wall and airways) of the system.[2,3] These mechanical structures can be described by their resistive and elastic properties. The study of the elastic properties of the respiratory system is relevant when the lungs are principally involved in a pathologic process. To eliminate the resistive, non-elastic factors included in the measured pressures, it is necessary to reach static or quasi-static conditions. The visco-elastic behavior of the respiratory system may also influence the P-V curve, at least when it is acquired under dynamic conditions.[4] To study the elastic properties of the respiratory system needs also to eliminate the influence of the respiratory muscles. Patients are therefore usually under deep sedation, and muscle relaxants are most often administered.

During mechanical ventilation, airway pressure depends both on flow, volume and positive end-expiratory pressure (PEEP) generated by the ventilator, and on the resistive and elastic properties of respiratory system. At each moment, the equation of motion allows to describe the relationship between airway pressure and the mechanical properties of the thoraco-pulmonary system:[1]

$$\text{(Equation 1) } P_{AW} = P_{RES} + P_{EL} + PEEP$$

where P_{AW} is the airway pressure, P_{RES} is the resistive pressure and P_{EL} is the elastic pressure. PRES varies with flow (?) and the resistances (R) of the respiratory system while PEL depends both on volume (V) and elastance (E) of the respiratory system.

Developing the P_{RES} and P_{EL} terms in Equation 1:

$$\text{(Equation 2) } P_{AW} = (? \ R) + (V \ ? \ E) + PEEP.$$

Based on these preliminary considerations, the techniques used to trace the P-V curve can be distinguished in static and quasi-static, or dynamic. With the static methods (the supersyringe and the multiple occlusion techniques), the airway pressure is measured during an end-inspiratory pause, i.e, at zero flow. Therefore, the first term in the Equation 2 (?R) is zero and both the flow-resistive and inertial pressures are eliminated. Thus, if the insufflated volume is known, it is possible to calculate the elastance (and its inverse, the compliance) of the respiratory system. Conversely, with the dynamic, quasi-static method (the low flow technique), the resistive and the viscoelastic phenomena are present during the measurement of the curve; thus, for a correct evaluation of the elastic properties of the respiratory system it is essential to take these phenomena into account. Nevertheless, if a sufficiently low flow is used, P_{RES} becomes negligible and the P_{AW} approximates the P_{EL}. Another approach is to use a sinusoidal flow allowing the exact computation of resistive airway pressure. In this case, the P_{RES} can be subtracted by the P_{AW} to precisely calculate the P_{EL}.

Whatever the technique, the simultaneous measurement of the airway and esophageal pressure allows the investigator to trace the P-V curves of the lung and of the chest-wall.

Techniques for P-V curve measurement

The supersyringe technique

The supersyringe technique was the first described for the static P-V curve measurement in ARDS patients.[5,6] It consisted of an insufflation, starting from the resting volume of the respiratory system to the estimated total lung capacity, accomplished with a calibrated syringe of known volume ranging from 1.5 to 2 liters. Disconnection from the ventilator, allowing a complete exhalation to the resting volume of the respiratory system, was necessary before connection to the syringe. To trace the curve, the respiratory system was inflated in a stepwise manner from the starting volume, considered to be the relaxation volume of the system, to a maximum of 40 to 50 cmH_2O and then deflated.[7,8]

The supersyringe technique has been largely used to describe the modifications of the elastic properties of the respiratory system related to the severity and the stage of the ARDS.[6] Nevertheless, this method has drawbacks and limitations that have been well described.[9,10] The procedure is relatively long and the results may be influenced by oxygen consumption, as well as by the changes in gas temperature and humidity. The artifacts and the cumbersome nature of the procedure have limited the use of this method to the research field.

The multiple occlusion technique

The multiple occlusion technique uses the ventilator capabilities to perform volume and static pressure measurements, during occlusions performed with different inflated volumes delivered during constant-flow controlled ventilation.[11 12] After ensuring the absence of leaks in the system, the intrinsic PEEP is measured during an end-expiratory pause before each studied insufflation to ensure the stability of the end-expiratory volume and pressure.[13] The multiple end-inspiratory occlusions are performed using different tidal breaths starting from the same lung volume. The static pressure values, obtained after a few seconds pause, and the exhaled volume after the occlusion release are read on the ventilator monitor. The curve is then constructed by plotting the volumes against the corresponding static pressures. By acquiring the P-V curves from zero end-expiratory pressure (ZEEP) and from PEEP together with the measurement of the lung volume retained by PEEP, it is possible to quantify the alveolar recruitment.[12 14 15]

Compared to the supersyringe technique, the multiple occlusion method offers the advantages of not requiring special equipment, and the disconnection of the patient from the ventilator, also allowing the computation of alveolar recruitment, when curves from PEEP and ZEEP are performed. Apart from the relative complexity of the technique, however, the sequential insufflation of the different tidal breaths can modify the lung volume history, thus potentially influencing the shape of the curve. The need to ventilate at ZEEP for a prolonged period can also be poorly tolerated.

The low flow technique

The technical complexity and the cumbersome nature of the apparatus needed with the above described methods, led to an effort to simplify the measurement of the P-V curve. A dynamic technique using a low constant flow for the curve recording was proposed in the 1970s.[16] This technique is based on the concept that, during passive lung inflation with constant inspiratory flow, the rate of the airway pressure change is inversely related to the compliance of the respiratory system.[17] However, for a correct analysis of the elastic properties of the system, it is necessary to take into account the resistive pressure. Different authors have found that results obtained with a sufficiently low flow were highly reproducible and similar to those found with the supersyringe and multiple occlusion methods.[14 18] The initial part of the P-V curve was also better visualized compared to other techniques. Recently, Servillo *et al.*[19] described a new automated low flow inflation method, performed with a commercial ventilator (Servo Ventilator 900C, Siemens-Elema AB, Sweden) controlled by a computer. This method allowed a complete control of the events preceding the measurements as well as the calculation of the resistances related to the endotracheal tube and to the airway. After subtracting the resistive pressure to the measured airway pressure, the authors showed a good agreement between the results of the automated low flow inflation method (15 L/min) and of the multiple occlusion technique, concerning the chord compliance and the lower inflection point (LIP) values. In some patients slight differences existed at high lung volume concerning the upper inflection point (UIP), usually above the tidal range and thus with small practical relevance. This probably reflected the importance of the viscoelastic and/or of the pendelluft phenomena at the higher lung volume.[4]

Rodriguez *et al.*[20] compared the multiple occlusion with the low constant flow method performed with the standard equipment of a commercial ventilator. They found a good agreement between the P-V curves and the linear compliance values with the two techniques, with a slight tendency to overestimate static pressure with the low flow. The time needed for a complete P-V curve acqui-

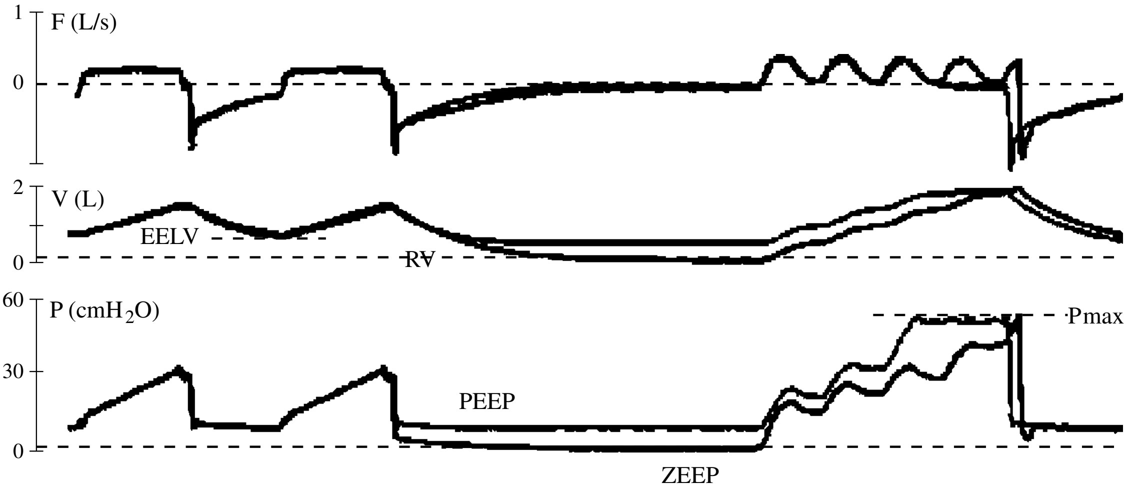

Fig. 1.—Maneuvers for P-V curves acquisition using the low sinusoidal flow technique. Schema illustrating the recording maneuvers for the acquisition of the pressure-volume curves at ZEEP and at PEEP with the low sinusoidal flow technique. The flow (F), volume (V) and pressure (P) signals preceding the insufflations, *i.e.* the lung volume history, from ZEEP and from PEEP are superimposable. After a prolonged expiration at ZEEP or at PEEP, the insufflation at modulated flow allows to trace the elastic P-V curves at ZEEP and at PEEP, respectively. EELV: end-expiratory lung volume, at PEEP; Pmax: safety maximal pressure; RV: the resting or elastic equilibrium volume, at ZEEP (adapted from [23]).

sition was 38 minutes with the multiple occlusion *versus* 3 minutes with the low flow technique.

The previous findings have been confirmed in a recent study of Lu *et al.*[21] These authors compared the P-V curves obtained with two constant flows of 3 and 9 L/min with those acquired with the supersyringe and with the multiple occlusion methods. The constant flow technique was performed with a standard intensive care unit ventilator, allowing the simultaneous display of the curve on the screen and the subsequent analysis using cursors. The pulmonary and thoraco-pulmonary P-V curves measured with the supersyringe, the multiple occlusions, and the constant flow of 3 L/min were superimposed. The curves obtained with 9 L/min were slightly shifted to the right because of the flow-dependent resistive component, but this shift seemed to have little clinical relevance (the resistive airway pressure resulting from the administration of the 9 L/min constant flow was less than 1 cmH_2O). The thoracic P-V curves as well as the values of chord compliance and LIP were identical with all the methods.

Svantesson *et al.*[22] recently evaluated a similar technique in healthy anesthetized subjects, using a low sinusoidal flow to allow the exact computation of resistive airway pressure. The inspiratory airway resistance determined with the flow interruption technique and with the sinusoidal flow was similar. Also, an excellent agreement between the values of chord compliance, LIP, and UIP measured with a low constant flow and with the sinusoidal flow was found. The modulated flow seems to permit an accurate measurement of P-V curve in different situations, even in case of high resistances, and without needing a very low flow.

The automated low sinusoidal flow technique has been used in patients with acute lung injury (ALI) by Jonson *et al.*[23] (Fig. 1). The authors found that the measurement of inspiratory airway resistance by the flow modulation allowed an accurate determination of the elastic recoil pressures. Because it is a dynamic measurement, viscoelastic forces especially present at high lung volumes are included in the calculated elastic pressures.[24] For this reason, P-V curve acquired under dynamic conditions may give more realistic information about the pressures really exerted on peripheral lung units during insufflation, and be more clinically relevant than the static recordings. The control of the lung volume history preceding the measurements, together with the automated and unbiased successive analysis of results, allowed to minimize methodological errors.

In conclusion, the low flow technique offers many advantages compared to the other methods. It is easy to use at the bedside, requires much less time than the multiple occlusion technique, and can be done without special equipment other than a modern ventilator that supplies a low constant flow (<15 L/min), without the patient's disconnection from the ventilator. Airway pressure measurement allows the investigator to estimate the alveolar pressure when the resistive pressure is minimized or subtracted. Moreover, this technique allows P-V curves to be drawn for different ventilatory settings and different levels of PEEP, and can be used to quantify alveolar recruitment.

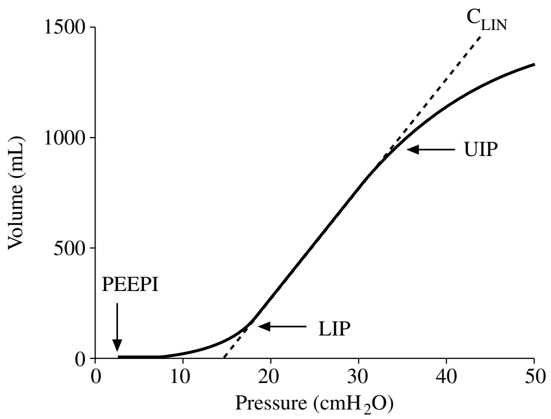

Fig. 2.—Typical inspiratory pressure-volume curve of the respiratory system in a patient with acute respiratory distress syndrome. Volumes are expressed relative to the end-expiratory lung volume at ZEEP. The curve consists of three segments identified by the presence of a lower inflection point (LIP) and an upper inflection point (UIP). The intermediate linear segment has the steeper slope (interrupted line), *i.e.*, the greater compliance (chord compliance or C_{LIN}). Note the presence of intrinsic positive end-expiratory pressure (PEEPi) causing a rightward shift of the curve.

Interpretation of the inspiratory thoraco-pulmonary P-V curve

In healthy humans, the P-V curve from residual volume to total lung capacity has usually a sigmoidal shape. However, above the functional residual capacity (the lung volume at the end of a normal, quiet expiration), in the volume range of tidal ventilation, the slope of the curve is usually linear. In ARDS patients, the reduction of the number of normally ventilated alveolar units decreases the range where tidal ventilation occurs, resulting in small variation of volume per unit of pressure change, and the entire curve is flattened.

The S-shaped inspiratory P-V curve of ARDS patients can be thought as consisting of three segments (Fig. 2). At low lung volumes, the initial flat segment with very low compliance reflects the collapse of peripheral airways and lung units.[6] 16 As insufflation proceeds, an intermediate linear segment with a steeper slope, *i.e.*, a greater chord compliance (C_{LIN}), is observed. Compliance remains stable over the linear portion,[25] reflecting the progressive opening of collapsed alveoli all along the inflation. At high pressures, but at much smaller lung volumes than in healthy subjects, the P-V curve flattens again with a fast decrease of the slope, i.e. the compliance, in its third segment. The transition between the first flat portion and the linear part of the curve is termed the LIP. In ARDS patients, the LIP is usually supposed to represent the mean critical pressure needed to reopen the previously collapsed airways and alveolar units, a phenomenon defined as alveolar recruitment.[6 26] Similarly, the point at the transition between the linear part and the third segment where the compliance begins to fall is termed the UIP. The UIP may correspond to the volume at which overdistension of certain lung units happens [25] and/or at which tidal alveolar recruitment ends.[27] Then, lung ventilation occurring below the LIP or above the UIP carries the risk of generating repetitive collapse/reopening or overdistension phenomena, both of which are associated with the appearance and the progression of lung injury. Hence, the segment between the LIP and the UIP may represent the zone over which tidal ventilation in ARDS patients should preferentially occur in order

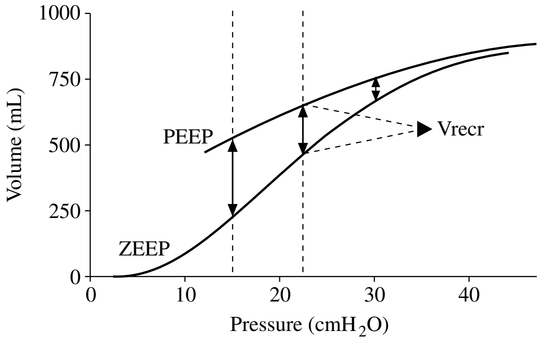

Fig. 3.—Inspiratory elastic pressure-volume curves recorded from ZEEP and from PEEP in a patient with ARDS. Volumes are expressed relative to the end-expiratory lung volume at ZEEP. The chord compliance of the P-V curve from PEEP is lower than the curve from ZEEP. Recruitment of collapsed alveolar units causes an upward shift of the curve from PEEP. Thus, the recruited lung volume (Vrecr) is expressed by the volume difference between the P-V curve at ZEEP and the P-V curve at PEEP for the same amount of airway pressure. ARDS: acute respiratory distress syndrome; PEEP: positive end-expiratory pressure; ZEEP: zero end-expiratory pressure.

to protect the lung from further injury.[28] The pressure at LIP has been suggested for the setting of the "best" PEEP in order to optimize recruitment and to prevent end-expiratory collapse, while the tidal volume should be set so that the end-inspiratory pressure (plateau pressure) does not overstep the pressure at UIP.

The above described shape and characteristics of the P-V curve is greatly influenced by a number of factors including the pathophysiologic mechanism and the stage [6] of the lung disease, changes of the chest-wall mechanics [29, 30] the presence of intrinsic PEEP,[31] the ventilatory mode and parameters preceding the measurement as well as the technique used to acquire the P-V curve. Other factors make the interpretation of the inspiratory thoraco-pulmonary P-V curve quite difficult. Recent data suggest that the LIP does not indicate either the beginning or the end of recruitment [23, 27] which proceeds far above the LIP. Moreover, recent experimental studies [32] have shown that tidal ventilation with PEEP does not occur on the inspiratory limb of the whole P-V envelope but on its deflation limb. This implies that the "best" PEEP to prevent end-expiratory collapse has to be set according to the alveolar closing pressures and not the opening pressures.[33] To analyze the relationship between the LIP and the alveolar closing pressures we have quantified the derecruitment induced by progressively decreasing PEEP levels (20, 15, 10 and 5 cmH_2O) with a constant tidal volume (7 mL/kg) in 16 ARDS patients.[34] The hypothesis was that, if PEEP was set above the closing pressure of the alveoli, a small decrement in PEEP would not result in substantial derecruitment, until the PEEP was set below the closing pressure. If derecruitment was found uniformly distributed, the closing pressures might be considered to be spread over a large range of pressures. We found that derecruitment was quite well distributed over the range of the studied PEEP but was predominant at the highest PEEP levels and no correlation existed between the amount of derecruitment and the LIP. These results suggest that the closing pressures range is quite large and the alveolar closure begins already at pressures as high as 20 cmH_2O. Finally, the LIP seems to be poorly correlated with the alveolar closure and might not to be the best parameter to set the optimal PEEP in ARDS. The presence of a marked LIP might simply indicate an homogeneously diseased lung and/or the need to recruit the lung,[27, 35] while the measurement of the alveolar closing pressures and/or the quantification of the derecruitment which could be prevented with the application of PEEP might be more relevant for an appropriate PEEP setting. Also the C_{LIN} of the P-V curve from ZEEP gives information about lung recruitment. Indeed, the P-V relationship between LIP and UIP reflects progressive recruitment. This has been recently suggested using a mathematical model [27] and shown in patients with ALI.[23, 34] We found that the application of PEEP induced a decrease in C_{LIN} of the P-V curves from PEEP compared to the C_{LIN} at ZEEP. A relatively low value of C_{LIN}, as observed at high PEEP, may indicate that the lung is well recruited while a high value of C_{LIN} on the curve from ZEEP may be interpreted as a sign of ongoing recruitment occurring during the tidal insufflation. This may explain the good correlation that was observed between the C_{LIN} at ZEEP and the recruitment measured at PEEP 15 cmH_2O.[34] Therefore, when a high C_{LIN} is present at ZEEP, it may indicate a highly recruitable lung. The UIP was initially related to the beginning of overdistension of certain lung units.[25] New data show that the UIP may indicate that recruitment has ceased during inflation, and does not necessarily indicate only overdistension.[27] Depending on the range of opening pressures, the UIP caused by overdistension can be masked if concomitant recruitment continues above the pressure at UIP.

Chestwall mechanics may affect the P-V relationship of the respiratory system. Mergoni *et al.*[29] investigated the impact of chestwall mechanics on the shape of the P-V curve of the respiratory system, particularly concerning the LIP, in patients with acute respiratory failure. The authors found that only in the majority of patients the LIP on the P-V curve of the respiratory system was due only to the characteristics of the P-V curve of the chestwall. These results suggest that, in some cases, the impairment of the chestwall may explain the LIP seen on the P-V curve of the respiratory system, even if this seems to have limited clinical implications (the higher LIP value found on the P-V curve of the chestwall was 4.8 cmH_2O). Recently, Ranieri *et al.*[30] studied the relative contribution of the chestwall, the lung and the abdomen to the overall impairment of the P-V relationship of respiratory system in ARDS patients. They found that the impairment of the elastic properties of the respiratory system varied with the underlying disease responsible for ARDS. By contrast to patients with medical ARDS, in patients with surgical ARDS, the inflating volume caused a decrease in the respiratory system and the lung compliance associated with alveolar overdistension. While the medical ARDS patients had normal chestwall and abdominal curves, patients with surgical ARDS had chestwall and abdominal P-V curves shifted rightward and flattened. This suggested that the flattening of the P-V curves of the respiratory system observed in some ARDS patients might be secondary to abdominal distension.

Alveolar recruitment

Alveolar recruitment, *i.e.*, the reopening of the previously collapsed lung units, is the cornerstone of some recent protective ventilatory strategies in ARDS.[36] Indeed, it has been suggested that the presence of atelectasis may have an injurious effect on the lung, causing the maintenance and the increase of lung and systemic inflammation ("atelectrauma").[37, 38] Reversing such a deleterious phenomenon and recruiting the collapsed alveoli, together with reducing the intrapulmonary shunt and improving oxygenation, are goals of lung protection.[28] Therefore, the analysis of the effect of PEEP on alveolar recruitment is of special concern in the management of ARDS patients. No precise quantification of recruitment had been made before the studies by Ranieri and coworkers.[12, 14, 15] The authors simply measured the end expiratory lung volume above the relaxation volume both during ZEEP and during PEEP ventilation. They could therefore place both curves (traced on ZEEP and on PEEP) on the volume axis referencing similarly the two curves to the relaxation volume of the respiratory system. Recruitment of lung units was calculated as the difference in lung volume between ZEEP and PEEP at the same airway pressure (Fig. 3). Using this method, Jonson *et al.*[23] have recently evaluated the amount of volume recruited by PEEP in ALI patients. The low sinusoidal flow technique was used to measure the P-V curves at PEEP and, after a single prolonged expiration during which PEEP was eliminated, at ZEEP. A significant recruitment occurred with PEEP, ranging from 205 mL at 15 cmH_2O to 78 mL at 30 cmH_2O, and a correlation was found between the LIP value and the amount of alveolar recruitment. As previously reported, linear compliance was always lower on the curve recorded at PEEP than on the curve at ZEEP, related to the PEEP-induced lung recruitment phenomena. If the effect of PEEP on recruitment is well known, relatively scarce data exist in the literature about the influence of tidal volume (Vt). We have recently investigated the effect of two tidal volumes (low, 6 mL/kg, and conventional, 10 mL/kg) on the alveolar recruitment in 15 ARDS patients.[39] The PEEP was kept constant with the two Vt and set at the LIP on the P-V curve recorded from ZEEP. Compared to the conventional Vt, the low Vt induced a significant alveolar derecruitment. The alveolar derecruitment induced by the low Vt was completely reversed by a re-expansion maneuver and prevented by the application of the higher level of PEEP (4 cmH_2O above the LIP). These results show that both PEEP and Vt have an influence on the recruitment of the lung. When Vt is reduced, setting the PEEP at LIP does not guarantee an optimal recruitment suggesting that LIP may not be the best parameter for PEEP setting. Thus, these results reinforce the idea that the Vt reduction, actually recommended in order to protect the lung,[40] has to be associated with higher levels of PEEP, often well above the LIP, to prevent derecruitment. Concerning the effect of a re-expansion maneuver, it is interesting to note that it was effective in increasing alveolar recruitment only when performed during low tidal ventilation, while it produced no effect with the conventional Vt. In other terms, if the lung is well recruited a re-expansion maneuver is relatively ineffective while, when effective, it may simply indicate that the lung was not optimally recruited.

Conclusions

Adapting ventilatory settings to individual characteristics of the patients in terms of respiratory mechanics, gas exchange and hemodynamics may be an extremely important aspect for a better management of the most difficult to ventilate patients with ALI or ARDS.[41] There is considerable experimental evidence that both the opening-collapse phenomena and the excessive lung stretch may cause damage to the lungs. Therefore tools allowing an individual titration of ventilatory settings taking into account the constraints of the respiratory system seem highly desirable. The pressure-volume curve might be easily achievable at the bedside as a monitoring tool. The low-flow technique using ventilator technology has several potential advantages. It is thus tempting to think that in the future the measurement of the P-V curve and the quantification of alveolar recruitment may be easily provided at the bedside and may help for the titration of the PEEP level and of the tidal volume.

Riassunto

La curva pressione-volume del sistema respiratorio è una metodica fisiologica utilizzata a scopo diagnostico per descrivere le proprietà meccaniche statiche del sistema respiratorio. Un rinnovato interesse nella curva pressione-volume è stato recentemente suscitato dai dati sperimentali riguardanti le informazioni che si possono ottenere dalla curva, da una migliore comprensione dei fattori fisiopatologici che influenzano la sua interpretazione e dai risultati positivi degli studi clinici che hanno basato sull'uso della curva pressione-volume il trattamento ventilatorio della acute respiratory distress syndrome. Adattare i parametri della ventilazione alle caratteristiche individuali dei pazienti, in termini di meccanica respiratoria, può costituire un aspetto estremamente importante nel trattamento dei pazienti con danno polmonare acuto che presentino gravi difficoltà di ventilazione. Esiste una considerevole evidenza sperimentale che sia i fenomeni di apertura-collasso sia un eccessiva distensione del parenchima polmonare siano in grado di provocare un danno polmonare. Tutti gli strumenti che consentano una regolazione individuale dei parametri ventilatori, che tenga conto dei limiti entro cui è costretto il sistema respiratorio, sembrano essere, quindi, altamente desiderabili. La curva pressione-volume può essere facilmente ottenuta al letto del paziente, come un sistema di monitoraggio abituale. La tecnica a basso flusso, che sfrutta la tecnologia del ventilatore, ha diversi potenziali vantaggi. È auspicabile che nel futuro la misurazione della curva P-V e la quantificazione del reclutamento alveolare possano essere facilmente accessibili al letto del malato e possano aiutare la regolazione dei parametri ventilatori nella pratica clinica. Questa review presenta brevemente il background fisiologico, la descrizione della tecnica e i più recenti progressi nell'interpretazione della curva P-V nei pazienti critici.

Parole Chiave: Curva pressione-volume - Acute respiratory distress syndrome - Meccanica respiratoria.

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