Respiratory Mechanics Derived From Signals in the Ventilator Circuit

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The aim of this article is to identify and interpret the data provided by modern ventilators that provide the greatest clinical help in evaluating respiratory mechanics during mechanical ventilation. In intensive care, respiratory mechanics can be assessed in dynamic conditions (no flow-interruption) or static conditions (occlusion techniques) to record compliance and resistance and to monitor pressure, flow, and volume. Real-time visualization of the pressure curve is crucial for monitoring during volume-controlled ventilation, in which pressure is the dependent variable. Analysis of the pressure curve has little clinical utility during pressure-controlled ventilation, in which the dependent variable is the flow waveform, which varies according to changes in the mechanics of the respiratory system. Pressure-volume loops and flow-volume loops provide useful information on the dynamic trends of the respiratory system compliance and resistance, respectively. Modern ventilators provide complete monitoring of respiratory system mechanics, which is our guideline for optimizing ventilatory support and avoiding complications associated with mechanical ventilation. *Key words: respiratory mechanics, lung compliance, lung resistance, mechanical ventilation, waveforms.* [Respir Care 2005;50(1):55–65. © 2005 Daedalus Enterprises]

Introduction

The aim of this review is to identify and interpret the data provided by modern ventilators that provide the greatest clinical help for a correct evaluation of respiratory mechanics during artificial ventilation.

Respiratory mechanics measurements can be assessed in a dynamic condition (no flow interruption) or a static condition (occlusion techniques). Ventilators provide analog (waveform) and digital (numeric) output to record compliance and resistance and to monitor pressure, flow, and volume at the bedside.

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Equation of Motion

The pressure applied to the respiratory system of a ventilated patient is the sum of the pressure generated by the ventilator (measured at the airway opening [ie, mouth] (P_{AO}) and the pressure developed by the respiratory muscles, as described by the equation of motion:

$$P_{RS} = P_{AO} + P_{mus} = \dot{V} \times R + \frac{V}{C} + k \quad (1)$$

in which P_{RS} is the respiratory-system pressure, P_{AO} is the pressure at the airway opening, P_{mus} is the pressure developed by the respiratory muscles, V is volume, $\dot{V}$ is flow, R is the airways resistance, C is the respiratory-system compliance, and *k* is a constant that represents the alveolar end-expiratory pressure.

P_{AO}, and $\dot{V}$ can be measured by the pressure and flow transducers in the ventilator. Volume is derived mathematically from the integration of the flow waveform.

The term $\dot{V} \times R$ corresponds to the pressure dissipated across the airway and the endotracheal tube, to overcome the frictional forces generated by gas flow, which, along with $\dot{V}$ determine the resistance of the respiratory system (R_{RS}).

The term V/C, on the other hand, corresponds to the pressure that must be applied to overcome elastic forces, and V/C depends on both the volume insufflated in excess of resting volume and on the respiratory system compliance (C_{RS}). The respiratory system is linear in the range of the tidal volume (V_T), with constant R_{RS} and C_{RS} throughout the respiratory cycle.[1]

The constant *k* indicates the P_{AO} value when both flow and volume are zero, and it has the practical utility of absorbing any error that might be made in estimating functional residual capacity from an integrated flow signal.[2] The constant *k* takes into account the application of positive end-expiratory pressure (PEEP) or the presence of intrinsic PEEP (auto-PEEP), if present.

When the patient's breathing activity is entirely passive (full ventilatory support), pressure developed by the respiratory muscles is negligible, and the driving pressure necessary to move air in and out of the thorax can be described by the simplified equation of motion:

$$P_{RS} = P_{AO} = \dot{V} \times R + \frac{V}{C} + k \quad (2)$$

The equation of motion can be applied when single-compartment models of respiratory system (ie, pipe-balloon) are used to describe the dynamic relationship between pressure, flow, and volume.

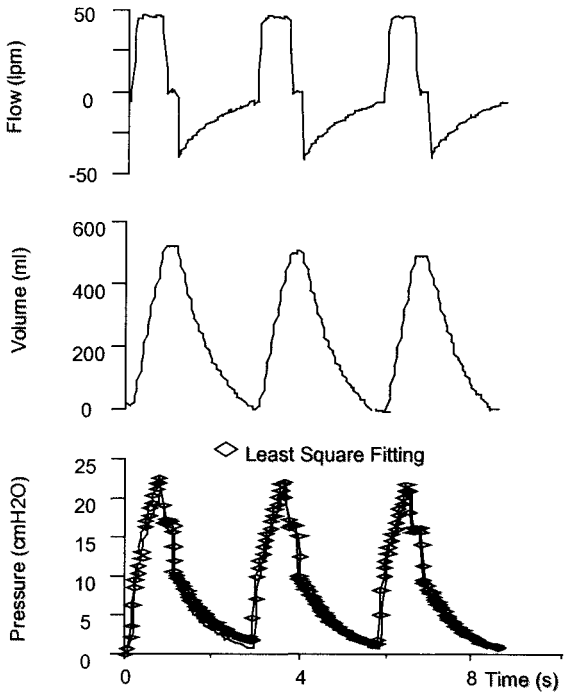

Fig. 1. Data collected during volume-controlled ventilation with constant flow. The pressure curve provided by Equation 2 (rhomboidal dots) (see text) is compared to measured airway pressure displayed on the ventilator.

Dynamic Mechanics

Dynamic mechanics can be derived during partially-supported ventilation (pressure-support ventilation) or totally-supported ventilation (volume-controlled ventilation [VCV]) with intubated patients, without flow interruption.[3,4]

Therefore, the respective values of the R_{RS}, C_{RS}, and *k* can be obtained by fitting Equation 2 to the sample values of P_{AO}, V, and $\dot{V}$ with a multiple linear regression analysis, or linear least squares fitting.[5–8]

With the use of a computer, the P_{AO}, V, and $\dot{V}$ variables, which change throughout the respiratory cycle, can be digitized at high speed (100 Hz), so C_{RS} and R_{RS} can be calculated from 100 or more equations per breath. The least squares fitting method does not require a peculiar inspiratory flow pattern; it can be applied during the whole breathing cycle or only in the inspiratory or expiratory phase. To avoid unrealistic results due to expiratory flow limitation in chronic obstructive pulmonary disease patients, Volta et al[9] suggest restricting the least squares fitting analysis to the inspiratory phase. That approach allows the superimposition of the "fitted" pressure curve on the P_{AO} curve, to give a visual impression of the "goodness of fit" between the 2 pressure waves (Fig. 1). The least square fitting method is less valid if the patient is actively breathing, because it assumes that P_{mus} is zero.

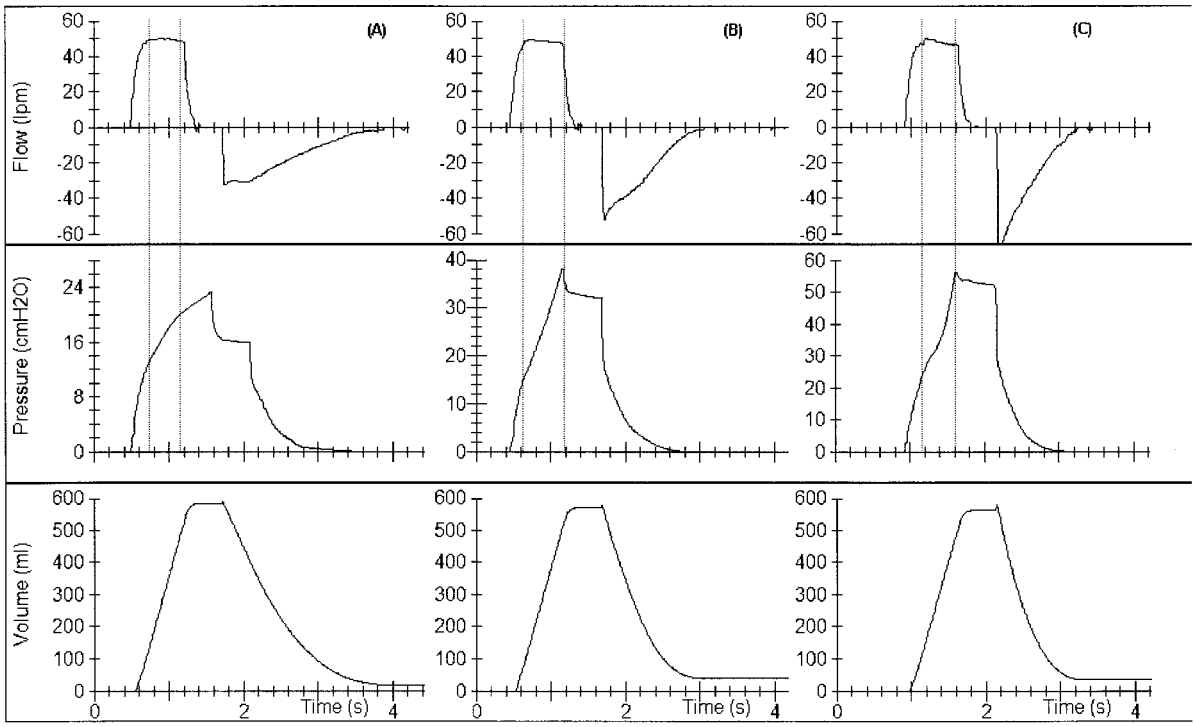

Fig. 2. Time-course of static airway pressure, flow, and volume during volume-controlled ventilation. From panel A to panel C, respiratory system compliance decreases. The constant-flow phase is defined by the dotted lines, which show the elastic load on the pressure curve. As peak inspiratory pressure increases, the morphology of the curve changes, turning from concave to linear to convex.

One of the most-important methodological limits of the single linear model is that it does not take into account the variation of R_{RS} and C_{RS} with lung volume, and it neglects flow turbulence and inertial forces. Moreover, R_{RS} can be underestimated during low-level pressure-support ventilation with high respiratory effort.[4]

Time Course of Airway Pressure During Constant-Flow Inflation

The pressure-time curve has a characteristic feature during constant-flow VCV. At the beginning of flow, an almost-vertical pressure increase occurs (because of the frictional forces generated by gas flow), which is necessary to overcome the resistance of the airways and the endotracheal tube. The curve shape then changes to a linear increase and follows a given slope to its maximum value (peak inspiratory pressure [PIP]), which occurs at end-inspiration. That course, which is normally linear, depends on the respiratory system compliance alone. As seen in Figure 2, in isovolumetric conditions the curve loses its linearity and becomes either convex or concave, according to the increase or decrease in C_{RS}.[10]

Similarly, during lung overdistention the curve shape is concave, and becomes first linear and then convex as the pre-set V_T decreases (Fig. 3).

This method, despite implying a complete muscle palsy, allows for real-time monitoring, with an immediate visualization of hyperinflation and lung recruitment.[11] From mathematical analysis of the shape of the dynamic pressure-time waveform during constant-flow ventilation in an animal model, Ranieri et al[12] derived the so-called *stress index*, to minimize the risk of ventilator-induced lung injury.

Static Mechanics

The dynamic mechanical properties of the respiratory system pertain to the situation where flow varies over time, whereas the static mechanical properties are emphasized during the absence of flow.

The assessment of C_{RS} and R_{RS} in paralyzed patients can be made during constant-flow, volume-controlled ventilation, with the end-inspiratory occlusion technique.

Many intensive-care ventilators can provide rapid airway occlusion at end-inspiration. During the pause, the flow drops rapidly to zero, the volume (V_T) is briefly trapped inside the lung, and static airway pressure can be measured.

The static airway pressure waveform (Fig. 4) has a characteristic trend, with the highest peak at end-inspiration (peak inspiratory pressure [PIP]), followed by a rapid drop

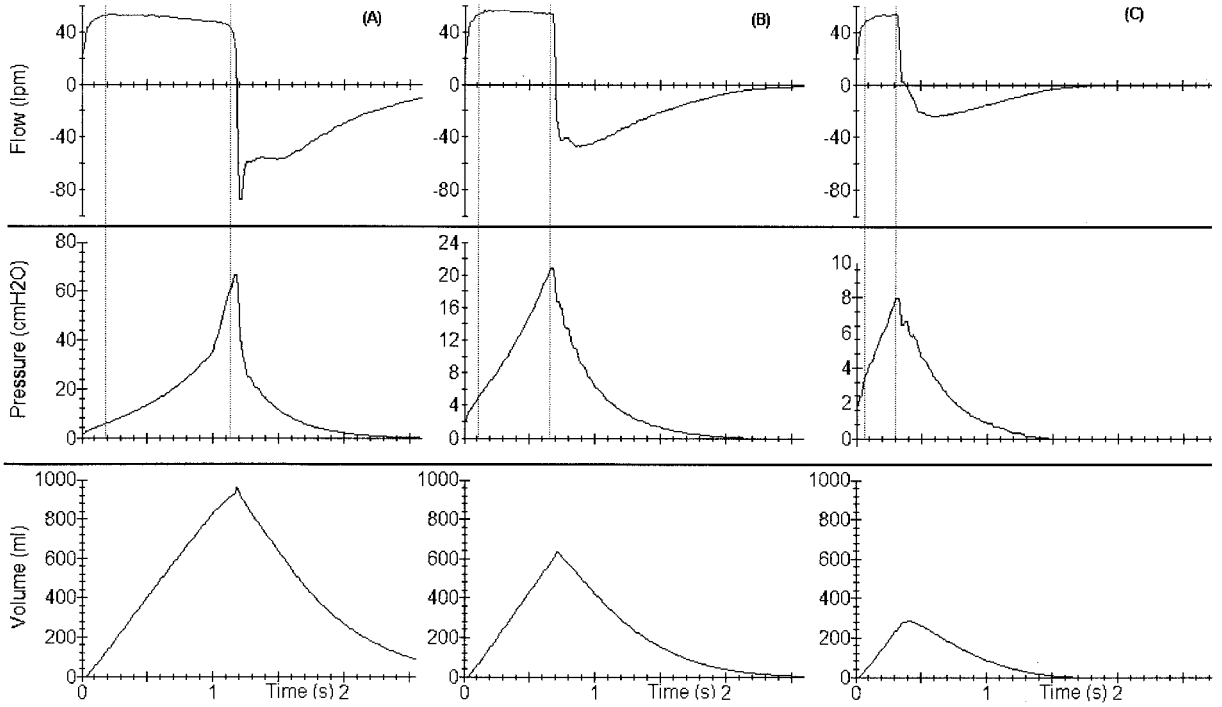

Fig. 3. Time-course of static airway pressure, flow, and volume during volume-controlled ventilation. From panel A to panel C, volume decreases and the shape of the airway-pressure curve changes. Panel A shows the typical airway-pressure-curve aspect during lung overdistention.

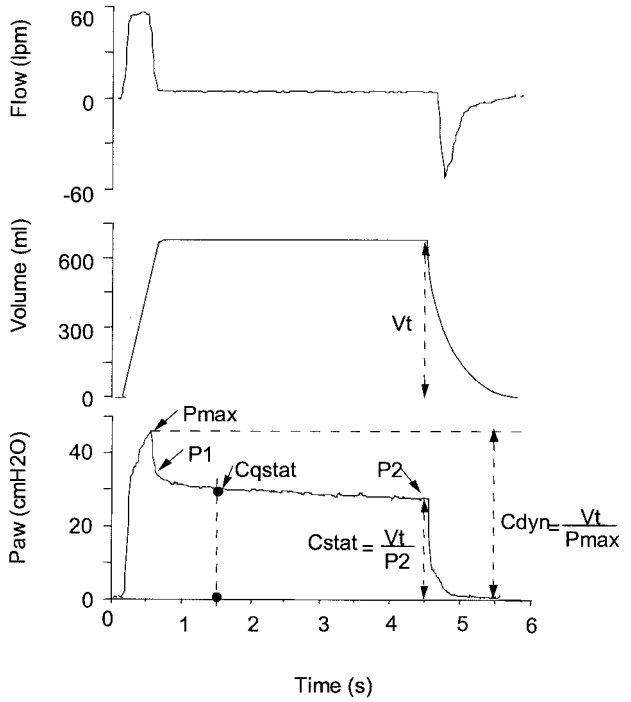

Fig. 4. Flow, volume, and airway pressure (P_{aw}) during end-inspiratory occlusion. P_{max} = maximum (peak) airway pressure. P1 points to end of the rapid post-occlusion pressure-drop. P2 points to the end of the slow pressure-decay plateau. C_{stat} = static compliance of the respiratory system. V_T = tidal volume. C_{dyn} = dynamic compliance of the respiratory system.

Table 1. Relationship of Compliance to Set Inspiratory Pause Time

Compliance	Pause time (s)
Static	2–3
Quasistatic	1–1.5
Dynamic	< 1

after the occlusion (P1 in Fig. 4), and a slow decay until a plateau is reached (P2 in Fig. 4).

P2 is the static pressure of the respiratory system, which in the absence of flow equals the alveolar pressure, which reflects the elastic retraction of the entire respiratory system. The pressure drop from PIP to P1 represents the pressure required to move the inspiratory flow along the airways without alveolar interference, thus representing the pressure dissipated by the flow-dependent resistances.

The slow post-occlusion decay from P1 to P2 (see Fig. 4) depends on the viscoelastic properties of the system and on the pendulum-like movement of the air (pendelluft). During the post-inspiratory occlusion period there is a dynamic elastic rearrangement of lung volume, which allows the different pressures in alveoli at different time constants to equalize, and depends on the inhomogeneity of the lung parenchyma. The lung regions that have a low time constant (ie, rapid zones), where the alveolar pressure rises rapidly, are emptied in the lung regions that have higher time constants (ie, slow zones), where the pressure rises

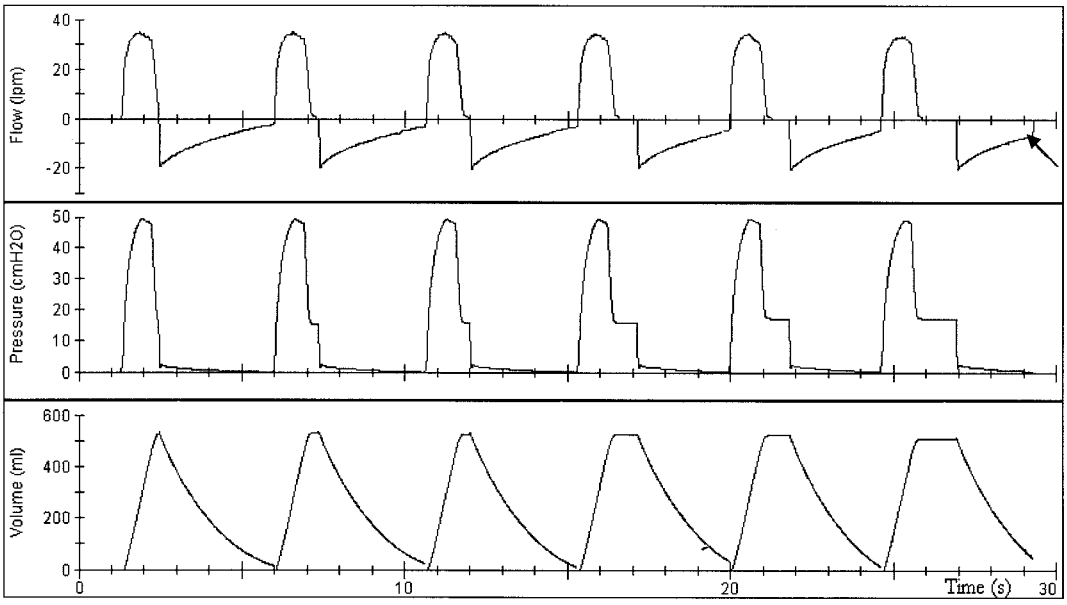

Fig. 5. Progressive increase in the inspiratory pause during volume-controlled ventilation, with a consequent decrease in expiratory time, which causes intrinsic positive end-expiratory pressure, which is evidenced by the fact that the expiratory flow wave fails to reach zero (arrow).

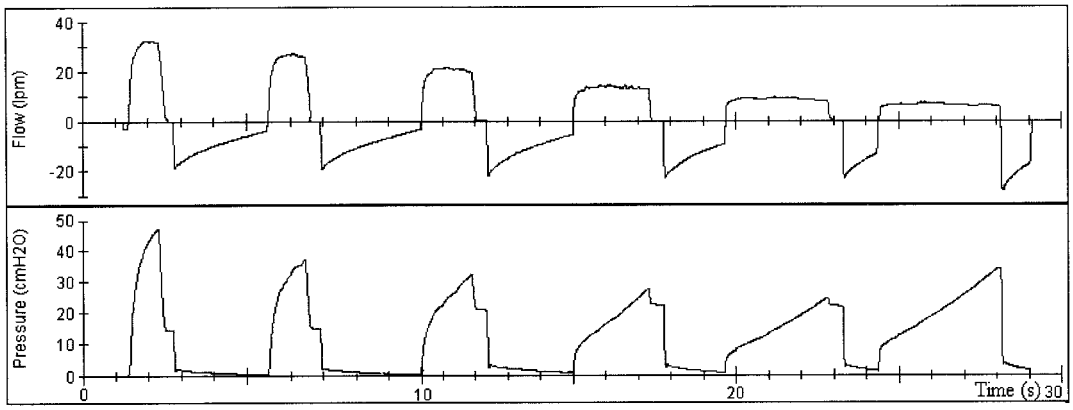

Fig. 6. Volume-controlled ventilation with progressive increase in the inspiratory time, but with a constant pause. During the 2 first cycles the flow-decrease decreases the PIP-P1 (see text) pressure gradient, while P2 (see text) remains unchanged, because the expiratory time is enough to avoid hyperinflation. Starting at the third cycle there is a progressive increase in P2, which tends to the peak inspiratory pressure value. During the last cycle the pause is no longer recognizable, because of the extreme decrease in expiratory time, which leads to air trapping, increase in PIP, and risk of barotrauma.

more slowly because of higher resistance or lower compliance (see Fig. 4).[13–18]

The concepts that we just expressed are indispensable to understanding the difference between the static compliance (C_{stat}) and the dynamic compliance (C_{dyn}) of the respiratory system. For static compliance the *volume variation* refers to the static plateau pressure (P2). For dynamic compliance the *volume variation* refers to PIP. Thus:

$$C_{stat} = V_T/P2 \quad (3)$$

and

$$C_{dyn} = V_T/PIP \quad (4)$$

The static compliance of the respiratory system mirrors the elastic features of the respiratory system, whereas the dynamic compliance also includes the resistive (flow-dependent) component of the airways and the endotracheal tube. The pressure gradient P1-P2 is based on the intrapulmonary post-occlusion dynamic phenomena that we described above.

When the inspiratory pause is shorter than 2 seconds, P2 does not always reflect the alveolar pressure. The compliance value thus measured is called quasi-static compliance

(see Fig. 4). In healthy subjects the difference between static compliance and quasi-static compliance is minimal, whereas it is markedly higher in patients who have acute respiratory distress syndrome or chronic obstructive pulmonary disease.

Table 1 shows how the respiratory system compliance varies in relation to the length of the inspiratory pause set on the ventilator.[19] Compliance based on the length of the set inspiratory pause varies from a static to a dynamic condition, and takes on different values and clinical importance.

If the operator wishes to include an inspiratory pause in the ventilatory setting, attention must be paid to the possible effects on the expiratory phase. Complete lung emptying may be prevented by decreasing expiratory time (Fig. 5).

At the end of a normal expiration in a normal subject, the alveolar pressure is nearly zero. Expiratory flow limitation, or an inadequate respiratory pattern (high V_T or high respiratory rate) cause auto-PEEP, due to volume trapping. Auto-PEEP is detectable during the post-expiratory occlusion maneuver.

Therefore, compliance must be calculated with the following formula, which also takes into consideration the possible use of PEEP:[20]

$$C_{RS} = \frac{V_T}{P_{aw} - (PEEP + PEEPi)} \quad (5)$$

The rapid-occlusion technique during constant-flow inflation allows measurement of respiratory system resistance when the respective pressure gradients (PIP-P2), (PIP-P1), and (P1-P2) are divided by the flow value ($\dot{V}$) immediately before occlusion. The ratio of (PIP-P1) to $\dot{V}$ is the *airway flow-dependent resistance*, which is commonly called "initial" or "minimal" or "ohmic" resistance (initial R_{RS}). When the (P1-P2) pressure gradient is taken into account, the *additional lung resistance* (ie, the change in R_{RS}) is obtained, which reflects the pendelluft phenomenon and the visco-plasto-elastic lung and thorax behavior. The total resistance, which takes into consideration the PIP-P2 pressure gradient, represents the sum of initial R_{RS} plus the change in R_{RS}.

In clinical practice, it is important to remember that the PIP-P1 pressure gradient is flow-dependent, whereas P2 is affected only by variations in volume and/or compliance. Figure 6 shows that during constant-flow VCV the inspiratory time is gradually increased, which decreases the height of the flow wave. There is a progressive decrease in the PIP-P1 gradient and in initial R_{RS}, and a contemporaneous increase in the plateau pressure, which reflects the establishment of a ventilatory pattern with inverted ratios, which generates auto-PEEP.

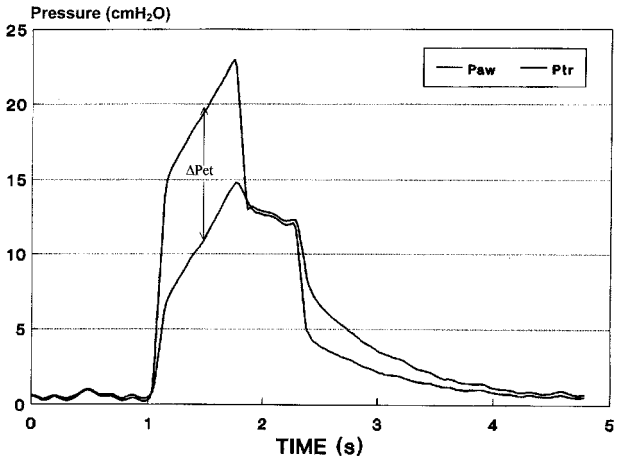

Fig. 7. Pressure curve measured at the mouth (static airway pressure [P_{aw}]) and at the end of the endotracheal tube (P_{tr}). The similar trend of the 2 curves only differs in the amount of pressure dissipated to overcome the resistance of the endotracheal tube (ΔP_{et}). During the end-inspiratory pause, the pressure gradient at the extremities of the endotracheal tube is zeroed, and both curves settle on the same P2 value (see text).

PIP represents the sum of the pressures applied by the ventilator to overcome the elastic and resistive forces of the respiratory system (airways and endotracheal tube). In isovolumetric conditions, variations in PIP depend on the pressure dissipated to overcome that resistance, so airway secretions, bronchospasm, and the diameter of the endotracheal tube affect PIP. The resistance exerted by the endotracheal tube can be calculated by a modern ventilator or measured with a dedicated catheter. In this case, PIP measured at the trachea is lower than the corresponding value measured at the mouth, because of the amount of pressure dissipated by the endotracheal tube, as shown in Figure 7. That amount of pressure should not be considered in the assessment of the risk of barotrauma, because it does not have any consequence at the alveolar level.

It must be emphasized that P2 (an indicator of compliance) may also be measured with a nonconstant-flow wave, as shown in Figure 8.[21] Furthermore, the end-inspiratory-occlusion maneuver can identify a leak in the respiratory circuit, because if there is a leak, the plateau pressure cannot be reached. In short, real-time visualization of the pressure curve is crucial for monitoring during VCV, because pressure is a dependent variable in the system. Volume-controlled ventilation also includes the use of descending-ramp and sinusoid flow waves, which correspond to the respective pressure waves seen in Figure 9.

On the other hand, the analysis of the pressure curve has limited clinical utility during pressure-controlled ventilation (PCV). In that case the dependent variable is flow, which changes as the features of the respiratory system change: the ventilator will constantly adjust flow so that the inspiratory pressure is maintained during the entire set

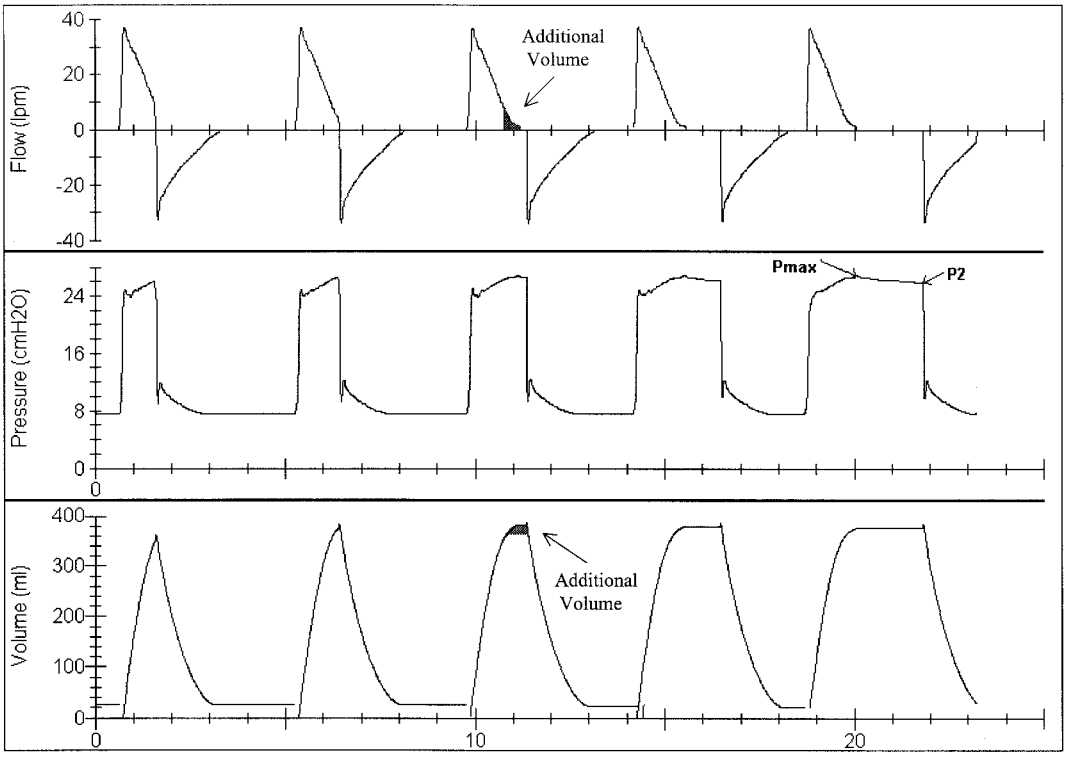

Fig. 8. Pressure controlled ventilation (PCV) with the characteristic flow-descent. The presence of an adequate pause at end-inspiration allows identification of the plateau pressure. The prolongation of inspiratory time in the third cycle produces an additional amount of volume.

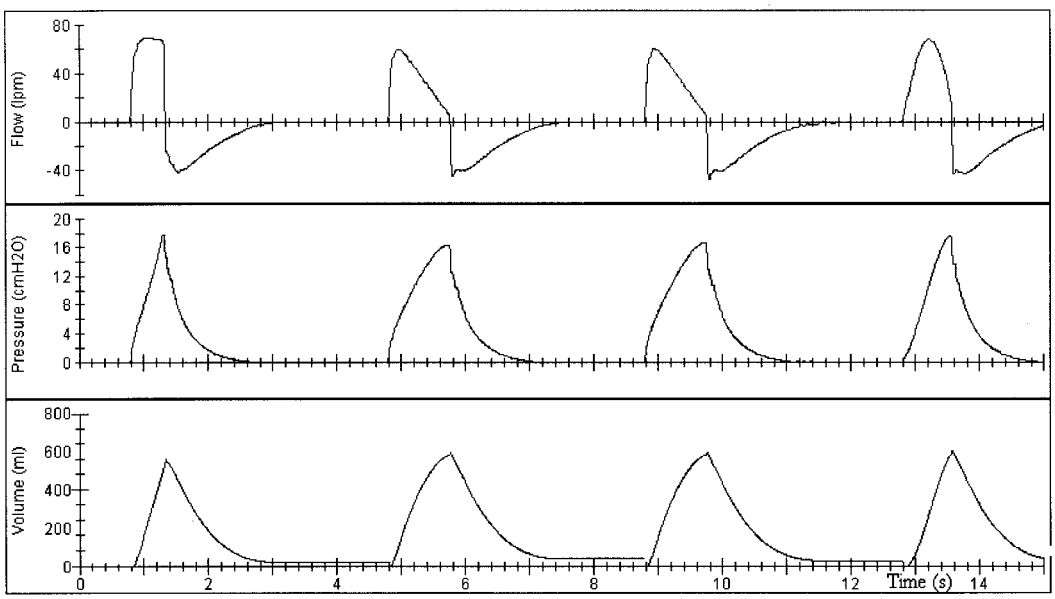

Fig. 9. Volume-controlled ventilation waveforms, with a constant, descending ramp and sinusoid flow. In the 2 last situations, measuring resistance becomes useless, whereas estimating the P2 value (see text) remains clinically valid.

inspiratory time. During PCV the flow wave in Figure 8 has the characteristic descending shape that reaches zero at the third breath, with a prolongation of the inspiratory time. In that case the working pressure set on the ventilator corresponds to the alveolar pressure, which is only linked to the elastic component of the system, as the elastic component is zero, because of the absence of flow. Furthermore, an additional amount of volume is obtained, as com-

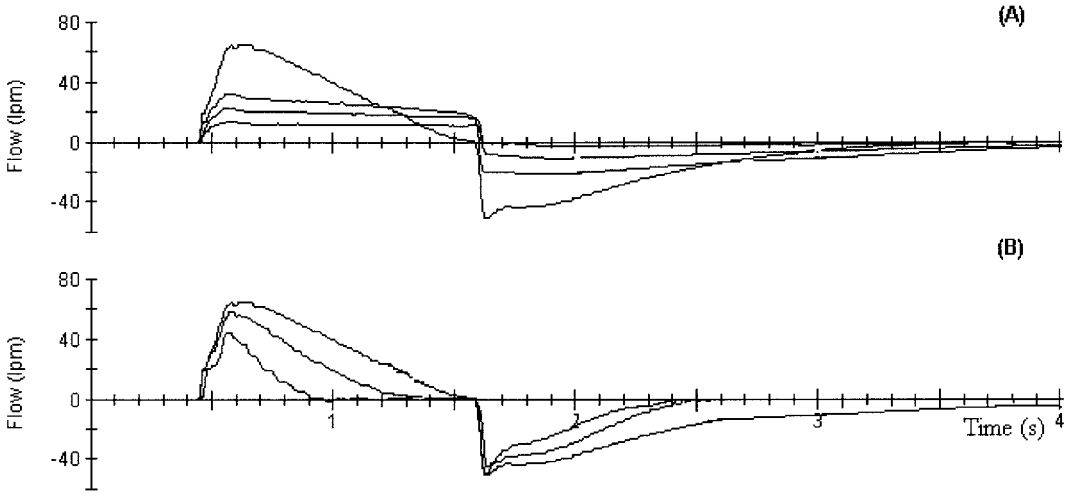

Fig. 10. Different flow shapes at different mechanical loads during pressure-controlled ventilation. A: Resistance variations. B: Compliance variations.

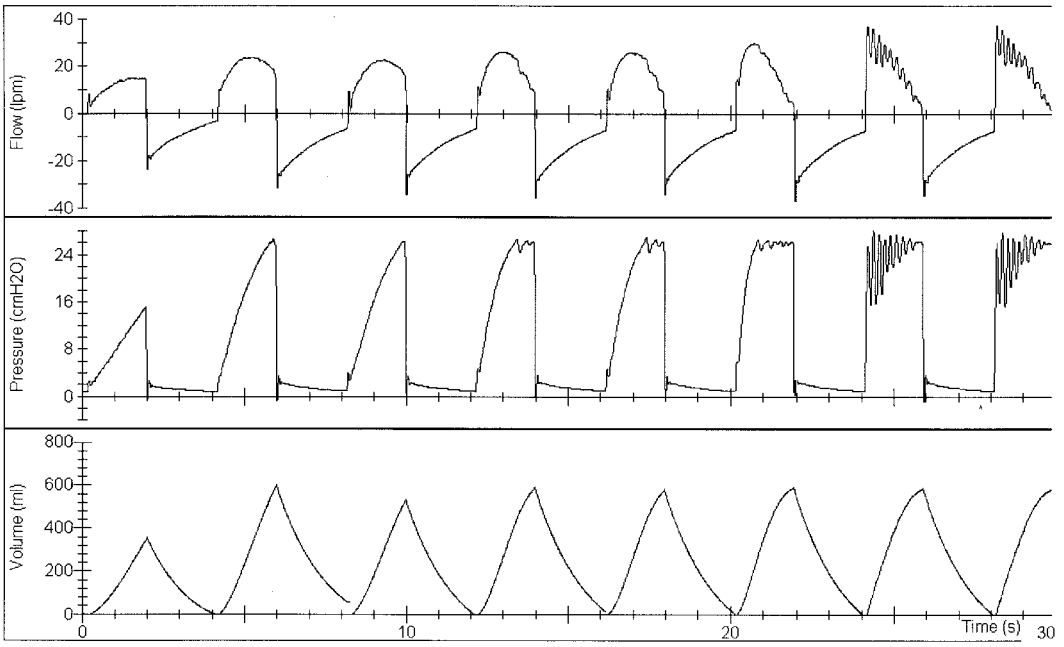

Fig. 11. The first cycle is characterized by slow attainment of the set working pressure (low slope) that stops at 16 cm H_2O, causing hypoventilation. Set working pressure (25 cm H_2O) is reached by increasing pressurization in the following breath. Dampening waves are recognizable in the last cycle.

pared to the 2 first breaths, where the inspiratory flow is truncated. The respective volume waveforms show the volume increases. Further prolonging the inspiratory pause decreases the expiratory time, which leads to gas-trapping.[22]

In the inspiratory flow waveform, 2 phases can be observed: the initial peak, which corresponds to the system pressurization, and a second segment that descends with a variable slope. The latter is a function of both the respiratory system compliance and the resistance. In Figure 10A, the flow wave varies as resistance increases and the compliance remains constant. As the resistive load increases, the flow loses its characteristic descending aspect and becomes almost constant. On the other hand, when compliance decreases as the resistive load remains stable, the morphologic features of the flow wave are not altered, but the wave is interrupted early, which leads to a decrease in the tidal volume (see Fig. 10 panel B).

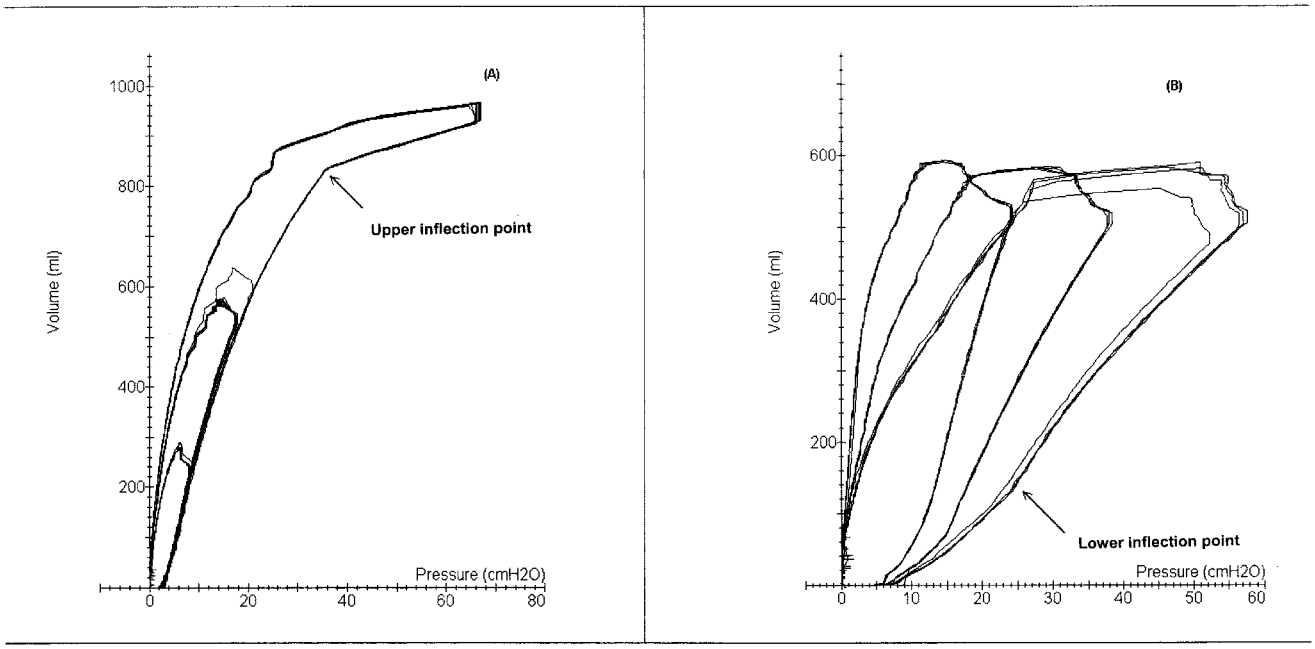

Fig. 12. Pressure-volume loops from volume-controlled ventilation. A: Pressure increase related to the volume increase, with identification of the upper inflection point. B: Pressure increase related to the decrease in compliance. On the inspiratory limb, the lower inflection point (arrow) occurs at higher pressure as the compliance decreases.

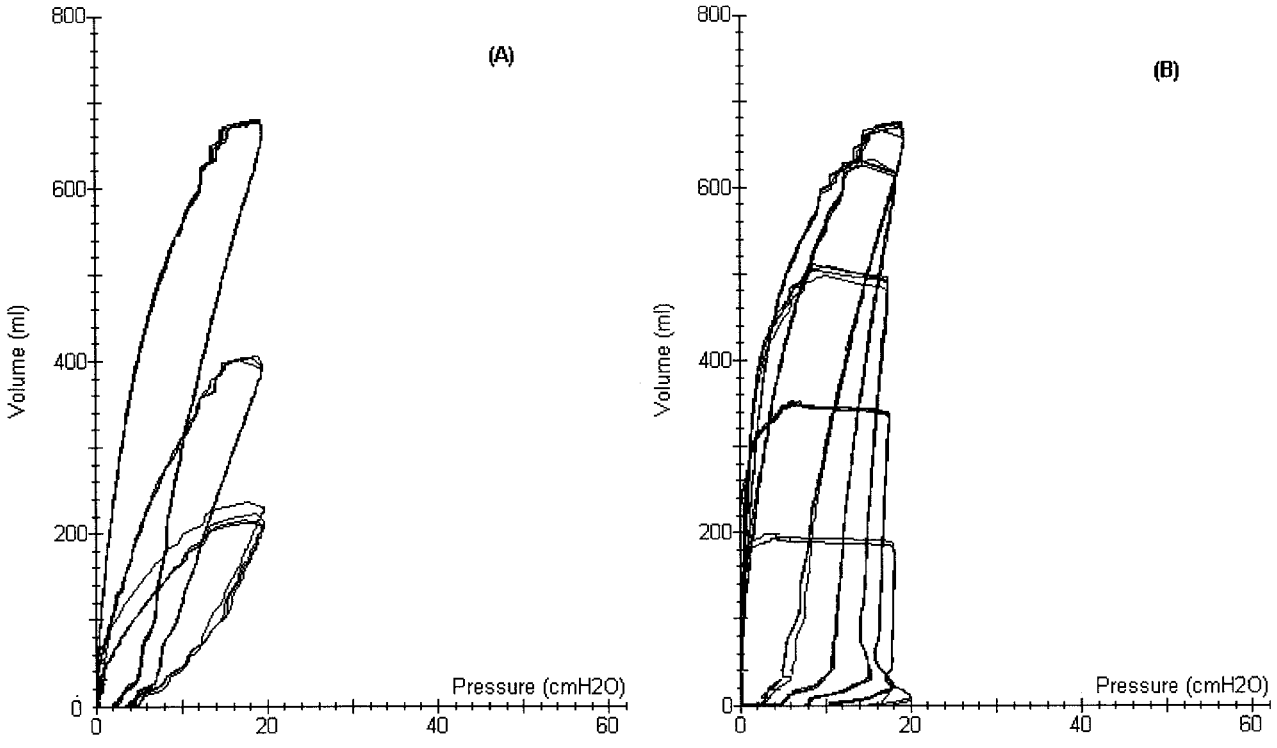

Fig. 13. Pressure-volume loops from pressure-controlled ventilation. A: Changes in loop caused by changes in compliance. B: Changes in loop caused by changes in resistance.

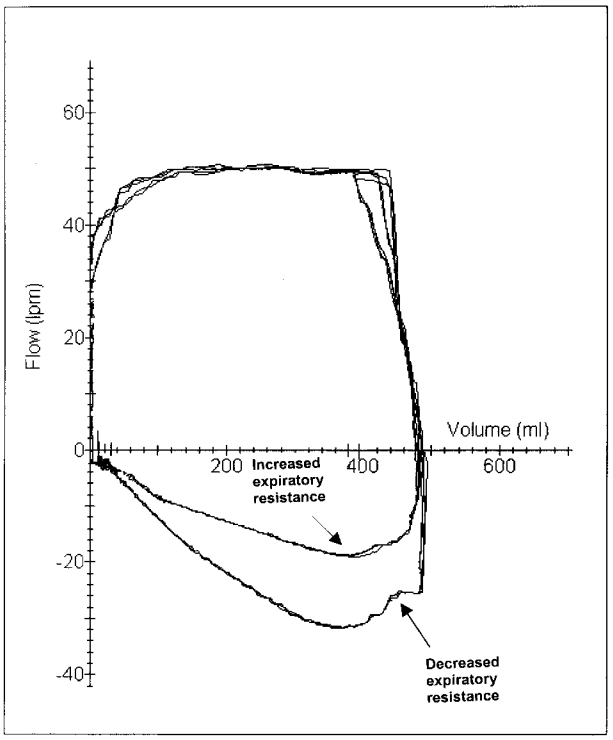

Fig. 14. The effect of resistance is only evidenced on the expiratory limb of the flow-volume loop, with a decrease in the expiratory flow peak, without affecting the inspiratory flow pattern.

Pressure-controlled ventilation strategies, such as PCV and pressure-support ventilation, depend on the rapid achievement of the working pressure set on the supplying system. Modern ventilators allow a variation in the slope of the pressure curve. Figure 11 shows that during PCV at 25 cm H_2O a slow attainment of the set working pressure does not guarantee an adequate volume. Increasing the rate of pressurization creates the characteristic "square wave" pressure curve. An exaggerated pressurization may be evidenced by the presence of dampening waves during the remaining inspiratory time.[23]

Pressure-Volume and Flow-Volume Loops

Pressure-volume (P-V) and flow-volume (F-V) loops provide information on the dynamic trend of, respectively, the respiratory system compliance and resistance. In particular, the P-V loop is useful for detecting lung overdistention during constant-flow VCV. As shown in Figure 12A, increasing V_T to $>$ 800 mL produces a sharp change in the slope of the inspiratory P-V curve. That bend in the curve is known as the *upper inflection point*, above which small volume increases produce large pressure increases.

The difference in location of the 3 loops in Figure 12B is a consequence of the change in the respiratory system compliance, in isovolumetric conditions, as evidenced by the rightward shift of the loop.

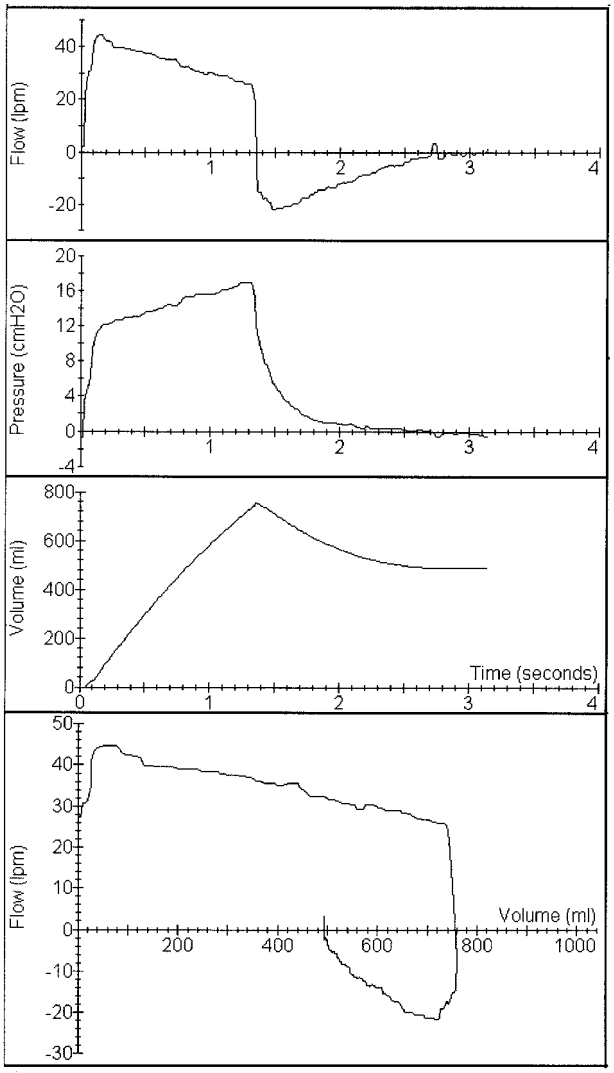

Fig. 15. Air leak is evidenced by the failed closure of the flow-volume loop (bottom panel) and by failure to reach zero on the expired volume curve.

During PCV the P-V loop provides a real-time visualization that the V_T depends on compliance and on resistance. In this case the supplied volume changes, while the working pressure remains constant (Fig. 13).

F-V loops are particularly important for assessing the response to bronchodilator administration during VCV (Fig. 14). F-V loops enable clinical analysis of the therapeutic effect.[24–26] The F-V loop is also useful for identifying air leaks that would not be identified with dynamic monitoring of the flow and pressure curves. In Figure 15 the F-V loop is incomplete because of early truncation of the expiratory phase, caused by a loss in volume.

When the loops are used to monitor respiratory function, it is clinically useful to freeze a baseline curve to use as a reference for following modifications.

Summary

Thanks to new technologies, modern ventilators allow complete monitoring of respiratory mechanics, which must become a guideline for optimizing and personalizing ventilatory support, in order to avoid the risk of barotrauma and volutrauma.

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Discussion

Harris: I have a difficult time with the concept of static compliance without knowing absolute lung volume. You can imagine a situation where, for instance, the static compliance is low and you happen to be ventilating a patient, perhaps with a high lung volume, and they're on the descending limb of the P-V curve. Or take the opposite situation, where you have a pneumothorax, and you are ventilating a very low lung volume, and the compliance is also low. Those are 2 very different situations, and yet that number may look very similar, so I have a difficult time interpreting it.

Do you think static compliance is a useful number without knowing what the lung volume is?

Blanch: Static compliance reflects the behavior of the total respiratory system. Assessment of regional lung volume is not possible at the bedside. In ARDS [acute respiratory distress syndrome] different lung regions have different mechanical properties and different time constants, and some regions might be collapsed or consolidated. Therefore, it is rather difficult to set the ventilator according to the compliance. I think there are other simple variables that we need to look at; one of the most important is airway plateau pressure. With a patient who has ARDS, plateau pressure higher than 30–32 cm H_2O invariably causes overdistension in some lung regions. Interestingly, some investigators have found overdistended areas at end-expiration with PEEP of 12 cm H_2O, perhaps reflecting overpressurized trapped gas. A relatively normal compliance measurement would reflect the ability to accept more tidal volume at reasonable plateau pressure. Whether or not some patients with moderate lung injury would benefit from this breathing pattern is a matter of debate.

MacIntyre: This question is probably going to come up multiple times in this conference, but I'll introduce it here on the first day. Are global measurements of compliance—or any other mechanical properties—really what we ought to be aiming for? In ARDS the lung is heterogeneously injured: some regions are very sick, while other regions, although not very healthy, are at least less sick. I'm afraid that a lot of times our measurements are only looking at what we're doing with the sick region. If we pop them open, overall compliance looks a lot better. But we're not so good at looking at what we're doing to the healthier regions of the lung when we try to open the sick regions.

The ARDS Network trial[1] was very eye-opening for me, because the small-tidal-volume group had the best mortality, but they also had the worst gas exchange and the worst compliance. I think that's a very important lesson for all of us—that these mechanical and gas exchange targets we've grown up with and spend a lot of time studying are only part of the story. Your presentation was excellent in reviewing the physiologic principles, but I think we've got to be really careful in looking at these global measurements of lung function and making the leap that they should be the guide to mechanical ventilation strategies.

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Blanch: I agree. My presentation was focused to explain the physiologic principles behind those measurements, although to set the ventilator accordingly remains difficult. I agree with you that ventilation with low PEEP and low tidal volume favors lung collapse, so compliance measurements are affected in the same direction, because less lung is available for ventilation. At the bedside we need a tool to assess whether there is alveolar over-stretching or repetitive closing-and-opening during each tidal breath. Looking at static P-V curves, it is easy to identify a pressure point beyond which overdistention occurs. However, in setting a PEEP level higher than the lower inflection point, clinicians cannot rule out the possibility of repetitive alveolar opening-and-closing.

Recent experimental research suggests that the shape of the pressure-time curve during volume-controlled ventilation at constant square flow is a useful tool to optimize mechanical ventilation. A straight pressure-time waveform during inspiration is associated with minimal lung injury, and this phenomenon was independent of the total lung volume. Perhaps this is what happened in the patients in the treatment arm in the ARDS Network study. A tidal volume of 6 mL/kg combined with a moderate PEEP did not allow both complete lung recruitment at end-expiration and tidal recruitment and derecruitment, thus avoiding further damage inflicted by positive-pressure ventilation.

Hess: Neil [MacIntyre] brought up a very important point, because there are, I think, 2 schools of thought about managing ARDS. One is, "If I keep the plateau pressure less than 30 cm H_2O, then everything is OK." The other is, "I should try to lower the tidal volume to 6 mL/kg and have a plateau pressure as low as possible." I think, Neil, that's what you were getting at, and I think Neil's point was that maybe there are some problems if we just say, "Let's just keep the plateau pressure less than 30 cm H_2O regardless of the tidal volume and everything will be OK."

MacIntyre: That was part of what I was getting at. However, I would like to challenge an even more fundamental concept, and that is this concept that if the radiograph or CT [computed tomogram] shows that we've recruited the lung units that were collapsed, then we've done a good thing. I'm challenging that assumption because it ignores the effect on the already-open units. Maybe—just maybe—we may have to "sacrifice" a few alveoli, if you will; let them stay collapsed because it protects the healthier regions of the lung from overdistention. Perhaps the concept and accepted standard of a wide-open, beautiful, black CT chest scan may in fact be masking regional overdistention of healthier units and producing a worse outcome.

Dhand: I think the question that was raised by Scott [Harris] is really the

central issue, because we don't know the lung volume at which we are ventilating a patient. If we knew that, then we would know where in the P-V curve we were and what the limitations are. Why is it so difficult for us to measure or even estimate what the lung volume is at which the patient is being ventilated? The plateau pressure is just a surrogate measurement. When we are giving a small tidal volume to a patient who is on the flat portion below the lower inflection point, then that might be harmful. Likewise, if the patient is at the very top of the P-V curve, then it could be harmful, so what we really need to know is where on that curve we are when ventilating the patient.

Blanch: I agree with you. We performed recruitment maneuvers in patients with ARDS and found that at high PEEP, recruitment maneuvers had little or no effect.[1] Interestingly, when the lung-volume increase induced by PEEP was high, a recruitment maneuver had little effect on oxygenation. Alveolar units not stabilized with high PEEP might be recruited at high airway pressure, although they are prompt to collapse unless very high PEEP levels are applied at end-expiration.

REFERENCE

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Sanborn: I wanted to come back to Neil's issue. It's an issue of compromise, it seems to me. If you believe the lung should be completely recruited—forgetting for the moment that we recognize primary and secondary ARDS—you could inflate the lung to maximum inspiratory capacity and make the CT scan all black, and we know that lung units would be overdistended. So by ventilating in the upper region of the P-V curve with high PEEP we are probably accepting some overdistention. If you choose to ventilate the lower, gravity-down region with low PEEP, then we are accepting that, with each inflation-deflation cycle, the consolidated lung units will undergo excessive and destructive shear stress. So it seems to me that somewhere in between the maximum-recruitment strategy and the conventional, low-PEEP strategy—which will most certainly subject *some* closed units to high stress as they open—lies an optimum strategy. The problem is that we don't know exactly where we are on the P-V curve. That's the big issue. There's no tool at the bedside you can pull out of your pocket that will say, "This is where I should be to satisfy that compromise."

MacIntyre: I agree with you completely. The point I was trying to make is exactly that—that we don't know where we are, and that this oversimplification of the CT scan or the steep part of the deflation limb of the curve as the ideal place to place the lung is, I think, just that—an oversimplification.

Hess: I am reminded of something I heard someone say a couple of years ago. We know that if we overdistend the lung, that's bad. We know if we allow alveoli to open and close, that's bad. But if it's closed and we just leave it alone, maybe that's OK. We just don't know.