

— Special Articles —

## Mean airway pressure: Physiologic determinants and clinical importance—Part 1: Physiologic determinants and measurements

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**Purposes:** To discuss the theoretical relationship of mean alveolar pressure to its most easily measured analog, the mean airway pressure, and to describe the key determinants, measurement considerations, and clinical implications of this index.

**Data Sources:** Relevant articles from the medical and physiologic literature, as well as mathematical arguments developed in this article from first principles.

**Study Selection:** Theoretical, experimental, and clinical information that elucidates the physiologic importance, measurement, or adverse consequences of mean airway pressure.

**Data Extraction:** Mathematical models were used in conjunction with data from the published literature to develop a unified description of the physiological and clinical relevance of mean airway pressure.

**Synthesis:** Geometrical and mathematical analyses demonstrate that shared elements comprise mean airway pressure and mean alveolar pressure, two variables that are related by the formula: mean alveolar pressure = mean airway pressure +  $(\dot{V}_E/60) \times (R_E - R_I)$ , where  $\dot{V}_E$ ,  $R_E$ , and  $R_I$  are minute ventilation and expiratory and inspiratory resistances, respectively. Clear guidelines can be developed for selecting the site of mean airway pressure determination, for specifying technical requirements for mean airway pressure measurement, and for delineating clinical options to adjust the level of mean airway pressure. Problems in viewing mean airway

pressure as a reflection of mean alveolar pressure can be interpreted against the theoretical basis of their interrelationship. In certain settings, mean airway pressure closely relates to levels of ventilation, arterial oxygenation, cardiovascular function, and barotrauma. Because mean airway pressure is associated with both beneficial and adverse effects, a thorough understanding of its theoretical and practical basis is integral to formulating an effective pressure-targeted strategy of ventilatory support.

**Conclusions:** Mean airway pressure closely reflects mean alveolar pressure, except when flow-resistive pressure losses differ greatly for the inspiratory and expiratory phases of the ventilatory cycle. Under conditions of passive inflation, mean airway pressure correlates with alveolar ventilation, arterial oxygenation, hemodynamic performance, and barotrauma. We encourage wider use of this index, appropriately measured and interpreted, as well as its incorporation into rational strategies for the ventilatory management of critical illness. (Crit Care Med 1992; 20:1461-1472)

**KEY WORDS:** mechanical ventilation; monitoring; hemodynamics; barotrauma; work of breathing; critical care; positive end-expiratory pressure; tidal volume; pulmonary emergencies

Few concepts relate more closely to the clinical effects of mechanical ventilation than mean alveolar pressure. Mean alveolar pressure relates intimately to ventilation, arterial oxygenation, cardiovascular function, and ventilator-induced barotrauma. Despite its central importance, mean alveolar pressure is generally neglected in clinical practice; this variable is often incompletely understood and seldom estimated outside of the research setting. The primary purpose of this article is to define the relationship of mean alveolar pressure to its most easily measured analog, mean airway pressure, and to describe how mean alveolar pressure and mean airway pressure can be assessed

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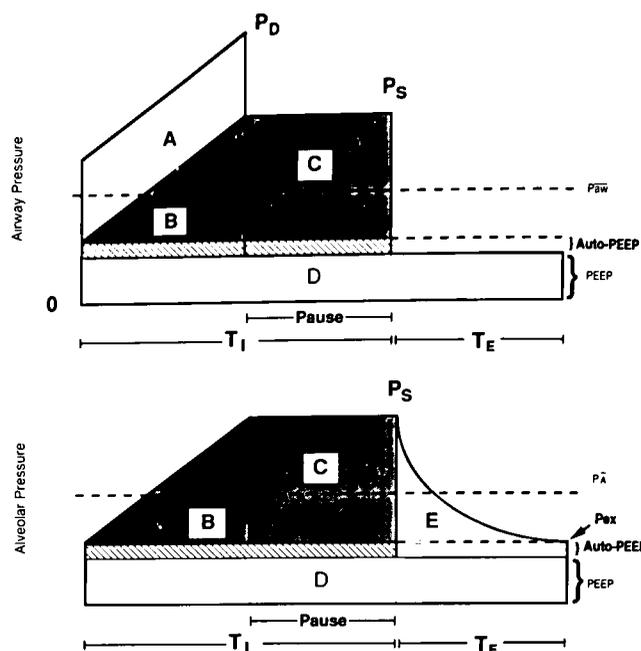
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and adjusted to achieve important clinical objectives. Better appreciation of these important variables should be encouraged, since microprocessor-based equipment now in widespread use displays mean airway pressure continuously and can provide data essential for mean alveolar pressure estimation. Part 1 of this two-part article concentrates on theory and measurement. Part 2 reviews the clinical implications of the mean airway pressure measurement. Unless otherwise noted, this discussion assumes that inflation occurs passively by the cyclic application of positive pressure.

### COMPONENTS OF MEAN AIRWAY AND MEAN ALVEOLAR PRESSURES

**Mean Airway Pressure.** Mean airway pressure is measured near the airway opening and is averaged over the entire ventilatory cycle. If mean airway pressure is not automatically assessed by the ventilator, airway pressure can be tapped in the connecting tubing of the external circuit, near the endotracheal tube. Unless positive end-expiratory pressure is applied, mean airway pressure is influenced solely by changes in the inspiratory pressure waveform or duty-cycle length. Ideally, the expiratory valve opens widely to allow unimpeded deflation, so that the pressure in the external circuit remains nearly atmospheric (0 cm H<sub>2</sub>O) throughout exhalation. Any external positive end-expiratory pressure adds to atmospheric pressure during exhalation, thereby contributing to mean airway pressure. Figure 1 (top) is a schematic airway pressure tracing for volume-cycled, constant-flow ventilation, which depicts all frictional and elastic elements that affect mean airway pressure (flow resistive, tidal elastic, end-inspiratory pause pressures, auto-positive end-expiratory pressure, and positive end-expiratory pressure). Numerically, mean airway pressure is the quotient of the area under the airway pressure-time tracing, divided by total cycle duration.

**Mean Alveolar Pressure.** In theory, tracings for mean alveolar pressure and mean airway pressure should differ in three respects (Fig. 1, bottom). First, mean alveolar pressure is not influenced by the frictional inspiratory airway pressure component, which dissipates proximal to the alveolus (Fig. 1, area A). Secondly, because alveolar pressure drives expiratory flow, mean alveolar pressure *does* include an expiratory flow resistive component (Fig. 1, area E). Finally, unlike mean airway pressure, auto-positive end-expiratory pressure adds to mean alveolar pressure throughout the entire respiratory cycle. Because mean airway pressure and mean alveolar pressure share many components, mean airway pressure closely approximates mean alveolar pressure under certain conditions. However, this identity is not consistent; at times, mean airway pressure and mean alveolar pressure may differ significantly.



**Figure 1.** Schematic drawings of an idealized pressure tracing for one respiratory cycle during constant flow mechanical ventilation. *Top:* A, nonelastic (frictional); B, tidal elastic; C, pause; D, positive end-expiratory pressure (PEEP) component. The cross-hatched area corresponds to auto-positive end-expiratory pressure (auto-PEEP). Note that auto-PEEP adds to airway pressure only during inspiration, since airway pressure decreases to the set PEEP level during exhalation. *Bottom:* A, B, C, D, and cross-hatched areas are as defined above; E, expiratory frictional component.  $P_D$ , peak dynamic pressure;  $P_S$ , peak static pressure;  $P_{\bar{a}w}$ , mean airway pressure;  $P_{\bar{a}}$ , mean alveolar pressure;  $P_{ex}$ , end-expiratory alveolar pressure;  $T_I$ , inspiratory time;  $T_E$ , expiratory time.

### HOW ARE MEAN AIRWAY AND MEAN ALVEOLAR PRESSURE RELATED?

In the medical literature, the term "mean airway pressure" is often considered synonymous with mean alveolar pressure. However, the validity of this assumption depends on accurate pressure measurement and equivalence between inspiratory and expiratory flow-resistive pressure losses. For the discussion that follows, the units are: flow (L/sec), compliance (L/cm H<sub>2</sub>O), resistance (cm H<sub>2</sub>O·sec/L), pressure (cm H<sub>2</sub>O), and tidal volume (L). Total airway and alveolar pressures are considered the sum of two components: pressure conserving (elastic) and pressure dissipating (non-elastic). The nonelastic or frictional portion (also termed the "resistive" component) includes all sources of dissipated pressure. Inertance, viscoelastance, pendelluft, and true airway resistance are not separately considered. Three simplifying assumptions are also made. First, unique values can be specified for inspiratory and expiratory resistances. This assumption implies either purely laminar flow, or the ability to specify a lumped constant (inspiratory or expiratory resistances) that

represents the average quotient of the nonelastic pressure/flow for the relevant portion of the ventilatory cycle. Except where otherwise noted, inspiratory and expiratory resistances are assumed to be flow-independent in the following discussion. Second, the static pressure-volume curve of the respiratory system does not demonstrate hysteresis. Third, the system is self-contained; all gas enters and exits via the airway opening. Inspiratory and expiratory tidal volumes are equal.

*Inflation-Deflation Cycle.* When the passive respiratory system is mechanically inflated, pressure is cyclically applied and released from the airway opening. During inflation, the applied pressure drives gas to the alveolar level and expands the lungs and chest wall against their combined recoil. In this closed system, the applied pressure must always equal the sum of the elastic counterpressure within the alveolus (Fig. 1, *top and bottom, area B*) and the pressure required to overcome flow impedance (Fig. 1, *area A*). (A similar principle applies during spontaneous breathing, where the difference between atmospheric and intrapleural pressures drives flow and expands the passive lung.) Three components comprise total elastic pressure. The first component relates to any end-expiratory residual pressure ( $P_{ex}$ ) distending the alveolus relative to the equilibrium position (where alveolar as well as airway opening pressures would be zero). The second component is the additional elastic pressure that accrues during tidal inflation. The third component is the elastic pressure added by the application of an end-inspiratory pause. At any moment, the difference between instantaneous alveolar pressure and end-expiratory alveolar pressure (the sum of positive end-expiratory pressure and auto-positive end-expiratory pressure) defines the tidal elastic pressure. The pressure dissipated against resistance (the nonelastic or "frictional" component) accounts for the entire difference between the pressure applied at the airway opening and the simultaneous alveolar pressure.

During passive exhalation, alveolar pressure serves both to counterbalance lung and chest recoil and to drive expiratory flow. Assuming no expiratory valve resistance distal to the site of measurement, airway pressure theoretically remains constant during expiration at atmospheric pressure or positive end-expiratory pressure (PEEP). The entire pressure difference existing across the system during exhalation ( $P_A$ ) must be accounted for in "frictional" losses ( $P_A - PEEP$ ) and residual pressure (PEEP). The  $P_A - PEEP$  difference approximates the pressure dissipated across the flow resistance of the airways, endotracheal tube, and external circuitry during exhalation (Fig. 1, *area E*). However, note that  $P_A$  is the total pressure (flow-

resistive pressure plus positive end-expiratory pressure) pushing gas through the circuit when expiratory flow is under way. End-expiratory alveolar pressure may be higher than the set level of end-expiratory pressure (positive end-expiratory pressure) when dynamic hyperinflation drives flow until the exact point of expiratory valve closure, creating a component of auto (or intrinsic) positive end-expiratory pressure (Fig. 1, *bottom*) (1, 2).

*Relationship Between Airway and Alveolar Pressures During Passive Ventilation.* A pressure difference represents a gradient of potential energy per unit volume. In a closed system, the first law of thermodynamics (energy can neither be created nor destroyed) ensures that any potential energy difference will convert to another form of energy (kinetic energy or heat) when an open pathway is established between the two points under consideration (3). It follows that all pressure applied to the airway opening must be accounted for as dissipated (flow-resistive) pressure or as elastic pressure conserved in the alveolus. The simplified equation of motion for the respiratory system concisely expresses this principle of pressure accounting (4). When a pressure difference (airway pressure [ $P_{aw}$ ]) is applied cyclically across the respiratory system, the potential energy represented by airway pressure must appear in the form of its total elastic ( $P_{EL}$ ) or resistive ( $P_R$ ) equivalents:

$$\begin{aligned} P_{aw} &= P_{EL} + P_R \\ P_{aw} &= P_{EL} + \dot{V}R \\ P_{aw} &= \left[ \frac{V_I}{C} + P_{EX} \right] + \dot{V}R \end{aligned}$$

Here,  $C$ ,  $R$ ,  $\dot{V}$ ,  $P_{EX}$ , and  $V_I$  indicate compliance, resistance, flow, end-expiratory alveolar pressure, and volume inspired relative to the end-expiratory position, respectively.  $V_I/C$  (the "tidal elastic" pressure) is the elastic pressure associated with any lung volume ( $V_I$ ) above the end-expiratory position, and the product of flow and resistance ( $\dot{V}R$ ) is the frictional (dissipated) component of airway pressure. Because the potential energy of the system eventually returns to its end-expiratory value, the energy stored during tidal expansion (the quotient of tidal volume and compliance) must fully dissipate against the frictional impedance of expiration.

Tracings of airway opening and alveolar pressures can be viewed as records of the changes in potential energy per unit volume that occur at those sites during the respiratory cycle. When divided by total cycle time ( $T_T$ ), the integrals of these time-based pressure records (areas under the pressure-time tracings) define the respective mean pressures. During the inspiratory

period, the integrals of residual, tidal elastic, and pause pressures are common to both airway opening and alveolus (Fig. 1). During exhalation, any set level of end-expiratory airway pressure (positive end-expiratory pressure) is reflected in both the alveolar and airway compartments. The frictional pressure-time integral of inspiration (Fig. 1, *area A*) is an element of the total airway pressure-time area, whereas the frictional pressure-time integral of exhalation (Fig. 1, *area E*) comprises the entire alveolar pressure-time area that exceeds central airway pressure at the point of measurement. (Assuming negligible expiratory valve resistance, pressure in the central airway is positive end-expiratory pressure throughout exhalation. The effect of valve resistance on airway pressure and alveolar pressure is considered below.) Therefore, from a purely geometrical standpoint, the mean pressures in the alveolus and central airway (averaged over the entire cycle) must be equal if the frictional pressure-time integrals of inspiration and expiration can be shown to be equivalent (Fig. 1, *area A* and *area E*).

In a system with linear pressure-flow characteristics, inspiratory and expiratory resistances are flow-independent. It follows that the frictional (flow-resistive) component of the airway pressure-time integral for inspiration (Fig. 1, *area A*) is the product of tidal volume ( $V_T$ ) and the constant inspiratory resistance ( $R_I$ ):

$$\begin{aligned} \int_0^{T_I} P_{Rdt} &= \int_0^{T_I} (P_{aw} - P_A) dt \\ &= \int_0^{T_I} R_I \dot{V} dt \\ &= R_I \int_0^{T_I} \dot{V} dt \\ &= R_I V_T \end{aligned}$$

Here  $T_I$  is inspiratory time,  $P_R$  represents the resistive pressure, and  $\dot{V}$  is flow. Similarly, the frictional pressure-time integral for expiration (Fig. 1, *area E*) is the product of tidal volume ( $V_T$ ) and expiratory resistance ( $R_E$ ):

$$\begin{aligned} \int_{T_I}^{T_E} P_{Rdt} &= \int_{T_I}^{T_E} P_A dt \\ &= \int_{T_I}^{T_E} R_E \dot{V} dt \\ &= R_E \int_{T_I}^{T_E} \dot{V} dt \\ &= R_E V_T \end{aligned}$$

In these expressions,  $R_I$  and  $R_E$  refer to resistance in the pathway connecting the sites of airway pressure and alveolar pressure measurement, and  $T_I$  and  $T_E$  correspond to the inspiratory and expiratory times, respectively. Assuming that inspiratory and expiratory tidal volumes are identical, the ratio of frictional pres-

sure-time integrals for inspiration and expiration is simply the ratio of their respective resistances:

$$\frac{\int_0^{T_I} P_{Rdt}}{\int_{T_I}^{T_E} P_{Rdt}} = \frac{V_T R_I}{V_T R_E} = \frac{R_I}{R_E}$$

It follows that mean alveolar pressure must equal mean airway pressure when inspiratory and expiratory resistances are equal. (Although flow is not a precisely linear function of driving pressure in practice, the ratio of mean flow-driving pressure to mean flow does quantify the average pressure dissipated per unit of flow and, in this sense, characterizes "average" resistance.)

*Effect of Inspiratory and Expiratory Resistance on the Relationship Between Mean Alveolar and Mean Airway Pressures.* The following analysis demonstrates that the difference between mean alveolar pressure and mean airway pressure is the product of minute ventilation and the difference between the expiratory and inspiratory resistances of the path that connects the sites of pressure measurement. For example, the difference between mean alveolar pressure and the mean airway pressure measured at the airway opening can be computed by considering their mean inspiratory (mean inspiratory alveolar pressure [ $\bar{P}_{A_I}$ ], mean inspiratory airway pressure [ $\bar{P}_{aw_I}$ ]), expiratory (mean expiratory alveolar pressure [ $\bar{P}_{A_E}$ ], and mean expiratory airway pressure [ $\bar{P}_{aw_E}$ ]) components:

#### Mean Alveolar Pressure

$$\begin{aligned} \bar{P}_A &= \frac{\int_0^{T_T} P_A dt}{T_T} = \bar{P}_{A_I} + \bar{P}_{A_E} \\ \bar{P}_A &= \frac{(\int_0^{T_I} P_A dt)}{T_T} + \left[ \frac{R_E V_T}{T_T} + PEEP \left( \frac{T_E}{T_T} \right) \right] \quad [1] \end{aligned}$$

#### Mean Airway Pressure

$$\begin{aligned} \bar{P}_{aw} &= \frac{\int_0^{T_T} P_{aw} dt}{T_T} = \bar{P}_{aw_I} + \bar{P}_{aw_E} \\ \bar{P}_{aw} &= \frac{(R_I V_T + \int_0^{T_I} P_A dt)}{T_T} + PEEP \left( \frac{T_E}{T_T} \right) \\ \bar{P}_{aw} &= \left[ \frac{R_I V_T}{T_T} + \frac{\int_0^{T_I} P_A dt}{T_T} \right] + PEEP \left( \frac{T_E}{T_T} \right) \quad [2] \end{aligned}$$

Using these expressions for mean alveolar pressure (Eq. 1) and mean airway pressure (Eq. 2)—with

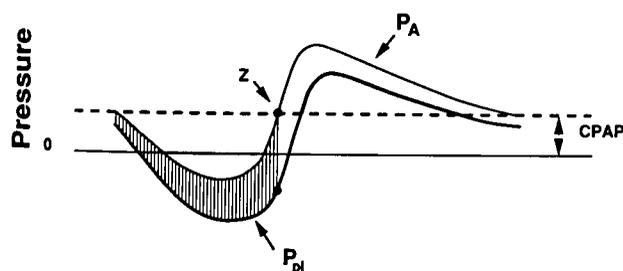
frequency expressed in cycles/min, minute ventilation ( $\dot{V}_E$ ) in L/min, and total cycle time ( $T_T$  in seconds ( $T_T = 60/\text{frequency}$ ))—the difference in mean pressures can be calculated as follows:

$$\begin{aligned} (\bar{P}_A - \bar{P}_{aw}) &= \frac{R_E V_T}{T_T} - \frac{R_I V_T}{T_T} = \frac{V_T (R_E - R_I)}{T_T} \\ &= \frac{V_T f (R_E - R_I)}{60} \\ (\bar{P}_A - \bar{P}_{aw}) &= \frac{\dot{V}_E}{60} (R_E - R_I) \end{aligned} \quad [3]$$

This useful relationship (Eq. 3) holds whenever linear pressure-flow kinetics apply to both inspiration and expiration, whether or not dynamic hyperinflation or an end-inspiratory pause is present.

*Relationship of Mean Airway and Alveolar Pressures During Spontaneous Breathing.* Similar principles apply to the mechanics of spontaneous breathing (Fig. 2). However, mean airway pressure and mean alveolar pressure both theoretically equal the pressure measured at the airway opening (atmospheric pressure or any set level of end-expiratory pressure), provided that inspiratory and expiratory resistances are equal and the same simplifying assumptions apply as previously outlined. This theoretical equivalence holds equally as well for instances in which there is vigorous expiratory muscular activity as for those instances in which exhalation is passive, provided that expiratory resistance and the exhaled tidal volume are not changed by the effort. In the case of spontaneous breathing, therefore, mean airway pressure provides no index of the effort or pressure cost of breathing.

*Estimating Inspiratory and Expiratory Resistance.* As Equation 3 indicates, mean alveolar pressure may



**Figure 2.** Schematic comparison of pressures at the airway opening (dashed line), alveolus ( $P_A$ ; fine solid line), and pleural space ( $P_{pl}$ ; heavy solid line) during a spontaneous breath with continuous positive airway pressure (CPAP). Shaded areas represent frictional pressure losses. The transition between inspiration and expiration occurs at the zero flow point (Z), where alveolar and airway opening pressures are equal. The difference between  $P_{pl}$  and  $P_A$  (cross-hatched area) corresponds to the inspiratory tidal elastic pressure. Mean alveolar pressure approximates that pressure of the airway opening (CPAP).

significantly exceed mean airway pressure when expiratory resistance exceeds inspiratory resistance, particularly if minute ventilation is high. Dynamic airway compression may accentuate any innate difference between the flow-resistive properties of inspiration and expiration. In actuality, the frictional pressure/flow ratio can seldom be typified by a single number that pertains to all flows observed during inflation or deflation. However, the inspiratory and expiratory resistance inputs for Equation 3 can also be considered mean (lumped or composite) values that indicate the quotients of mean nonelastic pressures and mean flow rates. Under such conditions, Equation 3 remains qualitatively instructive, if not quantitatively precise.

In clinical practice, the resistance measured at end-inspiration ( $[\text{peak pressure} - \text{static pressure}]/\text{flow}$ ) is usually a good reflection of the average inspiratory resistance value during passive inflation with constant flow. However, describing the flow-resistive properties of expiration is more problematic, as flow is a continuously changing function of exhaled volume. One approach is to use "stop flow" methods for the multiple point measures of alveolar pressure needed to fully characterize expiratory resistance (5). Whenever the chest empties quasi-exponentially, a "mean" value for expiratory resistance ( $R_E$ ) can also be approximated from the quotient of the measured values of the unexponential time constant ( $\tau$ ) and respiratory system compliance ( $C$ ) (6):  $R_E = \tau/C$ . (The  $R_E C$  product,  $\tau$ , is the time required to expire to  $1/e$  [or 37%] of the tidal elastic pressure.) Although the deflation time constant is usually evaluated by analyzing an exhaled spirogram, it is better assessed from sequential stop-flow measurements of alveolar pressure when dynamic hyperinflation is present (7).

*Effect of Hysteresis on the Mean Airway to Mean Alveolar Pressure Relationship.* Given the powerful and direct influence of the resistance relationship to the accuracy of mean airway pressure in estimating mean alveolar pressure, it is interesting to note that the precise contours of the alveolar pressure-volume curve and flow pattern, as well as the presence of hysteresis, do not (in theory) affect the validity of mean airway pressure as a reflection of mean alveolar pressure. As Equation 3 predicts, discrepancies between mean airway pressure and mean alveolar pressure are not influenced by tissue elastic properties. Whatever contour the alveolar pressure tracing might assume, the time-averaged amount by which alveolar pressure exceeds airway pressure during exhalation must equal that amount by which airway pressure exceeds alveolar pressure during inspiration, adjusted for any differences in resistance between them.

## CLINICAL OPTIONS FOR INCREASING MEAN AIRWAY PRESSURE

For illustrative purposes, the inflation/deflation cycle can be divided into three portions: a) the inflation segment—during which alveolar volume steadily accumulates; b) the end-inspiratory plateau (inspiratory hold or pause) segment—during which expiratory flow is prevented and end-inflation pressure is held; and c) the deflation segment—spanning the period of expiratory flow (Fig. 1). Apart from changes in respiratory system impedance (resistance and compliance), several setting options are available during volume-controlled and pressure-preset ventilation to increase or decrease mean airway pressure and mean alveolar pressure.

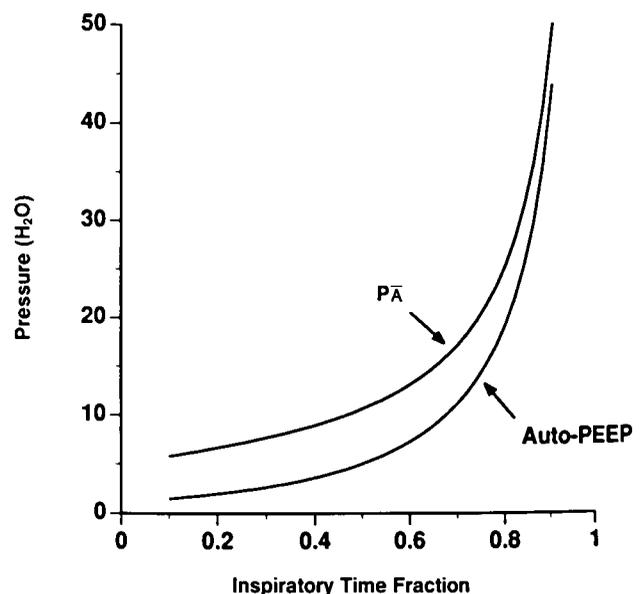
**Volume-Cycled Ventilation.** The inspiratory duty cycle (inspiratory time fraction) is the ratio of minute ventilation to inspiratory flow rate. Therefore, once minute ventilation is determined, setting either the mean inspiratory flow rate or duty cycle determines the other variable. Conversely, once inspiratory duty cycle or inspiratory flow rate is selected, variations in minute ventilation may influence the calculated values of the "unset" variable. In some machines currently being used, duty cycle is a selected setting; in others, inspiratory flow rate is chosen.

**Influence of Tidal Volume on Mean Airway Pressure and Mean Alveolar Pressure.** An increase in tidal volume will increase mean airway pressure and mean alveolar pressure by increasing the tidal elastic pressure requirement (Fig. 1, top and bottom, area B). The tidal elastic pressure required to deliver a given tidal volume is determined by the compliance of the respiratory system. Depending on the rate of inspiratory flow, area B may enlarge, and auto-positive end-expiratory pressure will develop if expiratory time is insufficient to allow for full deflation of the increased volume.

**Influence of Frequency on Mean Airway Pressure and Mean Alveolar Pressure.** With tidal volume held constant, increases in frequency influence mean airway pressure and mean alveolar pressure, primarily through associated alterations in the end-expiratory alveolar pressure, which is produced by dynamic hyperinflation and caused by shortening of expiratory time. The effect of increasing frequency depends strongly on any associated change in the inspiratory time fraction. For example, lung pressures can increase dramatically at high frequencies if inspiratory flow rate is held constant (while the inspiratory time fraction lengthens), especially if an unvarying end-inspiratory pause has been applied. Conversely, the effect of increasing frequency will be modest if the inspiratory time fraction remains unchanged (while inspiratory flow increases). Even before the induction of auto-positive end-expiratory

pressure, increasing frequency increases mean airway pressure due to shortening of total cycle time. Since mean pressure is calculated by dividing the pressure-time area by the total cycle time, a decrease in total cycle time increases both mean airway pressure and mean alveolar pressure.

**Influence of Flow Rate or Inspiratory Time Fraction on Mean Airway Pressure and Mean Alveolar Pressure.** At fixed values for frequency and tidal volume, variations of the mean inspiratory flow rate ( $V_T/T_I$ ) alter the mean inspiratory airway pressure by affecting the magnitude of the dissipated pressure loss and, at slower flows, by the development of auto-positive end-expiratory pressure. Although slowing the average rate of inspiratory flow invariably lengthens the inspiratory time fraction and increases mean airway pressure and mean alveolar pressure, it does so in an alinear, hyperbolic fashion. As the inspiratory time fraction increases, mean airway pressure increases slowly until the time allowed for exhalation is short enough to produce clinically important dynamic hyperinflation. Auto-positive end-expiratory pressure and mean airway pressure then increase dramatically with further increases in the duty cycle (Fig. 3). Even when both tidal volume and frequency are fixed (constant minute ventilation), small variations of the inspiratory flow setting made over a critical range can produce large changes in



**Figure 3.** The relationship of mean alveolar pressure ( $P_A$ ) and auto-positive end-expiratory pressure (Auto-PEEP) to inspiratory time fraction during volume-cycled ventilation. With tidal volume held constant,  $P_A$  increases as a hyperbolic function of the inspiratory time fraction, driven by the generation of auto-PEEP. With tidal volume fixed, a similar hyperbolic relationship holds for all other inspiratory flow profiles. (Computer simulation for tidal volume = 1 L; frequency = 20 cycles/min; inspiratory and expiratory resistances = 15 cm H<sub>2</sub>O·sec/L; and compliance = 0.08 L/cm H<sub>2</sub>O.)

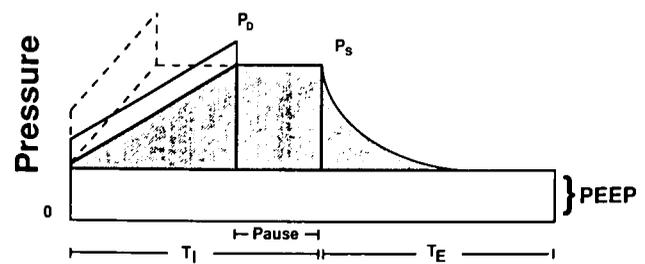
end-expiratory alveolar pressure and, therefore, in mean alveolar pressure. The sensitive flow region is easily entered when a high frequency or significant end-inspiratory pause contracts the time available for exhalation and the inspiratory time fraction lengthens. Although auto-positive end-expiratory pressure may develop at any inspiratory-to-expiratory ratio (depending on end-inspiratory volume, the expiratory time constant, and the duration of expiration), ratios that exceed 1:1 (inverse ratios) and high levels of minute ventilation are usually required to produce clinically important gas trapping in patients with acute lung injury.

*Influence of Pause, Positive End-Expiratory Pressure, and Inspiratory Flow Profile on Mean Airway Pressure and Mean Alveolar Pressure.* An end-inspiratory pause holds the lung volume and alveolar pressures achieved at end-inflation for the specified duration of the pause. Therefore, an end-inspiratory pause adds directly to mean airway pressure and mean alveolar pressure (Fig. 1, *top and bottom, area C*). A pause may further accentuate the mean pressure increase if it simultaneously causes the development of auto-positive end-expiratory pressure by curtailing expiratory time.

Positive end-expiratory pressure adds equally to mean airway pressure and mean alveolar pressure (Fig. 1). Alternative inspiratory flow profiles may alter mean airway pressure and mean alveolar pressure. For example, a decelerating flow profile, delivered in the same inspiratory time as an identical tidal volume delivered with constant flow, has a higher mean airway pressure and mean alveolar pressure, since a greater proportion of the average flow (and therefore, more volume and alveolar pressure) is delivered earlier in the inspiratory period. (In Figure 1, *area B* becomes larger.) Another option to increase mean airway pressure is to "square up" the inspiratory pressure (Fig. 4) by delivering the tidal volume more quickly, using a pause to maintain the same inspiratory time.

*Pressure-Preset Ventilation.* In pressure-preset (pressure-controlled) ventilation, tidal volume depends on the resistance and compliance of the respiratory system, as well as on the selected values of the applied airway pressure, cycling frequency, and inspiratory time fraction. With the applied pressure fixed, the relationships of duty cycle and frequency to mean airway and alveolar pressures are fundamentally different in comparison with those relationships during volume-cycled ventilation.

In pressure-preset ventilation, mean airway and mean alveolar pressures are constrained to values lower than the preset pressure ( $P_{SET}$ ) (8). Mean airway pressure can be estimated by the simple formula:

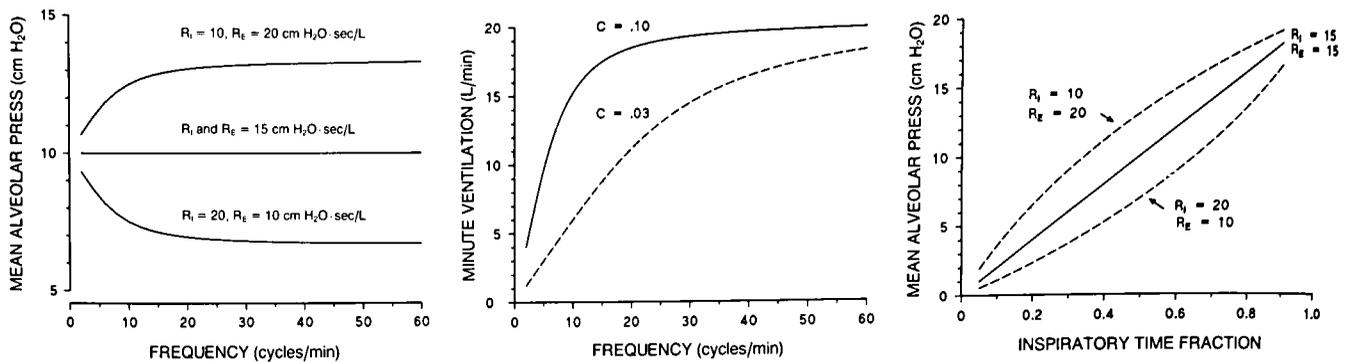


**Figure 4.** Options for increasing mean airway and alveolar pressures during passive inflation with constant flow. The shaded areas represent alveolar pressure. For the same inspiratory time ( $T_i$ ) and tidal volume, both mean airway pressure and mean alveolar pressure can be increased by increasing the positive end-expiratory pressure (PEEP) or creating auto-PEEP, by lengthening the end-inspiratory pause, or by "squaring-up" the inflation pressure profile (dashed lines). Lengthening the pause period may accentuate auto-PEEP if it shortens the deflation period ( $T_e$ ). PEEP and plateau areas can be considered static pressures, which together with the inspiratory tidal elastic pressure, are shared in common by airway pressure and alveolar pressure.  $P_b$ , dynamic pressure;  $P_s$ , static pressure.

$$P_{aw} = P_{SET} \left( \frac{T_i}{T_T} \right) + PEEP \left( \frac{T_e}{T_T} \right)$$

where  $T_i$ ,  $T_e$ , and  $T_T$  are inspiratory, expiratory, and total cycle times. When inspiratory and expiratory resistances are equal, Equation 3 in conjunction with this last expression, predicts that mean airway pressure and mean alveolar pressure are also equal and independent of frequency and compliance so long as  $P_{SET}$  and the inspiratory time fraction remain unchanged (Fig. 5, *left*). However, when these resistances differ, mean alveolar pressure approaches a plateau set higher or lower than mean airway pressure as the frequency is varied, because minute ventilation—the driver of the mean alveolar pressure minus mean airway pressure difference—is a hyperbolic function of frequency (Fig. 5, *middle*) (8). Note that when there is equivalence between the inspiratory and expiratory resistances, compliance fails to influence mean airway pressure or mean alveolar pressure so long as  $P_{SET}$ , frequency, and the inspiratory time fraction are held constant. When resistances differ, and all machine settings are kept the same, compliance affects mean alveolar pressure but not mean airway pressure. Mean alveolar pressure bears a more or less linear relationship to the inspiratory time fraction, depending on the ratio between inspiratory and expiratory resistance (Fig. 5, *right*). As the inspiratory time fraction approaches 1.0, mean airway pressure and mean alveolar pressure approach the preset airway pressure.

*Inverse Ratio Ventilation.* Inversion of the inspiratory-to-expiratory ratio can be achieved during time-cycled, pressure-controlled ventilation by appropriate adjustment of the settings for inspiratory time and cycling frequency. Airway and alveolar mean pressures



**Figure 5.** *Left:* Relationship of mean alveolar pressure to frequency during pressure-preset ventilation. Mean alveolar pressure is independent of frequency when inspiratory ( $R_i$ ) and expiratory ( $R_e$ ) resistances are equal, but mean alveolar pressure is a hyperbolic function of frequency when they differ. In this example, the applied pressure is 20 cm H<sub>2</sub>O; compliance ( $C$ ) is 0.10 L/cm H<sub>2</sub>O, and inspiratory time fraction is 0.5. *Middle:* Relationship of minute ventilation to frequency during pressure-preset ventilation, depicted here for inspiratory time fraction = 0.5, applied pressure = 20 cm H<sub>2</sub>O, and  $R_i$  and  $R_e = 15$  cm H<sub>2</sub>O-sec/L. This relationship between minute ventilation and frequency, in conjunction with Equation 3 in the text, explains the hyperbolic relationships of mean alveolar pressure to frequency that are depicted in the left-hand portion of this figure. Changing the compliance ( $C$ , expressed in L/cm H<sub>2</sub>O) affects the rate of approach of minute ventilation to its plateau, but not the plateau itself. *Right:* Relationship of mean alveolar pressure to inspiratory time fraction during pressure-preset ventilation. Mean alveolar pressure is a linear function of the inspiratory time fraction, unless  $R_i \neq R_e$ . In this example, the applied pressure is 20 cm H<sub>2</sub>O; compliance is 0.10 L/cm H<sub>2</sub>O, and frequency is 20 cycles/min. Resistance is expressed in cm H<sub>2</sub>O-sec/L.

increase during inverse ratio ventilation, both because mean inspiratory pressures increase and because auto-positive end-expiratory pressure is created (9). During flow-controlled volume-preset ventilation, ratio inversion can be achieved by increasing the cycling frequency or tidal volume, by using an end-inspiratory pause, or by slowing the average inspiratory flow rate. For example, during volume-preset ventilation with decelerating flow, pressure and flow profiles similar to those profiles of pressure-preset, inverse ratio ventilation can be achieved by appropriate selection of the peak and end-inspiratory flow settings (Fig. 6).

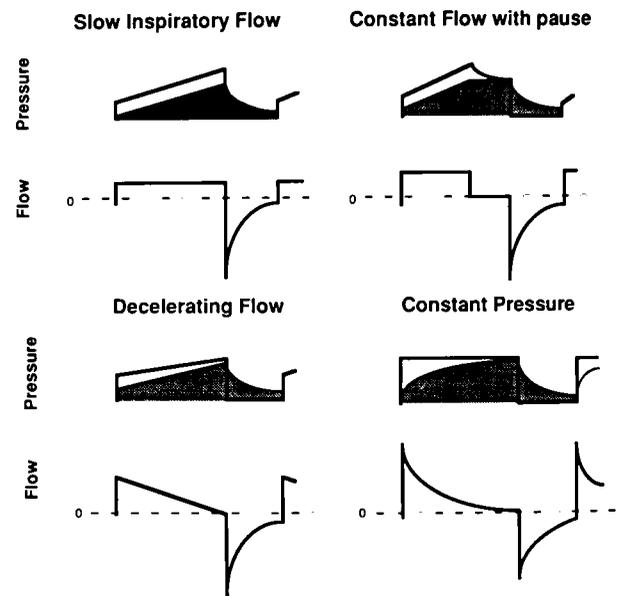
**ESTIMATION OF MEAN AIRWAY PRESSURE**

Although a variable of considerable scientific and clinical interest, alveolar pressure is generally inaccessible to direct measurement. Yet, despite this difficulty, mean airway pressure provides an easily determined and often accurate reflection of mean alveolar pressure. However, care must be exercised in both the acquisition and interpretation of the airway pressure signal.

*Practical Aspects of Airway Pressure Measurement.*

*Where Should Airway Pressure Be Measured? Practical Considerations.* In the clinical setting, airway pressure is usually tapped within the ventilator itself or near the Y-piece of the external circuit, often using an auxiliary port intended for airway temperature measurement.

*Where Should Airway Pressure Be Measured? Theoretical Considerations.* The airway can be considered to extend from the true airway opening to the alveolus. Although mean airway pressure is often described as if



**Figure 6.** Inverse ratio ventilation applied by four techniques. Airway pressure, alveolar pressure (shaded areas), and inspiratory flow are depicted for pressure-preset ventilation and three forms of volume-preset ventilation. Similar alveolar pressure profiles can be generated using pressure-preset ventilation, volume-controlled decelerating flow, or volume-controlled constant flow with an end-inspiratory pause.

it were a single number, the value measured for mean airway pressure may well vary with sampling site. Whether or not the mean pressure value measured at one position agrees with that value at another position depends on the symmetry of pressure dissipation in the airway segment that separates the two positions (Fig. 7). It can be shown that during laminar flow, the mean

pressures at any two sites in the airway (e.g., from alveolus to airway opening [segment C to A of Fig. 7]) differ by the value, (minute ventilation/60) × (RE - RI), where RI and RE are the total inspiratory (RI<sub>1</sub> + RI<sub>2</sub>) (Fig. 7) and expiratory (RE<sub>1</sub> + RE<sub>2</sub>) resistances of the segment between them. This equation is the same expression developed earlier as Equation 3, which, in effect, considered the alveolus as one specific point along the airway. Considering any intermediate site (Fig. 7, position B), the difference between the mean pressures measured at that site and the airway opening is: (mean airway pressure at B - mean airway pressure at A) = [(minute ventilation/60) × (RE<sub>1</sub> - RI<sub>1</sub>)], where RI<sub>1</sub> and RE<sub>1</sub> are the inspiratory and expiratory resistances of the segment in question. These relationships imply that if resistive pressure losses were perfectly symmetrical from airway opening to alveolus in both directions, then mean airway pressure would be the same at all sites of measurement.

In the external circuit, the flow-resistive characteristics of the tubing are similar for inspiration and exhalation (both values being almost negligible). Therefore, mean airway pressure does not vary significantly wherever measured proximal to the endotracheal tube. When appropriately adjusted for asymmetry of pressure dissipation in the segment between the tap and the alveolus, mean airway pressure accurately estimates mean alveolar pressure. However, all sites within the circuit tubing lie upstream of the exhalation valve, which frequently offers clinically important resistance to airflow. The effect of valve resistance (Rv) can be appreciated by examining the airway pressure tracing, and can be quantified by planimetry (Fig. 8):

$$R_v = \frac{\int_{T_i}^{T_E} (P_{aw} - PEEP) dt}{V_T}$$

where T<sub>i</sub>, T<sub>E</sub>, and V<sub>T</sub> are inspiratory time, expiratory time, and tidal volume. Valve resistance boosts transduced mean airway pressure by a small amount

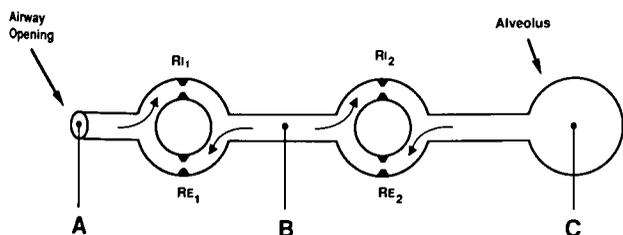


Figure 7. Schematic representation of the respiratory system from airway opening (point A) to alveolus (point C). Point B indicates another site of airway pressure measurement along the pathway. RI<sub>1</sub>, RI<sub>2</sub>, RE<sub>1</sub>, and RE<sub>2</sub> represent segmental inspiratory and expiratory resistance components.

(valve resistance × minute ventilation/60 ≈ 0 to 3 cm H<sub>2</sub>O). However, valve resistance “downstream” from the airway pressure tap influences both airway pressure and alveolar pressure equally, and therefore, does not affect the validity of mean airway pressure as an indicator of mean alveolar pressure, so long as the appropriate segmental correction (minute ventilation × [RE - RI]) is applied (Fig. 8).

*Technical Requirements for Airway Pressure Measurement.* Because the transduced pressure can be influenced by kinetic energy transfer and Bernoulli effects at the sampling location, pressure should be tapped at an appropriate site and at right angles to axial flow (10, 11). The sensitivity of the recorded value to such disturbances has been well documented for high-frequency jet ventilation, in which setting, the pressure tap must be positioned considerably downstream from the injector orifice (12).

Display of the pressure signal can be accomplished with any transducer of the appropriate range linked to the bedside monitor that is normally used to display cardiovascular data. A disposable transducer intended for measurement of pulmonary artery pressure serves well, with gas rather than liquid employed as the coupling agent (13). Used in this way, such transducers must be dedicated to gas pressure measurement and must not be shared with a vascular catheter, so as to avoid the possibility of iatrogenic gas embolism.

Mechanical or electronic damping, manual or computer-aided planimetry, and electronic signal conditioning by mathematical algorithms each effectively average the pressure signal and can be made arbitrarily accurate for that purpose. Mean airway pressure can be closely approximated without resorting to planimetry of the undulating airway pressure waveform by sufficient mechanical damping or electronic filtering

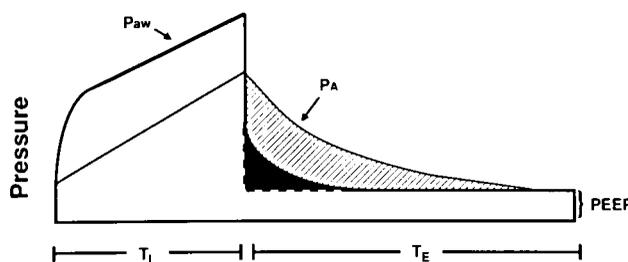


Figure 8. Airway pressure tracing (heavy line) obtained from a single site within the ventilatory circuit. Darkly shaded region reflects the resistance presented by the exhalation valve downstream from the site of airway pressure measurement. The cross-hatched region represents the dissipated pressure-time product required for computation of the segmental expiratory resistance (RE) that is used in the estimation of mean alveolar pressure (P<sub>A</sub>) from mean airway pressure (P<sub>aw</sub>) (Eq. 3): P<sub>A</sub> = P<sub>aw</sub> + (minute ventilation/60) × (RE - RI). PEEP, positive end-expiratory pressure; T<sub>i</sub>, inspiratory time; T<sub>E</sub>, expiratory time; R<sub>i</sub>, inspiratory resistance.

(14, 15). In the recording of a fluctuating pressure, the frequency response and damping factor of the mechanical and electronic components bear directly on the fidelity of the tracing. Yet, however badly the contours of the original waveform may be distorted, symmetrical damping of the fluctuating waveform causes the mean pressure of the damped signal to approach the same value as that value of the original tracing. Mechanical damping can be achieved by introducing a variable compliance into the pressure-sensing circuit that is large in comparison with the unmodified catheter-transducer system. This technique is most practical when cycling frequencies are high. Thus, such methods have been successfully used in the transduction of mean airway pressures during high-frequency oscillation (16).

Modern recording equipment can easily provide mean pressure data by "low pass" electronic filtering of the raw airway pressure signal. Most systems are capable of good fidelity to frequencies 10 to 30 times greater than the fundamental frequency defined by the breathing rate (17). More importantly, the frequency response of the recording system is largely immaterial for the recording of mean pressures, since a poor mechanical frequency response serves only to act as a mechanical "low pass" filter. However, as indicated by Simon et al. (13), both the linearity and symmetry of the measuring system are important. Prefiltered pressures that are outside the linear range of the equipment can cause signal distortions and measurement error. Fortunately, most transducers used in the clinical and research setting have a wide linear range that easily spans the spectrum of clinical input. Designed for high-fidelity recording of pressures and frequencies that are more extreme than those frequencies encompassed by the range of airway pressure (17), the transducers used in pulmonary arterial pressure measurement are adequate for airway pressure assessment. Although simple electronic filtering can provide a good "on-line" approximation of mean airway pressure, microprocessor-controlled bedside monitors usually accomplish integration by an averaging algorithm that samples a multicycle period. (Such a strategy minimizes the beat-to-beat variation inherent in the cardiovascular signals for which they are designed, and can prove helpful in mean airway pressure measurement when respiratory pressure ( $T_i/T_T$ ) and/or frequency vary. However, as discussed in Part 2 of this article, the accuracy of these averaging algorithms for respiratory applications varies widely, and is often influenced by breathing frequency and intercycle variations of pressure waveform.)

#### Indirect Estimation of Mean Airway Pressure and Mean Alveolar Pressure From Selected Pressure

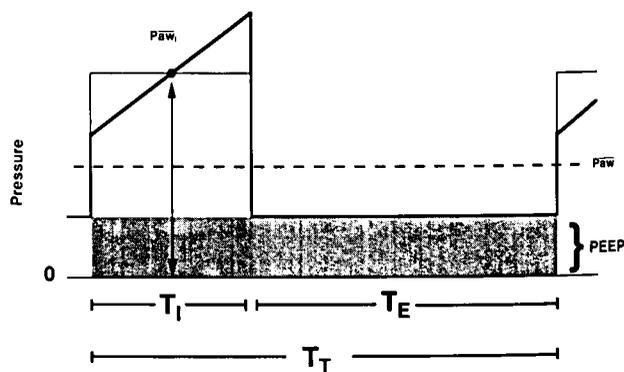
**Measurements.** During passive inflation with constant flow, the pressure measured halfway through the inspiratory portion of the cycle closely estimates the time-averaged mean inspiratory airway pressure (Fig. 9). The same estimation method holds true for pressure-preset ventilation. However, for most other inflation patterns, the mean inflation pressure cannot be readily estimated from a single-point determination on the airway pressure tracing. Ideally, the pressure at the airway opening decreases to the positive end-expiratory pressure level as soon as the expiratory valve opens. Under this assumption, mean airway pressure can be computed from knowledge of the mean inspiratory airway pressure ( $\overline{Paw}_i$ ), positive end-expiratory pressure, and the inspiratory and expiratory time fractions ( $T_i/T_T$  and  $T_e/T_T$ ):

$$\overline{Paw} = \overline{Paw}_i \left( \frac{T_i}{T_T} \right) + PEEP \left( \frac{T_e}{T_T} \right)$$

If a pressure tracing is not readily available, a crude estimate of mean airway pressure during constant flow can still be made:

$$\overline{Paw} = \left( P_D - \frac{(P_S - P_{EX})}{2} \right) \frac{T_i}{T_T} + PEEP \left( \frac{T_e}{T_T} \right)$$

where  $P_D$ ,  $P_S$ , and  $P_{EX}$  represent manometer-measured values for peak dynamic, peak static, and end-expiratory alveolar pressures (e.g., as measured by a hand-held gauge or the manometer readout of the



**Figure 9.** Airway pressure tracings during inflation with pressure-preset ventilation (*thin solid line*) and with volume-cycled, constant inspiratory flow ventilation (*thick solid line*). For both types of pressure waveforms, mean inflation pressure ( $\overline{Paw}_i$ ) can be accurately approximated as the pressure recorded at the midpoint of the inflation half cycle. Mean airway pressure for the total cycle ( $\overline{Paw}$ , *dashed line*) can then be estimated as:

$$\overline{Paw} = \overline{Paw}_i \left( \frac{T_i}{T_T} \right) + PEEP \left( \frac{T_e}{T_T} \right)$$

where  $T_i$ ,  $T_e$ , and  $T_T$  are inspiratory, expiratory, and total respiratory cycle times, respectively. *PEEP*, positive end-expiratory pressure.

ventilatory system). If inspiratory and expiratory resistances are known, Equation 3 can be used in conjunction with mean airway pressure to estimate mean alveolar pressure as well.

*Estimation of Mean Airway Pressure and Mean Alveolar Pressure Without Pressure Measurements.* Assuming that positive end-expiratory pressure and other key variables have been approximated, estimates for mean airway pressure and mean alveolar pressure can be attempted without measuring airway pressures. When inspiratory and expiratory resistance, compliance, tidal volume, and the inspiration time fraction ( $T_i/T_T$ ) are known, the instantaneous inflation pressure ( $P_i$ ) for the respiratory system can be computed as

$$P_i = \frac{V}{C} + R_i \dot{V} + P_{EX}$$

*Constant (Square-Wave) Flow.* During ventilation with a constant flow of  $\dot{V}$  liters/min and no positive end-expiratory pressure applied, the mean inflation pressure ( $\overline{P_{aw}}$ ) measured at the airway opening would be:

$$\overline{P_{aw}}_1 = \frac{R_i \dot{V}}{60} + \frac{V_T}{2C} + P_{EX}$$

(In general, mean inspiratory flow ( $\overline{\dot{V}}$ ) is the quotient of tidal volume to inspiratory time or of minute volume to the inspiratory time fraction. During constant mean inspiratory flow,  $\dot{V}$  theoretically equals the peak flow setting.) Assuming uniexponential decay of alveolar pressure and volume during exhalation, the end-expiratory alveolar pressure ( $P_{EX}$ ) of this expression can also be estimated without specific measurement (8):

$$P_{EX} = \frac{V_T}{C(e^{T_E/R_E C} - 1)}$$

As before, mean airway pressure =  $\overline{P_{aw}}_1 (T_i/T_T) +$  positive end-expiratory pressure ( $T_E/T_T$ ). If inspiratory and expiratory resistances are unequal and the discrepancy is known, mean alveolar pressure can be approximated as:

$$\begin{aligned} \overline{P_A} &= \overline{P_{aw}} + \left( \frac{\dot{V}_E}{60} \right) (R_E - R_i) \\ \overline{P_A} &= \left( \frac{R_i \dot{V}}{60} + \frac{V_T}{2C} + \frac{V_T}{C(e^{T_E/R_E C} - 1)} \right) \left( \frac{T_i}{T_T} \right) \\ &+ PEEP + \left( \frac{\dot{V}_E}{60} \right) (R_E - R_i) \end{aligned}$$

*Other Flow Waveforms.*  $\overline{P_{aw}}_1$ , mean airway pressure, and mean alveolar pressure for sinusoidal inspiratory flow are theoretically identical to those

variables predicted for constant flow, assuming the same input values for inspiratory and expiratory resistances, compliance, frequency, tidal volume, and inspiratory time fraction (during sinusoidal flow;  $\dot{V} = V_T/T_i$ ) (18). However, mean airway pressure differs for pressure-preset ventilation (8), where it can be computed using the applied pressure ( $P_{SET}$ ) and the inspiratory and expiratory time fractions:  $P_{SET} \times (T_i/T_T) +$  positive end-expiratory pressure  $\times (T_E/T_T)$ . Finally, for volume-preset ventilation with linearly decelerating flow, mean airway pressure would be:

$$\overline{P_{aw}} = \frac{R_i \dot{V}_E}{60} + \frac{f V_T^2}{90C} \left[ \frac{2\dot{V}_P + \dot{V}_F}{(\dot{V}_P + \dot{V}_F)^2} \right] + \frac{f V_T^2}{30C(\dot{V}_P + \dot{V}_F)(e^{T_E/R_E C} - 1)}$$

where  $\dot{V}_P$  and  $\dot{V}_F$  are the set rates for peak and end-inspiratory (final) flow, expressed in liters per second. Other variables are as previously defined (19).

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