Stepwise Ventilator Waveform Assessment to Diagnose Pulmonary Pathophysiology

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Electronically displayed ventilator waveforms provide a wealth of insight into the physiology of the respiratory system. Pressure and flow values can be independent variables that reflect control by the ventilator or dependent variables that demonstrate the respiratory system's response to mechanical ventilation. Diagrams of time-based pressure and flow curves may reveal underlying pathophysiology beyond more commonly assessed parameters such as peak airway pressure, respiratory rate, and tidal volume. In this narrative review, we introduce a stepwise approach clinicians can take to diagnose pulmonary pathophysiology by using ventilator waveforms in patients receiving pressure control ventilation or volume control ventilation. Understanding a patient's pathophysiology (*i.e.*, increased airway resistance) does not distinguish pathology (*i.e.*, kinked endotracheal tube or bronchospasm). A pathology such as pulmonary edema may have more than one pathophysiologic process (*i.e.*, increased airway resistance and low respiratory compliance). To interpret ventilator waveforms, we assume a one-compartment lung model with a linear response to a range of tidal volumes.[1] We do not discuss identification of ventilation mode or adaptive ventilation.[2]

The Equation of Motion

The equation of motion is an equation of pressures. In this equation, the pressure applied to the patient's respiratory system is the sum of ventilator pressure (P_{VENT}), a positive pressure, and the patient's respiratory muscle pressure (P_{MUS}), a negative pressure. The combination of P_{VENT} and P_{MUS} must overcome the patient's intrinsic resistance to flow, elastic response to volume, and retained positive end-expiratory pressure (PEEP) to deliver a ventilated breath.[3]

$$P_{MUS} + P_{VENT} = \text{resistive pressure} + \text{elastic pressure} + \text{PEEP} \quad (1)$$

or

$$P_{MUS} + P_{VENT} = R \star \left(\dot{V}\right) + E \star \left(V\right) + \text{PEEP} \quad (2)$$

Resistive pressure or load is a function of airway resistance ($R = \Delta\text{pressure}/\text{flow}$) and airflow ($\dot{V} = \Delta\text{volume}/\Delta\text{time}$). Elastic pressure or load is a function of airway and chest wall elastance (E) and volume of air in the respiratory system (V). Because elastance ($\Delta\text{pressure}/\Delta\text{volume}$) is the inverse of compliance ($\Delta\text{volume}/\Delta\text{pressure}$), a patient with poorly compliant lungs or a stiff chest wall has an increased elastic load. The gradient between the left and right sides of the equation of motion determines the direction of airflow. Clinically, this is shown as changes in airway pressure and flow waveforms over time as a volume of air cycles in or out of the airways. For example, a patient with high respiratory elastance (poor compliance) will have a large elastic load and thus will need comparable increases in P_{MUS} or P_{VENT} to drive air into the lungs.

In a paralyzed patient, the ventilator generates the entire positive pressure received by the patient's lungs (*i.e.*, P_{MUS} is 0). As patient effort increases (reflected as a negative pressure), airway pressure decreases, and the ventilator generates less pressure, which is also known as "work shifting." If the ventilator delivers more pressure than the patient demands, then airway pressure is above baseline, and the patient is "assisted." From a waveform perspective, there would be no observed increase in pressure on the pressure waveform if the ventilator delivered a pressure that *exactly* matched (did not assist) the patient's demand over time.

We can implement the equation of motion at the bedside by assessing the flow and pressure waveforms over time in the context of patient and ventilatory activity. The following presents a stepwise approach to diagnosing respiratory pathophysiology using ventilator waveforms. In the first step, we generate a pathophysiologic hypothesis based on the shape and duration of the expiratory flow waveform.

Expiration: Below the Baseline

In pressure control ventilation and volume control ventilation, the cessation of ventilator-applied flow initiates expiration. Expiration is dependent on patient effort and

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underlying lung physiology. In the absence of patient effort, expiration is a passive process that reflects the resistive and elastic loads of the respiratory system. Passive expiratory flow waveforms demonstrate exponential decay to baseline as thoracic elastic recoil forces air out of the lung until PEEP is reached (fig. 1A).[4] This period is especially useful for the bedside clinician who is attempting to generate a hypothesis of intrinsic pulmonary pathophysiology, as this passive period reflects the influence of variables on the right side of the equation of motion only.

Step 1: Assess Expiratory Flow Waveform to Generate a Pathophysiologic Hypothesis

Passive Exhalation during Ventilation. The gradient between alveolar pressure (plateau pressure) and PEEP drives expiratory flow and is maximal at the beginning of expiration when alveolar pressure is highest. To assess the expiratory flow waveform, examine the patient's peak expiratory flow in relation to the expiratory time constant. The expiratory time constant is a product of the relationship between resistance and compliance and represents the time for the flow waveform to return to baseline or the patient's functional residual capacity (fig. 1, B to H). Bedside interpretation of waveforms assumes a linear one-compartment model. In this context, the time constant is defined as the time to exhale 63% of lung volume.[1]

Prolonged expiratory time constants (more than 0.7 s with an expiratory flow time of more than 2.5 s) should prompt bedside clinicians to hypothesize that lung compliance is high (*e.g.*, in chronic obstructive pulmonary disease [COPD]) or airway resistance is elevated (*e.g.*, mucous plugging, kinked endotracheal tube, bronchial resistance). In these instances, the expiratory time constant will be prolonged and expiratory peak flow will be reduced during passive exhalation (fig. 1B).[5,6] In patients who have a high resistive load (*e.g.*, bronchospasm), use of bronchodilators may increase the peak expiratory flow rate and shorten the time for flow to return to baseline.

Short expiratory time constants (less than 0.5 s with an expiratory flow time of less than 1 s) along with an increase in peak expiratory flows are observed in patients with decreased compliance (*e.g.*, acute respiratory distress syndrome [ARDS], cardiogenic pulmonary edema, restrictive lung disease, chest wall stiffness, intra-abdominal hypertension, kyphoscoliosis; fig. 1C).[7] In these pathophysiologic states, the diseased alveoli empty rapidly.

Active Exhalation (Patient Effort) during Ventilation. It is important to identify patient effort during expiratory waveform analysis because the presence of patient effort can confound the diagnosis of pathophysiologic conditions that impact the right side of the equation of motion. Patient effort should also be analyzed in the context of bedside

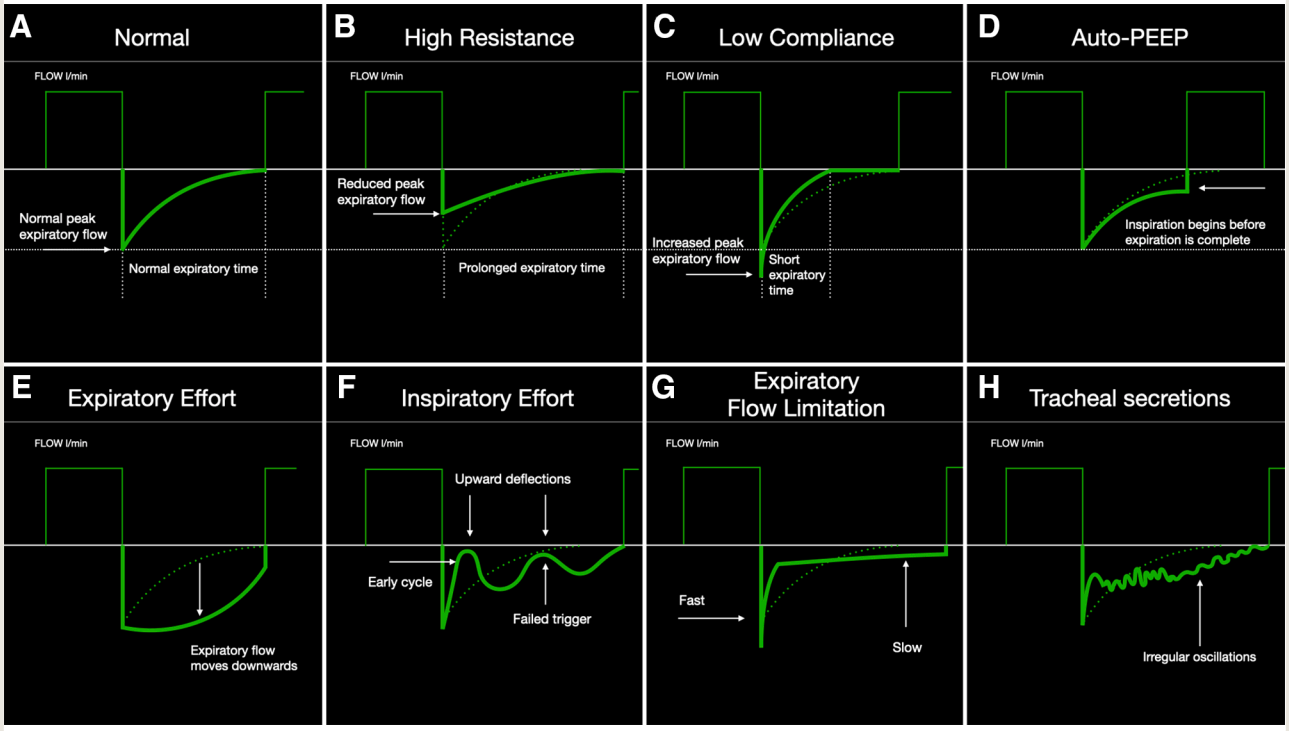

Fig. 1. Expiratory flow waveforms (*bold lines*) in volume control ventilation. Note that the expiratory portion of the waveforms would be similar in pressure control ventilation. PEEP, positive end-expiratory pressure.

evaluation of the patient's respiratory effort and receipt of sedatives or paralytics.[8] Inspiratory patient effort during expiration is identified on the expiratory waveform by the movement of flow upwards, toward baseline (fig. 1F). An upward deflection without a subsequent inspiratory breath may also represent a failed trigger, an autotrigger (see the sections "Failure to Trigger" and "False Triggering"), or relaxation of expiratory effort. An upward deflection that occurs immediately after an inspiratory cycle suggests a premature cycle (patient inspiratory effort beyond the set inspiratory time) to expiration (fig. 1F). Expiratory effort during expiration is characterized by the movement of flow downwards "obliterating" exponential decay; this may be confused with auto-PEEP (fig. 1, D and E).

Auto-PEEP. Auto-PEEP occurs when incomplete exhalation leads to retained air in the alveoli. It is important to assess for the presence of auto-PEEP during expiratory waveform analysis, as its presence influences the rate of expiratory airflow. It is often present in patients with high compliance (*e.g.*, COPD), increased resistance (*e.g.*, bronchospasm), rapid respiratory rate, large tidal volumes, or a combination of these entities. Auto-PEEP is measured in the absence of spontaneous respiratory activity by performing a static expiratory hold maneuver.[9] The detected airway pressure represents total PEEP, from which applied PEEP is subtracted to identify the auto-PEEP contained in the system.

$$\text{Auto} - \text{PEEP} = \text{total PEEP} - \text{applied PEEP} \quad (3)$$

The expiratory flow waveform of patients with substantial auto-PEEP does not return to baseline but may have a typical decay appearance (fig. 1D). Decreasing the respiratory rate can reduce auto-PEEP.

Expiratory Flow Limitation. Expiratory flow limitation occurs from dynamic small and distal airway collapse when expiratory flow cannot rise with a high expiratory driving pressure. This leads to a bicompartmental expiration phase, in which expiratory volume return from heterogeneous lung areas is both rapid and slow. This can cause *pendulluft-ing*, in which air flows between lung compartments rather than directly returning to the ventilator in one linear, continuous motion. Expiratory flow limitation causes auto-PEEP and is observed in patients with COPD, congestive heart failure, obesity, and ARDS, especially with low PEEP. Because flow may be close to 0, an expiratory pause can detect the presence of auto-PEEP. This phenomenon is also observed in patients with single-lung transplant who have two different compartments for exhalation where the transplanted lung empties faster than the native, overly compliant COPD lung.

Hypothesize the presence of expiratory flow limitation when an inflection point is observed instead of a single exponential decay in the expiratory flow waveform (fig. 1G).[10] In these circumstances, an initial flow spike represents dynamic compression and exhalation of gas in the central airways. This rapid expulsion leads to a reduction in expiratory flow from the other airways and regional volutrauma. In contrast to auto-PEEP from a short expiratory time, expiratory flow limitation does not respond to prolonging expiratory time or reducing respiratory rate.[10] If PEEP is reduced to 0, flow increases in normal lung zones but decreases in zones with expiratory flow limitation. Increasing external PEEP can alleviate intrinsic PEEP during expiratory flow limitation.

Secretions. Hypothesize the presence of airway secretions or condensate in the ventilator circuit when faced with a sawtooth expiratory flow (fig. 1H).

Inspiration: Above the Baseline

The inspiratory period reflects the interaction between the ventilator (*i.e.*, P_{VENT}) and the patient's lungs. Inspiratory flow waveforms reflect dynamic flow and appear as square, descending, or decelerating (fig. 2, A to C). In volume control ventilation, flow is controlled, and pressure waveforms represent the respiratory system's response to inspiration. In pressure control ventilation, pressure is controlled, and inspiratory flow waveforms represent the respiratory response to inspiration.

Step 2: Assess Inspiratory Flow Waveform to Corroborate Pathophysiologic Diagnosis

Volume Control: Square and Descending Flow Waveforms. Assessment of the inspiratory flow waveform in volume control ventilation is limited because flow is controlled; the shape of the inspiratory waveforms changes little with patient effort. With a square waveform, flow quickly rises and remains constant until the target tidal volume has been delivered before cycling into exhalation (fig. 2A). A descending waveform decreases linearly after maximum flow (fig. 2B). Although analysis of the inspiratory flow waveform during volume control ventilation is limited, it is necessary to understand the geometric shape of the inspiratory flow waveform to interpret concurrent pressure waveforms (see Step 3).

Pressure Control and Pressure Support: Decelerating Waveforms. Pressure control ventilation is a pressure-targeted, inspiratory time-cycled mode with variable flow and volume. Pressure control ventilation characteristically utilizes a decelerating inspiratory flow waveform with exponential decay (fig. 2C).[6] Inspiratory flow changes dynamically owing to patient effort, airway resistance, and the gradient between proximal airways and alveoli (mathematically represented by the equation of motion), which can vary from breath to breath (fig. 2D).

If the presence of increased compliance or increased airway resistance has been hypothesized in Step 1, next confirm the presence of decreased peak inspiratory flow. Compared with peak inspiratory flows in patients with normal lung physiology (fig. 2E), those in patients with

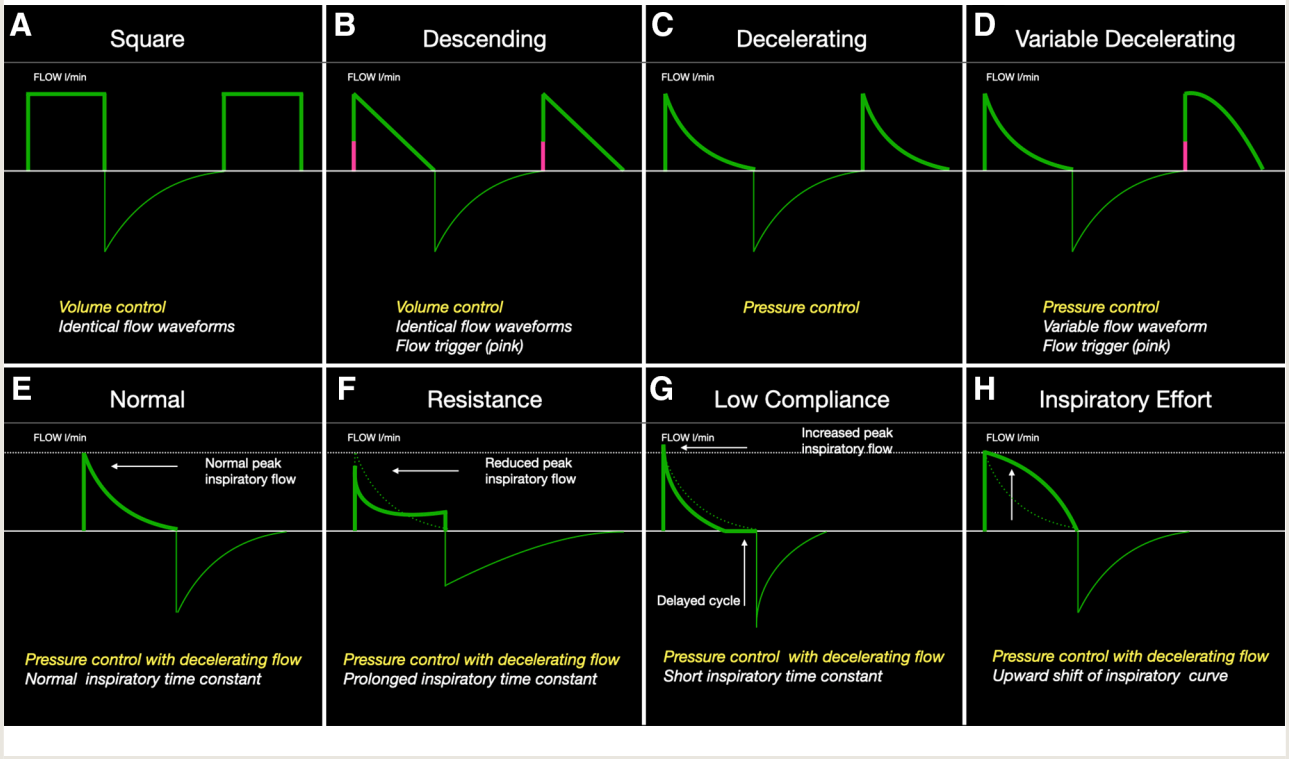

Fig. 2. Inspiratory flow waveforms (*bold lines*) in volume control ventilation (*A*, *B*) and pressure control ventilation (*C* to *H*).

increased compliance (COPD) and increased airway resistance are lower, and time constants are prolonged. Flow may not reach baseline because the lung inflates slowly (fig. 2F). In lung units with prolonged inspiratory time constants (*i.e.*, bronchospasm), alveolar inflation time is prolonged, and tidal volumes may be reduced if inspiratory time is not increased. Conversely, the hypothetical presence of low compliance states can be corroborated by the observation of short inspiratory time constants and decreased insufflation times (fig. 2G).

If the decelerating flow waveform has a linear or upward concavity shape (*vs.* an exponential decay), inspiratory patient effort is likely, and inspiratory pressures or flow may be inadequate (fig. 2H). Shortening the rise time or the time to achieve a target pressure can manage inadequate flow. A leak or an increase in flow from lung recruitment also shifts the waveform upwards.

Step 3: Assess Inspiratory Pressure Curves to Confirm Pathophysiologic Diagnosis

Flow is the independent variable controlled by the ventilator in volume control ventilation, and assessing the dependent pressure waveform can confirm the pathophysiologic hypothesis generated by analysis of the expiratory flow waveform. In contrast to volume control ventilation, the pressure waveform of pressure control ventilation is the independent variable, and the flow waveform is the dependent variable.

Calculate the Plateau Pressure and the Driving Pressure. Plateau pressure (P_{PLAT}) reflects lung compliance and can be calculated in both volume control ventilation and pressure control ventilation. Calculating plateau pressure during pressure control ventilation is important when resistive load is high or inspiratory time is short. Plateau pressure is assessed at end inspiration with the inspiratory hold maneuver, when alveolar and circuit pressure have reached equilibration (fig. 3A). The difference between peak inspiratory flow (PIP) and P_{PLAT} reflects airway resistance. Escalating [PIP – P_{PLAT}] values suggest resistive pathologies such as mucus plugging, bronchospasm, or circuit obstruction (fig. 3B). Increased P_{PLAT}, indicative of poor compliance, suggests ARDS, pulmonary edema, pneumonia, or pneumothorax (fig. 3C). Additionally, auto-PEEP in the setting of bronchospasm is associated with elevated plateau pressures and an increased [PIP – P_{PLAT}] (fig. 3D). Perform a static expiratory hold maneuver to measure the presence of auto-PEEP, which may influence compliance measurements.

Volume Control: Pressure Waveform with Square Flow. Flow is controlled by the ventilator after the clinician selects the desired volume in volume control ventilation. Assessing the resulting pressure waveform can confirm the pathophysiology hypothesis. The initial rise in pressure reflects the resistive load in a passive patient. The end inspiratory pressure is

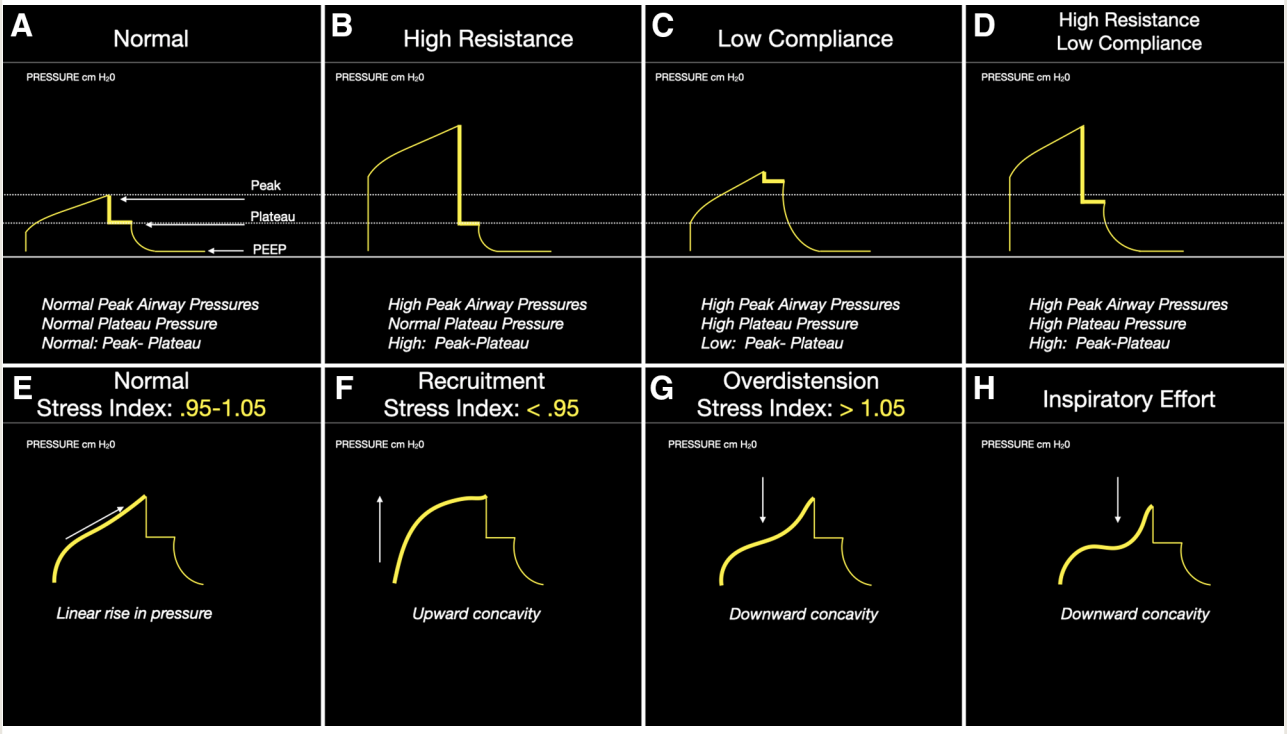

Fig. 3. Pressure waveforms in volume control ventilation with a square waveform flow pattern. PEEP, positive end-expiratory pressure.

a function of the elastic load in the airways. In contrast to volume control ventilation, inspiratory pressure waveforms add little information to inspiratory flow waveform analysis during pressure control ventilation. With constant flow in a passive patient, the slope of the pressure curve (after the initial rise in pressure) reflects lung compliance.

The stress index is derived from the airway pressure–time curve ($P_{aw} - t$) and has been validated to assess compliance by quantifying recruitment and overdistention during square waveform flow.[11,12] The shape of the airway pressure–time curve with constant inspiratory flow is related to compliance and is represented by the equation:

$$P_{aw} = a \star tb + c \quad (4)$$

where a is the slope of curve of steady flow between $time_0$ and $time_1$, c is the P_{aw} at $time_0$, and b (stress index) is a dimensionless number that describes the shape of the curve.

Experimental models and clinical trials have suggested that a stress index of 0.95 to 1.05 is ideal. This calculated value is not commonly available on most anesthesia ventilators but can be reliably assessed with visual analysis of the ventilator $P_{aw} - t$.[13] If the slope is linear (stress index or b =1) throughout inspiration, compliance is linear and reflects noninjurious alveolar distention (fig. 3E). If compliance is worse at the beginning of the breath and improves as the lung recruits, the inspiratory pressure curve bows out (downward concavity: stress index or b < 0.95; fig. 3F). If the initial portion of the pressure curve is flat and then appears scooped as the breath is delivered, compliance decreases and overdistention is likely (upward concavity; stress index or b > 1.05; fig. 3G). The pressure waveforms of a patient with overdistended alveoli or a patient with an active inspiratory effort in the setting of inadequate flow are similar with an upward concavity (fig. 3H). Distinguish overdistension from active effort by observing the patient at the bedside and noting the presence or absence of a patient-triggered breath.[14]

Volume Control: Pressure Waveform with Decelerating Flow. Switching the ventilator from a square to a descending flow waveform in volume control ventilation may reduce the effect of increased airway resistance and peak inspiratory pressure. Additionally, decelerating flow waveforms decrease peak inspiratory pressures, dead space ventilation, the A-a gradient, and potentially patient respiratory effort by increasing mean airway pressures and improving patient-to-ventilator synchrony.[15] Switching from a square waveform to a decelerating flow waveform may increase inspiratory time unless the flow rate is increased, predisposing patients to reduced expiratory times and potential auto-PEEP. When inspiratory flow is delivered in a descending pattern, the initial pressure rise is from the resistive load in a passive patient and is also reflected at end inspiration by the difference between the peak and plateau pressures (fig. 4A). This initial rise is higher with increased resistive load. A high initial rise with a significant drop in the pressure waveform to a lower end

inspiratory pressure once the flow reaches 0 can confirm the presence of a resistive lung load (bronchospasm; fig. 4B). As flow descends to 0, the pressure waveform reflects the plateau pressure, such that the end of inspiratory pressure with descending flow reflects elastance. Patients with a high elastic load (low compliance) will see an elevated pressure at the end of inspiration as the flow reaches 0 (fig. 4C). Poor lung compliance should be distinguished from exhalation during a mechanical inspiratory breath, which also increases pressure at the end of inspiration (fig. 4D).

Volume Control: Inspiratory Patient Effort. Pressure waveforms can provide evidence of patient effort. If the patient is actively inhaling during inspiration, the inspiratory pressure curve moves down toward baseline (fig. 4E). Paradoxical motion of the patient's thorax and abdomen may be observed. The phenomenon of flow starvation occurs if the inspiratory pressure drops below baseline to generate negative inspiratory pressure. Manage inadequate flow by increasing the ventilator flow rate, which will decrease the inspiratory time.

Asynchronies: Absence of Patient Effort or Ventilator Response

Step 4: Identify the Triggering Source

Triggering. Patient effort or time triggers inspiration. Patient effort can trigger a breath by a change in pressure of typically 1 to 2 cm H_2O of patient effort (represented by a negative deflection in the pressure waveform; fig. 4A) or a change in the continuous flow of the circuit (often represented by a change in color of the initial portion of inspiratory flow; fig. 2, B and D). Identifying a patient-triggered breath and a passive or machine-triggered breath can unmask pathology in the presence of patient effort. If both breaths are identical, there is no P_{MUS}. A deep and wide negative deflection of pressure during the trigger phase suggests a strong respiratory drive. Triggering asynchronies reflect either an absence of ventilator response, known as "failure to trigger," or an absence of patient effort, known as "false trigger."[16]

Failure to Trigger. Mechanical ventilators recognize patient inspiratory effort as negative deflections in either circuit pressure or airflow. An upward deflection during the expiratory phase (below the baseline) suggests inspiratory effort. If this effort is insufficient to decrease alveolar pressure, the corresponding decrease in circuit pressure or flow will not occur (fig. 1F). Respiratory effort may be inadequate to trigger a breath if alveolar pressure is elevated (*i.e.*, auto-PEEP). Setting a flow trigger compared to a pressure trigger may reduce the work of breathing by decreasing the sensitivity to trigger a breath.[6,17] Flow triggers, however, do not completely avoid the challenges associated with high alveolar

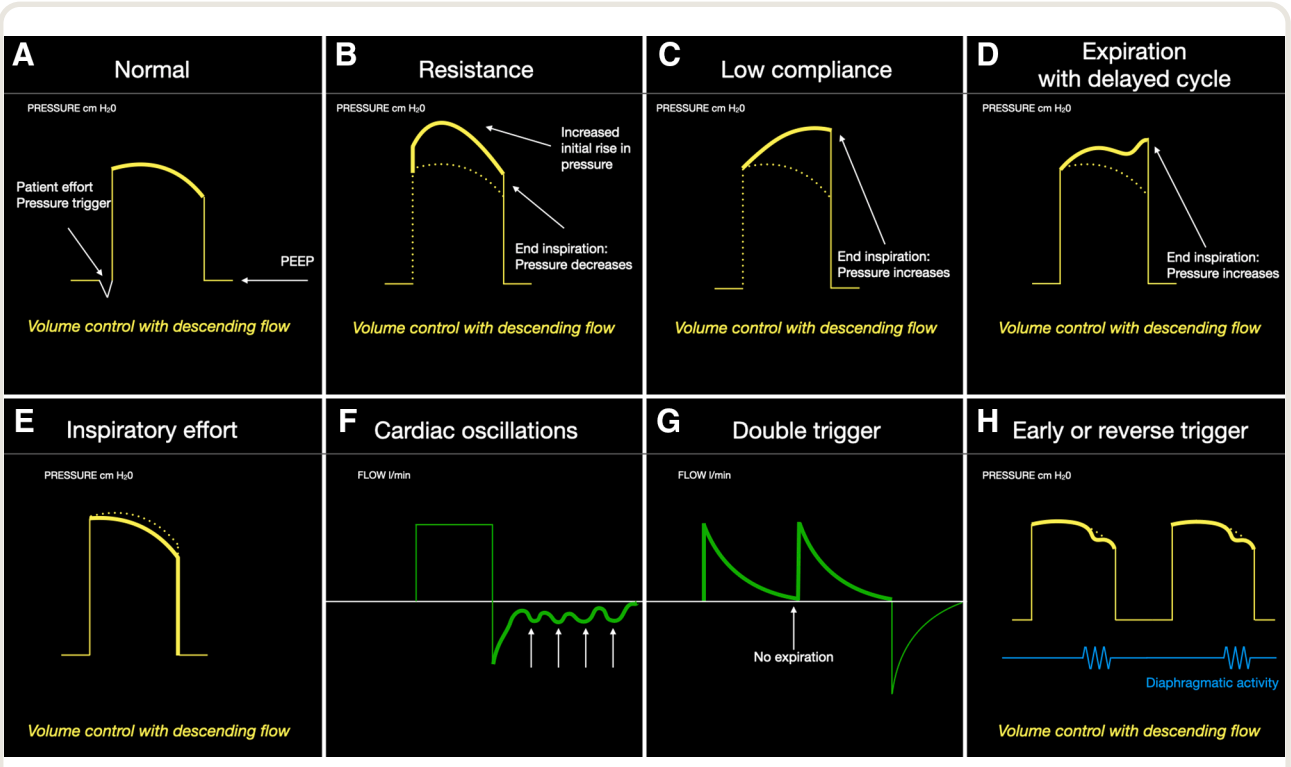

Fig. 4. Pressure waveforms in volume control ventilation with a descending waveform flow pattern (*A* to *E*, *H*); cardiac oscillations during expiratory flow waveform (*F*); and double triggering with inspiratory flow waveform (*G*). PEEP, positive end-expiratory pressure.

pressure because alveolar pressure must still be overcome to negative to initiate flow into the lungs (as described in the equation of motion).[18]

False Triggering (Autotriggering). False triggering or autotriggering occurs when the ventilator provides a breath in response to a stimulus unrelated to patient effort. Entities such as cardiac oscillations, condensation in ventilator tubing, continuous negative flow from the ventilator circuit or chest tube leaks, highly sensitive (easy) triggering thresholds, or external vibrations (*i.e.*, renal replacement therapy, chest tube, gastric suction tubing) can cause deflections in pressure and flow that are interpreted by the ventilator as patient effort to trigger a breath. Cardiac oscillations manifest as rapid distortions at the frequency of the patient's heart rate in the expiratory flow and pressure waveforms and should be distinguished from ineffective triggering[19] (fig. 4F). Autotriggering can be diagnosed by fixing circuit leaks, removing sources of external vibrations, or switching the mechanical ventilator to a pressure trigger mode. Autotriggering has been observed after brain death and may confound the clinician's ability to verify that death has occurred.[20]

Dyssychronies: Mismatch between Patient Demand and Ventilator Response

Step 5: Identify the Time between Inspiration and Expiration

Cycling describes the transition from inspiration to expiration. Flow, time, or pressure determine cycling, and cycling dyssynchronies occur when there is mismatch between the length of time allowed by the ventilator for inspiration and expiration and the patient's actual inspiratory and expiratory cycles.

Premature Cycling (Double or Multiple Triggering). If an inspiratory effort continues beyond the ventilator's set inspiratory time, a second breath, without expiration from the first breath, can be triggered.[21] Patients desiring a longer breath or who have a fast pressure rise time or inadequate pressure support may experience premature cycling (fig. 4G). The delivery of a second breath during the expiratory phase of the machine-initiated breath may sequentially "stack" breaths, generating large tidal volumes and increased inspiratory pressures. Extending machine inspiratory time can correct premature cycling and omit breath stacking.

Delayed Cycling. Delayed cycling occurs when the set inspiratory time exceeds a patient's desired intrinsic inspiratory time. This results in patient exhalation while a machine-delivered breath is still occurring. This may be caused by unnecessarily long inspiratory time or very slow expiratory flow deceleration, as occurs with obstructive pulmonary disease (fig. 2C). Spontaneous exhalation during machine insufflation may generate very high airway pressures and increases the risk of barotrauma. Abrupt increases in airway pressure late in the machine-delivered inspiratory cycle suggest this dyssynchrony (fig. 4G). As for premature cycling, correct delayed cycling by modifying inspiratory time to match the patient's desired inspiratory length more precisely, in this case, by shortening the machine's inspiratory epoch.[21]

Early Trigger or Reverse Trigger. With an early or reverse trigger, a reflexive inspiratory effort follows a machine-triggered breath (fig. 4H). When coupled with inspiratory cycles over time, this phenomenon is referred to as "entrainment." It typically occurs near the transition from the inspiratory to the expiratory phase. Reverse triggering is speculated to occur most frequently during transitional states between intense sedation and spontaneous patient-driven triggering events that occur with milder sedation, leading to increased respiratory drive. Strategies to prevent or correct reverse triggering are uncertain, although increasing the respiratory rate or reducing sedation may reduce the propensity for entrainment to occur.[22]

Conclusions

Clinicians can use mechanical waveform analysis as a diagnostic tool to identify pulmonary pathophysiology. Flow and pressure waveforms can be interpreted to confirm diagnosis and to optimize ventilator management. This review offers tools for clinicians to use when developing a mental model hypothesis of a patient's lung pathophysiology and offers a stepwise approach to either accepting or rejecting the hypothesis.

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Competing Interests

Dr. Moitra is the American Society of Anesthesiologists' (Schaumburg, Illinois) liaison to the American Heart Association (Dallas, Texas). He provides expert testimony. Dr. Moitra is also a Director of the American Board of Anesthesiology (Raleigh, North Carolina). The other authors declare no competing interests.

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