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Mechanical Ventilation

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In many practice environments, the act of intubating a patient sets off a cascade of events that quickly take the patient out of the emergency department and into the intensive care unit (ICU) or operating room. Increasingly, hospital overcrowding leads to a delay in transfer and ventilator management falls upon the emergency medicine (EM) physician. In addition, during nights and weekends in some facilities, the EM physician may be called upon to troubleshoot or stabilize mechanically ventilated patients in the ICU. This article reviews the common modes of mechanical ventilation, new technologies, and specific ventilator strategies that have been shown to be beneficial. Indications for mechanical ventilation, ventilator associated pneumonia (VAP) and liberation from mechanical ventilation are beyond the scope of this article.

Perhaps one of the most confusing aspects of mechanical ventilation is the plethora of terms and acronyms that are used. However, this subject can be simplified by keeping in mind some key questions.

- Why is the patient on the ventilator?
- Is the patient breathing spontaneously?
- Who is doing the greater work of breathing: the patient or the machine?
- Is a volume-targeted strategy in place? Or is a pressure-targeted strategy being used?
- Is it a dual controlled mode?
- What is the set respiratory rate?

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- What is the total respiratory rate?
- What is the set extrinsic/applied positive end-expiratory pressure (PEEP)?
- Is there intrinsic PEEP (auto-PEEP) present?
- What is the inspiratory/expiratory ratio, flow rate, and trigger mode?
- What do the ventilator graphics indicate?

By convention, many clinicians order a standard "AC/RR14/Vt600/PEEP 5/FiO_2 100%," however, these orders ignore important facets of care that might prove to be beneficial to our patients. Most ventilators can be set to achieve goals of spontaneous breathing, volume-targeted ventilation, pressure-targeted ventilation, or some combination. In volume-targeted (also known as volume-cycled or volume assist/control) ventilation, the ventilator is set to reach a preset volume regardless of the pressure required to do so. Pressure-targeted modes are set to reach a preset pressure independent of volume generated. Dual modes combine the benefits of both strategies.

The respiratory rate and tidal volume determine a patient's minute-ventilation. Patients intubated for airway protection due to trauma or toxicology often do well with a normal minute-ventilation and as such, initially setting respiratory rates at 10–14 breaths/minute and the tidal volume at 7–8 mL/kg ideal body weight (IBW) is usually sufficient. Adjustments can be made based on arterial blood gas (ABG) analysis, end-tidal CO_2, or venous blood gas and pulse oximetry. Patients who are septic or have a severe acidosis often require a higher minute-ventilation. Respiratory rates can be increased, as can tidal volume; however, volumes above 10 mL/kg IBW should not be used due to the risk of inducing ventilator associated lung injury. In addition, in special scenarios such as acute lung injury (ALI) and acute respiratory distress syndrome (ARDS), initial tidal volumes should be set as low as 6 mL/kg IBW. Some of these specific scenarios are discussed later in this article.

Although the fraction of inspired oxygen (FiO_2) is usually initially set at 1.0, most agree that quickly titrating down is beneficial due to the theoretic risk of oxygen toxicity. Adjustments can be made based on ABG or pulse oximetry, with a goal of keeping an arterial PO_2 above 60 mmHg or a arterial oxygen saturation above 90%. Extrinsic PEEP is typically set at 5–8 cmH_2O. It is used to offset the gradual loss of functional residual volume (FRC) in the supine mechanically ventilated patient. Care should be exercised when PEEP levels >8 cmH_2O are used in the setting of elevated intracranial pressures [1], unilateral lung processes, hypotension, hypovolemia, or pulmonary embolism.

Flow rate and waveform should be considered in the discussion with the respiratory therapist. The flow waveform is usually set to decelerate in an effort to optimize recruitment due to different time constants in the lung. Trigger mode can be set to detect either pressure or flow gradient. This

should be set such that the patient can trigger the ventilator without great effort. If the trigger is set too high (not sensitive enough), the work of breathing incurred by the patient can be substantial. By convention, many ventilators are set to a flow trigger with a sensitivity of 1–3 cmH_2O [2]. If, the sensitivity is set too low (too sensitive), the ventilator can ''auto-trigger'' because of oscillating water in the ventilator tubing, or by hyperdynamic heartbeats, or when the patient moves.

Spontaneous breathing

Spontaneously breathing patients can be supported on the ventilator by pressure support ventilation (PSV). In this mode, the ventilator provides a supplemental inspiratory pressure to each of the patient generated breaths. The clinician sets the FiO_2 and PEEP. The patient sets the respiratory rate and generates their desired flow rate. The applied pressure is turned off once the flow decreases to a predetermined percentage. The tidal volume is dictated by the pressure support given, patient effort and the lung compliance. There is no minimal rate, although there is a back-up apnea rate in most modern ventilators.

Volume-targeted (control) modes

Volume-targeted modes are the most commonly used and the most familiar mode of mechanical ventilation. As its name implies, ''volume'' (ie, tidal volume) is the ventilator's targeted parameter. The ventilator will generate the necessary driving pressure to reach this ''target.'' In addition to tidal volume, the clinician sets a desired respiratory rate, FiO_2, and PEEP. It should be noted that other important aspects of the mechanical ventilator can be controlled in this setting, such as waveform (decelerating or square), inspiratory/expiratory ratio (I:E ratio), flow rate, and trigger mode. However, many clinicians fail to consider these factors in their ventilator management.

Both assist control (AC) and synchronized intermittent mechanical ventilation (SIMV) can be set as volume-targeted modes. In these modes, both will provide the desired tidal volume at a preset, minimum respiratory rate; however, if the patient triggers the ventilator at a rate greater than the set rate, these two strategies diverge. In AC, each breath above the set respiratory rate will result in a full mechanically supported breath at the set tidal volume. In SIMV, the ventilator will only give the set number of breaths at the clinician selected tidal volume and each additional breath will require the patient to generate a spontaneous tidal volume without mechanical assistance. Furthermore, a patient-generated breath must overcome any resistance caused by the artificial airway and/or ventilator circuitry. Pressure support can be added to SIMV for patient-generated breaths in efforts to alleviate or reduce any increase in work of breathing related to resistance imposed by the ventilator circuit and endotracheal tube (see below). Assist

control and SIMV, set at rates greater than the patient's intrinsic respiratory rate, are virtually identical.

Caution should be exercised to avoid auto-PEEP (also known as breath-stacking) when using volume-targeted AC modes. Because each mechanically delivered breath is given at full tidal volume, patients with a high respiratory rate on AC may not have sufficient time to completely exhale between breaths. This results in progressive air trapping, leading to an increase in auto (intrinsic) PEEP.

Pressure-targeted (control) modes

As its name implies, "pressure" is the ventilator's targeted parameter. The ventilator will generate an inspiratory pressure that has been set by the clinician. The resulting tidal volume will be a function of respiratory mechanics. In addition, the clinician sets the desired PEEP, respiratory rate, FiO_2, and inspiratory time or I:E ratio, and trigger mode. It should be noted that the clinician does not control waveform or peak inspiratory flow. As with volume-targeted ventilation, pressure-targeted ventilation can be set to either an AC or SIMV mode. Pressure-targeted modes might have better pressure distribution and improved dissemination of airway pressures [3] and their use has been growing in popularity. In this mode, the decelerating waveform optimizes distribution of ventilation.

The main drawback with this strategy is that any change in the system compliance and/or resistance will affect the generated tidal volume. For example, if the patient bites on the endotracheal tube or a mucus plug develops, the set pressures that were generating an adequate volume will no longer do so. In contrast, a sudden increase in system compliance might result in the generation of tidal volumes that may be considerably larger than desired. Thus, instead of the traditional pressure alarm limits, one must adjust and be cognizant of tidal volume and minute-ventilation alarm settings. Uncertainties like these have led many clinicians to favor volume targeted strategies or dual controlled strategies in the acute care setting.

Alternative ventilator strategies

Synchronized intermittent mechanical ventilation plus pressure support ventilation

Due to the increased work of breathing imposed by the ventilator circuit and endotracheal tube, SIMV plus pressure support ventilation (PSV) was developed. This strategy supplies inspiratory pressure during spontaneous breaths. In a way, this became the first use of a dual targeted mode. This mode was initially recommended by those who felt that as a patient's need for mechanical ventilatory support decreased, the set respiratory rate could be decreased and thus the patient "weaned" to pressure support

ventilation alone and ensuing extubation. However, subsequent data has shown that this method of liberation actually increases the number of ventilator days (patient days on mechanical ventilation) [4].

Dual control modes

These new modes use a closed-loop ventilator logic that combines the features of volume and pressure targeted ventilation. These modes automatically alter control variables, either breath-to-breath or within a breath, to ensure a minimum tidal volume or minute-ventilation [5].

Breath-to-breath

Pressure-Regulated Volume Control (PRVC) (MAQUET, Servo 300 and Servo-i; VIASYS Healthcare, AVEA), AutoFlow (Drager Medical, Evita 4), Volume Ventilation Plus (VV+) (Covidien, Purtian Bennett 840), Adaptive Support Ventilation (ASV) (Hamilton Medical- Hamilton G5, Hamilton C2, Galileo, Raphael), and Variable Pressure Control (VPC) and Variable Pressure Support (VPS) (Cardiopulmonary Corporation, Venturi Ventilator) are all very similar proprietary modes. All use a pressure-targeted logic in which the ventilator determines after each breath if the pressure applied to the airway was sufficient to deliver the desired tidal volume. If, for example, the tidal volume did not meet the set target, the ventilator will adjust the pressure applied to the airway on the next breath. These modes can be thought of as pressure control on ''autopilot.'' Some have described this as having a computerized respiratory therapist at the bedside at all times. Unfortunately, guidelines for the use of these modes have yet to be established. A concern with these modes is that they might spend an inordinate amount of time chasing the patient in an effort to deliver a desired tidal volume. In addition, if the patient begins to generate higher flows, as with pain or anxiety, the ventilator may misinterpret this effort and inappropriately decrease its output, thus increasing the patient's work of breathing.

Within a breath

Volume Assured Pressure Support Ventilation and Pressure Augmentation

These modes, Volume Assured Pressure Support Ventilation (VAPSV) (VIASYS Healthcare, Bird 8400STi) and Pressure Augmentation (VIASYS Healthcare, BEAR 1000), alter the control parameter ''within'' the inspiratory cycle of a breath. The clinician sets a pressure-support level, peak flow and tidal volume. Thus, the ventilator provides an inspiratory support and inspiratory demand flow in the same manner as with PSV, but will adjust the inspiratory pressure to obtain assured tidal volume delivery if the set tidal volume is not achieved with initial settings.

Other modes

High frequency ventilation

High-frequency ventilation (HFV) attempts to achieve adequate gas exchange by using asymmetrical velocity profiles when combining very high respiratory rates with tidal volumes that are smaller than the volume of anatomic dead space. It is used more commonly in neonates and infants with neonatal respiratory failure. There has been a renewed interest in using HFV in adult patients with ALI/ARDS, with the rationale that the small tidal volumes may cause less ventilator associated lung injury. However, more trials are necessary to determine if HFV can improve mortality outcomes in these patients [6].

Airway Pressure Release Ventilation and BiLevel Ventilation

Both of these modes, Airway Pressure Release Ventilation (APRV) (Drager Medical, Evita 4) and BiLevel Ventilation (Covidien, Puritan Bennett 840), are proprietary names, yet function essentially in the same manner. The clinician sets a pressure high, pressure low, and a time at each level (time high and time low). Ventilation occurs during the release from pressure high to pressure low. The time low is typically 0.2–0.8 seconds in restrictive lung disease and 0.8–1.5 seconds in obstructive lung disease. It is probably most prudent to start at 0.8 and titrate to meet individual patient requirements. The time low is also referred to as the release phase [7]. A common occurrence in this mode is setting the time low too long. This essentially mimics a pressure-targeted SIMV strategy. The patient typically spends 4–6 seconds in time high. In the paralyzed patient APRV/BiLevel is identical to pressure targeted inverse ratio ventilation. For these reasons, some have described this mode as inverse ratio ventilation (IRV). However, a major difference between APRV/BiLevel and IRV is that IRV typically requires chemical paralysis or heavy sedation. APRV/BiLevel allows for spontaneous breathing throughout both pressure levels, making it relatively more comfortable and typically does not require heavy sedation or paralysis. APRV/BiLevel has gained popularity in patients with hypoxemic respiratory failure because it improves oxygenation by optimizing alveolar recruitment and ventilation/perfusion (V/Q) matching [8].

Proportional Assist Ventilation Plus and Proportional Pressure Support

Proportional Assist Ventilation Plus (PAV+) (Covidien, Puritan Bennett 840) and Proportional Pressure Support (PPS) (Drager Medical, Evita 4) are forms of synchronized ventilatory assistance, where the ventilator generates pressure in proportion to the patient's effort, ie, the greater the effort, the more pressure the ventilator generates. Therefore the clinician determines the level of resistive and elastic unloading, irrespective of patient volume or flow requirements.

Specific scenarios

Asthma and chronic obstructive pulmonary disease

Approximately 4% of patients that are hospitalized for asthma require mechanical ventilation [9]. Patients with obstructive pulmonary processes that require intubation can at times be very difficult to manage. Mortality rates for respiratory failure due to status asthmaticus before 1990 were reported to be as high as 38% [10]. However, there has been a dramatic drop to an estimated 7% in 2000 [9]. It is thought that the acceptance of permissive hypercapnia and improved understanding of the potential complications from mechanical ventilation, ie, auto-PEEP and ventilator induced lung injury (VILI), has lead to this decline [11].

The peri-intubation period can be quite dangerous and requires caution to prevent fatal events. These patients are often volume depleted and induction agents can precipitously drop blood pressures. Pre-induction crystalloid boluses of 500 mL to a liter are often prudent. In addition, care should be taken to prevent breath stacking and auto-PEEP. Patients typically have some level of hyperinflation and auto-PEEP at the time of intubation. Overzealous bag ventilation can lead to worsening of this auto-PEEP, potentiating hypoxia, hypotension, and VILI. In addition, auto-PEEP causes the patient's work of breathing to increase because the patient has to generate a larger drop in airway pressure to initiate a breath. For example, a patient with an auto-PEEP of 10 cmH_2O and a set pressure trigger of -2 cmH_2O (and no applied PEEP) has to generate an alveolar pressure of 12 cmH_2O to generate a breath. Assisted breaths should be limited to less than 8–10 breaths per minute and adequate time should be allowed for exhalation. The primary goal is to allow expiration of trapped air and minimize airway pressures. This is done by decreasing the inspiratory time and increasing the expiratory time. Strategies that can be implemented to achieve this goal include reducing the respiratory rate, reducing tidal volume (initially setting 6–7 mL/kg IBW), increasing the flow-rate (initially setting at 80–100 L/min) and using a square waveform. Several of these options will subsequently increase the peak inspiratory pressure; however, this is well tolerated below pressures of 50 cmH_2O [11,12] and is not a reliable marker of hyperinflation or the risk of VILI [13]. In addition, this strategy may worsen hypercapnia due to a decrease in minute-ventilation. This is termed ''permissive hypercapnia''.

As the treatment (β-agonists, steroids) takes affect, the peak pressures will begin to lower, the peak expiratory flow rate (PEFR) should increase and the expiration time (T_E) should become shorter (Fig. 1). These strategies to increase the expiratory time and shorten inspiratory time must be balanced with hypercapnia. Allowing patients to develop severe respiratory acidosis from permissive hypercapnia can result in cardiac arrhythmias and death. Sodium bicarbonate infusion or the administration of THAM (tris-hydroxymethyl aminomethane) may be required to keep the arterial pH at

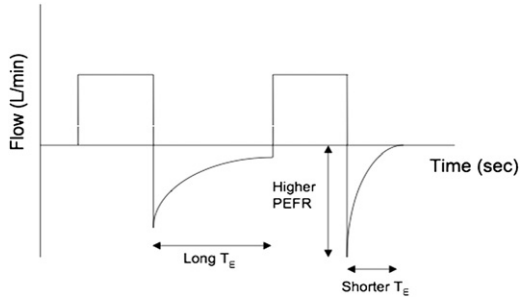

Fig. 1. Schematic of flow-time curve showing improvement in obstructive process. Note that peak expiratory flow rate (PEFR) increases and expiratory time (T_E) decreases with improvement.

a safe level above 7.15 to 7.20. Finally, it is necessary to adequately sedate these patients, as their hypercapnic state is a powerful stimulus to breath rapidly. Opiates such as fentanyl and sedatives such as propofol have gained increased roles in these patients. Occasionally, one will be required to chemically paralyze these patients to keep their respiratory rate and thus their expiratory time controlled. This should be a final option as steroids are part of the treatment in obstructive airway disease and the side effects of steroids and paralytics can be quite devastating [14,15]. However, paralysis cannot be avoided in certain cases.

Valuable information can be gained from flow-time curves and pressure-time curves. Improvement (see Fig. 1) or worsening of airway obstruction and air trapping (Fig. 2) can often be seen before becoming clinically apparent. In ideal situations, intrinsic PEEP (auto-PEEP) can be measured by using an end-expiratory hold (Fig. 3). This measurement is often inconsistent and difficult to obtain. Some authors have recommended that extrinsic PEEP be applied at 80% of the measured auto-PEEP [16]. However, due to the difficulties of accurately measuring auto-PEEP and the potential hazard of adding too much extrinsic PEEP [17], others have suggested that it should

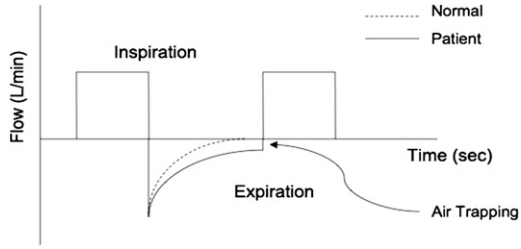

Fig. 2. Schematic of flow-time curve showing air trapping. Note that patient has not finished full exhalation prior to the initiation of the next breath. Square flow waveform is being used in this example.

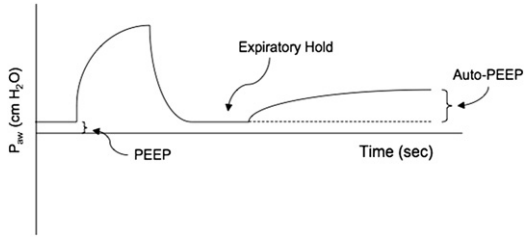

Fig. 3. Schematic of pressure-time curve showing an expiratory hold and subsequent auto-PEEP measurement.

be set at 50% of the measured intrinsic PEEP. If auto-PEEP becomes severe enough, it will begin to affect the end-inspiratory plateau pressures. Current recommendations for obstructive airway disease are to keep the plateau pressures below 35 cmH_2O [18,19]; however many clinicians follow the ALI/ARDS recommendations and keep the plateau pressures less than 30 cmH_2O. Extrinsic (applied) PEEP can be used to decrease some of the negative pressure that the patient has to generate to initiate a breath. Going back to the previous example, a patient with an auto-PEEP of 10 cmH_2O and a set pressure trigger of -2 cmH_2O (and no applied PEEP) has to generate an alveolar pressure of 12 cmH_2O to generate a breath. Adding an extrinsic PEEP of 5 cmH_2O means that the ventilator will trigger a breath when the alveolar pressure is 3 cmH_2O. Therefore, the patient now only has to generate a decrease of 7 cmH_2O (instead of 12 cmH_2O), thus reducing the required inspiratory effort.

Finally, if sudden hypotension occurs, a first step in troubleshooting is to remove the patient from the ventilator. Often times this is both diagnostic and therapeutic for air trapping. In addition, tension pneumothorax must be considered (see below).

Acute lung injury and acute respiratory distress syndrome

The American–European Consensus Conference on Acute Respiratory Distress Syndrome (ARDS) defines this entity as a PaO_2/FiO_2 ratio <200 with bilateral pulmonary infiltrates on a chest radiograph consistent with pulmonary edema and no clinical evidence of left atrial hypertension or (if measured) a pulmonary capillary wedge pressure <18 mm Hg. Acute lung injury (ALI) has a similar definition with the exception being that it is a PaO_2/FiO_2 ratio <300 [20]. As recently as the 1990s, the mortality for patients with ARDS had been as high as 40%–50%. However, with the advent of lung-protective ventilation, mortality rates have decreased significantly. The ARDS Network showed that this strategy lead to a significant decrease in mortality [21]. Essentially, lung-protective ventilation attempts to prevent further damage to a patient's lung by avoiding overdistention

of alveoli. Tidal volumes of 6 mL/kg IBW are used in conjunction with higher respiratory rates (initially set at 18–22 breaths/minute) to achieve an adequate minute-ventilation. Plateau pressures are limited to 30 cmH_2O [22], and tidal volumes can be further incrementally decreased down to 4 mL/kg IBW to achieve this goal. Permissive hypercapnia is allowed and tolerated as long as arterial pH is kept above 7.20. Sodium bicarbonate infusion or the administration of THAM may be required to keep the arterial pH at a safe level above 7.15 to 7.20. In addition, FiO_2 and PEEP are adjusted in a step-wise fashion to assure adequate oxygenation. It should be noted that although both volume-targeted and pressure-targeted modes can be used, the only firm data showing improved outcomes in ARDS to date involved volume-targeted ventilation. Finally, central venous pressure monitoring should be used to guide fluid management [23,24].

In the early phases of a resuscitation, it is often difficult to ascertain if pulmonary edema is cardiogenic or noncardiogenic. However, in light of the mortality benefit seen with a lung protective ventilation strategy, its relative simplicity, and minimal complications, one should consider implementing this strategy early in a patient's clinical course.

A common finding in lung protective ventilation is the occurrence of patient–ventilator dysynchrony. This dysnchrony is thought to be due to the patient wanting a higher flow rate than the ventilator is providing while on a volume-targeted strategy. This situation occasionally leads to double or triple cycling of the ventilator. It should be noted that in this situation, the patient is actually receiving higher tidal volumes and not benefiting from lung protective ventilation. Sedation needs to be optimized and, at times, different modes like pressure-targeted modes may be attempted. In addition, temporarily weakening the patient with paralytics may be considered.

To date there are several areas of uncertainty in the mechanical ventilation of patients with ALI/ARDS. Patients with traumatic brain injury, intracranial hemorrhage, fulminant hepatic failure, and elevated intracranial pressures (ICP) who develop ARDS must be managed carefully as lung protective ventilation may induce hypercapnia. Acutely hypercapnia may lead to cerebral vasodilation and an increase in ICP. In addition, there is little evidence to support the recommendation of any particular rescue therapy in patients with severe refractory hypoxia, such as recruitment maneuvers, high dose albuterol, inverse ratio ventilation, high frequency ventilation, prone ventilation and extracorporeal membrane oxygenation. In dire circumstances, these modalities may be used based on clinician preference and expertise.

Troubleshooting

Respiratory distress in mechanically ventilated patients has a broad differential that includes anxiety, pain, inadequate ventilator settings,

air-leak, endotracheal tube malfunction, pulmonary parenchymal process and extrapulmonary causes. Included in this list are: the acute life-threatening entities of tension pneumothorax and severe auto-PEEP. The troubleshooting process will be guided by the severity of the distress and the stability of the patient.

If the patient is hemodynamically stable a systematic approach can be taken. Obtain a focused history from the bedside nurse and respiratory therapist, perform a focused physical examination, check the ventilator, the circuit and respiratory mechanics (note current and past peak and plateau airway pressures), evaluate the chest x-ray and examine the pleural cavity with bedside ultrasound. This systematic approach will typically yield the cause of the distress.

Respiratory mechanics can be used to guide the troubleshooting process. Current peak pressures (P_{peak}) and plateau pressures (P_{plat}) should be compared with previous values when the patient was stable. This should be done in volume-targeted modes. Airway pressures are a function of volume and respiratory system compliance. A set volume and compliance results in a specific pressure. Peak pressures are obtained during inspiration and thus incorporate resistance to airflow. Plateau pressures are obtained during an inspiratory pause eliminating airflow and thus reflect pulmonary system compliance. An isolated increase in peak pressures (ie, an increase in $\Delta P_{peak}–P_{plat}$) points to a problem with airflow (Fig. 4). An increase in the plateau pressures leading to an unchanged or decreased $\Delta P_{peak}–P_{plat}$ points to a change in lung compliance (Fig. 5). Table 1 lists common causes of each.

Patients who are simply asynchronous with the ventilator should be evaluated for sufficient sedation, appropriate trigger mode and sensitivity, inspiratory time, tidal volume, and flow rate. Double-cycling (back-to-back ventilator delivered breaths) is typically seen in volume-targeted modes when the patient desires a higher flow rate than the set rate. Increasing

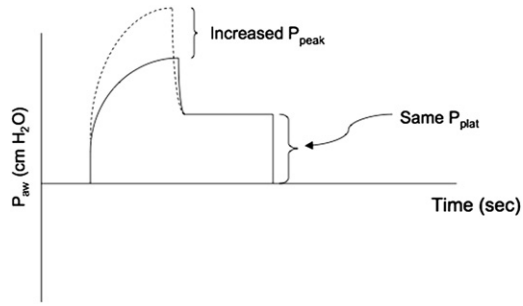

Fig. 4. Schematic of two superimposed pressure-time curves showing an isolated increase in peak inspiratory pressures (P_{peak}) with no change in plateau pressures (P_{plat}). This is characteristic of obstruction and increased airway resistance (see Table 1).

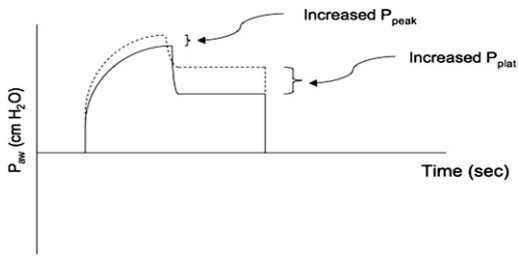

Fig. 5. Schematic of two superimposed pressure-time curves showing a small increase in peak inspiratory pressures (P_{peak}) with a greater increase in plateau pressures (P_{plat}). This is characteristic of decreased lung compliance (see Table 1).

the flow rate may alleviate this "air hunger." It is also commonly seen in patients on lung protective ventilation with low set tidal volumes. If increasing the flow rate does not improve synchrony and sedation is deemed to be adequate, one must decide between increasing the tidal volume or changing to a pressure-targeted mode. In addition, temporarily weakening the patient with paralytics may be considered.

Patients who become acutely hemodynamically unstable while on mechanical ventilation should immediately be removed from the ventilator and hand-ventilated with 100% oxygen. Special care should be taken with this maneuver in patients on high PEEP or on nitric oxide (NO). PEEP valves can be used to maintain recruitment if auto-PEEP has been ruled out and NO should not be acutely discontinued. Severe decompensation or continued deterioration requires quick action and empiric treatment of potentially life-threatening problems. Severe auto-PEEP should be considered and care should be taken not to worsen this process. Patients on volume-targeted modes, with an obstructive or reactive airways disease, receiving a high minute-ventilation, or on inverse ratio ventilation are at high risk for auto-PEEP. Patients should not be hyperventilated and special attention should be paid when the patient is initially disconnected from the ventilator. A prolonged expiration of trapped air with clinical improvement can be diagnostic of this process. Tension pneumothorax should also be considered and addressed by needle decompression, followed directly by tube thoracostomy. If time allows, a quick history and physical examination can potentially guide the decision of which side of the chest to needle decompress. Knowledge of a recent central line attempt, chest tube placed on water seal, or decreased breath sounds on one side may be helpful. Often times the history and physical examination is not be helpful and decompressing both sides may be necessary based on the condition of the patient.

If the patient quickly improves with manual ventilation (and auto-PEEP is excluded), then the likely culprit is the ventilator settings or circuit. Consider adjusting the settings and check the circuit for air leaks or condensation that has accumulated and is causing interference.

Table 1
Respiratory mechanics in mechanically ventilated patients

Increase in ΔP_{peak}–P_{plat}	Unchanged or decreased ΔP_{peak}–P_{plat}
• Increased airway resistance caused by heat and moisture exchanger (HME)	• Pneumonia
• Patient biting endotracheal tube	• Atelectasis
• Kinked or twisted endotracheal tube	• Mucus plugging of one lung
• Obstruction of endotracheal tube by secretions, mucus, blood	• Unilateral intubation
• Bronchospasm	• Pneumothorax
• Obstruction of lower airways	• Pulmonary edema (noncardiogenic and cardiogenic)
	• Abdominal distention/pressure

If the patient stabilizes with manual ventilation, note if any air is escaping through the mouth or nose and pay close attention to the resistance faced with each manual ventilation. Escaping air or an expiratory volume significantly smaller than the set tidal volume should lead to an inspection of the endotracheal tube positioning (ie, has it migrated out) and inspection of the pilot balloon. A deflated pilot balloon typically indicates a deflated cuff. Occasionally, adding more volume to the cuff may correct the problem. However, more commonly either the cuff has ruptured or the cuff apparatus has failed. At times the pilot balloon mechanism is the culprit and some respiratory therapists can easily replace the pilot balloon with commercially available kits. This may be a good starting point in a stable patient deemed difficult to reintubate, yet failure of the cuff apparatus typically requires replacement of the endotracheal tube. If there is a lot of resistance with manual ventilation—and auto-PEEP and pneumothorax have been ruled out—attempt to pass the suction catheter. Difficulty passing the suction catheter should prompt one to evaluate if the patient is biting on the endotracheal tube, or if the tube is kinked, twisted, obstructed, or out of position.

Finally, patients who are accidentally extubated (for example, during transport) or have an unplanned extubation (self-extubate) should promptly be reintubated if they are felt to be at high risk for respiratory failure or airway compromise. Of note, these patients should be treated as presumed difficult intubations, as accidental and unplanned extubations are notorious for causing trauma to the glottis leading to vocal cord edema. In a recent series, 22% of patients who failed unplanned extubation required multiple laryngoscopic attempts, 14% suffered hypoxemia, and 14% suffered esophageal intubations. One patient was unable to be ventilated and subsequently died [25]. Careful planning and preparation for difficult airway management can avert disasters and the need for emergent surgical airway.

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