CHAPTER 3

The Crashing Ventilated Patient

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IN THIS CHAPTER

Evaluation of hemodynamically unstable ventilated patients in distress
Management of hemodynamically unstable ventilated patients
Special scenarios: pediatric patients and patients with a tracheostomy

Introduction

Critically ill patients present to the emergency department every day. Some of them required intubation in the prehospital setting; others are intubated on arrival or during emergency department evaluation. After their airways have been secured and their conditions stabilized, patients can remain in the emergency department because of a lack of critical care beds. In some hospitals, a dedicated intensivist will take charge of these patients, but in others this type of coverage is not available at all or not around the clock. Thus, acute complications or deteriorations must be handled by emergency physicians.

This chapter provides a framework for managing the crashing mechanically ventilated patient. The information provided will assist the practitioner in determining if the patient’s condition is related to the underlying pathology that necessitates mechanical ventilation or if it is being caused by mechanical ventilation itself. This chapter does not fully address basic ventilator management, noninvasive positive-pressure ventilation, advanced trauma life support, or advanced cardiac life support (ACLS).

Determine Hemodynamic Stability

The initial step in managing the crashing ventilated patient is to determine the patient’s hemodynamic stability. The ventilated patient is, by definition, critically ill. However, he or she can fall anywhere on the spectrum from being ventilated for airway protection, with normal vital signs, blood pressure, and oxygen saturation, to being ventilated and in cardiac arrest. Determining where the patient is on this spectrum, including assessing for hypotension and hypoxia, will dictate how much time the practitioner has to implement rescue strategies. In addition, it is important to anticipate the patient’s clinical course. The approach to a patient who is intubated for hypoxia stemming from pneumonia and whose blood pressure and oxygenation gradually trend down over hours or days is different from the approach to a patient who is declining over a span of minutes (Figure 3-1).

As a general rule, the following evaluation should be performed within 1 hour on patients with new unexplained hypotension (systolic blood pressure [SBP] <90 mm Hg), new unexplained hypoxia (SaO₂ <90%), or a new marked change in vital signs (a drop in SBP by more than 20 points or a drop in SaO₂ by more than 10%). Patients with stable SBP between 80 and 90 mm Hg and SaO₂ between 80% and 90% should be evaluated expeditiously, with the hope of halting the decline. Those with SBP <80 mm Hg or an SaO₂ <80% and those who continue to decline rapidly should be evaluated quickly, with consideration given to entering the cardiac arrest/near arrest algorithm. These
FIGURE 3-1.
The crashing ventilated patient algorithm. The steps are discussed in more detail in the main text. For the differential diagnosis in difficult-to-ventilate patients, see Table 3-2.

- Cardiac arrest/near arrest
  - Determine hemodynamic stability
  - Rush of air, improvement
    - Step 1: Disconnect from ventilator.
      - No improvement
        - Step 2: Hand ventilate with 100% oxygen.
          - Look for unequal chest rise
          - Listen for air leak and unequal breath sounds
          - Feel for difficulty to ventilate and crepitus
        - No improvement
          - Step 3: Determine that the ET tube is functioning and in proper position.
            - Pass suction catheter
            - Pass intubating stylet or direct visualization
          - ET tube functioning and in proper position
            - Step 4: Special procedures: ultrasound, chest radiograph, needle decompression
          - ET tube not functioning or not in proper position
            - Evaluate for ET tube position adjustment, exchange, or re-intubate.
    - Likely auto-PEEP*: check settings and ventilator
      - Improvement, unclear if auto-PEEP
        - Check settings and ventilator.
      - Improvement
        - Step 1: Obtain a focused history.
          - Step 2: Perform physical examination.
        - Step 3: Check gas exchange.
          - Step 4: Check respiratory mechanics.
            - Step 5: Observe waveforms.
            - Step 6: Evaluate chest radiograph and ultrasound image.
            - Step 7: Evaluate sedation.

*For auto-PEEP, see Table 3-1.
*For air leak, see Figure 3-2.
*For needle decompression, see Key Point, page 20.
FIGURE 3-2.
Approach to the ventilated patient with an air leak

Cardiac arrest/near arrest

Determine whether the ET tube is in the trachea
Intubating stylet
Direct visualization
Fiberoptic scope (if time allows)
Be prepared to re-intubate.

Not in the trachea
Re-intubation is required.

Stable/near stable

Determine hemodynamic stability.

Feel the pilot balloon.
Note if it is deflated.

Add air (2-5 mL) to the pilot balloon.
If this stops the air leak, document that air was added to the balloon.

If air leak persists, the pilot balloon does not inflate, or the pilot balloon deflates with time and the air leak returns with time, there is a defect in the pilot balloon-cuff apparatus or the ET tube has migrated out of the trachea.

Determine the ability to repair the pilot balloon mechanism with commercially available kit.

If air leak persists after repair or if repair is not possible, the ET tube must be replaced.
values are arbitrary demarcation points and do not take precedence over clinical judgment.

PEARL

The initial step in managing the crashing ventilated patient is to determine the patient’s hemodynamic stability.

KEY POINT

Important questions to ask:
- How stable is the patient?
- How rapidly is the patient deteriorating?
- How much time is there to determine the cause of the instability and address the problems?

The Cardiac Arrest/Near Arrest Patient

Time is of the essence in the patient with cardiac arrest or near arrest. ACLS algorithms should be implemented quickly. Additionally, there are some key points to remember in the ventilated patient who has a cardiac arrest or becomes acutely hemodynamically unstable. The emergency department practitioner should develop a step-wise approach in this situation. During each step, the practitioner should “look, listen, and feel” to run through the differential diagnosis.

During the stabilization of these patients, it is important to keep in mind the original pathology that necessitated intubation. The crashing ventilated patient could simply be growing worse from the primary pathology. The multi-trauma patient could have an intrathoracic or intra-abdominal catastrophe, and the septic patient could be deteriorating clinically from lack of source control.

However, it is also important to determine and address special circumstances that the ventilator can precipitate. The most significant of these are tension pneumothorax and severe auto-positive end-expiratory pressure (auto-PEEP). Tension pneumothorax can lead to marked hypotension because of decreased cardiac output and marked hypoxia from ventilation perfusion mismatch. Auto-PEEP (also referred to as intrinsic PEEP, breath stacking, or dynamic hyperinflation) is caused by trapped volume in the pulmonary system. If severe enough, it will eventually lead to increased intrathoracic pressure. This can cause hypotension and decreased cardiac output from decreased venous return as well as marked hypoxia from ventilation perfusion mismatch.

KEY POINT

Patients on volume-targeted modes with obstructive or reactive airway disease, those on volume-targeted modes receiving a high minute ventilation, and those receiving inverse-ratio ventilation are at risk for auto-PEEP (breath stacking).

PEARL

Tension pneumothorax and severe auto-PEEP are important causes of ventilator-induced hemodynamic instability.

In critically ill ventilated patients who develop respiratory distress and are hemodynamically unstable, the following steps will assist the emergency physician in determining the cause of decompensation (Figure 3-1).

Step 1: Disconnect the patient from the ventilator.

This is perhaps the easiest step to perform. It can be both diagnostic and therapeutic in the crashing ventilated patient. A quick rush of air or a prolonged expiration of trapped air from the endotracheal (ET) tube can be diagnostic of ventilator-induced auto-PEEP (Table 3-1). A few seconds of observation can determine if this is the case. Return of hemodynamic stability implies that the maneuver was successful.

Patients undergoing cardiopulmonary resuscitation (CPR) should not be connected to a ventilator. The intrathoracic pressure variations caused by CPR will trigger ventilator breaths at high rates if the ventilator is set on assist-control. Patients on inhaled nitric oxide should not be removed from the nitric oxide abruptly, and efforts should be made to quickly reestablish the supply through the bag-valve system. In addition, care should be taken when disconnecting patients who are on high PEEP such as those with acute respiratory distress syndrome (ARDS). Although it is important to disconnect the patient from the ventilator to address causes of auto-PEEP, derecruitment could occur and hypoxia could be worsened. Once auto-PEEP has been ruled out, PEEP valves may be used to maintain the extrinsic PEEP levels and thus avoid derecruitment. PEEP valves can be problematic in the markedly hypotensive patient, as they could increase intrathoracic pressures and thereby decrease venous return.

PEARL

Inhaled nitric oxide should not be discontinued abruptly because this could cause rebound pulmonary hypertension. Administration should be reestablished quickly through the bag-valve system.

| TABLE 3-1. |
| Managing auto-PEEP |

| Determine what caused the auto-PEEP |
| - High set respiratory rate, high patient respiratory rate, obstructive airway disease |

| Consider decreasing the tidal volume in patients with obstructive or reactive airway disease |
| - Consider decreasing the set respiratory rate |
| - Will be ineffective in assist-control mode with a high intrinsic rate |

| Optimize sedation |
| - Use opiates to control respiratory rate |

| Monitor ventilator flow-time waveform |
| Consider changing to synchronized intermittent ventilation |
| Consider chemical paralysis |
Step 2: Breathing—Hand ventilate with 100% oxygen.

Ensure that 100% oxygen is being delivered and limit the respiratory rate to 8 to 10 breaths per minute. Particular attention should be given to the delivery of hand ventilation. Inadvertent rates as high as 40 breaths per minute are often used in codes. Excessive rates will increase intrathoracic pressures, leading to a decrease in venous return and cardiac output. Look at both sides of the chest to determine if there is equal chest rise. Unequal chest rise can signify a main-stem intubation, pneumothorax, or mucus plug. Listen for air escaping from the mouth or nose (a sign of an air leak). Listen over the epigastriac area and in both axillas. Decreased breath sounds could indicate main-stem intubation, pneumothorax, or atelectatic lung. Feel for subcutaneous crepitus (a sign of pneumothorax) and assess for difficulty in hand ventilating (a sign of low dynamic or static respiratory system compliance [Table 3-2]).

Step 3: Airway—Determine that the endotracheal tube is functioning and in the proper position.

The ET tube function by providing a conduit to the lower trachea. Its cuff attempts to create a seal between it and the inner wall of the trachea. To determine if it is functioning properly, pass the suction catheter and listen for an air leak (Figure 3-2). Easy passage of the suction catheter does not guarantee that the ET tube is in the trachea because the catheter could be passing down the esophagus; however, if it is difficult or impossible to pass the suction catheter, the tube is either dislodged, obstructed, or twisted or the patient is biting the tube. Attempt to correct a twisted or bent ET tube by repositioning the patient’s head: if the patient is biting on the tube, insert a bite block. Dislodged or obstructed ET tubes require re-intubation. Patients with dislodged tubes should be treated as difficult intubations because unplanned extubations are notorious for causing trauma to the glottis, eading to vocal cord edema.

In the cardiac arrest or near arrest patient, the best choice for determining that the ET tube is in the proper position is direct visualization of the tube passing through the cords. This step is often omitted in the crashing ventilated patient because of the belief that the tube has not migrated. Unfortunately, unrecognized ET tube migration can occur during routine care of the critically ill patient. Patients are frequently moved in and out of EMS vehicles, transferred to and from stretchers for imaging studies, and turned for procedures or bathing, all of which can dislodge the tube. This visualization step can be performed while providing hand ventilation.

Other simple techniques may be used to confirm that the ET tube is in the trachea. Direct visualization of the carina with a fiberoptic scope is an option, but this device is typically not readily available in an emergency department. Another quick and readily available technique is to pass an intubating stylet (gum elastic bougie or Eschmann introducer) gently through the ET tube. If resistance is met at 30 cm, the ET tube is in the trachea. If, however, the stylet passes beyond 35 cm without resistance, the tube is likely in the esophagus. If resistance is met too soon, the intubating stylet may be catching on the tube.

At least one of these techniques to determine proper positioning should be employed early enough in the code to correct any airway issues. In addition, improper positioning should be confirmed before simply removing the tube and re-intubating the patient, particularly if the patient is thought to have a difficult airway (unless it is glaringly evident that the patient is extubated).

PEARL

If it is difficult or impossible to pass the suction catheter, the ET tube is either dislodged, obstructed, or twisted or the patient is biting the tube.

KEY POINT

Passage of an intubating stylet (gum elastic bougie or Eschmann introducer) is a quick, simple, and readily available technique for confirming that the ET tube is in the trachea.

- Gently pass the intubating stylet through the ET tube—do not force it.
- Resistance should be encountered at approximately 30 cm.
- Passage of the stylet beyond 35 cm without resistance implies that the ET tube is in the esophagus.

Step 4: Special Procedures

If the patient is still in cardiac arrest or near arrest after being disconnected from the ventilator, ensuring proper placement of

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**TABLE 3-2.**

Causes of decreased respiratory system compliance

<table>
<thead>
<tr>
<th>Causes of high peak pressures (increased airflow resistance, decreased dynamic compliance)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Airway</strong></td>
</tr>
<tr>
<td>• Biting on the ET tube</td>
</tr>
<tr>
<td>• Bronchospasm</td>
</tr>
<tr>
<td>• Obstruction of the ET tube by secretions, mucus, blood</td>
</tr>
<tr>
<td>• Twisted ET tube</td>
</tr>
<tr>
<td><strong>Pulmonary</strong></td>
</tr>
<tr>
<td>• Partial mucus plugging</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Causes of high plateau pressures (low respiratory system compliance, decreased static compliance)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pulmonary</strong></td>
</tr>
<tr>
<td>• ARDS/acute lung injury (ALI)</td>
</tr>
<tr>
<td>• Atelectasis</td>
</tr>
<tr>
<td>• Auto-PEEP</td>
</tr>
<tr>
<td>• Mucus plugging</td>
</tr>
<tr>
<td>• Pneumonia</td>
</tr>
<tr>
<td>• Pneumothorax</td>
</tr>
<tr>
<td>• Pulmonary edema</td>
</tr>
<tr>
<td>• Unilateral intubation</td>
</tr>
<tr>
<td><strong>Chest wall</strong></td>
</tr>
<tr>
<td>• Chest wall rigidity</td>
</tr>
<tr>
<td>• Circumferential chest wall burn</td>
</tr>
<tr>
<td>• Obesity</td>
</tr>
<tr>
<td><strong>Other</strong></td>
</tr>
<tr>
<td>• Abdominal distention/pressure</td>
</tr>
</tbody>
</table>
the ET tube, and hand ventilating with 100% oxygen, a clinical decision will be required regarding needle decompression of the chest. If time permits, a focused history from the bedside nurse, respiratory therapist, or paramedic and a focused physical examination will indicate which side of the chest to decompress. In addition, depending on the urgency of the situation, bedside ultrasonography and chest radiography may be employed. The presence of a "lung-slide" artifact on bedside ultrasonography excludes pneumothorax. The lung slide artifact appears in M-mode as the "seashore sign" (Figure 3-3); its absence appears as the "stratosphere sign/bar-code sign" (Figure 3-4).8-10

At times, the clinical situation does not allow for imaging studies, and the focused history and physical examination may not be helpful. In these cases, needle decompression of both sides of the chest should be considered if other more likely causes of acute decompression are not found. It is important to remember that chest tube placement is required in patients after needle decompression.11-13

**PEARL**
Use ultrasonography to quickly evaluate for pneumothorax.

**KEY POINT**

**Needle Decompression**

- Determine which side to decompress first.
- Identify the second intercostal space, in the midclavicular line.
- Prepare the area with chlorhexidine if time permits.
- Anesthetize the area if the patient is conscious and time permits.
- Insert an over-the-needle catheter over the rib.
  - A 14-gauge catheter, at least 5 cm is preferred
  - May need a different size needle depending on the size of the patient
- Puncture the parietal pleura while listening for a sudden escape of air.
- Remove the needle while leaving the catheter in place.
- Secure the catheter with a bandage or small dressing.
- Prepare for chest tube thoracostomy.

**The Stable/Near Stable Patient**

If the patient is deemed stable or near stable or quickly regains stability after disconnection from the ventilator and hand ventilation, the event should be approached in a systematic manner (Figure 3-1). The patient should be placed on 100% oxygen during this evaluation.

**Step 1: Obtain a focused history.**

A focused history should be obtained from the practitioners most involved with the patient’s care (bedside nurse, respiratory therapist, resident, and paramedic). Valuable information includes the indication for intubation, the difficulty of the intubation, the depth of the ET tube, the ventilator settings, and recent procedures or moves (central line insertion; chest tube placement; removal or transition to water seal; thoracentesis; endotracheal tube manipulation; transport off stretcher; rotation for cleaning, a procedure, or chest radiograph).

**Step 2: Perform a focused physical examination.**

Take a general survey of the patient. Observe for agitation, attempts to pull at the ET tube and line, gasping for breath (the patient will have the mouth open and appear dyspneic), and tearing of the eyes.

**Airway.** Look at the ET tube, and determine if it has migrated from its previous position. It is possible that it has migrated out of the trachea or into a main bronchus. Adjust if necessary. Listen for escaping air (an air leak) from the mouth or nose (Figure 3-2). This typically signifies that the tube has lost its seal with the trachea and occurs in extubation or cuff failure. Feel the pilot balloon; if it is deflated, the cuff is deflated. Add air to the pilot balloon. If this stops the air leak, make a note that air was added to the balloon. If the pilot balloon does not inflate or deflates with time, there is a defect in the pilot balloon-cuff apparatus and the ET tube will likely need to be exchanged. Occasionally it may be possible to repair the pilot balloon mechanism with commercially available kits. This is a good option in patients who are difficult to intubate.

**PEARL**

If the pilot balloon does not inflate or deflates with time, there is a defect in the pilot balloon-cuff apparatus, and the ET tube will likely need to be exchanged.

Determine that the ET tube is functioning properly by passing the suction catheter. If it is difficult or not possible to pass the suction catheter, the endotracheal tube is either dislodged, obstructed, or twisted, or the patient is biting the tube. Attempt to correct a twisted or bent ET tube by repositioning the head; insert a bite block if the patient is biting on the tube. Dislodged or obstructed tubes require re-intubation.

If extubation is suspected at any point in the evaluation, determine that the ET tube is in proper position. Any of the techniques discussed in the previous section may be used.

**TABLE 3-3.**

<table>
<thead>
<tr>
<th>Initial ventilator settings for ALI/ARDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume-targeted, assist control</td>
</tr>
<tr>
<td>Tidal volume: 6-8 mL/kg ideal body weight</td>
</tr>
<tr>
<td>Can start at 8 mL/kg ideal body weight and work down to 6 within 4 hours</td>
</tr>
<tr>
<td>Respiratory rate: Set to approximate baseline minute ventilation (not to exceed 35 breaths/min)</td>
</tr>
<tr>
<td>PEEP: 5-8 cm H₂O</td>
</tr>
<tr>
<td>• Titrated up based on protocol</td>
</tr>
<tr>
<td>Fio₂: 100%</td>
</tr>
<tr>
<td>• Titrated down based on protocol</td>
</tr>
<tr>
<td>Flow rate: 60 L/min</td>
</tr>
<tr>
<td>Keep plateau pressures &lt;30 cm H₂O</td>
</tr>
</tbody>
</table>
KEY POINTS

- Is the ET tube in proper position?
  - Did it migrate out of the trachea?
  - Did it migrate down into the main bronchus?
- Is it functioning properly?

**Breathing.** Look at both sides of the chest to determine if there is equal chest rise. Unequal chest rise can signify a main-stem intubation, pneumothorax, or mucus plug. Look at the ventilator tubing and determine if there is an oscillating water collection. Listen for air escaping from the mouth or nose (a sign of an air leak). Listen over the epigastric area and in both axilla. Decreased breath sounds may provide clues regarding main-stem intubation, pneumothorax, or atelectatic lung. Feel for subcutaneous crepitus (a sign of pneumothorax).

**KEY POINT**
Dealing with whole-lung atelectasis:

- Use recruitment maneuvers
  - Disconnect from ventilator and provide hand ventilation at higher tidal volumes
- Provide frequent suctioning
- Rotate patient
- Perform chest percussion
- Administer bronchodilators
- Perform bronchoscopy

**Circulation.** Check for pulses, and cycle the blood pressure cuff frequently. If the patient has an arterial line, make sure the transducer is level. Determine the need for fluid bolus and/or vasopressors.

**Step 3: Assess gas exchange.**

Hypoxia can be diagnosed based on pulse oximetry if the waveform is reliable. The waveform should not be highly variable, and the frequency of the waveform should match the heart rate on the cardiac monitor. In a few instances, such as carbon monoxide poisoning, pulse oximetry is not reliable. In these cases, or if the pulse oximeter is not picking up, an arterial blood gas (ABG) sample should be obtained. Patients with a PaO2/FiO2 ratio less than 200 should be evaluated for ARDS. Those with a ratio between 200 and 300 should be evaluated for acute lung injury (AL). A lung-protective strategy should be implemented in those found to have ALI or ARDS (Table 3-3). Hypoventilation cannot be identified based on pulse oximetry; ABG measurement is beneficial in this event.

**PEAL**

Hypoventilation cannot be identified based on pulse oximetry; ABG measurement is beneficial in this event.

**Step 4: Check respiratory mechanics.**

Determine if the peak pressures and plateau pressures have changed from their previous values. These values should be obtained on volume-targeted modes. Airway pressures are a function of volume and respiratory system compliance. The respiratory system incorporates the ventilator circuit, ET tube, trachea, bronchi, pulmonary parenchyma, and chest wall. A set volume with a set system compliance results in a specific pressure. Peak pressures are a function of the volume, resistance to airflow, and respiratory system compliance. The plateau pressure is obtained during an inspiratory pause, thus eliminating airflow, and therefore reflects only the respiratory system compliance. An isolated increase in the peak pressure is indicative of increased resistance to airflow; an isolated increase in the

**FIGURE 3-3.**

Seashore sign. The lung-slide artifact appears in ultrasound M-mode as the seashore sign. This excludes pneumothorax. Image courtesy of Dr. Christine Butts and Dr. Matthew Bernard, Louisiana State University Health Sciences Center.

**FIGURE 3-4.**

Stratosphere/barcode sign. Absence of the lung-slide artifact is identified in ultrasound M-mode as the stratosphere/barcode sign. Pneumothorax cannot be excluded. Image courtesy of Dr. Christine Butts and Dr. Matthew Bernard, Louisiana State University Health Sciences Center.
plateau pressure is indicative of a decrease in respiratory system compliance (Table 3-2). Note that the plateau pressure can never be higher than the peak pressure and that if the plateau pressure rises, so will the peak pressure. It is important to keep in mind the relationship of the Δ (peak pressure – plateau pressure). Also note that these measurements assume a comfortable patient. Peak pressures and plateau pressures are not reliable in the “bucking” patient.\textsuperscript{17,18}

PEARL
Peak pressures and plateau pressures can be obtained only in volume-targeted modes.

**Step 5: Observe ventilator waveforms.**

The two most helpful ventilator waveforms are the flow-time curve and the pressure-time curve. The flow-time curve can be used to detect air trapping. The pressure-time curve can be used to determine plateau pressures with an inspiratory hold (Figure 3-5).

A notching in the pressure-time curve during inspiration can signify air hunger. In this situation, the patient desires a higher flow rate than the ventilator is delivering (Figure 3-6). It is commonly seen in volume-targeted modes. Increasing the flow rate will often alleviate this phenomenon. Another solution is to change to a pressure-targeted mode.

Double triggering can also be seen on ventilator waveforms. This occurs when the patient desires a higher tidal volume than the ventilator is set to deliver. The patient is still inspirating when the first breath has finished cycling and the ventilator immediately gives a second mechanical breath (Figure 3-7). This is frequently seen in low-tidal-volume ventilation, as used for patients with ARDS and status asthmaticus. It is important to recognize because the actual tidal volume being provided is essentially twice the set tidal volume. This has important ramifications for patients with ARDS and obstructive processes such as asthma and chronic obstructive pulmonary disease in which the goal is lower tidal volumes. Typically, improved sedation with emphasis on blunting the respiratory drive with opiates alleviates double triggering. Other adjustments that could help are increasing the flow rate, increasing the tidal volume by 1 mL/kg predicted body weight up to 8 mL/kg, or changing from a volume-targeted mode to a pressure-targeted mode.

**Step 6: Imaging Studies—Chest Radiograph and Bedside Ultrasonography**

Evaluate the chest radiograph for ET tube position, mainstem intubation, lung atelectasis, pneumothorax, and worsening parenchymal process. Bedside ultrasonography, if available, is typically quicker in evaluating for pneumothorax; however, it will not provide information on the location of the ET tube, lung atelectasis, or parenchymal processes (Figures 3-3 and 3-4).

**Step 7: Evaluate sedation.**

Some patients, such as those with drug overdoses or traumatic head injuries, may not require any sedation. Others may tolerate intubation quite well while almost fully awake. However, most patients require some form of sedation or analgesia to make the ET tube and ventilation tolerable.

Agents should be chosen based on the desired effect. If a patient appears agitated, sedative-hypnotics such as benzodiazepines, propofol, and dexmedetomidine should be used; however, it is important to note that these agents do not provide an analgesic component. If a patient is being given adequate sedative doses and still appears agitated, consider pain as a cause. For example, the agitated patient with a femur fracture and receiving high-dose benzodiazepines may simply need an opiate for pain control. Opiates that can usually be used are fentanyl, hydromorphone, and morphine. The goal of sedation and anesthesia in ventilated patients who are not being evaluated for extubation is to achieve a state in which the patient will arouse to gentle stimulation but will return to a sedated state when left alone. Patients who are being sedated and require deep stimulation to get a response are oversedated.

Patients who display air hunger and a high respiratory rate can be given a trial of opiates to relieve symptoms. Proper sedation and analgesia are paramount in patients being treated with a strategy that allows or induces hypercapnia such as those with status asthmaticus and ARDS. Hypercapnia is a powerful stimulus to the respiratory drive, and opiates are often required to control respiratory rates. Patients who tend to be difficult to control (besides those with status asthmaticus and ARDS) include those with hepatic encephalopathy or intracranial processes such as mass effect and hemorrhage. Chemical weakening with intermittently desired paralytics can be required if patients have had a good trial of sedation, analgesia, and ventilator changes and are still markedly tachypneic. Careful consideration should be given prior to this step, as prolonged paralysis
has been implicated in critical illness polyneuropathy. In addition, expert consultation should be obtained prior to prolonged paralysis of neurosurgical patients. The goal of chemical paralysis in these patients is to weaken them enough to control their interaction with the ventilator. Usually, this does not require a full dose of the paralytic.

Hemodynamic instability in mechanically ventilated and sedated patients can be a result of medication; sedatives and analgesics can precipitate or worsen hypotension. As a general rule, continuous infusions should be held in these cases. Patients who are hypoxic and agitated, but not hypotensive, could benefit from improved sedation. It is possible that their pulmonary status is so tenuous that they are agitated from the hypoxia and their condition is worsened by the oxygen consumption caused by their agitation. Patients who are agitated and hypotensive may respond well to a low-dose benzodiazepine and opiate if the agitation is a precipitant of hypotension. In all these cases, it is imperative to determine if sedation is a factor in the decompensation. Chemical paralysis should be reserved as a final option.

PEARL
Hemodynamic instability in mechanically ventilated and sedated patients could be a result of medication.

KEY POINT
The goal of sedation is a patient who arouses to gentle stimulation but returns to a sedated state when left alone. If deep stimulation is required to get a response, the patient is oversedated.

Special Scenarios
Two special scenarios should be mentioned. One is the crashing intubated pediatric patient, and the second is the patient with a tracheostomy. The previously described approach can be used in pediatric patients; however, there are a few caveats that can improve the approach. The first is to recognize that ET tube migration is common with small movements of the head and neck. A simple solution is to use a cervical collar for immobilization. Second, small ET tubes are often uncuffed and do not have a pilot balloon. Air leaks in this scenario should prompt the clinician to consider that the tube is either dislodged or too small. Finally, specialized equipment such as intubating stylets and fiberoptic scopes are typically not available for pediatric sizes.

Important questions that have ramifications for the care of the crashing ventilated patient with a tracheostomy are these: 1) Does the patient have a laryngectomy? 2) Why does the patient have a tracheostomy? and 3) How old is the tracheostomy? These are important questions because patients with a laryngectomy cannot be intubated orally, patients with a tracheostomy secondary to anatomic considerations or difficult/failed airway can be difficult to intubate orally, and the track in a patient with a recent tracheostomy (less than 1 week) may not have matured enough to safely reintroduce a tracheostomy tube.

<table>
<thead>
<tr>
<th>TABLE 3-4.</th>
<th>Special scenarios</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Children</strong></td>
<td>• Commonly, ET tubes migrate in and out of position with small manipulations of head position.</td>
</tr>
<tr>
<td></td>
<td>• Place a cervical collar to help stabilize head position.</td>
</tr>
<tr>
<td></td>
<td>• Document that the purpose is not for cervical spine protection.</td>
</tr>
<tr>
<td></td>
<td>• Small ET tubes are uncuffed.</td>
</tr>
<tr>
<td></td>
<td>• Most readily available intubating stylets and fiberoptic scopes are too large for pediatric ET tubes.</td>
</tr>
</tbody>
</table>

| **The Tracheostomy Patient – Unintentional Extubation** | • Determine if patient had a laryngectomy. |
| --- | – Oral intubation is an option if the patient did not have a laryngectomy. |
|  | • Determine reason for tracheostomy. |
|  | – Anatomic reason, difficult or failed airway – oral intubation may not be an option |
|  | – Traumatic brain injury, chronic respiratory failure – oral intubation may be an option |
|  | • Determine age of tracheostomy. |
|  | – <1 week – site may not be mature enough for manipulation; high risk of creating false tract |
|  | – Gently place a 6.0 ET tube in the stoma. |
|  | • May confirm with fiberoptic visualization. |
|  | • Stop if there is any resistance. |

| **The Tracheostomy Patient – Obstruction** | • Remove inner cannula and replace with same-sized cannula. |
Conclusion

Mechanically ventilated patients are typically the most critically ill patients that the emergency department practitioner will manage. The underlying disease process that required intubation is typically life threatening. When patients become unstable, the physician should take a step-wise approach toward determining if the patient is deteriorating because of the underlying disease process or because of interaction with the ventilator. It is hoped that the approach presented here will assist practitioners with a framework to evaluate and stabilize the crashing ventilated patient.

References