Workshop

on

Mechanical Ventilation

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Workshop on Mechanical Ventilation

Date: January 31, 2009
Time: 1.00 pm – 5.00 pm

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1. How to Intubate the Trachea in the Intensive Care Unit?

*MH Rao, Bippy Kath*

Tracheal intubation is one of the most common and important life-saving interventions performed in the intensive care units (ICUs). Securing the airway is the first step in all resuscitation protocols and forms the cornerstone in the management of critically ill patients. Intubation can be performed either through the oral or nasal route. Tracheal intubation presents unique problems when performed in the ICU setup, as most of the patients are decompensated and the procedure itself may need to be performed on an emergent basis, further jeopardizing the already existing critical scenario. The management of airway in these patients requires a high degree of knowledge, skill, expertise, and comfort in managing patients with limited physiological reserves.

**Assessment of the Patient Prior to Intubation**

Assessment and adequate preparation of the patient prior to intubation is crucial to ensuring successful and safe intubation. The pre-intubation assessment of patient consists of assessment of the underlying medical condition and airway assessment.

**Assessment of underlying medical conditions**

1. Cardiovascular factors
   - Myocardial infarction or ischemia
   - Hypovolemia
   - Cardiomyopathy
   - Arrhythmias
2. Neurologic factors
   - Raised intracranial pressure
   - Presence of intracranial bleeding, arteriovenous malformation, or aneurysm
   - Cervical spine disease
3. Drug allergies
4. Pulmonary factors
   - Hypoxemia, airway obstruction, or lung restriction
5. Aspiration risk
   - Nothing-by-mouth (NPO) status
   - Morbid obesity
   - Impaired gastric emptying or gastroparesis
   - Ileus or intestinal obstruction
   - Pregnancy
6. Coagulation parameters

**Assessment of airway**

Before approaching a patient requiring intubation, it is prudent to understand the airway anatomy. The term “airway” is used to denote the upper airway consisting of the nasal and oral cavities, pharynx, and larynx up to glottis. The normal airway may be altered by either congenital syndromes like Pierre-Robin syndrome, Treacher-Collins syndrome etc or acquired abnormalities like tumor, edema, trauma, etc. Knowledge of the normal anatomy helps us to identify patients with anticipated difficult airway and also facilitates uneventful tracheal intubation in routine cases.

Patients coming to the ICU may require tracheal intubation at one or other stage during their ICU stay, hence it is always prudent to do airway assessment and document the findings and make a note of the anticipated difficulties, if any, for future
The term 'difficult airway' is used to denote two situations - i) difficult mask ventilation; and ii) difficult intubation. Different methods of airway assessment have been devised over the years with different sensitivities and specificities but no single method has been shown to be infallible. Cooperative patients can be assessed using the Mallampati/Samsoon–Young classification of the oropharyngeal view (Table 1). ‘Cannot ventilate and cannot intubate scenario’ is a life-threatening situation and patient may rapidly desaturate, leading onto hypoxia, bradycardia, and cardiac arrest. This is a nightmare for the person who is attempting to intubate.

**Table 1: Samsoon Young’s modification of Mallampati classification**

<table>
<thead>
<tr>
<th>Class</th>
<th>Direct visualization, patient seated</th>
<th>Laryngoscopic view</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Soft palate, fauces, uvula, pillars</td>
<td>Entire glottis</td>
</tr>
<tr>
<td>II</td>
<td>Soft palate, fauces, uvula</td>
<td>Posterior commissure</td>
</tr>
<tr>
<td>III</td>
<td>Soft palate, uvular base</td>
<td>Tip of epiglottis</td>
</tr>
<tr>
<td>IV</td>
<td>Hard palate only</td>
<td>No glottic structures</td>
</tr>
</tbody>
</table>

For airway assessment in unconscious and uncooperative patients, the methods available to exclude difficult airway include:

1. Thyromental distance: measured along a straight line from tip of mentum to thyroid notch in neck-extended position (normal > 6 cm)
2. Head and neck movement: the range of motion from full extension to full flexion
3. Presence of a beard
4. Body-mass index > 26 kg/m²
5. Lack of teeth
6. Age > 55 years
7. History of snoring (from relatives)
8. Facial or cervical spine trauma

**Indications for Intubation**

The decision to intubate a patient should be taken by the clinician at the bedside after doing a scrupulous study of the clinical profile and the relevant laboratory data available. Decision on intubating patients receiving non-invasive ventilation is even more difficult.

**Table 2: Indications for tracheal intubation in ICU**

<table>
<thead>
<tr>
<th>System</th>
<th>Presentation</th>
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<tbody>
<tr>
<td>Circulatory</td>
<td>Cardiac arrest</td>
</tr>
<tr>
<td></td>
<td>Shock</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Pulmonary edema (not responding to non-invasive ventilation)</td>
</tr>
<tr>
<td></td>
<td>Respiratory failure</td>
</tr>
<tr>
<td></td>
<td>Acute respiratory distress syndrome</td>
</tr>
<tr>
<td></td>
<td>Refractory hypoxemia</td>
</tr>
<tr>
<td></td>
<td>Respiratory fatigue</td>
</tr>
<tr>
<td></td>
<td>Failed extubation</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Diminished mental status (Glasgow coma score &lt; 8)</td>
</tr>
<tr>
<td></td>
<td>Elevated intracranial pressure</td>
</tr>
<tr>
<td>Airway</td>
<td>Upper airway obstruction</td>
</tr>
<tr>
<td></td>
<td>Diminished airway reflexes</td>
</tr>
<tr>
<td></td>
<td>Pharyngeal instability</td>
</tr>
</tbody>
</table>
Endotracheal Intubation

Patient positioning
Alignment of the three axes viz., oral, pharyngeal, and laryngeal enables the visualization of glottic opening. An improperly positioned patient is a common cause of failed intubation. The head should be placed on a small pillow (about 10 cm). The head is then extended and the neck flexed to achieve the ‘sniffing position’. The bed should be moved away from the wall and the head of the bed removed.

Technique of orotracheal intubation
To a casual observer, the technique looks pretty simple, but it takes a fair degree of experience to accomplish it, especially in the not-so-ideal ICU setup. Thorough suctioning of oral secretions and nasogastric tube should be performed.

A team of four people is required to perform the procedure and skilled assistance is mandatory.
- Intubator: coordinates the procedure
- One person for drug administration, if required
- One person for applying cricoid pressure
- One person for manual in-line stabilization (cervical trauma)

Based on the patient’s hemodynamic and medical status, intubation can be performed by one of the three techniques:
1. Sedation with muscle paralysis
   - For conscious or semiconscious patients
   - Rapid sequence for emergency or full-stomach patients
2. Sedation with or without topical anesthesia
   - Hemodynamically unstable
   - Diminished mental status
   - Anticipated difficult airway
3. Without any adjuncts
   - Cardiac arrest
   - Unconscious patient

Pre-oxygenation
Critically-ill patients are highly oxygen dependent and hence to build up the oxygen reserves, patients are administered 100% oxygen for 3-5 minutes through a tight fitting face mask on spontaneous ventilation. Pre-oxygenation allows the patient to withstand an apneic period of 3-5 minutes during laryngoscopy and intubation, but this time may be reduced in critically-ill patients. In unconscious patients, this can be achieved by manual ventilation using a self inflating bag (Ambu bag) or Mapleson D circuit.

Sedation and paralysis
The commonly used drugs for sedation are benzodiazepines, opioids, propofol, thiopentone, ketamine, and etomidate. The two benzodiazepines used are midazolam (2-10 mg) or diazepam (5-10 mg). The opioids used include fentanyl (1-2 µg/kg) and morphine (5-10 mg). Propofol (1-2.5 mg/kg), thiopentone (2-5 mg/kg), ketamine (1-2 mg/kg), and etomidate (0.2-0.6 mg/kg) are anesthetic induction agents. Except etomidate, all these agents can affect the hemodynamics adversely and must be administered with caution and in titrated doses according to the response. The patient should be manually ventilated after administration of sedation to prevent hypoxia and hypercapnia.

The choice of muscle relaxants depends on whether the intubation is rapid sequence or elective. For emergency intubations succinylcholine hydrochloride (1-2 mg/kg) has been considered the gold standard till now. Intubation can be performed after one minute of drug administration. However, this drug is associated with many adverse effects like hyperkalemia, malignant hyperthermia, increased intraocular and intragastric pressures, bradycardia, and even asystole. Recently rocuronium bromide (0.6-1.2 mg/kg) has been shown to provide clinically acceptable intubating conditions and holds good promise in rapid sequence intubations. For elective intubations, vecuronium bromide (0.15-0.2 mg/kg) or atracurium besylate (0.4-0.5 mg/kg) is commonly used.
mg/kg) are equally effective and allow intubation to be performed after three minutes of drug administration. Muscle relaxants should be administered only after confirming that the patient can be manually ventilated.

Loss of muscle tone following sedation and paralysis can often lead to difficulty in ventilation due to tongue fall and loss of laryngeal tone. This can be effectively managed by triple maneuver of Rees (head tilt, chin lift, and jaw thrust) or by insertion of a proper sized oropharyngeal airway. (Guedel’s airway)

**Rapid sequence intubation**

This method of intubation is usually employed for intubating full-stomach patients and emergency intubations. Pressure is applied over the cricoid cartilage (Sellick’s maneuver) to compress the esophagus against the vertebrae to prevent aspiration. Conscious patients do not allow excessive pressures as it leads to coughing and respiratory distress. Hence, it is prudent to apply a force of around 10 Newtons when the patient is conscious and increase it to around 30-40 Newtons once the patient loses consciousness. The force is applied using the forefinger, and the thumb and middle finger are used to prevent lateral displacement of cricoid cartilage. Pre-oxygenation is a must and at no time patient is ventilated till the patient is intubated. Once patient is intubated and the tracheal tube cuff is inflated, cricoid pressure is released.

For visualization of the cords, the lips are gently separated and mouth opened. The laryngoscope is held in the left hand and inserted through the right side of mouth to displace the tongue to the left under the laryngoscope blade. Blade is inserted till the tip of blade rests in the vallecula and the scope is lifted at 45 degree angle to lift the lower jaw, tongue, and epiglottis and bring the vocal cords into view. The laryngoscope blade should not be levered against the upper incisors. In case of non-visualization or partial visualization of vocal cords, pressure exerted over the larynx externally brings the cords in view.

On visualization of the cords, the endotracheal tube is inserted through the right side of laryngoscope blade to prevent blocking the view. It should be inserted gently along the curve without exerting too much pressure. The tube should be inserted till the cuff goes inside the cords and the black circular line on the tube is visible at the level of vocal cords. This usually corresponds to the ideal position, that is, between the glottis and the carina. Cuff should be inflated. Proper positioning should be checked by watching the chest excursions, auscultation of breath sounds equally on both sides of chest and capnography (if available). The depth of insertion of tube from the incisors usually corresponds to 20-22 cm in adult males and 18-20 cm in females for Indian population. The size (internal diameter) of tracheal tube is 7 to 8.5 mm for female and 8 to 9.5 mm for male population. Smaller-sized tubes are used in children and infants.

The tube must be properly fixed to prevent dislodgement or accidental extubation. Fixing the tube with cloth tapes is the preferred method as secretions reduce the adhesiveness of plaster. Trimming the length of the tube helps to reduce the dead space and perform effective and deep suctioning.

The intubating conditions are suboptimal when the procedure is performed under sedation or topical anesthesia. However, the main advantage with these techniques is that control of airway is not lost in case intubation fails. Nasal intubation is reserved only for cases where oral intubation is not possible as it is more traumatic with a higher incidence of ventilator-associated pneumonia and sinusitis.

**Complications**

Laryngoscopy and intubation are very stressful events and can lead to both immediate and delayed complications. Immediate complications include cardiac arrest and death, hypotension, arrhythmia, myocardial ischemia, hypoxia, airway trauma, regurgitation and pulmonary aspiration, laryngospasm, bronchospasm, tachycardia, and hypertension. Delayed complications are tracheal stenosis, tracheomalacia, sinusitis and otitis (nasal intubation), granuloma formation, and rarely tracheoesophageal fistula.

**Approach to a Difficult Airway**

The American Society of Anesthesiologists’ Difficult Airway Algorithm (Figure) provides a simple general guideline for management of an anticipated and unanticipated difficult airway. The protocol includes other supraglottic airway management tools like laryngeal mask airway (LMA) and even invasive methods for successful control of airway. This drill must be well rehearsed and a poster or chart kept for ready reference in the ICU. Fibreoptic intubation is the preferred choice in patients.
with 'difficult airway' and can be performed under sedation and topical anesthesia.

**Figure: ASA difficult airway algorithm**

- **A.** Awake intubation
  - Airway approached by non-invasive intubation
  - Invasive airway access
  - Succeed
  - Fail
  - Cancel case
  - Consider feasibility of other options
  - Invasive airway access

- **B.** Intubation attempts after induction of general anaesthesia
  - Initial intubation attempts successful
  - Initial intubation attempts unsuccessful from this point onwards consider:
    1. Calling for help
    2. Returning spontaneous ventilation
    3. Awakening the patient

- Face mask ventilation adequate
  - Non-emergency pathway ventilation adequate, intubation unsuccessful
  - Alternative approaches to intubation
    - Successful intubation
    - Fail after multiple attempts
      - Invasive airway access
      - Consider feasibility of other options
      - Awaken patient

- Face mask ventilation not adequate
  - Consider/attempt LMA
  - LMA adequate
    - Ventilation not adequate, intubation unsuccessful
    - Call for help
      - Emergency non-invasive airway ventilation
    - Successful ventilation
      - Awake patient
    - Fail
      - Emergency invasive airway access

  - LMA not adequate of not feasible

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**References**

2. How to Tune the Ventilator to the Patient’s Needs?

Acute respiratory failure is defined as any condition in which pulmonary gas exchange becomes impaired such that normal arterial blood gas tensions are no longer maintained and hypoxemia is present with or without hypercapnia. Acute respiratory failure is of two types - i) hypoxic respiratory failure (Type 1) where PaO₂ is <60 mmHg (8 kPa) while breathing room air at sea level. It is caused by the diseases that directly impair the alveolar function, ii) hypercapnic respiratory failure (Type 2) where PaO₂ is <60 mmHg (8 kPa) and PaCO₂ is >60 mmHg in the absence of metabolic alkalosis. It is caused by failure of alveolar ventilation whence the lung parenchyma may itself be normal like in central nervous system disturbances or neuromuscular diseases. Most commonly it occurs in patients with chronic obstructive airway disease (COAD).

Common clinical conditions leading to acute respiratory failure are listed in Table 1. Respiratory insufficiency is a condition wherein adequate gas exchange is maintained at a great expense to the breathing mechanism. This condition can eventually lead to respiratory failure.

Table 1: Common causes of acute respiratory failure

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of respiratory drive</td>
<td>CVA/ brain injury, Metabolic encephalopathy, Effects of drugs, CO₂ narcosis, Inappropriate oxygen therapy</td>
</tr>
<tr>
<td>Neuropathy and neuromuscular disease</td>
<td>Spinal cord injury, Phrenic nerve injury, Guillain-Barre syndrome, Myasthenia gravis, Botulism, Tetanus, Poliomyelitis, Muscular dystrophy, Drugs (muscle relaxants, nerve gas, OPC)</td>
</tr>
<tr>
<td>Chest wall abnormality</td>
<td>Trauma, flail chest, Obesity, Kyphoscoliosis</td>
</tr>
<tr>
<td>Airway obstruction</td>
<td>Foreign body, Tumor, Infection, Sleep apnea</td>
</tr>
<tr>
<td>Lung pathology</td>
<td>Asthma/COAD, Pneumonia, Acute and chronic fibrosing conditions, ARDS, Pulmonary edema of different etiologies, Pulmonary embolism, Postoperative pulmonary complications</td>
</tr>
</tbody>
</table>

CVA = cerebrovascular accident; OPC = organophosphorus compounds; COAD = chronic obstructive airway disease; ARDS = acute respiratory distress syndrome.
However, not every patient with a condition that could cause respiratory failure needs mechanical ventilatory support. Also, in patients with severe organ dysfunction for eg. renal failure or liver failure, mechanical ventilatory support becomes necessary to maintain the pH in the normal range, even though the lungs are otherwise normal. The assessment of a patient with acute respiratory failure is primarily clinical along with certain physiological indices which when deranged, warrant initiation of mechanical ventilation in any patient. These are shown in Table 2.

Table 2: Indices warranting initiation of mechanical ventilatory support

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Normal range</th>
<th>Critical value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consciousness level</td>
<td>Alert</td>
<td>Progressive deterioration</td>
</tr>
<tr>
<td>Activity level</td>
<td>Normal</td>
<td>Signs of exhaustion</td>
</tr>
<tr>
<td>Accessory muscle usage</td>
<td>None</td>
<td>Present</td>
</tr>
<tr>
<td>Respiratory rate (breaths per minute)</td>
<td>12 – 16</td>
<td>&gt;35 or 25 -35 but labored breathing</td>
</tr>
<tr>
<td>Heart rate (beats per min)</td>
<td>70 – 80</td>
<td>Tachycardia, bradycardia</td>
</tr>
<tr>
<td>Blood pressure (mm Hg)</td>
<td>120/80</td>
<td>Hypertension/hypotension</td>
</tr>
<tr>
<td><strong>Ventilatory mechanics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximum inspiratory pressure</td>
<td>50 – 100 mmHg</td>
<td>&lt;20 mmHg</td>
</tr>
<tr>
<td>Vital capacity</td>
<td>65 – 75 ml/kg</td>
<td>&lt;15 ml/kg</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>5 – 8 ml/kg</td>
<td>&lt;5 ml/kg</td>
</tr>
<tr>
<td>Forced expiratory volume</td>
<td>50 – 60 ml/kg</td>
<td>&lt;10 ml/kg</td>
</tr>
<tr>
<td><strong>Arterial blood gas analysis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.35 – 7.45</td>
<td>&lt;7.30 and worsening</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>35 – 45 mmHg</td>
<td>&gt;50 and rising</td>
</tr>
<tr>
<td>PaO₂</td>
<td>80 – 100 mmHg</td>
<td>&lt;50 on O₂ therapy</td>
</tr>
</tbody>
</table>

When it becomes necessary to provide mechanical ventilatory support for a patient the following basic ventilatory settings must be determined: mode, respiratory rate, tidal volume, FiO₂, inspiratory:expiratory ratio (I:E ratio), inspiratory flow rate and pattern and various alarm limits. With the advent of more sophisticated and user friendly ventilators, it is easy to turn on the knobs on the settings panel of the ventilator and initiate the mechanical ventilation even by a non-medical person. However, as intensivists we should know some basic principles of mechanical ventilation in the critically-ill patient. One should not get confused with the various terminologies described by different manufacturers. Referring to the manual or seeking a senior’s help who is familiar with that particular ventilator avoids experimenting on the patient who is critically-ill already. The following is a brief description of different operating modes available on most of the current modern ventilators.

**Types of Mechanical Ventilation**

Mechanical ventilation could be either negative-pressure ventilation or positive-pressure ventilation. Negative-pressure ventilation creates a transairway pressure gradient by decreasing the alveolar pressures below the atmospheric pressure. Unless airway obstruction is present, negative pressure ventilation does not require an artificial airway. Two classical devices that provide negative-pressure ventilation are the iron-lung and chest cuirass or chest shell.

We are basically interested in positive-pressure ventilation. This ventilation is achieved by applying positive pressure at the airway opening which is more than atmospheric pressure. This creates a transairway pressure gradient that generates an inspiratory flow resulting in delivery of tidal volume.

**Modes of Positive-Pressure Mechanical Ventilation**

A ventilator mode can be defined as a set of operating characteristics that control how the ventilator functions. An operating mode can be described by the way a ventilator is triggered into inspiration and cycled into exhalation, what variables are limited during inspiration, and whether or not the mode allows only mandatory breaths, spontaneous breaths or both. The
different ventilator modes currently available on modern ventilators are shown in Table 3.

Table 3: List of different ventilator modes available on modern ventilators

- Spontaneous
- Positive end-expiratory pressure (PEEP)
- Continuous positive airway pressure (CPAP)
- Bi-level positive airway pressure (BiPAP)
- Controlled mandatory ventilation (CMV)
- Assist control (AC)
- Intermittent mandatory ventilation (IMV)
- Synchronized intermittent mandatory ventilation (SIMV)
- Mandatory minute ventilation (MMV)
- Pressure support ventilation (PSV)
- Pressure control ventilation (PCV)
- Airway pressure release ventilation (APRV)
- Inverse ratio ventilation (IRV)
- Proportional assist ventilation (PAV)

The first step in selecting the ventilator mode is to decide whether the patient should receive full ventilatory support or partial support (i.e., if you want to retain the patient’s spontaneous respiratory efforts). Full ventilator support is achieved by any mode that assumes essentially all the work of breathing. The majority of patients initially require full support. This could be achieved with either a volume-controlled or pressure-controlled mode. Following is a brief account of how each mode delivers the necessary ventilation to the patient.

**Spontaneous mode**

Spontaneous mode is not an actual mode on the ventilator since the rate and tidal volume during spontaneous breathing are determined by the patient. The role of the ventilator during the spontaneous mode is to provide 1) adequate inspiratory flow to the patient in a timely manner, and 2) provide adjunctive modes such as PEEP to compliment the patient’s spontaneous breathing effort.

**Positive end-expiratory pressure (PEEP)**

PEEP is an increase in the baseline airway pressure to a value greater than atmospheric pressure (Figure 1). This mode is not used as a stand alone mode but rather as an adjunct to other modes. If PEEP is used in a spontaneously breathing patient, it is called continuous positive airway pressure (CPAP). The main indications for PEEP are refractory hypoxemia and intrapulmonary shunt and decreased functional residual capacity (FRC) and lung compliance.

PEEP acts like a stent and prevents alveolar collapse. It also re-inflates the collapsed alveoli and thus reverses the atelectasis and maintains alveolar inflation during exhalation. Once “recruitment” of these alveoli has occurred (the volume of recruitable lung varies widely from 2%-25%), PEEP lowers the alveolar distending pressure and facilitates gas exchange.
diffusion and oxygenation. Thus it increases the number alveoli participating in the gas exchange. For this reason PEEP is used almost universally in ventilator-dependent patients unless there is a contraindication for its use.

**Disadvantages**

1. Too much of PEEP can cause decreased venous return and thus can cause fall in cardiac output and hypotension
2. Overdistention due to excessive PEEP can cause barotrauma.

**Contraindications to PEEP**

Untreated significant pneumothorax or tension pneumothorax is an absolute contraindication. Hypovolemia is a relative contraindication. Caution is required in patients with elevated intracranial pressure and it is not of benefit in cases with pulmonary hyperinflation e.g., emphysema.

**Controlled mandatory ventilation (CMV)**

This is the simplest form of volume-controlled ventilation which provides a predetermined amount of tidal volume at a preset rate (number of breaths per minute) e.g., tidal volume ($V_t$) 500 mL at 12 breaths per minute (Figure 2). In this mode the ventilator does all the work and provides no allowance for any spontaneous activity from the patient. Hence it is suitable for only those patients who do not have any spontaneous drive due to disease or who are heavily sedated and paralyzed. If the patient tries to breathe spontaneously, it would be very distressing and the patient may end up “fighting” the ventilator. Hence it is advisable to sedate the patient.

**Complications**

As the patient is totally dependent on the ventilator for ventilation and oxygenation, any disconnection from the ventilator or malfunction would be disastrous. Also trauma to the lungs is a possibility because of pressure variations while delivering the fixed tidal volume to non-compliant lungs.

**Alarms**

Low exhaled volume (disconnection alarm) and low inspiratory pressure alarm.

**Assist-control mandatory ventilation (ACMV)**

In this mode, the ventilator delivers a fixed tidal volume but the patient contributes to the respiratory rate in addition to preset mechanical breaths. Whether time-triggered by the ventilator (controlled) or spontaneously generated by the patient, sensed and supported by the ventilator (assisted) each breath delivers a preset tidal volume and there ends the inspiratory phase (Figure 3). The ACMV mode is suitable for patients who have a stable respiratory drive i.e., spontaneous inspiratory efforts to be able to trigger the ventilator but requiring full ventilatory support.
Advantages
1. The work of breathing required by the patient is minimal when the triggering sensitivity (pressure -1 to -2 cm H₂O or flow 2L/min) is set appropriately and ventilator supplies an inspiratory flow that meets or exceeds the patient’s inspiratory flow demand.
2. It is possible to retain the spontaneous respiratory drive of the patient and allows the patient to control the respiratory rate and therefore the minute volume required to normalize the patient’s PaCO₂.

Disadvantages
1. If the patient breathes too fast, work of breathing can be up to 33% to 50%.
2. Alveolar hyperventilation occurs because all the patient’s breaths are assisted.
3. Machine sensitivity adjustments could be a problem in a patient with active inspiratory efforts.

Intermittent mandatory ventilation (IMV)
In this mode the ventilator delivers control breaths (mandatory breaths) and also allows the patient to breathe spontaneously in between the mandatory breaths, at any tidal volume that patient is capable of (Figure 4).

Advantages
Patient is allowed to breathe spontaneously.

Disadvantages
Breath-stacking i.e., superimposition of a time-triggered mandatory machine breath over the patient’s spontaneous breath inappropriately so that there is a very high airway pressure and large tidal volume. Setting appropriate high pressure limit alarm will reduce the risk of barotrauma. Due to this problem, this mode is not offered on the new ventilators. Improvisation over this is the synchronization between the patient and ventilator.

Synchronized intermittent mandatory ventilation (SIMV)
In this, the ventilator delivers either assisted breaths to the patient at the beginning of a spontaneous breath or time-triggered mandatory breaths (Figure 5). Immediately before each mandatory breath there is a small time window (the synchronization window – usually 0.5 seconds) during which the ventilator can recognize a spontaneous breath and respond by delivering the set breath early. If there is no spontaneous inspiratory effort by the patient, then the ventilator delivers the controlled breath according to time – triggering mechanism. This mode is suitable for patients with spontaneous ventilatory drive. This is the most common mode to change over from full ventilatory support to partial ventilatory support. If the mandatory breath rate is set above 10 with an I:E ratio of 1:2, the SIMV mode is almost equal to full ventilatory support. Most modern ventilators offer SIMV mode as either volume-controlled (V-
SIMV) or pressure-controlled (P-SIMV).

**Advantages**
1. Promotes spontaneous breathing and maintains respiratory muscle strength and avoids muscle atrophy.
2. Reduces ventilation to perfusion mismatch as it allows spontaneous breathing which tends to spread the tidal volume more evenly through the lung.
3. Decreases mean airway pressure and therefore maintains more stable hemodynamics.
4. Facilitates weaning.

**Disadvantages**
1. Tendency to wean off too quickly and leading to failure of weaning.
2. Increased work of breathing due to the need to breathe through long ventilatory circuit tubing with high resistance. This can be overcome by adding inspiratory pressure support as described later.

**Mandatory minute ventilation (MMV)**
It is designed to prevent hypoventilation and respiratory acidosis by automatically increasing the number of mandatory breaths or in some ventilators by increasing inspiratory pressure level when the spontaneous effort becomes inadequate in patients who are on SIMV mode and are being weaned. It is a safety feature to ensure that the patient receives a preset minimum minute volume (the other name for this feature).

The modes described so far provide volume-controlled ventilation, in which the tidal volume, respiratory rate or the minute volume are all determined by the clinician and patient may or may not contribute to maintain the normocarbia. The peak pressure required to deliver the preset tidal volume depends upon the ventilator settings and the patient’s pulmonary compliance. The high peak airway pressures may cause lung damage or barotrauma as shown in Table 4. To reduce this risk, pressure-controlled modes of ventilation are preferred in patients with poor pulmonary compliance. Instead of setting a predetermined tidal volume, a peak inspiratory pressure is set. The tidal volume delivered becomes a function of the peak pressure, inspiratory time and the patient’s compliance, airway resistance.

**Table 4: Manifestations of pulmonary barotrauma**

- **Intrathoracic**
  - Pulmonary parenchymal damage
  - Pulmonary interstitial emphysema
  - Pneumothorax
  - Pneumomediastinum
  - Pneumopericardium
  - Bronchopleural fistula

- **Extrathoracic**
  - Pneumoperitoneum
  - Pneumoretroperitonium
  - Subcutaneous emphysema
  - Arterial and venous air embolism

**Pressure-controlled ventilation (PCV)**
PCV uses a constant pressure to inflate the lungs. Ventilation is completely controlled by the ventilator with no participation by the patient. This mode also requires that patients be sedated. The mandatory breaths are time-triggered by a preset respiratory rate. Once the inspiration begins, a pressure plateau is created and maintained by servo controlled inspiratory flow for a preset inspiratory time (time-cycled) decided by the clinician (Figure 6). If adequate inspiratory time is provided, oxygenation and normocarbia can be maintained in the presence of reduced peak inspiratory pressure. This mode is particularly suitable for patients with abnormal lungs as in acute respiratory distress syndrome (ARDS).
Advantages
1. As peak inspiratory pressure is fixed, risk of barotraumas is less.
2. The presence of plateau pressure helps better distribution of inspired gas to most of the alveoli and oxygenation is well maintained.

Disadvantages
1. Tidal volumes are not constant which may sometimes cause inadequate ventilation if sufficient time for inspiration is not provided.

Pressure support ventilation (PSV)
Some ventilators name this mode as assisted spontaneous breathing (ASB). This is a spontaneous mode of ventilation and constitutes partial ventilatory support. In this mode the breaths are patient-triggered (either by pressure or flow). A demand valve in the ventilator generates a high inspiratory flow to rapidly increase to a preset pressure limit and then maintains a plateau by servo control and demand valve for the duration of patient’s inspiratory effort. Once the flow decreases to a preset low flow limit (usually 5 L), inspiration is terminated (Figure 7). This mode is typically used in combination with SIMV mode of ventilation, or in combination with PEEP to facilitate weaning.

Advantages
The main advantage is that provision of inspiratory pressure helps
1. To overcome the resistance of endotracheal tube and ventilator circuit tubing. This decreases the work of breathing.
2. To increase the patient’s spontaneous tidal volume.
3. To decrease the patient’s spontaneous respiratory rate.

Airway pressure release ventilation (APRV)
In this mode, time-triggered mandatory breaths which are pressure-limited are given by the ventilator and patient is allowed to breathe spontaneously in between mandatory breaths. The spontaneous breaths are at an elevated baseline (CPAP) airway pressure which is released periodically to facilitate expiration (Figure 8). The time-triggered closing and opening of a release valve in circuit facilitates this airway pressure release. The tidal volume varies directly with changes in lung compliance and inversely with changes in airway resistance. This mode is relatively new and at present this mode is used as an alternative to the conventional volume-controlled ventilation in patients with significant decrease in lung compliance such as ARDS.

Advantages
APRV provides effective partial ventilatory support with lower peak airway pressures than that provided by PSV and SIMV modes.
Disadvantages

1. Synchronization with mechanical breaths may be a problem.
2. It may be less comfortable than PSV and SIMV modes.

Bi-level positive airway pressure (BIPAP)

It is very similar to APRV but it is used in spontaneously breathing patients. Two different levels of positive airway pressures are used during inspiration (IPAP) and end expiration (EPAP) (Figure 9). This mode appears to be of value in supporting patients with chronic respiratory failure. CPAP and BIPAP are dealt with in the chapter on “non-invasive ventilation.”

Inverse-ratio ventilation (IRV)

The ratio of inspiration to expiration is normally in the range of 1:1.5 to 1:3 during spontaneous breathing. If this ratio is altered so that inspiration is equal or longer than expiration (2:1 to 4:1) it is called inverse-ratio ventilation (Figure 10). To achieve the same oxygenation, IRV requires a lower peak airway pressure and PEEP but a higher mean airway pressure (during a prolonged inspiratory period to deliver the tidal volume) which may help in reducing intrapulmonary shunting and improve oxygenation in ARDS. Because there is less time for expiration, air trapping and auto-PEEP can occur and this also helps reducing shunt and improve oxygenation. IRV mode is usually combined pressure-controlled ventilation (PCIRV) and in some modern ventilators, it is also available in combination with volume-controlled ventilation (VCIRV). This mode requires heavy sedation and paralysis of the patient.

Disadvantages

1. Because of high mean airway pressures there is a high incidence of barotrauma and hence it is usually combined with PCV (PC-IRV)
2. Worsening of pre-existing pulmonary edema

Proportional assist ventilation (PAV)

It is a mode of assisted ventilation where pressure is applied by the ventilator in proportion to the patient generated flow (flow assist - helps to reduce inspiratory effort needed to overcome airflow resistance eg. obstructive defects) or volume (volume assist – helps

Fig. 8: Airway pressure release ventilation (APRV): Airway pressure tracing showing a CPAP level of 10 cmH₂O and pressure “release” to 0 cmH₂O creating a pressure gradient of 10 cmH₂O to facilitate exhalation. The release period is short at RR < 20 with a potential for prolonged I:E ratio.

Fig. 9: Bi-level positive airway pressure (BIPAP): Airway pressure tracing showing an Inspiratory Positive Airway Pressure of 12 cmH₂O and Expiratory Positive Airway Pressure of 5 cmH₂O, with a pressure gradient of 7 cmH₂O. This differs from APRV in that the release pressure is always more than the ambient pressure.

Fig. 10: Inverse ratio ventilation: Note inspiratory period I is longer than exhalation period E.
to reduce inspiratory effort needed to overcome the systemic resistance eg. restrictive defects). It is active in assist breaths only. With PAV the level of pressure applied is designed to change with patient's effort. This means, patient does only a part of the job to meet his ventilatory requirements and the machine completes the rest. A more uniform breathing pattern is possible and promotes patient-ventilator synchrony.

**Initial Ventilatory Settings**

These are mainly based on a patient's body size, diagnosis, pathophysiology and laboratory results. They serve as a starting point and should be adjusted according to changes in patient’s condition and requirements. The following parameters must be determined by the clinician:

1. Full ventilatory support or partial ventilatory support
2. Volume-controlled or pressure-controlled ventilation strategy: mode selection
3. Respiratory rate: Aim is to maintain normocapnia. Usually about 10 – 12 breaths per minute. An alternative method is estimating the patient’s minute volume requirement and dividing the same with tidal volume:

   \[
   \text{Respiratory rate} = \frac{\text{Estimated minute volume}}{\text{Tidal volume}}
   \]

   The estimated minute volume for males is 4.0 × BSA and 3.5 × BSA in females. This calculation assumes normal conditions and must be changed in case of hyperthermia or hypermetabolic states: for every degree Fahrenheit above 99°F by 5% or by 9% for every degree Celsius above 37°C. For metabolic acidosis it should increase by 20%. The BSA (m²) can be obtained from Dubois body surface area chart (Figure11). Changing the respiratory rate is required according to the PaCO₂ seen on ABG obtained after stabilizing the patient on the initial settings. High PaCO₂ requires increase in the respiratory rate and vice versa.

4. Tidal volume: usually set at 10 – 15 mL/ kg of ideal body weight. The calculation of the ideal body weight is shown in Table 5. This amount of tidal volume was found to be excessive causing pulmonary barotrauma and volutrauma in patients where the lung compliance may not be homogeneous. Hence the current trend is to use tidal volumes in the lower normal range of about 6 - 8 mL/kg ideal body weight in such patients (lung protective ventilation).

   **Table 5: Calculation of ideal body weight (IBW)**

   - Male IBW in lbs = 106 + [6 × (height in inches – 60)]
   - Female IBW in lbs = 105 + [5 × (height in inches – 60)]
   - Convert the patient’s body weight from lbs to kg: divide lbs by 2.2

   Also in patients with COAD, lessening the estimated tidal volume by 100-200 mL helps shorten the inspiratory time and leaving adequate time for exhalation. This prevents air-trapping and development of auto PEEP, V/Q mismatch, and hypoxemia. Using a high inspiratory flow rate also helps to shorten inspiratory time in these patients. In patients with reduced lung volumes due to lung resection, lower tidal volumes are necessary. In all these cases, increasing respiratory
rate to maintain normocarbia or accepting a little more that normocarbia (permissive hypercapnia) is helpful.

However, not all the tidal volume that was set may not reach the patient. Some amount of volume is lost in the form of gas leakage in the ventilatory circuitry, at the endotracheal tube cuff and circuit compressible volume loss. The latter loss is due to the compliant tubing which expand during a positive pressure breath and in doing so consume a part of tidal volume and that does not reach the patient. This is usually about 3 mL/cmH₂O of peak inspiratory pressure ranging from 120 – 150 mL depending upon the peak inspiratory pressure.

Major leakage (>5% of ventilator tidal volume) from the ventilatory circuit should be identified and corrected. Low exhaled minute volume alarm is of some help in this case. Minor leaks and circuit compressible volume loss are compensated by using larger tidal volumes. Some ventilators (Puritan Bennett 7200 series) can do so automatically and deliver a stable and set tidal volume. Others have a facility to detect the VT delivered to the patient at the airway opening and this allows detection of volume loss due to the above reasons.

5. FₐO₂: For most of the patients who require full support mechanical ventilation, especially with severe hypoxemia, the initial FₐO₂ is set at 100%. Once the patient is stabilized on the initial settings and ABG analysis is done and FₐO₂ is then reset according to the needs i.e. to maintain the PaO₂ between 80% - 100%, lower for patients with chronic CO₂ retention. For patients who otherwise have normal lungs e.g. drug overdose, Guillain-Barre syndrome (uncomplicated), the starting FₐO₂ could be set at 50 – 40% and taken from there. If very high oxygen concentrations are required, then adding PEEP or any other adjunctive modes would help to reduce the FₐO₂. Oxygen toxicity should be kept in mind. If the patient’s PaO₂ is not within the desired range, the following equation can be used to estimate FₐO₂:

\[
\text{Desired } F_{\text{a}O_2} = \frac{\text{PaO}_2 \text{ (desired)} \times F_{\text{a}O_2} \text{ (known)}}{\text{PaO}_2 \text{ (known)}}
\]

6. PEEP: As discussed before, PEEP helps improving oxygenation and reduction of FₐO₂. The initial level may be set at 5 cm H₂O and increased or decreased during the course of illness / recovery as per the PaO₂ on ABG report.

7. I: E ratio: This is usually kept in the range of 1:2 which is near physiological range. Longer expiratory time is required in patients with COAD to prevent air trapping and auto-PEEP (Some machines can record auto-PEEP automatically). Auto-PEEP is present when the end-expiratory pressure does not reach the baseline (zero cmH₂O when PEEP is not in use or the level of PEEP set by the clinician). In cases of refractory hypoxemia, inverse I:E ratios can be used but only after the conventional modes have been tried. The I: E ratio can be altered by manipulating any one or more of the following controls: (1) Inspiratory flow rate (increase in the inspiratory flow rate causes decrease in the inspiratory time and therefore increases the I:E ratio) (2) inspiratory time (3) inspiratory time % (4) respiratory rate (5) minute volume i.e. tidal volume (increase causes increase in the inspiratory time) and respiratory rate.

8. Inspiratory flow pattern: The main flow patterns (Figure 12) offered on most modern ventilators are (1) square wave (constant) flow pattern: this is the usual pattern to start with. This provides an even and constant peak flow during the entire inspiratory phase which helps to overcome the airway resistance and parenchymal elastance. This is the only flow pattern in which the peak flow rate equals the mean flow rate and all others have peak flow rate more that mean flow rate; 2) accelerating or ascending flow pattern: With its ascending flow throughout the respiratory cycle, this pattern may improve the distribution of ventilation in patients with partial airway obstruction; 3) decelerating or descending flow pattern; 4) sine-wave pattern: This is considered more physiologic because it is similar to the
normal spontaneous breathing inspiratory flow pattern.

9. Inspiratory flow rate: It is an estimated mean delivered flow of the inspired gas. Since flow is volume per unit time, high flow rates shorten the inspiratory time for a given tidal volume and vice versa. High flow rates result in high peak pressure and poor gas distribution due to less inspiratory time and slow flow rates result in low peak pressure and prolong inspiratory time but improve gas distribution at the expense of reversing the I:E ratio and its resultant cardiovascular effects. In patients with non-homogeneous lungs as in ARDS slow flow rates are preferred and in patients with increased airway resistance as in COAD, fast flow rates allowing longer time periods for expiration are preferred. For otherwise normal lungs, it is desirable to have physiological I:E ratio (1:2) and so higher flow rates with usually constant flow pattern to start with. Calculating flow rate from the tidal volume and inspiratory time is quite simple by dividing \( V_t \) by \( T_i \). Flows are set to meet the patient’s inspiratory demand and this is usually in the range of 50 – 60 L/min.

10. Periodic hyperinflation or sigh: Some ventilators offer this mode which was popular in 1960s. This provides the capability of giving one or more deep breaths at periodic timed intervals such as three or four times per hour. The volume for the sigh breath is about 1.5 to 2.0 times the regular low \( V_t \) setting. This is more useful during low tidal volume ventilatory support.

**Ventilatory Alarms**

Alarms are ways of warning of possible dangers. These alarms should be backed up by a battery source to prevent malfunction in the event of electrical failure. The following are the most common and basic to all ventilators.

**Low exhaled volume alarm**

Should be set at about 100 mL lower than the expired tidal volume. This alarm is triggered when the patient does not exhale an adequate tidal volume. This is typically used to detect a system leak or circuit disconnection.

**Low inspiratory pressure alarm**

Should be set at 10 – 15 cm H₂O lower than the observed peak inspiratory pressure. This alarm complements the low exhaled volume alarm and is also used to detect a system leak or circuit disconnection.

**High inspiratory pressure alarm**

Should be set at 10 – 15 cm H₂O higher than the observed peak inspiratory pressure. Once this alarm is triggered, inspiration is immediately terminated and the ventilator goes into expiratory cycle. The patient must be evaluated to determine the cause of the alarm. Common causes are: water in the ventilatory circuit, kinking or biting of the ET tube, secretions in the airway, coughing, bronchospasm, increase in the airway resistance, decrease in the lung compliance or tension pneumothorax.

**Low source gas alarm**

It is triggered when the high pressure source gas is not available and cannot be silenced if the gas is critical to the operation of the ventilator that does not have built-in microcompressor.

**Apnea alarm**

Should be set with a 15 – 20 second time delay (with less time delay at higher respiratory rate). This also triggers an apnea ventilation mode in which the ventilator provides full ventilatory support until the alarm condition no longer exists. Apnea ventilation is a safety feature incorporated with the spontaneous breathing mode. In the event of apnea or an extremely slow spontaneous respiratory rate, back up ventilation is invoked by this feature and it delivers a predetermined tidal volume, respiratory rate, \( F_{O_2} \), and other essential options to the patient.

**High respiratory rate alarm**

Should be set at 10 – 15 breaths per minute over the observed respiratory rate. This is especially important in the MMV mode. This alarm indicates that the patient is becoming tachypneic – a sign of respiratory distress.
Ventilation Strategies for Specific Conditions

Acute respiratory distress syndrome

The large tidal volumes used in conventional mechanical ventilation (10 – 15 mL/kg) are nearly twice the size the tidal volumes during normal quiet breathing (6-7 mL/kg). In patients with ARDS, the functional lung volume is significantly reduced due to lung infiltrates which are spread more in the posterior or dependent lung regions. If these patients are given large tidal volume breaths, gas distribution primarily occurs in the functional lung portion and causes overdistention and rupture of distal airspaces. This condition is called ventilator-induced lung injury. Injury due to excessive alveolar volumes is called volutrauma that due to excessive alveolar pressures is called barotrauma. The injury mediates release of cytokines and other inflammatory mediators from the lung into the systemic circulation reaching distant organs and producing widespread inflammatory injury and multi-organ failure. This is called biotrauma.

This ventilator-associated lung injury was also evident in conditions other than ARDS as well. This evidence promoted the development of lung protective ventilation strategy for such patients. ARDS clinical network study of 800 patients with ARDS showed that low tidal volumes (6 mL/kg of predicted body weight) were associated with a 9% reduction in mortality when end-inspiratory plateau pressure was <30 cm H₂O. The protocol for lung protective ventilation is represented in Table 6.

Table 6: Protocol for low volume ventilation in ARDS

<table>
<thead>
<tr>
<th>Goals</th>
<th>TV = 6 ml/kg, Ppl &lt;30 cm H₂O, pH = 7.30 – 7.40</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. First Stage:</td>
<td></td>
</tr>
<tr>
<td>1. Calculate patient’s <strong>predicted</strong> body weight (PBW)*</td>
<td></td>
</tr>
<tr>
<td>Males: PBW = 50 + [2.3x(height in inches – 60)]</td>
<td></td>
</tr>
<tr>
<td>Females: PBW = 45.5 + [2.3x(height in inches – 60)]</td>
<td></td>
</tr>
<tr>
<td>2. Set initial tidal volume (TV) to 8 ml/kg PBW</td>
<td></td>
</tr>
<tr>
<td>3. Add Positive end-expiratory pressure (PEEP) at 5 – 7 cm H₂O</td>
<td></td>
</tr>
<tr>
<td>4. Reduce the TV by 1ml/kg every 2 hours until TV = 6 ml/kg PBW</td>
<td></td>
</tr>
<tr>
<td>II. Second Stage:</td>
<td></td>
</tr>
<tr>
<td>1. When TV is down to 6 ml/kg, measure plateau pressure (Ppl).</td>
<td></td>
</tr>
<tr>
<td>A. Target Ppl &lt; 30 cm H₂O</td>
<td></td>
</tr>
<tr>
<td>B. If Ppl &gt; 30 cm H₂O, decrease TV in 1 ml/kg steps until Ppl drops below 30 cm H₂O or TV down to 4 ml/kg.</td>
<td></td>
</tr>
<tr>
<td>III. Third Stage:</td>
<td></td>
</tr>
<tr>
<td>1. Monitor arterial blood gases for respiratory acidosis.</td>
<td></td>
</tr>
<tr>
<td>A. Target pH = 7.30 – 7.45</td>
<td></td>
</tr>
<tr>
<td>B. If pH is 7.15 – 7.30, increase respiratory rate (RR) until pH &gt; 7.30 or RR = 35 bpm</td>
<td></td>
</tr>
<tr>
<td>C. If pH &lt; 7.15, increase RR to 35 bpm. If pH still &lt; 7.15, increase TV at 1 ml/kg increments until pH &gt; 7.15.</td>
<td></td>
</tr>
</tbody>
</table>

* The predicted body weight is the weight at which lung volumes are normal

Severe airflow obstruction (asthma and COAD)

1. Select ventilatory modes which would allow spontaneous breathing which helps in normal distribution of inspired gas in alveoli as soon as possible. Flow triggering of inspiration reduces work of breathing in these patients than pressure triggering.
2. Premature closing of the alveoli during normal tidal breathing leads to air-trapping so allow long expiratory time. The I:E ratio should be 1:3 or 1:4.
3. Minimal PEEP actually helps by counterbalancing the critical closing pressure of the small airways and so keeps the alveoli open. However, the extrinsic PEEP should not be more than intrinsic PEEP or auto-PEEP.
4. Use low tidal volumes and accept permissive hypercapnia.
5. Fast inspiratory flow rates allow more time for expiration and decelerating flow pattern with its high peak airway pressure.
may be more useful in overcoming the high airway resistance observed in these patients.

6. Start with low FiO₂ as higher concentrations remove the hypoxic ventilatory drive in these patients unless the patient is having severe hypoxemia before initiating mechanical ventilatory support.

7. Airway pressures are usually high in these patients. So pressure limits on the high pressure alarms may need to be readjusted.

Table 7 shows an example of initial ventilatory settings for an adult male patient who is 5'10" in height who required mechanical ventilatory support due to drug overdosage. His ideal body weight is 75.45 kg.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Settings</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mode</td>
<td>Assist-control or SIMV</td>
<td>Provides full ventilatory support</td>
</tr>
<tr>
<td>RR</td>
<td>10 - 12 /min</td>
<td>Primary control to alter ventilation and PaCO₂</td>
</tr>
<tr>
<td>Vₜ</td>
<td>10 – 15 mL per kg (~ 800 mL)</td>
<td>Peak airway pressure is directly related to the Vₜ setting. Use lower Vₜ to reduce the risk of pressure-related injuries.</td>
</tr>
<tr>
<td>FIO₂</td>
<td>100% for severe hypoxemia or compromised cardiopulmonary status; 40% for mild hypoxemia or normal cardiopulmonary status</td>
<td></td>
</tr>
<tr>
<td>I : E ratio</td>
<td>1 : 2</td>
<td>1 : 4 if there is an air trapping or COAD changes</td>
</tr>
<tr>
<td>Flow rate</td>
<td>~ 40 – 50 L/min</td>
<td></td>
</tr>
<tr>
<td>Flow pattern</td>
<td>Square wave</td>
<td>Other flow patterns for a lower peak airway pressures (reading on the ventilator screen) and better gas distribution (breath sounds on auscultation).</td>
</tr>
</tbody>
</table>

While providing the mechanical ventilation to patients one should remember that the clinical assessment is the most important factor and one should treat not the numbers but the patient. The above text is only a set of guidelines and decisions regarding individual patient depend upon the clinical condition and progress on a given ventilatory support scheme.

References
Patients require mechanical ventilatory support when there is a disparity between the ventilatory and gas exchange capabilities of their respiratory system which are either overwhelmed or have failed.

Identifying the precise time when spontaneous breathing capacity returns is difficult, but attempting to do so is important because the risks accompanying mechanical ventilation increase with time. Liberation from mechanical ventilation is usually undertaken only after the underlying pathologic process that prompted the initiation of mechanical ventilation has resolved or at least started improving. A focused, simple daily screening can identify patients who are potential candidates for weaning. These assessments are multifaceted, and usually include the overall patient condition, hemodynamic stability, neurological and muscular status and the adequacy of gas-exchange, among other variables.

Once the decision has been made to initiate the process of discontinuation, one must assess the readiness-to-wean at the bedside. Daily screening can reduce the number of patients receiving mechanical ventilation for more than 21 days and has been associated with reduced in-hospital mortality. The traditional approach to weaning has been a gradual reduction of ventilatory support - starting with cutting back on supplemental oxygen and eventually ending with extubation or tracheal decannulation.
Weaning Parameters

Patients receiving mechanical ventilation for respiratory failure should undergo a formal assessment of discontinuation potential and tolerate a spontaneous breathing trial. In addition to various indices, the clinical acumen of an experienced clinician is as good to predict the weaning ability of the patient. Several criteria which could be objective or subjective had been propounded to determine the readiness-to-wean. Following are some of them:

1. Evidence for some reversal of the underlying cause for respiratory failure.
2. Respiratory rate (f) – a respiratory rate closer to normal (12-18 bpm) is always a good sign to wean a patient from ventilator. A tachypneic patient breathing lower tidal volumes is not a good candidate to wean from the respiratory support.
3. Adequate oxygenation (PaO₂/FiO₂ ratio ≥ 150 to 200; requiring positive end-expiratory pressure [PEEP] ≤ 5 to 8 cmH₂O; FiO₂ 0.4 to 0.5)
4. Hemodynamic stability, as defined by the absence of myocardial ischemia and the absence of clinically significant hypotension (i.e., a condition requiring no vasopressor therapy or therapy with only low-dose vasopressors such as dopamine or dobutamine, ≤ 5 µg/kg/min); and
5. Clear sensorium and capability to initiate an inspiratory effort
6. Hemoglobin level, should be at least 8 g/dL
7. Magnesium, potassium, and phosphorus within normal range

Predicting Successful Weaning

Discontinuation assessments for patients receiving mechanical ventilation for respiratory failure should be performed during spontaneous breathing rather than while the patient is still receiving substantial ventilatory support. An initial brief period of spontaneous breathing can be used to assess the capability of continuing on to a formal spontaneous breathing trial (SBT). There are various clinical parameters which can predict the success of weaning with substantial sensitivity (Table 1)

Table 1: Clinical parameters that predict successful weaning

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Desired value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory rate</td>
<td>&lt; 30 breaths/minute</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>4-6 mL/kg</td>
</tr>
<tr>
<td>Minute ventilation</td>
<td>10-15 L/minute</td>
</tr>
<tr>
<td>Negative inspiratory force</td>
<td>-20 to -30 cm H₂O</td>
</tr>
<tr>
<td>Maximal inspiratory pressure</td>
<td>-15 to -30 cm H₂O</td>
</tr>
<tr>
<td>Tracheal occlusion pressure at 100 msec (P₀.1)</td>
<td>&lt; 2 cm H₂O</td>
</tr>
<tr>
<td>Rapid shallow breathing index (RSBI) (respiratory rate divided by tidal volume)</td>
<td>60-105/L</td>
</tr>
<tr>
<td>CROP score (an index including compliance, rate, oxygenation, and pressure)</td>
<td>13</td>
</tr>
</tbody>
</table>

Rapid shallow breathing index is the ratio of breathing frequency to tidal volume in liters measured under spontaneous breathing conditions. If the index is > 105/min/l, it predicts weaning failure. It effectively predicts the weaning outcome in patients with no underlying pulmonary disease who have been mechanically ventilated for < 8 days. Tracheal occlusion pressure at 100 msec (P₀.1) is defined as the inspiratory pressure generated 100 msec after an airway occlusion maneuver. A special manometer is needed to measure the airway occlusion pressure at 0.1 seconds from the onset of inspiration, with the airway occluded at the functional residual capacity. Since P₀.1 is measured at zero flow, it is independent of the compliance and resistance characteristics of the respiratory system. It is a good indicator of the adequacy of central respiratory drive. The CROP (compliance, rate, oxygenation, and maximum inspiratory pressure) index reflects the adequacy of pulmonary gas-exchange as well as the balance between respiratory demand and reserve.

Spontaneous Breathing Trial

The best way to assess whether the patient is likely to tolerate weaning is to perform a trial of spontaneous breathing.
Spontaneous breathing trials (SBTs) usually last 30 min - 2 hrs. SBTs can either be performed with the patient connected to the ventilator with minimal support or else the patient breathes on his/her own through a T-piece.

**Continuous positive airway pressure (CPAP)**

In CPAP mode, the machine delivers a positive pressure throughout the inspiratory and expiratory phases of the respiratory cycle. The constant pressure increases the functional residual capacity (FRC), which improves the gas-exchange. If the patient can tolerate a CPAP of 5-6 cm H2O for 30 min - 2 hrs, and meets other clinical criteria, the endotracheal tube can be removed. If not, the patient is returned to full ventilator support. The advantage of this method is the ability to monitor the tidal volume and respiratory rate during spontaneous breathing to detect the rapid and shallow breathing that often signals failure to sustain spontaneous breathing.

**Breathing through a T-piece**

Spontaneous breathing trials (SBTs) can be conducted with the patient disconnected from the ventilator using the T-shaped circuit. The inhaled gas is delivered at a high flow rate (greater than the patient’s inspiratory flow rate) through the upper arm of the apparatus. The high flow rate serves two purposes. First, it creates a “suction effect” that carries the exhaled gas out of the apparatus and prevents rebreathing of exhaled gas. Second, it prevents the patient from inhaling room air from the exhalation side of the apparatus. The disadvantage of T-piece trials is the inability to monitor the patient’s spontaneous tidal volume and respiratory rate. The assumed advantage is less work of breathing when compared to spontaneous breathing while connected to the ventilator.

**Monitoring during SBT**

The following parameters should be continuously monitored in patients undergoing an SBT - respiratory pattern, adequacy of gas exchange, hemodynamic stability, and subjective comfort. A trial of spontaneous breathing should be discontinued promptly if any of the signs/symptoms of intolerance mentioned below appear and persist.

**Indicators of failed SBT**

1. Inadequate gas exchange: arterial oxygenation saturation (SaO₂) < 85% - 90%; PaO₂ < 50 – 60 mmHg; pH < 7.32; increase in PaCO₂ > 10 mmHg
2. Unstable ventilatory/respiratory pattern: respiratory rate > 30 – 35 breaths/min; respiratory rate change > 50%
3. Hemodynamic instability: heart rate > 120 – 140 beats/min; heart rate change > 20%; systolic blood pressure > 180 mmHg or < 90 mmHg; blood pressure change > 20%; vasopressors required
4. Change in mental status: drowsiness; agitation; anxiety; unresponsiveness
5. Signs of increased work of breathing: nasal flaring; paradoxical breathing movements; use of accessory respiratory muscles; subjective discomfort; diaphoresis

**Extubation**

Although extubation is conditional upon successful weaning, decision to wean and decision to extubate are entirely different issues. While the decision to wean is based on a successful SBT, the decision to extubate should be based on assessments of airway patency and the ability of the patient to protect the airway. The quantitative cuff leak test which is the difference between the inspired and expired tidal volumes during volume-cycled ventilation with the endotracheal tube cuff deflated can identify the patients at risk of post-extubation stridor which may result in reintubation.

**Failed SBT**

Patients receiving mechanical ventilation for respiratory failure who fail an SBT should be put back on the full ventilatory support, and have the cause for the failed SBT determined. The factors responsible for a failed SBT are,

1. Related to respiratory load: bronchospasm; left ventricular failure; sepsis; seizures.
2. Related to respiratory drive: sedation; neurologic diseases; hypercapnia.
3. Capacity of respiratory pump: pain and discomfort; positioning; use of muscle relaxant; nutrition; electrolyte imbalances such as hypokalemia, hypophosphatemia, and hypomagnesemia.

After instituting the appropriate therapy, the patient can be screened again daily for an opportunity at the earliest to attempt an SBT. In difficult-to-wean patients that fail multiple SBTs, one might attempt to gradually taper off the ventilatory support over a period of days to weeks employing specific modes of assisted ventilation such as the pressure support ventilation (PSV) and synchronized intermittent mandatory ventilation (SIMV).

Role of Tracheostomy

Tracheostomy is commonly performed for critically-ill, ventilator-dependent patients to provide long-term airway access. The benefits commonly ascribed to tracheostomy, compared to prolonged endotracheal intubation include improved patient comfort, more effective airway suctioning, reduction in dead space, decreased airway resistance, enhanced patient mobility, increased opportunities for articulated speech, ability to eat orally, and a more secure airway. These advantages might result in fewer ventilator complications (e.g., ventilator-associated pneumonia), accelerated weaning from mechanical ventilation, and the ability to transfer ventilator-dependent patients from the ICU.

In summary, for a successful weaning attempt, it is mandatory that the primary cause that led to mechanical ventilation needs to be treated. The most crucial issue in discontinuing mechanical ventilation is recognizing at the earliest when the patient is ready for an SBT. The patient needs to be closely monitored during these trials, ensuring that the exercise is within the physiological reserves. These measures aid successfully liberating the patient from mechanical ventilation.

References

4. Non-Invasive Ventilation

Bimlesh Dhar Pandey

Non-invasive ventilation (NIV) is the provision of assisted ventilation without the use of an endotracheal or tracheostomy tube. The past decade has seen rapid expansion of its use especially in patients with chronic obstructive airways disease (COAD). This modality can be an excellent method of assisted ventilation both in acute and chronic settings, in-hospital as well as out-of-hospital settings. The advantages of NIV over the conventional ventilation are listed in Box 1.

Modes of Non-invasive Ventilation

Continuous-positive-airway pressure (CPAP)

CPAP delivers a constant level of positive pressure throughout the respiratory cycle. It does not actively assist inspiration. It is useful in patients with hypoxemic respiratory failure. It improves oxygenation by increasing the mean airway pressure. It increases the functional residual capacity by recruitment, and opens poorly ventilated and collapsed alveoli, thus enhancing gas exchange and oxygenation. CPAP decreases the work of breathing by improving the lung compliance. Initially, the CPAP is set at a low level and gradually increased to allow adequate oxygenation. It is important to check the mask for leaks, because this can be an impediment to provide adequate support.

Bi-level positive airway pressure (BiPAP)

BiPAP differs from CPAP in that rather than delivering a constant level of pressure throughout inspiration and expiration, cycling occurs between two different operator-specified pressure levels. The pressure delivered during inspiration is higher than that during expiration, thereby providing a degree of assistance during inspiration. Settings depend on the patient's tolerance of the positive pressure delivered. The primary utility of this mode over CPAP is in patients with ventilatory fatigue or failure. BiPAP not only provides the benefits of CPAP by increasing the airway pressure during expiration, but also provides an inspiratory assist, reducing the work of breathing and assists ventilation in patients at risk for hypercapnia. The inspiratory pressure most often is set at 8–20 cm H₂O and expiratory pressure is set at 0-15 cm H₂O.

NIV services can be provided only at designated areas, which depend on many factors like logistics and trained manpower. Each hospital can determine the location depending on its requirement and convenience like intensive care unit (ICU), high dependency unit, respiratory ward, NIV ward, emergency ward, or general ward.

Patient-Ventilator Interfaces

Approximately 20%–30% of patients with acute respiratory failure cannot be managed by NIV. Proper mask fit is important for patient comfort and effective ventilation. Choice of mask depends on familiarity and availability. Nasal masks are comfortable, allow patients to communicate verbally, but suffer from air-leaks unless the patient keeps the mouth closed. Full-face masks offer the tightest seal and prevent leaks. These masks can be hazardous in patients with altered sensorium who are unable to protect their airway or who are at risk for vomiting. In patients intolerant of NIV because of nasal obstruction, nasal stents can be inserted to restore the patency of the upper airway. Preferably, different sizes of nasal masks, full-face masks, and nasal pillows should be available to choose for an individual patient. In acute hypercapnic respiratory failure a full-face mask should be used initially. Later on, a nasal mask may be used once the patient improves.

Applications of Non-invasive Ventilation

There have been a number of clinical trials describing the use of NIV in different conditions. None of the trials have used “sham NIV” as control therapy. NIV should be undertaken as a therapeutic trial with recourse to tracheal intubation if it fails. Tracheal intubation should be contemplated before commencing NIV in every patient. The common conditions where NIV is used are COAD with respiratory failure, hypoxemic respiratory failure (acute lung injury), cardiogenic pulmonary edema, postoperative respiratory failure, severe community-acquired pneumonia, post-extubation and during weaning, obstructive
sleep apnea, neuromuscular diseases, chest wall deformities, and patients awaiting lung transplantation.

Applications in COAD

There are a number of randomized controlled trials (RCTs) of NIV in patients with acute exacerbation of COAD. These RCTs have been done in ICU and non-ICU settings. NIV is useful in patients with acute exacerbation of COAD and respiratory acidosis (pH < 7.35) despite maximum medical treatment and controlled oxygen therapy. However, CPAP may not be considered standard therapy in patients with COAD; patients with relatively mild exacerbation of COAD are not candidates for NIV. On the other hand, NIV should not be used as a substitute for tracheal intubation and invasive ventilation when the latter is clearly more appropriate.

Applications in pulmonary edema

NIV is recommended in addition to standard medical treatment in patients with cardiogenic pulmonary edema, particularly if associated with hypercapnia. CPAP and BiPAP are equally effective. CPAP is effective in patients with cardiogenic pulmonary edema who remain hypoxic despite maximal medical treatment. Use of BiPAP should be reserved for patients in whom CPAP is unsuccessful.

Applications in bronchial asthma

NIV is not recommended for routine management of asthma exacerbations. It may be tried in carefully selected patients of status asthmaticus who fail to respond quickly to medical treatment and have no contraindication to NIV.

Applications in weaning

NIV can be used to reduce the muscle fatigue and thus can serve as a bridge between invasive assisted ventilation and spontaneous breathing. In this context, NIV can be used i) as a part of early weaning strategy when spontaneous breathing trial fails, iii) to prevent post-extubation failure after conventional weaning, and iv) when signs of respiratory failure develop after weaning. NIV can also be used to expedite extubation in selected patients with COAD after intubation for acute respiratory failure. Rather than improving outcomes, early extubation shortens the duration of invasive ventilation without worsening outcomes. Patients should be selected carefully with attention to cooperativeness and ability to clear secretions. Patients who are difficult to intubate should be excluded. NIV can be used to reduce chances of re-intubation in COAD patients. However, routine use of NIV after extubation to prevent respiratory failure and re-intubation is not recommended.

Applications in sleep-disordered breathing

CPAP is the treatment of choice for patients with moderate to severe obstructive sleep apnea. However, BiPAP is preferred in patients requiring high CPAP pressures, those presenting with acute respiratory failure, and in those with hypercapnic respiratory failure. NIV is also useful in patients with obesity-hypoventilation syndrome.

Applications in other conditions

NIV has been used in a variety of other conditions (such as acute respiratory distress syndrome, postoperative or post-transplantation respiratory failure) with reduced intubation rates, ICU stay, and mortality. In this context, patients who would be considered for intubation if NIV fails should receive NIV only in an ICU. There is tendency for decreased length of stay in the ICU when patients are offered NIV early enough.

Factors Associated with Success of NIV

The following factors are associated with successful outcome following NIV.

- High $P_{a}CO_{2}$ with low (A - a) oxygen gradient, pH 7.25 - 7.35
- Improvement as expected after 1 hour of NIV
- Good sensorium

The following factors are associated with failure of NIV.
The most common reason for failure of NIV is patient-intolerance. Appropriate patient selection and acclimatization of the patient to the device are very important. Gradual initiation and slow increase of pressure helps in better tolerance.

**Contraindications for NIV**

It is very important to understand the limitations of NIV, use it judiciously, and reap the maximum benefits after careful patient selection. NIV should be avoided if any of the conditions listed in Box 2 are present. It is important to keep in mind that the 'gold standard' for the management of acute respiratory failure is endotracheal intubation and conventional mechanical ventilation.

**Complications of NIV**

Complications of NIV are minor and include nasal congestion, eye irritation, discomfort, and pressure necrosis caused by a tight-fitting mask. In patients with altered sensorium, risk of aspiration is high, especially with use of a full-face mask. Gastric distension may occur if pressures exceed 20–25 cm H₂O.

**Monitoring**

Periodic physiological as well as clinical assessment is required in patients receiving NIV (Box 3). The monitoring depends on the location of the patient, severity of disease, and presence of co-morbidities; however, the first few hours are critical. Arterial blood gases should be measured after 1 - 2 hours of NIV and after 4 - 6 hours or as clinically indicated. If there is no improvement in this period, NIV should be discontinued and patients should receive invasive ventilation. Oxygen saturation should be monitored continuously for at least 24 hours after commencing NIV, and supplementary oxygen is administered to maintain saturations between 85% and 90%.

**When to Discontinue NIV Early**

Early discontinuation of NIV and prompt intubation should be considered if any of the following events occur in patients on NIV - electrocardiographic changes suggestive of ischemia or arrhythmia; deterioration of arterial blood gas parameters; no improvement in mental status within 30 minutes of NIV in patients with COAD and type II respiratory failure; and development of patient-machine asynchrony.

**Weaning NIV Support**

Once the blood gases improve and patient is clinically stable, gradually reduce the pressures (IPAP and EPAP), and then reduce the duration of NIV. Night-time NIV should be the last to be discontinued. Regular physiological and clinical monitoring should continue through the process of weaning NIV.
Box 1: Advantages of NIV

- Speech and swallowing are preserved
- Effective cough is possible
- Avoids resistive work imposed by endotracheal tube
- Avoids complications of endotracheal tube such as trauma to upper airways and trachea, aspiration, sinusitis and nosocomial pneumonia

Box 2: Contraindications to NIV

- Respiratory arrest
- Agitated or uncooperative patient
- Severe encephalopathy (e.g., Glasgow coma score < 10) or confusion
- Excessive secretions
- Inability to protect airway
- Life-threatening hypoxemia
- Anatomic abnormalities interfering with mask fit
- Facial trauma or burns
- Recent surgery on face, airway, or upper gastrointestinal tract
- Medically unstable (hypotensive shock, cardiac ischemia or arrhythmias)
- Undrained pneumothorax

Box 3: Monitoring a Patient on NIV

- Vital signs: respiratory rate, heart rate, blood pressure
- Use of accessory muscles, level of consciousness, patient comfort
- Alleviation of symptoms: dyspnea
- Gas-exchange: pulse oximetry, arterial blood gases
- Patient-machine synchrony: chest wall movement, mouth leak

References

5. Ventilator-Associated Pneumonia

Varun Gupta

Ventilator-associated pneumonia (VAP) is the most common nosocomial infection in the intensive care unit (ICU). A significant number of beds in the ICU are occupied by patients suffering from VAP. Importantly, it adds significantly to morbidity and mortality. VAP is a cause of increased utilization of healthcare resources and excess cost. VAP is defined as pneumonia that arises more than 48-72 hours after endotracheal intubation. It is further subdivided into early-onset VAP (occurring within the first four days of intubation) and late-onset VAP (five days or more of intubation).

Epidemiology

One-day point prevalence study designed to determine the prevalence of ICU-acquired infections identified pneumonia as the most common infection with a prevalence of 10%. Up to 28% of patients receiving mechanical ventilation suffer from an episode of VAP. The risk of pneumonia is maximum during the first five days of mechanical ventilation. During this period, the risk increases by 3% each day; during days 5-10 of ventilation it increases by 2% each day, and after day 10 it increases by 1% each day.

The crude mortality rate for VAP may be as high as 30%-70%. Patients with late-onset VAP are more likely to be infected with multidrug-resistant (MDR) pathogens like Pseudomonas aeruginosa, Acinetobacter baumannii and methicillin-resistant Staphylococcus aureus (MRSA) and have higher crude mortality than patients with early-onset disease. However, patients with early-onset VAP who have recently received antibiotics or had an admission to a healthcare facility are also at risk for colonization and infection with MDR pathogens. The mortality related to the VAP or “attributable mortality” has been estimated to be between 33%-50%. Increased mortality rates are associated with bacteremia, medical rather than surgical illness, and treatment with ineffective antibiotic therapy.

Etio-pathogenesis

Common pathogens include aerobic Gram-negative bacilli, such as A. baumannii, P. aeruginosa, Escherichia coli, and Klebsiella pneumoniae. Pneumonia due to S. aureus (much of which is MRSA) is more common among patients with diabetes mellitus and head injury. Rates of polymicrobial infections are especially high in patients with acute respiratory distress syndrome (ARDS). Pneumonia involving anaerobic organisms may follow aspiration in non-intubated patients, but is rare in patients with VAP. Rates of Legionella pneumophila vary considerably between hospitals and disease occurs more commonly with serogroup 1 when the water supply is colonized or there is ongoing construction. Viruses and fungi are uncommon causes of VAP in immune-competent patients. Rates of VAP due to MDR pathogens have increased dramatically in intensive care patients. Risk factors for colonization and infection with MDR pathogens are summarized in Table 1.

Table 1: Risk factors for MDR pathogens causing VAP

- Antimicrobial therapy in preceding three months
- Current hospitalization of five days or more
- High frequency of antibiotic resistance in the community or in the specific hospital unit
- Presence of risk factors for HCAP
  - Hospitalization for two days or more in the preceding three months
  - Residence in a nursing home or extended care facility
  - Home infusion therapy (including antibiotics)
  - Chronic dialysis within 30 days
  - Home wound care
  - Family member with multidrug-resistant pathogen
- Immunosuppressive disease and/or therapy

HCAP = Healthcare-associated pneumonia
The major sources of infection in VAP are healthcare devices, environment (air, water, equipment, and fomites), and transfer of microorganism from healthcare provider to the patient. The major potential reservoirs for organisms in a human body are stomach and sinuses. The bacteria get entry into the trachea from these potential sites via aspiration of oropharyngeal pathogens or via leakage of bacteria around endotracheal tube cuff. From trachea, the microbial pathogens migrate to the lower respiratory tract and colonize. Infected in the endotracheal tube, with subsequent embolization to distal airways, may also play an important role in the pathogenesis of VAP. If the host defense mechanisms are weak due to any factor or are insufficient to deal with the number of organisms, infection of the lower respiratory tract takes place.

Risk factors for the development of VAP can be differentiated into modifiable and non-modifiable conditions. Modifiable risk factors are obvious targets for improved management and prophylaxis. Important risk factors are enumerated in Table 2.

### Table 2: Risk factors for the development of VAP

- Age ≥ 60 years
- APACHE II score >16, severe burns
- Acute or chronic lung disease, cigarette smoking
- Excessive sedation, use of muscle relaxants
- Enteral nutrition
- Supine body position
- Glasgow coma scale < 9

### Prevention

Recommendations for modifiable risk factors for VAP prevention are presented in Table 3. Enhancing trace element status and anti-oxidant defenses by selenium, zinc, and copper supplementation has been shown to decrease nosocomial pneumonia in critically-ill, severely burned patients. In a recently published trial, patients receiving a silver-coated endotracheal tube had a significant reduction in the incidence of VAP and delayed time to VAP occurrence compared with those receiving a similar, uncoated tube. Role of intensive glycemic control and early tracheostomy in reducing the risk of VAP remains unclear at present. Many ICUs are adopting the ‘ventilator bundle’, which is a group of interventions related to patients receiving mechanical ventilation that, when implemented together, result in better outcomes than when implemented alone.

### Table 3: Recommendations for modifiable risk factors of VAP

#### General prophylaxis

- Routine staff education, alcohol-based hand disinfection, and isolation to reduce cross-infection
- Surveillance of ICU infections

#### Intubation and mechanical ventilation

- Avoid intubation and re-intubation, if possible
- NIV should be used whenever possible
- Orotracheal intubation and orogastric tubes preferred over nasotracheal intubation and nasogastric tubes
- Continuous aspiration of subglottic secretions
- Endotracheal tube cuff pressure should be maintained at >20 cm H₂O
- Contaminated condensate should be carefully emptied from ventilator circuits
- Change ventilatory circuit only when visibly soiled or malfunctioning
- Passive humidifiers or heat-moisture exchangers cannot be regarded as a pneumonia prevention tool
- Sedation vacation
- Maintaining adequate staffing levels in the ICU

#### Aspiration, body position, and enteral feeding

- Semi-recumbent position (30-45°)
- Prefer enteral nutrition over parenteral nutrition
Modulation of colonization: oral antiseptics and antibiotics

- Prevention of VAP by selective digestive or oropharyngeal decontamination recommended to contain MDR outbreaks; but is not recommended for routine use
- Prophylactic administration of systemic antibiotics for 24 hours at the time of emergent intubation in patients with closed head injury; don’t use routinely
- Oral chlorhexidine in those undergoing coronary bypass grafting, but routine use not recommended

Stress-ulcer prophylaxis and transfusion

- Avoidance of H2 antagonist or proton pump inhibitors for patients who are not at high risk for developing gastrointestinal bleeding remains unresolved
- Follow a restricted transfusion policy

Box 1: Components of ‘ventilator bundle’

1. Elevation of the head of the bed to between 30 and 45 degrees
2. Daily “sedation vacation”
3. Daily assessment of readiness to extubate
4. Peptic ulcer disease prophylaxis
5. Deep vein thrombosis prophylaxis (unless contraindicated)

Diagnosis of VAP

The accurate diagnosis of VAP remains problematic. Standard diagnostic features of pneumonia such as fever, tachycardia, leukocytosis, purulent sputum, and consolidation on the chest radiograph are unreliable in the critically-ill mechanically ventilated patient. Fever, leukocytosis, and tachycardia are non-specific markers of inflammation, not necessarily infection. Purulent sputum may be caused by tracheo-bronchitis. Infiltrates on the chest radiograph can be caused by a number of non-infective conditions including pulmonary edema, hemorrhage, contusions, aspiration, atelectasis, and acute lung injury. Disappointingly the use of scoring systems such as the clinical pulmonary infection score (CPIS), seems to add little to diagnostic accuracy (Table 4).

**Table 4: The clinical pulmonary infection score (CPIS)**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Score</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature °C</td>
<td>≥ 36.1 to &lt; 38.4</td>
<td>≥ 38.5 to &lt; 38.9</td>
<td>≥ 39 to &lt; 36</td>
<td></td>
</tr>
<tr>
<td>WBC count per µL</td>
<td>≥ 4,000 to &lt; 11,000</td>
<td>&lt; 4,000 to &gt; 11,000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secretions Absent</td>
<td>Present, non-purulent</td>
<td>Present, purulent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PaO2/FiO2</td>
<td>&gt; 240 or ARDS</td>
<td>≤ 240 and no ARDS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest radiography No infiltrate</td>
<td>Moderate or patchy infiltrate</td>
<td>Localized infiltrate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Microbiology No or light growth</td>
<td>Moderate or heavy growth; add one point for same organism on gram stain</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Nevertheless, the diagnosis of VAP is suspected if the patient has a new or progressive infiltrate on the chest radiograph accompanied by fever, leukocytosis, or purulent secretions. This is often accompanied by deterioration in gas exchange. Clinical criteria for diagnosis of VAP are presented in Table 5. Although clinical suspicion is highly sensitive it lacks specificity, and hence further diagnostic tests are required for optimal management.

Microbiological diagnosis

Ideally, the microbiological data should be obtained before the start of antibiotic therapy. The optimal method of
microbiological diagnosis of VAP remains controversial. As the trachea rapidly becomes colonized with bacteria in the critically-ill patient, cultures of sputum or tracheal aspirates may simply yield colonizers. The argument therefore revolves around whether specimens of lower respiratory tract secretions should be collected in an invasive manner or whether analysis of non-invasively collected tracheal aspirates is sufficient. Analyzing samples using quantitative culture techniques theoretically permits differentiation between colonization versus infection.

Table 5: American College of Chest Physicians criteria for VAP

i. Presence of new/progressive pulmonary infiltrates on chest radiograph, which is not otherwise explained
   plus

ii. At least two of the following
   (1) Temperature of $>38\degree C$
   (2) Leukocytosis $>10,000/\mu L$ or leukopenia $<5,000/\mu L$
   (3) Purulent respiratory secretions

According to the latest guidelines, obtain one sample of lower respiratory tract secretions for direct examination and culture before initiating or changing antibiotics. Delays in the initiation of appropriate antibiotic therapy can increase the mortality of VAP, and thus therapy should not be postponed for the purpose of performing diagnostic studies in patients who are clinically unstable. Tracheal aspirate, or bronchoscopic or non-bronchoscopic alveolar lavage material can be used effectively. A negative tracheal aspirate (absence of bacteria or inflammatory cells) in a patient without a recent (within 72 hours) change in antibiotics has a strong negative predictive value (94%) for VAP and should lead to a search for alternative sources of fever. A decision to employ bronchoscopy should be considered based on case to case basis and the availability of local expertise. Respiratory secretions obtained for microbiological evaluation should be transported to the laboratory and processed in $<2$ hours. Quantitative cultures have not yet been sufficiently standardized nor have they been shown to alter outcome for this technique to be considered part of routine evaluation. Respiratory secretions that are judged to be appropriate samples by the laboratory should be evaluated by Gram's stain and cultured for routine aerobic and facultative bacteria.

Pleural fluid should be obtained for Gram's staining and routine culture (with other studies as clinically indicated) if there is an adjacent infiltrate or another reason to suspect infection and the fluid can be safely aspirated. Blood cultures have limited value in the diagnosis of VAP and have a very low sensitivity for detecting the pathogenic organism responsible for the pneumonia. However, blood cultures are obviously useful in any patient with signs of sepsis. Isolation of a microorganism in the blood does not confirm the pathogen responsible for VAP. It is recommended that all patients with suspected VAP should have blood cultures performed, recognizing that a positive result can indicate the presence of either pneumonia or extra-pulmonary infection.

Immunological methods for the diagnosis of VAP also hold promise for the future. The triggering receptor expressed on myeloid cells (TREM-1) is a member of the immunoglobulin superfamily, and is involved in the acute inflammatory response. Neutrophils express high levels of TREM-1 on exposure to infected tissues. The presence of soluble TREM-1 in the broncho-alveolar lavage (BAL) fluid has been shown to be a highly accurate method for the diagnosis of fungal or bacterial pneumonia with a sensitivity of 98% and a specificity of 90%.

Treatment

Optimal management of patients with suspected VAP requires prompt initiation of appropriate antimicrobial therapy and general supportive care. Although microbiological sampling should be performed before start of therapy, this must not delay the commencement of antibiotic. Several studies have shown that delay in administration of antibiotics is associated with an increase in mortality rate.

Appropriate antibiotics in adequate doses should be instituted promptly. Initial empiric therapy should be tailored to the local microbial epidemiology and susceptibility patterns. Because of the plethora of potential causative organisms, a broad spectrum antibiotic regimen should be given initially with activity against enteric Gram-negative organisms. Initial antibiotics should always be given intravenously in the most aggressive tolerated doses with a switch to oral/enteral therapy in selected
patients with a good clinical response and a functioning intestinal tract. Highly bioavailable agents, such as the fluoroquinolones and linezolid, may be easily switched to oral therapy in such patients. The guidelines for initial empiric therapy are summarized in Table 6. The efficacy of aerosolized antibiotics (tobramycin, polymyxin) is unproven in the therapy of VAP. However, they may be considered as adjunctive therapy in patients with MDR Gram-negatives who are not responding to systemic therapy.

Table 6: Initial empiric therapy for VAP

| Patients with no known risk factors for MDR pathogens, early onset, and any disease severity | Ceftriaxone  
or Levofloxacin, moxifloxacin, or ciprofloxacin  
or Ampicillin/sulbactam  
or Ertapenem |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Potential pathogens:</td>
<td></td>
</tr>
<tr>
<td>Streptococcus pneumoniae</td>
<td></td>
</tr>
<tr>
<td>Haemophilus influenzae</td>
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<tr>
<td>Methicillin-sensitive S. aureus</td>
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<tr>
<td>Antibiotic-sensitive enteric Gram-negative bacilli</td>
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</tbody>
</table>

| Patients with late-onset disease or risk factors for MDR pathogens and all disease severity | Anti-pseudomonal cephalosporin (Cefepime, ceftazidime)  
or Anti-pseudomonal carbapenem (imipenem or meropenem)  
or β-lactam/β-lactamase inhibitor (piperacillin-tazobactam)  
plus Anti-pseudomonal fluoroquinolone (ciprofloxacin or levofloxacin)  
or Aminoglycoside (amikacin, gentamicin, or tobramycin)  
plus Linezolid or vancomycin |
<table>
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<tbody>
<tr>
<td>Potential pathogens: Organisms listed above plus MDR pathogens:</td>
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<tr>
<td>P. aeruginosa</td>
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<tr>
<td>A. baumannii</td>
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<tr>
<td>K. pneumoniae (ESBL+)</td>
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<td>MRSA</td>
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De-escalation of the initial broad-spectrum therapy to the most focused regimen possible should be done on the basis of results of cultures and clinical response. This is necessary to reduce the excess antibiotic use and its attendant side effects, excess costs, and risk of emergence of MDR pathogens. The optimal duration of antimicrobial treatment for VAP is unclear. In a randomized clinical trial of over 400 patients, comparing 8 days versus 15 days of antibiotic therapy VAP, it was found that the efficacy of 8-day regimen was comparable to 15-day treatment regimen. In particular, there was no excess mortality or increase in microbiologically reported pulmonary infection recurrence in the 8-day group. Therefore, treatment can be stopped after 7-8 days in patients with uncomplicated VAP showing a good clinical response. However a longer duration (14-21 days) of therapy is required in the following circumstances:

- Multi-lobar pneumonia
- Malnutrition
- Cavitations on chest radiograph
- Gram-negative necrotizing pneumonia
- Isolation of P. aeruginosa, A. baumannii

Recommendations for MDR pathogens

If P. aeruginosa pneumonia is documented, combination therapy is recommended. If A. baumannii is documented, the most active agents are the carbapenems, sulbactam, colistin, and polymyxin. There are no data documenting an improved outcome if these organisms are treated with a combination regimen. If extended-spectrum β-lactamase (ESBL) producing Enterobacteriaceae are isolated, then monotherapy with a third-generation cephalosporin should be avoided. The most
active agents are carbapenems. Linezolid is an alternative to vancomycin for the treatment of MRSA-associated VAP and may be preferred if patients have renal insufficiency or are receiving other nephrotoxic agents. Antibiotic restriction can limit epidemics of infection with specific resistant pathogens. Formal antibiotic cycling might be able to reduce the overall frequency of antibiotic resistance. However, the long-term impact of this practice is unknown.

**Newer antibiotics**

Up to 70% of nosocomial infections are now caused by organisms resistant to one or more drugs. Medical personnel are more commonly dealing with organisms that are resistant to most broad spectrum agents. Unfortunately, the rapid rise in resistant organisms has outpaced the development of novel, effective, well-tolerated antimicrobial agents. It is therefore crucial that physicians attempt to use currently available antibiotics in a rational, judicious way to preserve their utility for future, critical infections. Colistin (polymyxin E) possesses concentration-dependent, bactericidal activity against a broad variety of Gram-negative organisms, including MDR strains. The primary toxicities associated with colistin are nephrotoxicity and neurotoxicity; both are possibly dose-related and reversible upon discontinuation. Due to the potential toxicity of this agent, the drug is generally reserved for MDR strains of *P. aeruginosa*, *K. pneumoniae*, and *A. baumannii* when few other options are available.

Tigecycline is a derivative of minocycline and covers both resistant Gram-negative and Gram-positive organisms. Additionally, it has good activity against anaerobic species including *Bacteroides fragilis*. Despite its broad spectrum Gram-negative activity, it is not active against *Pseudomonas*, *Morganella*, and *Providencia* species; that limits its usefulness as an empiric agent in the critically-ill. The drug has clinical utility for patients with penicillin allergies or renal dysfunction for the treatment of intra-abdominal and skin infections and infections caused by multiple resistant pathogens including MRSA, vancomycin-resistant *enterococci*, *A. baumannii*, and ESBL producing *K. pneumoniae*.

**Summary**

- VAP is the most common nosocomial infection in the ICU
- It is associated with substantial morbidity and mortality
- Duration of endotracheal intubation is the most important risk factor
- Late-onset VAP is frequently associated with multidrug-resistant pathogens
- Clinical diagnosis is unreliable; Microbiological diagnosis should complement clinical diagnosis
- Specimen from lower respiratory tract can be obtained bronchoscopically or non-bronchoscopically
- Empiric antibiotic treatment should be started pending bacteriological results
- Narrow down the antibiotic spectrum on the basis of microbiological data
- Hand-hygiene is the most important prevention strategy
- Apply other prevention strategies aimed at modifiable risk factors
- Introduce ventilator bundle in your ICU

**References**

6. Tracheostomy: When and How?

Henry B Nongrum, Alok Thakar, Aloka Samantaray, Abha Chandra

Tracheostomy is one of the most common procedures performed in the intensive care unit (ICU) for various indications. However, there has been much discussion about the type of tracheostomy procedure (percutaneous dilatational versus open surgical), the ideal locale to perform the procedure, effect of timing of tracheostomy on length of ICU stay, weaning from ventilator, cost of care, advantages over endotracheal intubation, complications and the incidence of nosocomial pneumonia related to tracheostomy. Over the years, the utilization of tracheostomy has expanded. It is also being commonly performed in the ICU by specialists other than otolaryngologists. This has led to the need for a larger number of personnel to be updated with the method of surgery, care of tracheostomy and procedure of decannulation.

Indications for Tracheostomy

Tracheostomy is generally indicated for
- Respiratory obstruction
- Respiratory failure
- Respiratory paralysis
- Removal of retained secretions
- Reduction of dead space

However in the ICU, the most common indications are
- Prolonged orotracheal intubation
- Difficult airway
- To facilitate tracheobronchial hygiene
- Multiple attempts to wean from mechanical ventilation have been unsuccessful for 14 to 21 days

Timing of Tracheostomy

The optimal timing to perform a tracheostomy in a critically-ill patient is controversial. Because there are no definitive guidelines available, the timing of tracheostomy depends on clinical conditions, physician judgment, and communication with the patient’s family. There is little consensus on the timing of tracheostomy. The 1989 American College of Chest Physicians (ACCP) Consensus Conference on Artificial Airways in patients receiving mechanical ventilation concluded that the appropriate duration of translaryngeal intubation could not be defined. It was suggested that if the anticipated need for mechanical ventilation is longer than 21 days then tracheostomy is preferable. For mechanical ventilation that is anticipated to last between 10 and 21 days, the decision was left to the physician, and a daily assessment was recommended.

However, a recently published systematic review of randomized controlled trials and meta-analysis found that early tracheostomy (up to seven days after ICU admission) significantly shortened the duration of mechanical ventilation and ICU stay. But, early tracheostomy did not decrease mortality or the risk of pneumonia. The ACCP suggests that “tracheostomy should be considered after an initial period of stabilization on the ventilator, when it becomes apparent that the patient will require prolonged ventilator assistance.” The obstacle remains the accurate prediction of patients that would need prolonged assisted ventilation. The best predictors appear to be nonspecific markers of poor outcome such as a high Acute Physiology and Chronic Health Evaluation II score (APACHE II score > 25) and the presence of shock at the time of ICU admission.

Tracheostomy Techniques

Tracheostomy can be performed by either a traditional open technique or a percutaneous dilatational technique (PDT). The open technique performed by a surgeon is generally done in the operating room or at bedside. However, PDT is increasingly
becoming a popular technique and could be performed by a trained physician at bedside. Below is a brief description of the surgical steps.

**Open surgical tracheostomy**

- Anesthesia: local or general anesthesia
- Make a collar incision 2 cm above suprasternal notch; elevate skin flaps
- Retract the strap muscles from the midline or divide medial fibers
- Thyroid isthmus may be avoided; otherwise, divide
- Palpate and expose the trachea
- Alert the anesthetist; keep suction ready
- Children – insert two stay-sutures on the trachea; use a vertical tracheal incision
- Adults – use a horizontal tracheal incision, in the third tracheal space; may need to excise part of one tracheal ring
- Insert the tube and connect to anesthetic circuit
- Tape stay sutures to the chest in a child
- Loose sutures on skin
- Suture and tape the tube

**Percutaneous dilatational tracheostomy**

The percutaneous method consists of percutaneous needle puncture of the trachea, followed by stepwise (Ciaglia technique) or one-stage (Griggs’s technique) dilatation and placement of a tracheostomy tube.

**Ciaglia technique (Figure 1a-e)**

- A 2-cm transverse incision is made at the level of T₁-T₂ or T₂-T₃ tracheal ring.
- A needle is passed into the trachea after the existing endotracheal tube has been withdrawn above the incision site.
- A guide wire is passed through the needle toward the carina. A guiding catheter is then passed over the guide wire.
- Multiple dilators or one–staged tracheal spreader or dilator are used to create a tracheostoma by passing it over the guiding catheter with the guide wire in-situ.
- Tracheostomy tube is finally passed over a guide wire.

**Griggs’s technique**

- Initial steps similar as above.
- Once the guide wire is introduced into the trachea the dilating forceps is threaded over the guide wire and introduced through the anterior tracheal interspace. This is done in two steps.
- First step - dilate the pre-tracheal tissue with the forceps at right angle to the tracheal wall.
- Second step - puncture the trachea, dilate the anterior tracheal wall with the forceps handle tilted towards the head of the patient, so that the tip lies inside and parallel
Fig. 1b: Introducing a guide wire through the needle into the trachea.

Fig. 1c: Guiding catheter inserted over the guide wire.

Fig. 1d: One-stage dilator inserted over the guiding catheter to create a tracheostoma.

Fig. 1e: Tracheostomy tube introduced over the guide wire.
to the tracheal wall.

- Remove the forceps and the selected tracheostomy tube is finally passed over the guide wire.

A fiberoptic bronchoscope passed through the endotracheal tube can prove useful. It helps to correctly position the endotracheal tube above the site of incision, prevent undue pressure of the inserted instruments on the posterior tracheal wall, ensure correct placement of the tube, and to enhance the safety of the procedure and also serves as a valuable teaching aid (Figures 2 and 3).

**Contraindications for percutaneous tracheostomy**

- Hemodynamic instability
- Infection and cellulitis of the neck
- Previous tracheostomy or neck surgery, anatomic distortion like thyromegaly, tumor, etc.
- Morbid obesity
- Cervical vertebrae injury or fracture
- Emergency situation
- Children
- High positive end expiratory pressure requirement

**Tracheostomy Care**

1. A well trained nurse should be in attendance at all times for the first 24 hours.
2. Secretions should be removed by suctioning every 30 minutes or more often if indicated, in the first 48 hours, after which it can be lengthened. This should be done with sterile gloves and minimal trauma to the tracheal and bronchial mucosa.
3. Artificial humidification is required to prevent crusting. This is done by a hot water bath humidifier or by nebulizer delivering cold air.
4. The tube is first changed after 48 hours and thereafter every 2 – 3 days.
5. The cuff should be deflated for 5 minutes every hour.
6. Daily dressing around the tracheostoma to prevent wound infection.

**Percutaneous versus Open Surgical Tracheostomy**

There remains a controversy as to which procedure is superior. Concerns center around each technique’s cost-effectiveness, complications, and safety. The open surgical technique is often performed in the operating room. It therefore increases the cost and carries with it the hazards of transporting critically-ill patients with the associated risks of accidental disconnection of the breathing circuit and inconvenience of operating room schedules. The percutaneous procedure, as it is performed at the bedside, has helped to resolve some of these issues. However, with adequate lighting and instrumentation, the open surgical method can be well performed at bedside. Even though it may appear old-fashioned, open bedside tracheostomy remains a safe and simple procedure. It is cheaper than other techniques and should be considered as an option for ICU patients.
A meta-analysis of randomized trials found that percutaneous tracheostomy is faster to perform and less costly than conventional open surgical tracheostomy. However, the percutaneous technique was associated with a slightly higher risk of early complications which are minor. Another meta-analysis comparing the percutaneous and surgical techniques in critically-ill patients found that the risk of wound infection and bleeding was significantly less with the percutaneous technique.

**Principles of Tracheostomy-Closure**

Prior to attempting decannulation the following need to be ensured

- Adequate muscle strength and respiratory power
- Reversal of initial chest pathology, if any
- Adequacy of supra-tracheostomal respiratory airway
- Ability to clear tracheal secretions/effective cough

**Procedure for decannulation**

- Initially, the tracheostomy tube size is reduced.
- Later, the tracheostomy tube is kept blocked for increasing periods.
- Once the patient can tolerate the corked tube and sleep adequately for at least one night with reasonable ventilation and clearance of respiratory secretions, the tube can be removed and the tracheostomy site is strapped.

**Complications of Tracheostomy**

**Early complications**

1. Bleeding
2. Tube obstruction
3. Tube displacement
4. Subcutaneous emphysema
5. Pneumothorax

**Late complications**

1. Innominate artery rupture
2. Infection
3. Aspiration
4. Granuloma formation
5. Subglottic stenosis
6. Tracheoesophageal fistula

**References**

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