

Chapter 2

The Indications for Mechanical Ventilation

Apart from its supportive role in patients undergoing operative procedures, mechanical ventilatory support is indicated when spontaneous ventilation is inadequate for the sustenance of life.

The word *support* bears emphasis, for mechanical ventilation is not a cure for the disease for which it is instituted: it is at best a form of support, offering time and rest to the patient until the underlying disease processes are resolved. Results with mechanical ventilation are consistently better when mechanical ventilatory support is initiated early and electively rather than in a crash situation.

The indications for mechanical ventilation may be viewed as falling under several broad categories (Fig. 2.1).

2.1 Hypoxia

Mechanical ventilation is often electively instituted when it is not possible to maintain an adequate oxygen saturation of hemoglobin. While optimization of tissue oxygenation is the goal, it is rarely possible to reliably assess the extent of *tissue* hypoxia. Instead, indices of *blood* oxygenation may rather need to be relied upon. Increasing the fraction of inspired oxygen (FIO_2) indiscriminately in an attempt to improve oxygenation may unnecessarily subject the patient to the danger of oxygen toxicity (these concepts will be addressed at a later stage). Mechanical ventilation enables better control

Indications for intubation	Indications for ventilation
• Need to secure airway • Depressed sensorium • Depressed airway reflexes • Upper airway instability after trauma • Decreased airway patency • Need for sedation in the setting of poor airway control • Imaging (CT, MRT) and transportation of an unstable patient	• Hypoxia: acute hypoxemic respiratory failure • Hypoventilation • Unacceptably high work of breathing • Hemodynamic compromise • Cardiorespiratory arrest • Refractory shock • Raised intracranial pressure • Flail chest

FIGURE 2.1. Indications for intubation & ventilation.

of hypoxemia with relatively low inspired O_2 concentrations, thereby diminishing the risk of oxygen toxicity.

2.2 Hypoventilation

A major indication for mechanical ventilation is when the alveolar ventilation falls short of the patient's requirements. Conditions that depress the respiratory center produce a decline in alveolar ventilation with a rise in arterial CO_2 tension. A rising $PaCO_2$ can also result from the hypoventilation that results when fatiguing respiratory muscles are unable to sustain ventilation, as in a patient who is expending considerable effort in moving air into stiffened or obstructed lungs. Under such circumstances, mechanical ventilation may be used to support gas exchange until the patient's respiratory drive has been restored, or tired respiratory muscles rejuvenated, and the inciting pathology significantly resolved (Fig. 2.2).

Hypoventilation results from decreased bulk flow in and out of the lungs				
Inspiration results in the bulk flow of air into the lungs, up to the level of the smallest bronchioles. Further progress of the gas molecules is by the mechanism of facilitated diffusion peripherally				
Disorders in which bulk flow to the lungs is compromised include				
<i>CNS depression</i> e.g., Sedative agents Cerebrovascular accidents Central sleep apnea Metabolic alkalosis Myxedema Hyperoxia (Hyperoxic hypoventilation)	<i>Spinal cord or peripheral nerve disorders</i> e.g., Spinal trauma Amyotrophic lateral sclerosis Polio Multiple sclerosis Guillain Barre syndrome Botulism	<i>Neuro-muscular disorders</i> e.g., Aminoglycosides Paralysing agents Steroid myopathy Myasthenia gravis Muscular dystrophies Dyselectrolyte mias Poor nutrition Respiratory muscle fatigue	<i>Disorders affecting the thoracic cage</i> e.g., Kyphoscoliosis Flail chest Ankylosing spondylitis	<i>Proximal airway (extra-pulmonary airway) obstruction</i> e.g., Tracheal obstruction by stenosis, tumor etc Epiglottitis Obstructive sleep apnea

FIGURE 2.2. Causes of Hypoventilation.

2.3 Increased Work of Breathing

Another major category where assisted ventilation is used is in those situations in which excessive work of breathing results in hemodynamic compromise. Here, even though gas exchange may not be actually impaired, the increased work of breathing because of either high airway resistance or poor lung compliance may impose a substantial burden on, for example, a compromised myocardium.

When oxygen delivery to the tissues is compromised on account of impaired myocardial function, mechanical ventilation by resting the respiratory muscles can reduce the work of breathing. This reduces the oxygen consumption of the respiratory muscles and results in better perfusion of the myocardium itself.

2.4 Other Indications

In addition to these major indications, mechanical ventilation may be of value in certain specific conditions. The vasoconstriction produced by deliberate hyperventilation can reduce the volume of the cerebral vascular compartment, helping to reduce raised intracranial pressures. In flail chest, mechanical ventilation can be used to provide internal stabilization of the thorax when multiple rib fractures compromise the integrity of the chest wall; in such cases, mechanical ventilation using positive end-expiratory pressure (PEEP) normalizes thoracic and lung mechanics, so that adequate gas exchange becomes possible.

Where postoperative pain or neuromuscular disease limits lung expansion, mechanical ventilation can be employed to preserve a reasonable functional residual capacity within the lungs and prevent atelectasis. These issues have been specifically addressed in Chap. 9.

2.5 Criteria for Intubation and Ventilation

While the prevailing criteria for defining the need for intubation and ventilation of a patient in respiratory failure have met general acceptance, these are largely intuitive and based upon the subjective assessment of a patient's condition (Fig. 2.3 and Table 2.1). *See also Chap. 12.*

Objective criteria that are in current use are a forced expiratory volume in the first second (FEV_1) of less than 10 mL/kg body weight and a forced vital capacity (FVC) of less than 15 mL/kg body weight, both of which indicate a poor ventilatory capability.

Similarly, a respiratory rate higher than 35 breaths/min would mean an unacceptably high work of breathing and a substantial degree of respiratory distress, and is recognized as one of the criteria for intubation and ventilation. A $PaCO_2$ in excess of 55 mmHg (especially if rising, and in the presence of acidemia) would likewise imply the onset of respiratory muscle fatigue. Except in habitual CO_2 retainers, a $PaCO_2$ of

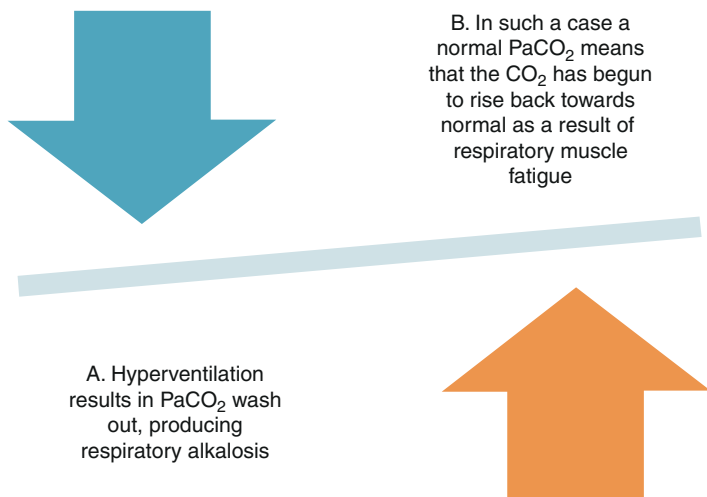


FIGURE 2.3. PaCO_2 in status asthmaticus.

55 mmHg and over would normally reflect severe respiratory muscle dysfunction.

Documented PaCO_2 from an earlier stage of the patient's present illness may have considerable bearing on the interpretation of subsequent PaCO_2 levels (Fig. 2.3). For example, in an asthmatic patient in acute severe exacerbation, bronchospasm-induced hyperventilation can be expected to "wash out" the CO_2 from the blood, producing respiratory alkalosis. If in such a patient, the blood gas analysis were to show a normal PaCO_2 level, this would imply that the hypoventilation produced by respiratory muscle fatigue has allowed the PaCO_2 to rise back to normal. It is important to realize here, that although the PaCO_2 is now in the normal range, it is actually on its way up, and if this is not appreciated, neither the PaCO_2 nor the patient will stay normal for very long. A supranormal PaCO_2 in status asthmaticus should certainly be a cause of alarm and reinforce the need for mechanical ventilatory support.

A PaO_2 of less than 55–60 mmHg on 0.5 FIO_2 or a widened A-a DO_2 gradient (of 450 mmHg and beyond on 100% O_2)

TABLE 2.1. Criteria for ventilation.

Criteria value	Normal range	Critical level	Comment
Respiratory muscle performance			
Maximum inspiratory pressure (MIP)	-50 to -100 cm H ₂ O	More positive than -20 cm H ₂ O	Useful in neuromuscular patients. Can be measured by a Bourdon manometer interfaced to the patient by a mask, mouthpiece, or ET adaptor. Ideally, MIP measurements should be made after maximal exhalation
Maximum expiratory pressure (MEP)	+100 cm H ₂ O	Less than 40 cm H ₂ O	
Vital capacity (VC)	65-75 mL/kg	<15 mL/kg	Measured at the bedside with a pneumotachometer or a hand-held spirometer
Tidal volume (V _I)	5-8 mL/kg	<5 mL/kg	Measured at the bedside with a pneumotachometer or a hand-held spirometer
Respiratory frequency (f)	12-20 breaths/min	>35 breaths/min	A high respiratory frequency indicates increased work of breathing, and may be indicative of impending respiratory muscle exhaustion
Forced expired volume at 1 s (FEV ₁)	50-60 mL/kg	<10 mL/kg	Important in evaluating the degree of airway obstruction in COPD/asthma. May be difficult or exhausting for the severely obstructed patient
Peak expiratory flow	350-600 L/min	<100 L/min	Important in evaluating the degree of airway obstruction in COPD/asthma. May be difficult or exhausting for the severely obstructed patient
Ventilation			
pH	7.35-7.45	<7.25	A falling pH from respiratory acidosis is a late feature of respiratory muscle fatigue

Criteria value	Normal range	Critical level	Comment
PaCO ₂	35–45 mmHg	>55 mmHg, and rising	A rising PaCO ₂ from respiratory acidosis is a late feature of respiratory muscle fatigue
VD/VT	0.3–0.4	>0.6	Dead-space ventilation can be easily calculated at the bedside using capnometry and blood gas analysis (see Chap. 3)
<i>Oxygenation (low values indicate the need for oxygen therapy or PEEP/CPAP; mechanical ventilation may be required if hypoxemia is nonresponsive to the above support, or is very severe)</i>			
PaO ₂	80–100 mmHg	<60 mmHg (on FIO ₂ 0.5)	A PaO ₂ of 60 mmHg represents the approximate point where the slope of the oxy-hemoglobin dissociation curve abruptly changes. As the PaO ₂ drops further below 60 mmHg, the SpO ₂ can be expected to fall sharply
Alveolar-to-arterial oxygen difference	3–30 mmHg	>450 mmHg (on high concentrations of O ₂)	The A-a DO ₂ is the difference between the alveolar O ₂ tension (PAO ₂) and the arterial oxygen tension (PaO ₂), and is a measure of the ease with which the administered oxygen diffuses into the pulmonary capillary blood
Arterial/alveolar PO ₂	0.75	<0.15	The PaO ₂ /PAO ₂ ratio is the proportion of oxygen in the alveolus that eventually gains entry into the pulmonary capillary blood. The PaO ₂ is easily read out from the ABG, but the PAO ₂ cannot be directly measured and needs to be calculated from the alveolar gas equation (see section 7.1)
PaO ₂ /FIO ₂	475	<200	The PaO ₂ /FIO ₂ ratio obviates the need to calculate PAO ₂ (which can be something of an effort for those who are mathematically challenged!)

means that the gas exchange mechanisms in the lung are deranged to a degree that cannot be supported by external oxygen devices alone, and that intubation and ventilation is required for effective support.

It is important to emphasize that the criteria for intubation and ventilation are meant to serve as a guide to the physician who must view them in the context of the clinical situation. Conversely, the patient does not necessarily have to satisfy every criterion for intubation and ventilation in order to be a candidate for invasive ventilatory management. Importantly, improvement or worsening in the trends within these numbers provide the key to judgment in a borderline situation. It must also be pointed out that with the advent of noninvasive positive pressure ventilation as a potential tool for the treatment of early respiratory failure, some of the criteria for the institution of mechanical ventilatory support may need to be revisited. These issues have been discussed in Chap. 13.

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