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

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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).

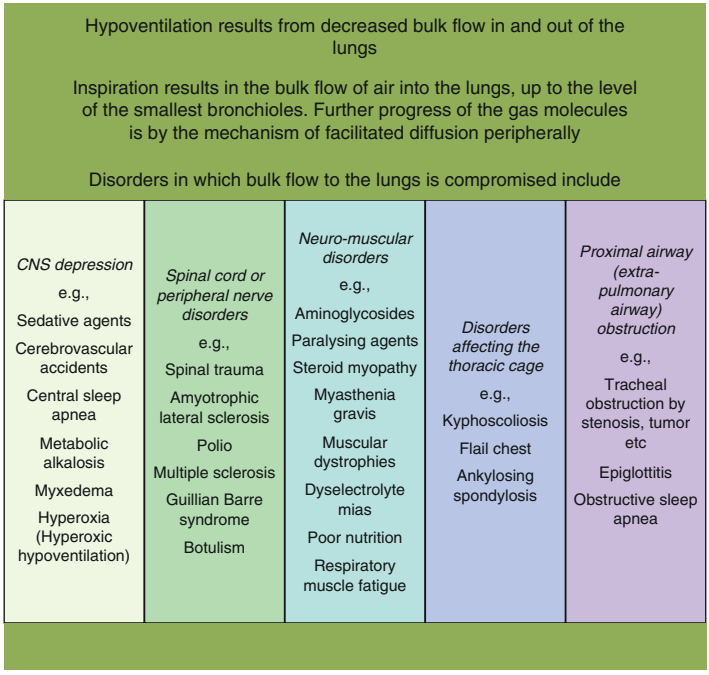

FIGURE 2.2. Causes of Hypoventilation.

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

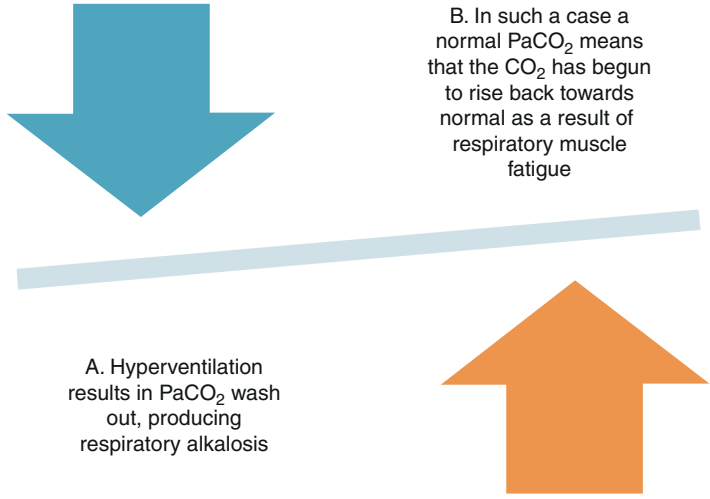

FIGURE 2.3. $PaCO_2$ in status asthmaticus.

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_2O	More positive than –20 cm H_2O	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_2O	Less than 40 cm H_2O	
Vital capacity (VC)	65–75 mL/kg	<15 mL/kg	Measured at the bedside with a pneumotachometer or a hand-held spirometer
Tidal volume (V1)	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)
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)			
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.

References

1. Brochard L. Profuse diaphoresis as an important sign for the differential diagnosis of acute respiratory distress. *Intensive Care Med*. 1992;18:445
2. Comroe JH, Botelho S. The unreliability of cyanosis in the recognition of arterial anoxemia. *Am J Med Sci*. 1947;214:1–6
3. Gibson GJ, Pride NB, Davis JN, et al Pulmonary mechanics in patients with respiratory muscle weakness. *Am Rev Respir Dis*. 1977;115:389–395
4. Gilston A. Facial signs of respiratory distress after cardiac surgery: a plea for the clinical approach to mechanical ventilation. *Anaesthesia*. 1976;31:385–397
5. Hess DR, Branson RD. In: Hess DR, MacIntyre NR, Mishoe SC, et al, eds. *Respiratory care: principles and practices.* Philadelphia: WB Saunders; 2003
6. Kacmarek RM, Cheever P, Foley K, et al Deterination of vital capacity in mechanically ventilated patients: a comparison of techniques. *Respir Care*. 1990;35(11):129

7. Lundsgaard C, Van Slyke DD. Cyanosis. *Medicine*. 1923;2:1–76
8. Manthous CA, Hall JB, Kushner R, et al The effect of mechanical ventilation on oxygen consumption in critically ill patients. *Am J Respir Crit Care Med*. 1995;151:210–214
9. Medd WE, French EB, McA Wyllie V. Cyanosis as a guide to arterial oxygen desaturation. *Thorax.* 1959;14:247–250
10. Mithoefer JC, Bossman OG, Thibeault DW, Mead GD. The clinical estimation of alveolar ventilation. *Am Rev Respir Dis*. 1968;98:868–871
11. Perrigault PF, Pouzeratte YH, Jaber S, et al Changes in occlusion pressure (P0.1) and breathing pattern during pressure support ventilation. *Thorax*. 1999;54:119–123
12. Semmes BJ, Tobin MJ, Snyder JV, Grenvik A. Subjective and objective measurement of tidal volume in critically ill patients. *Chest*. 1985;87:577–579
13. Slutsky AS. Mechanical ventilation. American College of Chest Physicians' Consensus Conference. *Chest*. 1993;104:1833
14. Strohl KP, O'Cain CF, Slutsky AS. Alae nasi activation and nasal resistance in healthy subjects. *J Appl Physiol*. 1982;52:1432–1437
15. Tobin MJ, Guenther SM, Perez W, et al Konno-Mead analysis of ridcage- abdominal motion during successful and unsuccessful trials of weaning from mechanical ventilation. *Am Rev Respir Dis*. 1987;135:1320–1328
16. Tobin MJ, Jenouri GA, Watson H, Sackner MA. Noninvasive measurement of pleural pressure by surface inductive plethysmography. *J Appl Physiol*. 1983;55:267–275
17. Tobin MJ, Mador MJ, Guenther SM, et al Variability of resting respiratory drive and timing in healthy subjects. *J Appl Physiol*. 1988;65:309–317
18. Tobin MJ. Respiratory muscles in disease. *Clin Chest Med*. 1988;9:263–286
19. Tobin MJ. Noninvasive monitoring of ventilation. In: Tobin MJ, ed. *Principles and Practice of Intensive Care Monitoring*. New York: NcGraw-Hill; 1998:465–495
20. Tobin MJ, Perez W, Guenther SM, et al Does rib cage-abdominal paradox signify respiratory muscle fatigue? *J Appl Physiol*. 1987;63:851–860

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