Mechanical Ventilation: History and Harm

Slutsky AS. History of mechanical ventilation. From Vesalius to ventilator-induced lung injury. *Am J Respir Crit Care Med.* 2015;191:1106-1115.

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Early Years

Contemporary approaches to mechanical ventilation began in the 1700s with the discovery of oxygen and subsequently the importance of respiration in the delivery of oxygen. Mouth-to-mouth resuscitation had been described by this time. Ironically, as Slusky notes, mouth-to-mouth resuscitation was discontinued after the discovery of oxygen because it was believed that exhaled air was lacking in oxygen because it had already been processed in another person's lungs.

Ventilator therapy based on contemporary appreciation of applied physiology began to appear in the 19th century. At this time, ventilation was provided using subatmospheric pressure delivered around the patient to replace or augment work done by respiratory muscles. Patients could sit or lay in a box that enclosed the body from the neck down or contained the entire patient. A plunger or other mechanism was used to decrease air pressure in the box, causing inspiration while the reverse produced expiration. The first workable iron lung was introduced in 1876 and replaced the previous box devices. The iron lung was used in Boston to treat polio patients in the early 20th century.

A major problem with all of these devices was the extreme difficulty associated with nursing patients because it was difficult to get access to the body. To address this problem, respirator rooms were developed in which the patient lay with their head outside the room while inside huge pistons generated pressure changes sufficient to cause air to move into and out of the lungs. The ventilator room had a door so that the medical staff could enter the ventilator to care for the patient. Ultimately, rooms were developed in which multiple patients could be treated. This technology again was used in Boston for patients afflicted by several epidemics.

When polio reappeared in the 1950s, it was a watershed event in the history of mechanical ventilation. Before this time, mechanical ventilation appeared to have usefulness but was not widely used. After the 1950s outbreak of polio, the benefits of ventilation were dramatic and obvious, leading to widespread use worldwide. In 1951, an international polio conference in Copenhagen was attended by most of the world's polio experts. The following summer, Copenhagen experienced a terrible polio epidemic likely triggered by the carriage of polio virus to Copenhagen during the previous year's conference. At the height of the epidemic, 50 patients per day were being admitted to the hospital, many with respiratory failure. Mortality in these patients exceeded 80%. At the time, most physicians believed that patients were dying from renal failure associated with systemic viremia.

Ibsen, an anesthesiologist trained in Boston, realized that death in these patients was associated with respiratory failure and recommended tracheostomy and positive pressure ventilation (PPV). With the introduction of PPV, mortality immediately dropped from greater than 80% to approximately 40%. Delivering care to these patients was a logistical problem because there were no mechanical positive pressure ventilators. Patients had to be hand bagged. It is estimated that at the height of the polio epidemic, 70 patients were simultaneously being manually ventilated. At the end of the polio epidemic, approximately 1,500 (medical) students provided manual ventilation for a total of 165,000 hours. One approach to the logistical challenge was to take care of these patients in 1 location. This led to the first intensive care units as we know them today.

The initial emphasis in providing support with assisted ventilation was replacement of respiratory muscle activity. Over ensuing decades, this changed with a greater focus on oxygenation failure stimulated in part by improved technology for the measurement of blood gases and identification of the acute respiratory distress syndrome (ARDS).

The 1960s were also a pivotal decade in the development of PPV influenced by advances in physiology and surgery and the need to address problems of postoperative lung collapse and traumatic lung injuries from battlefield conflict. Pressure-cycled devices were introduced, which delivered intermittent positive pressure not only to replace the work of respiratory muscles but to aid coughing, reduce basal lung collapse, and improve delivery of therapeutic aerosols. Improved control of inflation and deflation was developed so that these phases of each breath could be separately regulated. Treatment of ARDS was a central driver of new approaches to mechanical ventilation for respiratory failure but not the only one. Clinicians needed techniques to provide partial respiratory support, recondition respiratory muscles after pulmonary disease, and gauge readiness of the patient to assume the entire ventilatory workload.

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Tobin MJ. Advances in mechanical ventilation. *N Engl J Med.* 2001;344:1986-1996.

Contemporary Practice

PPV can be lifesaving in patients with hypoxemia or hypercarbia that cannot be addressed by other means. Mechanical

Table 1. Standard Ventilator Settings

Settings
• Rate 8-12 breaths/min
• Tidal (breath) volume 6-10 mL/kg (follow plateau pressure)
• FiO_2 100% → titrate to pulse oximetry (typical goal > 90% saturation)
• PEEP 5-10 cm H_2O
• Mode: see text
• If ventilator pressure is titrated to provide breaths or assist patient breaths, provide set pressure sufficient to achieve tidal volume in the above range

FiO_2 = fraction of inspired oxygen; PEEP = positive end-expiratory pressure (consistent airway pressure maintaining alveolar and airway patency). Tidal volume is the gas volume in each breath.

ventilation may also substitute for the action of respiratory muscles. In patients with severe respiratory distress, respiratory muscles may account for as much as 50% of total body oxygen consumption. Mechanical ventilation, in this situation, allows oxygen to be rerouted to other tissue beds that may be vulnerable. PPV may reverse and prevent lung collapse by allowing inspiration at a more compliant region of the pulmonary pressure-volume curve, and it may decrease the work of breathing. In its essence, the lung is given an opportunity to heal if mechanical ventilation improves pulmonary gas exchange and provides relief from excessive respiratory muscle work.

Simple controlled ventilation rapidly leads to atrophy of respiratory muscles. Assisted modes that are triggered by patient effort are preferred. Most common triggered modes are (volume) assist control ventilation, (pressure) assist control ventilation, (synchronized) intermittent mandatory ventilation, and pressure support ventilation. With assist control ventilation modes, the ventilator delivers a breath either when triggered by patient inspiratory effort or independently if such effort does not occur within a preset period. With synchronized intermittent mandatory ventilation, the patient receives periodic positive pressure breaths at a preset volume and rate. The ventilator is programmed to match patient effort as possible. Spontaneous breathing is also allowed by opening a demand valve. Unfortunately, spontaneous breathing with synchronized intermittent mandatory ventilation may considerably increase the work of breathing. Pressure support ventilation differs from the previously described modes in that the physician sets a level of pressure to augment every spontaneous effort. Airway pressure is maintained at a preset level until the inspiratory flow falls below a certain level (usually approximately 25% of peak flow). Tidal volume is determined by the level of pressure set, patient effort, and pulmonary mechanics. Typical ventilator settings are provided in Table 1.

Most ventilators are triggered by a change in airway pressure and sensitivity is set at -1 to -2 cm/H_2O. However, if the trigger setting is too sensitive, the ventilator will cycle too frequently, and severe respiratory alkalosis may result. Some patients, particularly those with chronic obstructive pulmonary disease and high minute ventilation, require additional management of triggering and timing of breaths. An inspiratory gas flow rate of 60 L/min is used with most patients during standard ventilatory modes. Again, underlying lung disease may require a change in these settings.

Slutsky AS, Ranieri VM. Ventilator-induced lung injury. *N Engl J Med.* 2013;369:2126-2136.

Potential Harm

Mechanical ventilation was indispensable during the polio epidemics of the 1950s. Despite obvious benefits with this therapy, many patients eventually died after the initiation of mechanical ventilation even with normal blood gas values. Multiple factors have been identified including mechanical trauma to the lungs, oxygen toxicity, and hemodynamic collapse with elevated intrathoracic pressures. During the polio epidemic, investigators noted that mechanical ventilation could cause structural damage to the lung. In the 1960s, the term "respirator lung" was coined to describe diffuse alveolar infiltrates and hyaline membranes that were found on postmortem examination of patients who had undergone mechanical ventilation. More recent studies characterize lung damage with mechanical ventilation to include inflammatory cell infiltrates, hyaline membrane formation, pulmonary edema, and increased vascular permeability. This constellation of pulmonary consequences of mechanical ventilation has been termed ventilator-induced lung injury (VILI). A more recent study (see later) showed the clinical importance of VILI by confirming that a ventilator strategy designed to reduce lung injury decreased mortality among patients with ARDS.

Regional lung overdistension is a key factor in generating VILI. Because there is no well-accepted clinical method of measuring regional overdistension in the lung, limiting inflation pressure during mechanical ventilation is a common surrogate strategy to limit lung overdistension. Alveolar pressure is relatively easy to estimate clinically as the airway pressure during a period of zero flow. In a patient undergoing mechanical ventilation who is not making spontaneous breathing efforts, airway pressure is measured during a period when airflow is stopped at end inspiration, which is called the plateau pressure. The measurement of plateau pressure, however, does not describe a second important factor—pressure in the pleural space around the lungs. Plateau pressure also has nuances. For example, the morbidly obese patient or the patient with a stiff chest wall requires increased airway pressure to maintain a given tidal volume.