Ventilator Management in the Cardiac Intensive Care Unit

Carlos Corredor, MRCP, FRCA,
Sian I. Jaggar, CertMedEd, MD, FRCA*

INTRODUCTION

The role of the cardiac intensive care unit (CICU) has evolved markedly from a purely observational unit dedicated to the monitoring and prompt resuscitation of patients with myocardial infarction to a unit treating an increasingly aging population with complex cardiac conditions and concomitant noncardiac comorbidities.1

Patients admitted to the CICU present with a variety of conditions, including complicated myocardial infarction, acute heart failure, refractory arrhythmias, and complications of adult congenital heart disease. Advances in early coronary intervention are reflected in decreasing rates of patients admitted with ST elevation myocardial infarction to the CICU. However, there is an increase in the prevalence of noncardiac critical illness, such as respiratory failure, sepsis, and acute kidney injury.2

This new paradigm has led to an increase in the number of patients requiring mechanical ventilation (MV) and with a longer duration of this therapy during their CICU stays. The observational study by Katz and colleagues1 looked at the characteristics of health care delivery in a coronary care unit at Duke University Hospital. It demonstrated a significant increase in the prevalence of patients requiring prolonged MV (>96 hours) in the 1989–2006 period.

KEYWORDS

- Mechanical ventilation
- Cardiac intensive care unit (CICU)
- Cardiopulmonary interactions
- Noninvasive ventilation
- Gas exchange
- PEEP (positive end expiratory pressure)
- Cardiac dysfunction
- Weaning

KEY POINTS

- The number of patients on mechanical ventilation has increased in the modern CICU as a result of increasing complexity and comorbidities in these patients, and a thorough understanding of ventilator management is essential for the practicing cardiac intensivist.
- The heart, lungs, and great blood vessels are contained within the thoracic cavity. Changes in intrathoracic pressure caused by different ventilatory modes (spontaneous or mechanical) have different effects on the determinants of cardiac output and overall cardiac performance.
- Positive end expiratory pressure and continuous positive airway pressure (CPAP) have beneficial effects in terms of the reduction of afterload, myocardial work, and oxygen consumption. Noninvasive mechanical ventilation is an effective and safe option for the management of cardiogenic pulmonary edema.
- The discontinuation of mechanical ventilation may reverse the beneficial effects of positive pressure ventilation in the failing heart. The identification and risk stratification of patients likely to fail weaning is crucial to ensure that interventions can be put in place preemptively.
PHYSIOLOGIC BASIS

A review of the physiology of breathing in health, along with cardiopulmonary interactions, is necessary to understand the effects of MV in patients with cardiovascular disease.

Gas exchange takes place between gas in the alveolus and blood. Carbon dioxide diffuses from the blood across the alveolar-capillary membrane and is exhaled; simultaneously, oxygen diffuses down a pressure gradient from the alveolus into the capillary blood. The volume of inhaled gas that participates in gas exchange is termed alveolar ventilation. Dead-space ventilation (V_D) is the fraction of inhaled gas that does not participate in gas exchange and together with alveolar ventilation (V_A) makes up total ventilation (V_T):

\[ V_T = V_A + V_D. \]

Tidal volume is the volume of gas that is inspired and expired during each breath; it is approximately 7 mL/kg in an adult. The volume of gas that remains in the lungs at the end of a normal expiration is the functional residual capacity (FRC). At FRC, the tendency of the lung tissue to collapse is counterbalanced by the tendency for the chest wall to expand (Fig. 1).

In addition, FRC has several important functions; it is a major oxygen store in the body and, thus, maintains a steady arterial P_O2 buffering the effects of an intermittent tidal delivery of oxygen with each breath. FRC prevents atelectasis by maintaining the lung in a state of partial inflation and by placing the respiratory system in the steep part of its compliance curve, minimizing the work of breathing.

FRC is determined by the compliance of the lung and chest wall. An example commonly encountered in the CICU is in patients with left ventricular failure and pulmonary edema. Alveoli flooded with fluid have diminished compliance and, therefore, reduced FRC. Pulmonary vascular resistance (PVR) is lowest at FRC and increases at both low or large lung volumes.3

EFFECT OF SPONTANEOUS BREATHING IN THE CARDIOVASCULAR SYSTEM

The heart, lungs, and great vessels are normally contained within a closed thoracic cavity and interact during each respiratory cycle. The right and left ventricles are connected in series through the pulmonary circulation and, like the great veins and thoracic aorta, are subjected to changes in the intrathoracic pressure. Dynamic mechanical properties of the lung and chest wall, such as compliance and elastance, can, therefore, have an effect on cardiac function.

![Fig. 1. Lung and chest wall pressure-volume curve. Continuous-line curves are pressure-volume diagrams for lung and chest wall. Dotted-line curve represents pressure-volume curve of the total respiratory system. At FRC, combined pressure of the total system is 0. RV, right ventricular.](image-url)
Inspiration occurs when inspiratory muscles generate a negative intrapleural pressure creating a pressure gradient, down which gas enters the lung from the atmosphere. The drop in intrapleural pressure expands the lungs but also affects the heart and intrathoracic blood vessels. The muscular cardiac wall is normally subjected to extramural forces (eg, intrathoracic pressure) in addition to intramural stress produced by the pressure inside the cardiac chambers. Transmural pressure equals the intrachamber pressure minus the extramural pressure. Inspiration produces a decrease in the extramural stress applied to the cardiac chambers and, thus, an increase in the transmural pressure. Inspiration increases diastolic filling of the right atrium as a consequence of the favorable pressure gradient created by the difference between the right atrial pressure and the intrathoracic pressure. The increased venous return is reflected in a higher right ventricular end-diastolic volume (RVEDV) (preload) and RV stroke volume. Conversely, during spontaneous inspiration, the left ventricle (LV) experiences a decrease in stroke volume caused by increased afterload. The afterload rises because as intrathoracic pressure decreases the extramural pressure of the LV also decreases and, as explained earlier, results in an increased transmural pressure for the LV. The LV must now generate a higher wall tension (afterload) against the increased transmural pressure.4,5

The right and left ventricles share a common septum and circumferential fibers. This anatomic and mechanical relationship makes the ventricles interdependent. Therefore, changes in end diastolic pressure of the RV will have an effect on the LV. The increased venous return and RV volume with inspiration produce a transient shift of the intraventricular septum toward the LV, decreasing LV end diastolic volume. The leftward shift can be particularly pronounced if the PVR is high, with concomitant increase in RV volume.6 During normal spontaneous breathing, the septum shift is only transient and there is little evidence that this effect is clinically significant.4,7 The effect of ventricular interdependence can, however, be pronounced in conditions when pleural pressure becomes extremely negative and lungs are overinflated, such as acute severe asthma or in cases of cardiac tamponade. This exaggerated effect of interdependence explains the pulsus paradoxus phenomenon.8

The net effect of inspiration is a small decrease in systolic blood pressure explained by the increased afterload of the LV and consequent decreased stroke volume. The decrease in systolic blood pressure is minimized by the increase in RV filling. Systolic blood pressure increases during expiration as the LV afterload returns to baseline and the inspiratory increased venous return reaches it.4

During spontaneous ventilation, normally the respiratory apparatus requires less than 5% of the total oxygen delivery (DO2). However, the metabolic demand for oxygen can reach up to 25% of the total DO2 in situations of respiratory distress because of the large O2 requirements of respiratory muscles.9 Patients with coronary heart disease and limited reserve can, therefore, experience myocardial ischemia with the increase in afterload and myocardial oxygen consumption that occurs with large decreases in intrathoracic pressure during strenuous spontaneous ventilation.10

EFFECTS OF INTERMITTENT POSITIVE PRESSURE VENTILATION ON THE CARDIOVASCULAR SYSTEM

The hemodynamic effects of MV can be continuous or occur cyclically during the respiratory cycle. The hemodynamic effects will be affected by the ventilation mode, the presence of spontaneous respiratory effort, and the addition of positive end expiratory pressure (PEEP).

During intermittent positive pressure ventilation (IPPV), the positive pressure delivered to the airway during inspiration is transmitted to the intrapleural space and intrathoracic structures causing different hemodynamic effects. The main mechanisms behind these effects are the following (Fig. 2)11:

- Reduced RV preload
- Increased RV afterload
- The effects of ventricular interdependence on LV preload
- Reduced LV afterload

The entirety of cardiac output returning to the right side of the heart depends on a very small pressure gradient. Guyton’s circulatory model states that venous return results from the interaction of the main systemic filling pressure, which is the degree of filling of the systemic circulation, the right atrial pressure, and the resistance to venous return.12

Initiation of positive pressure ventilation and the associated positive intrathoracic pressure alters both right atrial pressure and the mean circulatory systemic pressure. The venous return decreases, producing a reduction in RVEDV and stroke volume.13 The decrease in blood flow also reaches the LV after 3 to 4 beats, causing a reduction in LVEDV. RV afterload increases because of the
effect of positive pressure in the pulmonary vasculature.\textsuperscript{14}

Ventricular interdependence is evident when decreased RVEDV, in the presence of an increased transseptal pressure gradient, causes the septum to shift right, resulting in an increased LVEDV and LV stroke volume. The opposite is observed during expiration. Venous inflow transiently increases to the RV as positive pressure on the vessels is released, increasing both RVEDV and stroke volume. The septum shifts left, and there is a reduction in LVEDV and LV stroke volume.\textsuperscript{15}

Positive intrapleural pressure decreases LV afterload because of a reduction in LV transmural pressure. Under these conditions, the LV needs to generate less wall tension to eject blood.\textsuperscript{16} The reduction in afterload has potential benefits in the presence of left-sided heart failure because it facilitates ventricular emptying and is reflected in reduced myocardial work and oxygen consumption.\textsuperscript{17}

The net resulting effect of the aforementioned mechanisms on cardiac output depends on intravascular volume status and myocardial contractility. Failing ventricles, for example, are very sensitive to changes in afterload.\textsuperscript{17} Reduction of venous return, and the subsequent reduction in stroke volume and cardiac output that occur during the respiratory cycle, is the predominant mechanism. MV invariably causes a decrease in cardiac output in patients with hypovolemia, and a low-volume status exaggerates the cyclical changes in venous return and stroke volume. These cyclical changes in stroke volume and in surrogates, such as stroke volume variation or pulse pressure variation, form the bases of functional hemodynamic monitoring as a tool for assessing fluid responsiveness in mechanically ventilated patients.\textsuperscript{18}

\textbf{EFFECT OF PEEP ON THE CARDIOVASCULAR SYSTEM}

Maintaining positive airway pressure at the end of expiration can be achieved by the application of positive end expiratory pressure (PEEP) or continuous positive airway pressure (CPAP). The physiological principles behind these two terms are identical. Convention dictates that the term PEEP is used when positive pressure at the end of expiration is applied during a mechanical ventilation breath and CPAP during spontaneous breathing (Fig. 3). PEEP is commonly applied as part of a lung protective ventilation strategy. It promotes alveolar recruitment, prevents damage resulting from the cyclical opening and closing of alveolar units (atelectrauma), and improves oxygenation.

The addition of PEEP leads to a continuous increase in airway and intrapleural pressures. Compliance of the lung and chest wall determines the amount of PEEP that is transmitted from the airway to the intrapleural space.\textsuperscript{19}

The effects of PEEP are similar to those of positive pressure ventilation, differing only in that they are continuous and not cyclical. The predominant
The effect of PEEP is that of a reduction in venous return caused by an increase in the resistance to venous return. The reduction in RV venous return, RVEDV, and stroke volume is reflected in a secondary reduction in LV preload.

PEEP and continuous positive airway pressure (CPAP) reduce the afterload of the LV by producing a continuous increase in extramural pressure and, thus, a reduction of the transmural pressure of the LV during the whole respiratory cycle. The reduction in afterload is beneficial in terms of reduction of LV work and oxygen consumption in patients with congestive cardiac failure. Indeed, CPAP alone has been demonstrated to achieve respiratory muscle unloading and reduction in afterload without impairing overall cardiac performance and cardiac index (CI).

Noninvasive ventilation is associated with positive physiologic effects in spontaneously breathing patients with congestive heart failure. The reduced LV afterload associated with positive intrapleural pressure produced by CPAP improves CI and systemic oxygen delivery.

Controversy remains surrounding the possible relationship between PEEP and myocardial perfusion and ischemia. Although there is evidence that even clinical levels of PEEP can decrease myocardial blood flow, the overall effect of myocardial oxygen supply is difficult to predict because of the positive effects of PEEP in terms of reduced LV afterload and, thus, oxygen demand.

**TYPE OF CARDIAC DYSFUNCTION AND EFFECT OF POSITIVE PRESSURE VENTILATION**

As discussed earlier, positive pressure ventilation affects a variety of parameters of cardiac performance variably and can, therefore, have both positive and negative impacts on cardiac function. When IPPV is required, yet likely to induce negative cardiovascular effects, measures should be instituted to minimize these effects (Table 1).

**INDICATIONS FOR MV IN THE CICU**

A primary purpose of mechanical respiratory support is to promote alveolar ventilation and, therefore, carbon dioxide (CO2) elimination. Additionally, it is also used to correct impaired oxygenation (Box 1).

The interface used between the MV device and patients creates an artificial classification. When tracheal intubation is required, the term *invasive ventilation* is used; ventilation is termed *noninvasive ventilation* (NIV) when delivered via a tight-fitting face mask or hood. The boundary between the two terms is blurred in the modern CICU, and they are both now used in a continuum of respiratory support.

There is no single value of arterial PCO2, PO2, pH, or oxygen saturation that can predict the need for invasive ventilation. Apnea, severely depressed consciousness level, respiratory arrest, and severe cardiogenic shock are compelling indications for endotracheal intubation and, therefore, invasive MV. However, in other situations, the clinical assessment of underlying disease process and of the overall condition of patients will dictate the most appropriate type of respiratory support.

Esteban and colleagues carried out the largest prospective cohort study to date studying the characteristics and outcomes of patients receiving MV in ICUs worldwide. This study demonstrated that acute respiratory failure was by far the most common indication for initiation of MV, accounting for 68.8% of cases studied. Congestive heart failure was the third most common cause of acute respiratory failure (12% of the cases) behind pneumonia (16%) and postoperative respiratory failure (15%).

Congestive cardiac failure can arise from conditions affecting the myocardium, valvular disease, or rhythm disturbances. Regardless of the cause, pulmonary edema is the common denominator that arises from increased left atrial pressure and imbalance in the Starling forces across the
alveolar-capillary interface (Fig. 4). Lung compliance decreases as a result of fluid flooding the alveoli and interstitium, and the additional fluid in the capillary membrane creates a barrier for gas exchange.27 The resulting ventilation/perfusion mismatch causes hypoxia; the development of stiff, noncompliant lungs leads to an increased work of breathing, ventilatory failure, and CO2 retention. Severe cardiogenic shock or cardiac arrest may compromise the level of consciousness and the ability of patients to maintain a patent airway. Intubation and MV is, therefore, necessary to protect the airway from aspiration in these situations.

Patients admitted to the CICU often have concurrent acute or chronic pulmonary conditions that may compromise oxygenation and/or ventilation and require initiation of positive pressure ventilation. Both iatrogenic complications and those inherent to critical illness can develop in the CICU. Critical illness–induced weakness, pulmonary embolism, hospital-acquired pneumonia, and pneumothorax may compromise oxygenation or ventilation and mandate the need for respiratory support.

### OXYGENATION CONSIDERATIONS

As previously reviewed, spontaneous respiratory effort (in that it requires oxygen consumption) is a form of exercise. By supporting or taking over completely the work of breathing, respiratory support can minimize this and improve cardiovascular performance.

Oxygenation can be titrated by setting the inspired oxygen concentration (FiO2) and adding PEEP. Other therapeutic options commonly used to improve oxygenation are changing the pattern of ventilation and rescue and advanced oxygenation techniques, such as high-frequency oscillation and extracorporeal membrane oxygenation, which are beyond the scope of this article.

Adequate tissue oxygenation is crucial for maintaining efficient aerobic metabolism and tissue structure and function. Oxygenation is particularly important for highly oxidative organs, such as the heart, especially in the presence of conditions that limit oxygen delivery (eg, coronary disease).

Accepted normal values for PaO2 lie between 80 and 100 mm Hg (10.7–13.3 kPa) and SaO2 greater than 94% while breathing air at sea level.

<table>
<thead>
<tr>
<th>Preload-Related Ventricular Dysfunction</th>
<th>RV Dysfunction</th>
<th>LV Dysfunction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemia</td>
<td>Severe pulmonary hypertension</td>
<td>Ischemic cardiomyopathy</td>
</tr>
<tr>
<td>Ischemia</td>
<td>COPD</td>
<td>Cardiogenic pulmonary edema</td>
</tr>
<tr>
<td>Restrictive cardiomypathy</td>
<td>Acute PE</td>
<td>RV Infarct</td>
</tr>
<tr>
<td>Cardiac tamponade</td>
<td>RV</td>
<td></td>
</tr>
<tr>
<td>Valvular stenoses</td>
<td>afterload</td>
<td></td>
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<tr>
<td></td>
<td>RV O2 demand</td>
<td></td>
</tr>
</tbody>
</table>

**Box 1**

**Common indications for positive pressure ventilation in CICU**

- Congestive cardiac failure (cardiogenic pulmonary edema)
- Decreased level of consciousness requiring protection of the airway (eg, severe cardiogenic shock, cardiac arrest)
- Concurrent acute or chronic respiratory disease
- Complications of critical illness (eg, pulmonary embolism, pneumothorax, critical illness induced weakness)
Traditionally, these values have been used as cut-off points for the diagnosis of hypoxia and as targets for titration of oxygenation in critical care units.28 There is, however, a strong body of evidence suggesting that high concentrations of oxygen are potentially harmful and associated with poor outcomes in certain cardiovascular conditions. Arterial hyperoxia has been associated with undesirable cardiovascular responses, such as reduced stroke volume and cardiac output, increased systemic vascular resistance, coronary artery vasoconstriction, and reduced coronary blood flow.29 Two recent systematic reviews called into question the routine use of high-flow oxygen for the treatment of acute myocardial infarction. They found no evidence of oxygen therapy being beneficial in this setting; on the contrary, they found that it may result in greater infarct size and increased mortality.30,31 Hyperoxia can also be harmful in the context of resuscitation following cardiac arrest. A cohort study of 6326 patients admitted to the ICU following resuscitation from cardiac arrest found arterial hyperoxia (Pao2 >300 mm Hg/40 kPa) to be independently associated with increased in-hospital mortality compared with either hypoxia (Pao2 <60 mm Hg/8 kPa) or normoxia.32

Optimal oxygen delivery to the tissues (DO2) is the result of adequate cardiac output and arterial oxygen content.33 This relationship is described by the following equation:

\[
DO_2 \text{ (mL/min)} = CO \text{ (L/min)} \times CaO_2 \times 10
\]

where \(CaO_2\) is the total arterial oxygen content (the product of the oxygen bound to hemoglobin plus the oxygen carried in solution) and \(CO\) is cardiac output. The multiplier 10 is used because of the different measurement units used for \(CaO_2\) (mL.dL\(^{-1}\)) and cardiac output (L.min\(^{-1}\)).

Adequate \(DO_2\) ensures that aerobic metabolism is maintained and preserves tissue viability. The aforementioned equation makes clear that manipulating MV parameters only modifies one variable and that ensuring an adequate cardiac output and hemoglobin content are necessary for maintaining a satisfactory oxygen delivery. Together with clinical signs, lactate, base deficit, and mixed venous saturation (SvO2) and central venous saturation (SvO2) are commonly used as surrogate markers of adequacy of tissue perfusion. It is, therefore, reasonable to titrate inspired oxygen to the lowest possible levels that meet the oxygen requirement of the tissues and maintain aerobic metabolism.
High \( \text{FiO}_2 \) levels can be insufficient to reverse hypoxemia in the presence of pulmonary shunt. Shunt occurs when a substantial portion of the cardiac output is in contact with a nonventilated area of the lung. Patients with significant pulmonary edema, pneumonic consolidation, and compression of lung tissue by pleural effusion have poor lung compliance and can experience important degrees of shunt causing hypoxemia.\(^3^4\)

PEEP or CPAP is used in these situations to promote reopening of collapsed alveoli and to stent open the small airways opposing alveolar collapse and atelectasis. PEEP improves lung compliance by increasing the number of alveolar units available for ventilation, increasing the FRC, and reducing the work of breathing.\(^3^5,3^6\) PEEP may also protect from ventilator-induced lung injury by preventing damage from the repeated opening and closing of alveolar units (atelectrauma).\(^3^7\)

There are important caveats to the application of PEEP to patients with cardiovascular disease. PEEP in itself does not reduce lung water in patients with pulmonary edema. Indeed, it may actually increase the water content of the lungs by obstructing lymphatic drainage.\(^3^8\) The benefit of PEEP/CPAP in the setting of cardiogenic pulmonary edema derives from improvements in lung mechanics and LV performance and not from any effects on lung water. PEEP will only improve tissue oxygenation if any negative effects of positive pressure on cardiac output are considered and counterbalanced (see Table 1). As previously reviewed, PEEP can significantly affect ventricular preload; therefore, sufficient intravascular volume must be ensured to maintain an adequate cardiac output.

Other therapeutic options that can be considered to improve oxygenation are increasing the ratio of inspiratory to expiratory time (I/E ratio), recruitment maneuvers, and prone positioning. Under normal circumstances the I/E ratio is set at 1:2, which means that the expiratory time is twice the inspiratory time. Prolonging the inspiratory time increases the I/E ratio and improves oxygenation by increasing the mean airway pressure, generating recruitment of atelectatic alveolar units. However, inspiratory time equal or greater than expiratory time can generate significant gas trapping, decrease in venous return and hemodynamic compromise in patients with compromised cardiovascular function. An inverse ratio of ventilation occurs when the inspiratory time exceeds the expiratory time (eg, 2:1). This scenario can be very uncomfortable for patients and often requires deep sedation and paralysis.\(^3^9\)

Recruitment maneuvers involve the episodic delivery of high pressure and volume breaths with the aim of increasing the number of open alveoli and, therefore, oxygenation. There is no evidence at present that the use of recruitment maneuvers improves mortality or reduces length of ventilation in patients with acute respiratory distress syndrome (ARDS).\(^4^0\) Furthermore, they can be a source of hemodynamic instability in patients with cardiac pathologies.

Prone positioning may lead to improved oxygenation (but not mortality) in patients with ARDS. It has only been shown to reduce mortality in patients with acute hypoxemic respiratory failure and severe hypoxemia.\(^4^1\)

**VENTILATOR CONSIDERATIONS**

A mechanical ventilator is a device that delivers a controlled flow of gas to the airway. The magnitude, rate, duration, and triggering of the flow are determined by the operator. Advances in microprocessor technology have given the operator nearly limitless, and often confusing, options of flow delivery. Confusion in nomenclature of modes of ventilation arises from the coexistence of trademark names and historical abbreviations that do not always reflect the clinical application of the mode, potentially creating confusion that may adversely affect patient care.\(^4^2\) Modern definitions of ventilation modes are based on combinations of 3 parameters: control variable, breath sequence, and targeting scheme.\(^4^3\)

Control can be defined as the variable that the ventilator uses to control inspiration. The variable can be identified according to the relationship between peak inspiratory pressure and to the load experienced by the ventilator. In volume control, the peak pressure changes as the ventilator load changes, while maintaining a constant tidal volume. Pressure control maintains a constant peak inspiratory pressure as the ventilator load changes. In other words, pressure is the independent variable during pressure control, and volume is the independent variable during volume control.

Breath sequence can be:

- Continuous mandatory ventilation when the ventilator controls all breaths but may allow patient triggering
- Synchronized intermittent mandatory ventilation (SIMV), which allows patients to take spontaneous breaths between mandatory breaths
- Continuous spontaneous ventilation, when all breaths are spontaneous

Modern ventilators can respond to changes in patients’ lung compliance, lung resistance, and respiratory effort. The ventilator response is
determined by the targeting or feedback scheme programmed into the device. Feedback schemes range from simple static set points (eg, pressure limit in pressure control) with full operator input to complex dynamic automatic adjustment of set points based on complex algorithms. However, patients with severe cardiovascular physiology of positive pressure has been shown to reduce the deleterious effects degrees of spontaneous breaths. This variability and pressure support ventilation allow for different such as CPAP, SIMV, bilevel airway pressure, with fully controlled modes of ventilation. Modes pressure for a given minute volume, compared with different levels of mean airway pressure and intrapleural respiratory activity. They tend to produce lower with modes of ventilation that allow spontaneous activity. CPAP, SIMV, bilevel airway pressure, and pressure support ventilation allow for different degrees of spontaneous breaths. This variability has been shown to reduce the deleterious effects in cardiovascular physiology of positive pressure ventilation. However, patients with severe cardiogenic shock or LV dysfunction may benefit more from full ventilatory support that abolishes the effect of an increased oxygen consumption associated with spontaneous breathing.

Ultimately, ventilator settings will depend on local experience, type and brand of equipment, and familiarity with the device available in different CICUs. The clinician should choose a setting that ensures effective minute volume ventilation, optimizes the work of breathing, and minimizes hemodynamic adverse effects.

**NIV USE IN CONGESTIVE CARDIAC FAILURE AND ACUTE CARDIOGENIC PULMONARY EDEMA**

The last 3 decades have seen an increase in the use of NIV for the management of patients with acute cardiogenic pulmonary edema. The term NIV has been used by the literature to denote both CPAP ventilation and the true bilevel ventilation mode that provides noninvasive IPPV (NIPPV). Early interest was sparked by evidence from observational and small randomized studies demonstrating that CPAP improved oxygenation, reduced work of breathing, and increased cardiac output in patients with cardiogenic pulmonary edema. There has been controversy regarding which method of NIV is more effective. Chadda and colleagues performed a physiologic randomized crossover study suggesting that NIPPV was more effective than CPAP in terms of unloading respiratory muscles and improving cardiac performance. However, concern arose that bilevel ventilation was associated with a higher incidence of myocardial ischemia compared with CPAP. To date, this has not been supported by systematic reviews.

Results from recent meta analyses and systematic reviews suggest that the early institution of NIV is associated with a reduction in the rates of endotracheal intubation compared with standard medical therapy. NIV (both CPAP and NIPPV) was also associated with a reduction in mortality compared with standard therapy. None of the reviews found NIPPV to be superior to CPAP in terms of outcomes. The applicability of these findings is limited, however, by the small size of studies included and wide variations in study populations and interventions.

The largest multicenter randomized controlled trial to date studying the effect of NIV in acute cardiogenic pulmonary edema was performed by Gray and colleagues in 2008. The trial included 1069 patients with acute cardiogenic pulmonary edema and aimed to establish whether NIV improved survival when compared with standard medical therapy. A secondary outcome assessed the superiority of NIPPV over CPAP. The patients were randomized to 3 intervention groups: standard oxygen therapy, CPAP (5–15 cm of water), and NIPPV (inspiratory pressure of 8–20 cm H₂O and expiratory pressure of 4–10 cm H₂O). No difference was detected in 7-day mortality between patients receiving standard oxygen therapy (9.7%) and patients treated with NIV (9.5% \( P = .87 \)). There was no significant difference in the composite end point of short-term (within 7 days) mortality and intubation between CPAP (11.7%) and NIPPV (11.1% \( P = .81 \)). However, patient-reported dyspnea, tachycardia, hypercapnia, and acidosis were significantly improved in the NIV ventilation groups compared with standard oxygen therapy.

Despite the large number of patients, this article has some limitations. The trial was set in the emergency department, limiting its applicability to patients that develop pulmonary edema during other stages of their hospital stay. There was
<table>
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<tr>
<th>Mode of Ventilation</th>
<th>Other Names</th>
<th>Characteristics/Theoretical Benefits/Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Volume Modes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Assist-control ventilation</td>
<td>CMV with assist</td>
<td>Ventilator delivers preset volume; breaths can be either assist or control but of the same volume; may induce hyperinflation and respiratory alkalosis at high respiratory rates</td>
</tr>
<tr>
<td>SIMV</td>
<td></td>
<td>Mandatory breaths synchronized to coincide with spontaneous inspiration; guaranteed backup rate; cardiac output can decrease in patients with LV dysfunction because of increased afterload with unsupported spontaneous inspiratory efforts</td>
</tr>
<tr>
<td><strong>Pressure Modes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCV</td>
<td></td>
<td>Ventilator delivers set target pressure at a set respiratory rate; protects from barotrauma; volume delivered subject to lung compliance</td>
</tr>
<tr>
<td>PSV</td>
<td></td>
<td>Patients’ inspiratory effort assisted to a preset level; patient triggered, pressure limited, and flow cycled</td>
</tr>
<tr>
<td>CPAP</td>
<td>EPAP</td>
<td>Pressures set to remain constant during respiratory cycle while patients are allowed to breathe spontaneously</td>
</tr>
<tr>
<td>APRV</td>
<td>BiPap</td>
<td>Clinician set to level CPAP and time spent at each level (inspiratory and expiratory time); CPAP or pressure high and release pressure or pressure low; patients can breathe spontaneously at both levels; potential for hemodynamic compromise in preload-dependent conditions</td>
</tr>
<tr>
<td><strong>Dual Modes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PRVC</td>
<td>AutoFlow (Dräger Medical AG &amp; Co. KGaA, Germany) Adaptive pressure control</td>
<td>Closed-loop, pressure-controlled mode; patient or time triggered with tidal volume as the variable selected by operator; maintains a more stable tidal volume as lung compliance varies while protecting from barotrauma</td>
</tr>
<tr>
<td>ASV</td>
<td></td>
<td>Delivers pressure-controlled breaths using automatically calculated optimal settings (tidal volume and frequency) based on patients’ ideal body weight and percentage of minute volume ventilation; aims to minimize work of breathing while encouraging spontaneous breaths</td>
</tr>
<tr>
<td>PAV</td>
<td></td>
<td>Pressure-controlled output by the ventilator is adjusted to perform accordingly to patients’ effort; maximizes ventilator-patient synchrony and reduces work of breathing</td>
</tr>
<tr>
<td>NAVA</td>
<td></td>
<td>Electrical activity of diaphragm (Edi) is captured, fed back to ventilator, and breath assistance is delivered proportionally and in synchrony with patients’ Edi signal</td>
</tr>
</tbody>
</table>

**Abbreviations:** APRV, airway pressure release ventilation; ASV, adaptive support ventilation; BiPap, bilevel airway pressure; CMV, controlled mandatory ventilation; EPAP, expiratory positive airway pressure; NAVA, neutrally adjusted ventilator assist; PAV, proportional assist ventilation; PCV, pressure-controlled ventilation; PRVC, pressure-regulated volume control; PSV, pressure support ventilation.

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>No. Studies</th>
<th>No. of Patients</th>
<th>NIV Overall</th>
<th>CPAP</th>
<th>NIPPV (Bilevel)</th>
<th>Mortality NIV vs Standard Medical Care (RR [95% CI] P Value)</th>
<th>Mortality CPAP vs NIPPV (Bilevel)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masip et al, 2005</td>
<td>15</td>
<td>783</td>
<td>0.55 (0.40–0.78) P&lt;.001</td>
<td>0.53 (0.35–0.81) P&lt;.03</td>
<td>0.80 (0.35–1.05) P&lt;.07</td>
<td>6</td>
<td>219</td>
</tr>
<tr>
<td>Peter et al, 2006</td>
<td>18</td>
<td>877</td>
<td>N/A</td>
<td>0.59 (0.38–0.90) P&lt;.015</td>
<td>0.63 (0.37–1.10) P&lt;.11</td>
<td>9</td>
<td>406</td>
</tr>
<tr>
<td>Winck et al, 2006</td>
<td>16</td>
<td>815</td>
<td>N/A</td>
<td>0.13 (0.05–0.22) P&lt;.003</td>
<td>0.07 (0.01–0.14) P&lt;.08</td>
<td>7</td>
<td>297</td>
</tr>
<tr>
<td>Vital et al, 2008</td>
<td>17</td>
<td>930</td>
<td>0.62 (0.45–0.84)</td>
<td>0.58 (0.38–0.88)</td>
<td>0.70 (0.40–1.23)</td>
<td>6</td>
<td>230</td>
</tr>
<tr>
<td>Author/Year</td>
<td>No. Studies</td>
<td>No. of Patients</td>
<td>NIV Overall</td>
<td>CPAP</td>
<td>NIPPV (Bilevel)</td>
<td>Need for Intubation NIV vs Standard Medical Care (RR [95% CI] P Value)</td>
<td>Need of Intubation CPAP vs NIPPV (Bilevel)</td>
</tr>
<tr>
<td>------------------</td>
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</tr>
<tr>
<td>Masip et al, 2005</td>
<td>15</td>
<td>783</td>
<td>0.43 (0.32–0.57) P&lt;.001</td>
<td>0.40 (0.27–0.58) P&lt;.001</td>
<td>0.48 (0.30–0.76) P&lt;.002</td>
<td>6</td>
<td>219</td>
</tr>
<tr>
<td>Peter et al, 2006</td>
<td>19</td>
<td>928</td>
<td>N/A</td>
<td>0.44 (0.29–0.66) P&lt;.0003</td>
<td>0.50 (0.27–0.90) P&lt;.02</td>
<td>9</td>
<td>353</td>
</tr>
<tr>
<td>Winck et al, 2006</td>
<td>16</td>
<td>805</td>
<td>N/A</td>
<td>0.22 (0.10–0.34) P&lt;.0004</td>
<td>0.18 (0.04–0.32) P&lt;.01</td>
<td>7</td>
<td>299</td>
</tr>
<tr>
<td>Vital et al, 2008</td>
<td>17</td>
<td>930</td>
<td>0.53 (0.34–0.83)</td>
<td>0.46 (0.32–0.65)</td>
<td>0.68 (0.27–1.73)</td>
<td>7</td>
<td>257</td>
</tr>
</tbody>
</table>

Relative risk of death and intubation in NIV overall, CPAP, or NIPPV versus standard medical care and relative risk of death and intubation in CPAP versus NIPPV (bilevel).

NIV encompasses both CPAP and NIPPV.

Abbreviations: CI, confidence interval; N/A, not available; RR, relative risk.

Data from Refs.53-56
also significant crossover between the groups, with 56 patients in the standard-oxygen-therapy group being rescued with NIV. This crossover may have produced an artificially low rate of intubation in the standard-oxygen-therapy group.

The cumulative evidence suggests that NIV (both CPAP and NIPPV) are valuable and safe therapeutic options for the management of acute cardiogenic pulmonary edema in the CICU.

WEANING ISSUES IN PATIENTS WITH CARDIOVASCULAR DISEASE

The term weaning is used ubiquitously to describe the transition from full ventilatory support to spontaneous and unsupported breathing. Liberation and discontinuation have been suggested as more appropriate terms because they convey more urgency to the clinician to remove an intervention associated with morbidity and complications.58

Once the underlying process mandating ventilatory support has resolved or stabilized, an assessment should be made of the patient’s readiness to have ventilation discontinued. This assessment usually takes place during a spontaneous breathing trial (SBT). An SBT requires the patient to breathe completely unassisted (T-piece connected to endotracheal tube) or with low level CPAP and/or inspiratory pressure support. SBTs usually last for 30 min and during this time patients are closely monitored for signs of discomfort, excessive work of breathing and haemodynamic instability (Fig. 5).

With discontinuation, the beneficial effects of MV are effectively reversed, with an abrupt transfer from full ventilator support to spontaneous ventilation. This transition has been likened to performing an exercise stress test in patients with an already limited cardiopulmonary reserve.59 The effects of weaning in the cardiovascular system can be explained in terms of the effect of negative intrathoracic pressure, increase in the work of breathing, and increase in sympathetic tone.60

Spontaneous breathing causes a decrease in intrathoracic pressure that is commensurate with the degree of inspiratory effort. This decrease in pressure produces an increased LV afterload and venous return, resulting in increased RVEDV and LVEDV. Failing ventricles are particularly susceptible to afterload and filling pressure changes.61

The increased work of breathing related to the spontaneous respiratory muscle activity causes a surge in demand for oxygen delivery. Myocardial oxygen consumption increases as a result of the need for the higher cardiac output that is required to deliver this and the increased wall stress in the ventricles. Patients with coronary heart disease may experience significant ischemia during weaning that can lead to failure in the discontinuation of MV.62–65

There is significant sympathetic activation associated with the transition to spontaneous ventilation. Heart rate and blood pressure increase with noticeable effects in myocardial oxygen demand, and the associated venoconstriction causes an increase in venous return and preload. There is evidence that patients with chronic respiratory failure (eg, chronic obstructive pulmonary disease [COPD]) have more pronounced sympathetic activation with potential significant adverse effects during the weaning process.66

The potential adverse cardiovascular consequences of weaning have to be balanced against possible complications arising from MV (Table 4). Once the cardiac condition (eg, ischemia, cardiogenic shock) that prompted the initiation of MV has been treated/stabilized, every effort should be made to liberate patients from MV.

The identification and risk stratification of patients likely to fail weaning is crucial to facilitate preemptive interventions to avoid reintubation. Failed extubation is associated with poor patient outcomes and increased duration of hospital stay.76

IDENTIFICATION OF PATIENTS AT RISK OF WEANING FAILURE OF CARDIAC ORIGIN

Both clinical characteristics of patients and diagnostic modalities can be used to predict weaning failure in CICU patients. Awareness of this may help prevent such events by altering management strategies (Table 5).

Patients with a known history of LV disease, in isolation or in combination with COPD, are at risk of developing pulmonary edema leading to failure to wean. Anecdotal evidence suggests that a combination of tachycardia and hypotension is suggestive of cardiac failure during the weaning process.84

Increases in pulmonary artery occlusion pressure (PAOP) and decreases in mixed venous oxygen saturation values have been reported in patients that fail discontinuation of MV. However, the use of the pulmonary artery catheter solely for the purpose of monitoring weaning failure is probably not justified in view of its invasiveness and the lack of evidence of benefit associated with monitoring.86

Transthoracic echocardiography or transesophageal echocardiography are widely used in the CICU and are less-invasive methods of determining LV filling pressures. The measurement of
Fig. 5. Sample algorithm for institution of SBT in the CICU.

Is the patient ready for SBT trial?
- Cause of respiratory failure
  - Acute respiratory distress syndrome (ARDS)
- Perfusion status
  - Oxygenation: PaO₂/FiO₂
- Cardiovascular stability
- No or low ventilator weaning support
- Awake or level of sedation that allow own ventilatory efforts

NO

Perform SBT

YES

SBT failed:
- Insufficient tidal volume
- Worsening gas exchange
- Hemodynamic instability/myocardial ischemia
- Fatigue

Continue or resume mechanical ventilation
- Any unaddressed fluid, electrolyte, or metabolic issues?
- Review fluid balance

YES

Exhale

NO

SBT successful

Is patient ready for extubation?
- Effective cough and cooperation
- Awake and cooperative
- Secretion load

NO

Exhale

YES
Table 4
Common complications related to MV in the CICU and evidence-based strategies to reduce their incidence

<table>
<thead>
<tr>
<th>Complication</th>
<th>Strategies to Mitigate Complication</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>VALI</td>
<td>Small tidal volume (6 mL/kg) Plateau airway pressure &lt;30 cm H2O Permissive hypercapnia</td>
<td>ARDS network</td>
</tr>
<tr>
<td>VAP</td>
<td>Ventilator care bundle (peptic ulcer prophylaxis, DVT prophylaxis, daily cessation of sedation and 30° head elevation) Oral care Subglottic secretion drainage NIV</td>
<td>IHI</td>
</tr>
<tr>
<td>ventilation</td>
<td>Minimizing use of sedation in patients receiving MV Early physical and occupational therapy in patients receiving MV</td>
<td>Strom et al, Schweickert et al, 67 2010 71 2009</td>
</tr>
</tbody>
</table>

Abbreviations: DVT, deep-vein thrombosis; IHI, Institute for Healthcare Improvement; VAL, ventilator-associated lung injury; VAP, ventilator-associated pneumonia.

Data from Refs.67–75

Table 5
Therapeutic options for treatment of weaning failure of cardiac origin

<table>
<thead>
<tr>
<th>Likely Cause</th>
<th>Therapeutic Intervention</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑Preload</td>
<td>Diuretic therapy</td>
<td>Lemaire et al, 61 1998</td>
</tr>
<tr>
<td></td>
<td>Phosphodiesterase inhibitors (eg, enoximone, milrinone)</td>
<td>Paulus et al, 77 1994</td>
</tr>
<tr>
<td>↑Afterload</td>
<td>Vasodilators (eg, nitrates) NIV after extubation</td>
<td>Routsi et al, 78 2010</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ferrer et al, 79 2006</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
<td>Vasodilators</td>
<td>Demoule et al, 80 2004</td>
</tr>
<tr>
<td></td>
<td>Angioplasty</td>
<td></td>
</tr>
<tr>
<td>Impaired LV ejection fraction</td>
<td>Levosimendan</td>
<td>Sterba et al, 61 2008</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>Calcium channel blockers</td>
<td>Adamopoulos et al, 82 2005</td>
</tr>
</tbody>
</table>

Data from Refs.61,77–82
Finally, elective initiation of NIV immediately following extubation has been shown to reduce the incidence of respiratory failure in patients at risk, including those with underlying cardiac failure.79

SUMMARY

Patients admitted to the CICU are of increasing complexity in terms of cardiac conditions and noncardiac comorbidities and, as a consequence, require more and longer duration of ventilatory support. A deep understanding of respiratory physiology and the interactions between the cardiovascular and respiratory systems is essential for managing patients requiring MV in the CICU.

Both NIV and MV requiring an artificial airway are used in the modern CICU in a continuum of respiratory support. Congestive cardiac failure, resulting pulmonary edema, and severe cardiogenic shock are common indications for ventilatory support in the CICU.

A balance between maintaining adequate oxygenation, tissue perfusion, and aerobic metabolism and avoiding the deleterious effects of hyperoxygenation should be considered when titrating oxygen administration. The choice of ventilation modes should be tailored to the specific patient’s condition, ensuring effective minute ventilation, reducing the work of breathing and minimizing adverse hemodynamic effects. NIV is a valuable evidence-based therapeutic intervention for the management of patients with acute cardiogenic pulmonary edema.

Discontinuation of MV should be considered as soon as the cardiac pathology that prompted the initiation of respiratory support is stabilized. However, the weaning process can significantly stress the cardiovascular system, and cardiac failure is a common cause of failure to wean. The identification of patients likely to fail and prompt preemptive intervention is crucial for successful weaning and avoiding complications related to prolonged MV.

REFERENCES


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74. IHI proposes six patient safety goals to prevent 100,000 annual deaths. Qual Lett Healthc Lead Assoc 2005;294(24):11–2, 1.


