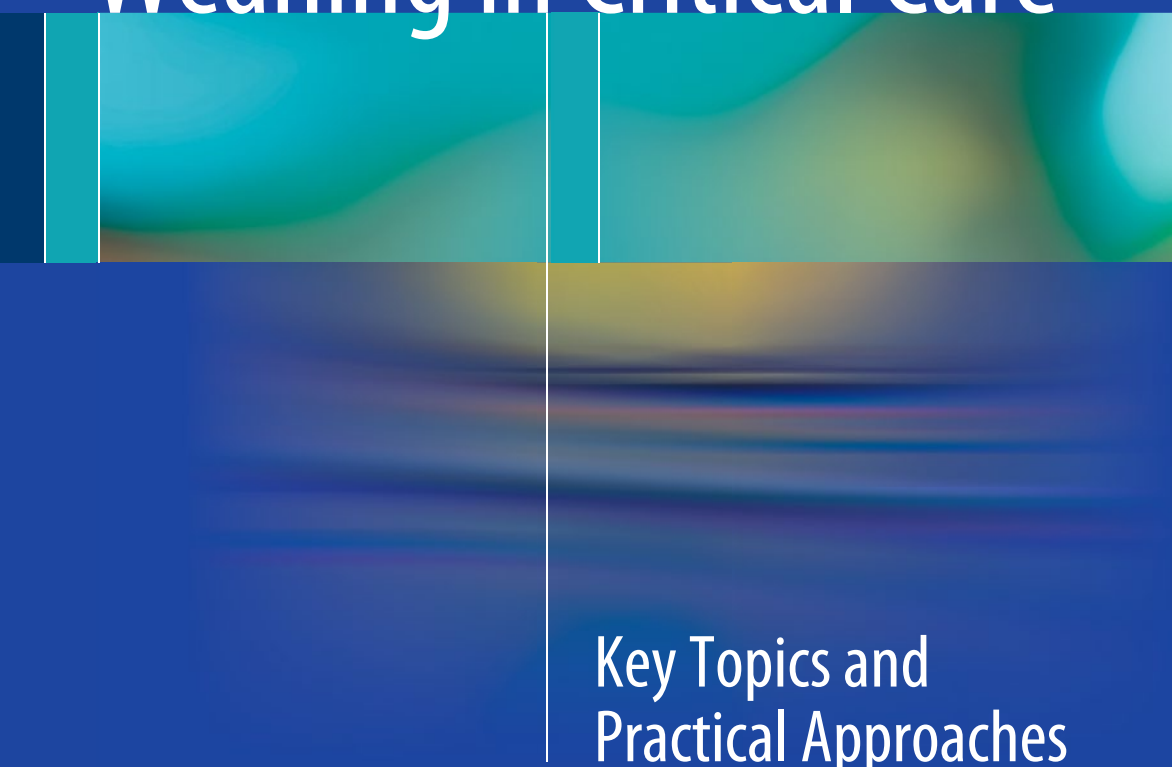
Antonio M. Esquinas *Editor*

Noninvasive Mechanical Ventilation and Difficult Weaning in Critical Care



Key Topics and Practical Approaches

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To wife Rosario, my daughters and Rosana Alba, inspiration and meaning

To the memory of my father

Preface

Ideally all strategies directed toward decreasing the duration of invasive mechanical ventilation (IMV) and reducing or avoiding its complications are useful in patients receiving IMV for different medical or surgical reasons. In the past decade advancement in protocols focusing on weaning from mechanical ventilation and new ventilation modes such as neutrally adjusted ventilatory assist (NAVA) and airway pressure release ventilation (APRV) has been developed along with improving the patient-ventilator interaction, advance monitoring, and strategies for early diagnosis and prevention of ventilator-associated pneumonia. However, there still remain a significant proportion of those who are dependent on IMV and develop difficulty in weaning from it even after their underlying acute respiratory failure (ARF) and other organ failure have resolved. This population represents weaning failure and ventilator dependence.

More and more advanced surgical procedures and medical management in the elderly population and those with multiple comorbidities also lead to failure to wean from IMV and impact healthcare delivery both due to persistent long-term illness and increasing cost of care.

Currently, noninvasive mechanical ventilation (NIV) is considered one of the alternatives to endotracheal intubation in selected patients who develop ARF of diverse etiology. Its establishment as a suitable, effective, and rational alternative is based not only for its strong and positive action on the respiratory muscles and gas exchange but also due to its positive influence on short- and long-term outcome in critically patients. This influence is significant particularly in patients with exacerbation of COPD and acute cardiac pulmonary edema and who are immunodepressed.

In the past decade there has been significant development in NIV equipment and interfaces and in the understanding of the patient-NIV interaction. This has led to physicians considering NIV as an alternate to endotracheal intubation and IMV, in the management of not only ARF but also failure to wean from IMV and extubation failure. The latter is defined as a condition where the patient is unable to sustain respiratory status postextubation from IMV. Is NIV a recognized alternative to IMV in these conditions? Will this strategy change patient outcomes and IMV-related complications? Will NIV influence healthcare delivery by improving quality of care and reduce cost of care?

In this book, sections and chapters are structured in response to these questions based on evidence, clinical practice, and expert recommendations.

The recognized chapters that we have contemplated on NIV have been divided into clinical conditions such as persistent weaning failure from prolonged mechanical ventilation, extubation post acute respiratory failure, and unplanned extubation and its use as alternative to short- and long-term IMV including those with tracheotomy. The use of NIV in these clinical conditions will look at the diverse medical and surgical (thoracic, cardiac, abdominal, lung transplants) population. Additionally, determinants of NIV response, comorbidities, equipments and interfaces, ventilatory modes, patient-ventilator interaction, and clinical monitoring will also be covered in this book.

We consider that this book represents a valuable tool for a practical approach by the rational use of NIV in prolonged mechanical ventilation, difficult weaning, and postextubation failure.

Murcia, Spain Antonio M. Esquinas, MD, PhD, FCCP

Contents

Part I

Weaning From Mechanical Ventilation. Determinants of Prolonged Mechanical Ventlation and Weaning

Physiologic Determinants of Prolonged Mechanical Ventilation and Unweanable Patients

1

Dimitrios Lagonidis and Isaac Chouris

1.1 Introduction

Unfortunately, there is no broadly accepted definition of prolonged mechanical ventilation (PMV). According to a consensus conference held in 2004, PMV is defined as ≥21 consecutive days of mechanical ventilation (MV) for ≥6 h/day [1]. This definition seems to have high sensitivity; most patients requiring MV for more than 21 days after acute critical illness or injury would meet the clinical phenotype of chronic critical illness syndrome (CCIS). Patients with CCIS have survived acute critical illness. Pathophysiologically, it consists of a metabolic, immune-neuroendocrine axis and nutritional derangements caused by the initial event (trauma, sepsis, surgery) and then maintained with unresolved critical illness, PMV, and chronic inflammation [3].

CCIS has been considered a distinct entity with a predictable constellation of clinical features and a course characterized by ongoing chronic inflammation, slow fluctuations in function and care needs, and slow (over weeks or months) progress or deterioration, which may be interrupted by acute events such as sepsis or acute heart failure [2, 3]. Apart from prolonged ventilator dependence, patients with CCIS have profound weakness (caused by myopathy, neuropathy, or loss of lean body mass); brain dysfunction (coma, delirium, depression, anxiety, cognitive impairment); distinctive neuroendocrine derangements (impaired secretion of anterior pituitary hormones, impaired anabolism); increased vulnerability to infections caused by multi-drug-resistant pathogens;, and skin disruption attributed to nutritional deficiencies, edema, and prolonged immobility.

CCSI has been considered a byproduct of medical technology and is increasingly recognized as an important problem in modern medicine and one of the growing

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challenges in health care [2, 3]. It is estimated that between 5 and 13 % of mechanically ventilated patients require PMV [4], and that about 50 % of these will be liberated from the ventilator. However, about 25 % of intensive care unit (ICU) survivors with CCIS and PMV are not weaned at the end of first year [2]. CCIS patients have poor prognosis and prolonged ICU and hospital stays (either in long-term acute care facilities or in specialized weaning centers), contributing to increased costs. It has also been estimated that 1-year mortality rates range from 48 to 68 % [3].

The ultimate goal for CCIS patients is liberation from a ventilator, because successful weaning is associated with improved survival, better quality of life, and less financial burden on health-care systems. Therefore, this review is intended not only to analyze the physiologic determinants of PMV and unweanable patients in the context of CCIS but also to guide physicians managing these patients in a comprehensive and structured way.

1.2 Physiologic Determinants

The adequacy of the respiratory function depends on the balance between the respiratory requirements (the "load") and the capability of the respiratory pump and its components (the respiratory motor drive and the neuromuscular system) to meet those requirements. A practical and methodical approach to the problem of difficult-to-wean and unweanable patients is to consider the various factors with the ability to "tip" the balance, thereby slowing down or even disallowing the weaning procedure.

1.2.1 Respiratory Physiological Determinants

1.2.1.1 Factors Determining Increased Respiratory Load

Control of Breathing

It has been long recognized that the hallmark of weaning failure is a rapid shallow breathing pattern, the combination of elevated frequency (f) and decreased tidal volume (V_T) [5–7]. Weaning failure patients exhibit marked shortening of both inspiratory and expiratory time, which results in increased breathing frequency. At the same time, the combination of decreased inspiratory time (Ti) and normal mean inspiratory flow leads to decreased V_T [8].

Acute hypercapnia has been consistently observed in many patients who failed to wean despite an increase, not a decrease, in respiratory drive, measured by using $P_{0.1}$ or the mean inspiratory flow. The hypercapnia is not caused by decreased minute ventilation. Instead, it is the consequence of the rapid shallow breathing pattern, resulting in dead-space ventilation [6].

Assessment of respiratory drive is determined by $P_{0.1}$, which is the airway occlusion pressure at the first 100 msec of inspiration (normal values: 0.5–1.5 cmH_2O). Although it is available with most ventilators, it is of limited value because of the wide normal range. The value of $P_{0.1}$ depends not only on respiratory drive but also

on inspiratory muscle capacity. It is worthy of consideration that in patients on PMV, the values of $P_{0.1}$ measured at the end-expiratory lung volume may be affected by further development of abnormal muscle length and chest wall distortion [11]. Values within the normal range practically exclude respiratory drive disorders as the source of difficult weaning, although considerable variability has been reported [11]. Nevertheless, $P_{0.1}$ remains a useful index when these limitations are recognized.

Impaired respiratory drive is only infrequently the cause of difficulties in weaning [5, 6]. It may involve defects in the peripheral and central chemoreceptors (carotid body dysfunction, prolonged hypoxia, metabolic alkalosis) or the brainstem respiratory centers (encephalitis, brainstem infarction, hemorrhage or trauma, demyelination, drug side-effects, endocrine disturbances – hypothyroidism or hyperthyroidism). Conversely, respiratory motor drive is increased in most patients who are unable to liberate from the ventilator [5, 6]. In ventilator-dependent patients, high $P_{0.1}$ associated with low V_T indicates the poor conversion of high drive to adequate ventilatory output. Accordingly, the demonstration of high drive to breathe has been found to predict weaning failure [11].

It is well known that the absence of high f/V_T breathing pattern can predict weaning success (WS), not only in heterogeneous ICU patients [7, 12, 13] but also in chronically ill patients [11]. Nevertheless, specific groups of patients on PMV, such as those with severe COPD, deserve special consideration. These patients may exhibit weaning failure (WF) despite a low f/V_T (shallow but not rapid breathing) during unassisted breathing [11]. The major mechanism responsible for WF is the combination of abnormal lung mechanics, specifically increased intrinsic positive end-expiratory pressure (PEEPi) and resistance, and the reduced pressure-generating capacity of inspiratory muscles resulting from dynamic hyperinflation. Interestingly, the respiratory drive is augmented to maintain adequate tidal volume but is poorly transformed into inspiratory flow because of the impaired respiratory muscles. As a result, the breathing effort leads to low V_T. The diminished V_T is therefore ineffective to meet metabolic demands and clear carbon dioxide. On the other hand, the high motor output drive charges the inspiratory muscles and forces them to use a significant amount (>40 %) of their maximal pressure-generating capacity to sustain spontaneous ventilation. Accordingly, unassisted breathing cannot be sustained without excessive dyspnea [11].

Respiratory Mechanics

In an acute setting, Jubran and Tobin [7] demonstrated that, during a spontaneous breathing trial (SBT), all passive respiratory mechanics (resistance, elastance, PEEPi) became more abnormal in WF patients than in WS patients. More specifically, respiratory resistance increased up to seven times the normal value at the end of the trial, whereas pulmonary elastance increased about five times the normal value. Moreover, PEEPi almost doubled during the trial. The same findings were also found by other investigators [9].

Airway resistance and respiratory load, that is, the work of breathing (WOB), are directly related. Significantly increased airway resistance that hinders the weaning procedure may arise from upper (obstruction of tracheotomy tube, secretions,

post-extubation tracheal injury) or lower airway pathology (bronchospasm, bronchial hyper-responsiveness, pulmonary edema). Increased elastance (decreased compliance) of the respiratory system correlates with increased WOB. Low thoracic wall compliance may arise from pathological states such as edema of the thoracic wall, rib cage deformities, pleural effusions, morbid obesity, increased intra-abdominal pressure. Additionally, decreased lung compliance may be the result of lung edema (cardiogenic or noncardiogenic), lung infections and atelectasis.

Expiratory flow limitation leads to inadequate expiratory time to achieve fully deflated lungs, hindering the lungs to reach the elastic equilibrium point. The result is the phenomenon of progressive air-trapping and dynamic lung hyperinflation, which is associated with the development of PEEPi. Dynamic hyperinflation may have hemodynamic consequences (decreased venous return and cardiac output) but is also a major cause of increased WOB. The positive pressure thus generated means that the threshold to initiate inspiratory flow is heightened and the patient's inspiratory efforts may be ineffective, leading to ineffective ventilator triggering and patient-ventilator asynchrony. Moreover, the presence of dynamic hyperinflation detrimentally affects the diaphragmatic force-generating capacity by displacing it to a suboptimal position of its length-tension curve.

In spontaneously breathing patients, dynamic measurement of PEEPi with an esophageal balloon delivers more precise results and thus is preferable. Elevated PEEPi may arise for the following reasons:

- increased expiratory flow resistance (bronchospasm, compromised endotracheal tube patency, heat and moisture exchange (HME) filters)
- loss of lung elastic recoil (emphysema)
- increased minute ventilation
- inadequate expiratory time

Gas Exchange

Inadequate gas exchange (hypoxemia, hypercapnia) exerts an additional load on the respiratory muscles because increased minute volume is required to restore gas exchange disturbances, resulting in muscle fatigue and WF. Hypercapnia results mainly from the following mechanisms: hypoventilation (e.g., neuromuscular diseases), severe low ventilation/perfusion mismatch (e.g., chronic obstructive pulmonary disease (COPD)), and, to a lesser extent, increased dead space (rapid shallow breathing, heat and moisture exchangers, connectors to the Y-point of the circuit).

Interestingly, studies using the multiple inert gas method showed that ventilation/perfusion maldistribution and hypercapnia were found in WF patients [10]. Specifically, acute hypercapnia was observed in many patients who failed to wean despite an increased respiratory motor output, measured by $P_{0.1}$ [7]. Acute hypercapnia is not caused by decreased minute ventilation. Instead, it is the consequence of a rapid shallow breathing pattern resulting in dead-space ventilation. Only in a minority of WF patients may hypercapnia be attributed to primary depression of respiratory drive [7].

1.2.1.2 Factors Determining Reduced Respiratory Capacity

Respiratory Muscle Weakness or Dysfunction

Spontaneous breathing during a weaning trial imposes a substantial load on the inspiratory muscles, which are considered the major part of the respiratory pump. Dysfunction of the respiratory pump may result from a defect anywhere between the respiratory centers in the medulla and the myocytes inside the respiratory muscles. Upon release of positive pressure ventilation and during unassisted breathing, patients have to make a greater inspiratory effort to compensate for the deteriorating respiratory mechanics. Using an esophageal balloon catheter, direct measurements of WOB and pressure-time product consistently showed that WF patients exhibit a greater effort compared with WS patients [7].

Respiratory muscle dysfunction is a major determinant of the degree of weaning difficulty. Clinical signs suggestive of respiratory muscle dysfunction, and thus of the respiratory pump, include tachypnea, dyspnea, and paradoxical respiratory movements. Respiratory muscle dysfunction may be caused by any condition that leads to:

- Impaired neurotransmission (amyotrophic lateral sclerosis, Guillain-Barré, myasthenia gravis, drugs, phrenic nerve dysfunction, critical illness polyneuropathy)
- Reduced muscle strength (malnutrition, sepsis-associated myopathy, acidosis, electrolyte disturbances, hypoxemia, low cardiac output states)

Global evaluation of inspiratory muscle strength includes the static measurement of *maximal inspiratory pressure* (*MIP*) during the Mueller maneuver, with lower normal values −75 cmH_2O in men and –50 cmH_2O in women younger than 65 years old. It can be measured either in mechanically ventilated or spontaneous breathing patients. Values that are more negative than normal essentially exclude significant inspiratory muscle weakness, whereas values that are more positive than normal do not prove muscle weakness. *MIP* depends on patient cooperation (it is a voluntary test) and lung volume and thus can falsely assess muscle weakness. Many studies have shown that *MIP* does not discriminate between WF and WS patients, suggesting that muscle weakness may not be a major determinant of weaning outcome [10].

A more reliable assessment of diaphragmatic strength is taken by recording *transdiaphragmatic pressure* (*Pdi*). *Pdi* is the difference between abdominal (gastric) and pleural (esophageal) pressure. It can be obtained after a forceful inspiration against a closed airway or after sniffing and both gastric and esophageal balloons are required. The energy expenditure of the diaphragm can be estimated by *the tension-time index and the pressure-time product*. These indices are too complicated for routine clinical use. Ideally, *Pdi* should be measured during a SBT, because it is influenced by positive pressure of the ventilator [27]

The involuntary evaluation of diaphragm strength is obtained by the measurement of twitch *transdiaphragmatic pressure* (Pdi_{tw}) or *twitch airway pressure* (Paw_{tw}) after magnetic phrenic nerve stimulation [25, 26]. These methods are not applicable in everyday practice because they are fairly invasive and technically difficult in critically ill intubated or tracheostomized patients [27]. Values of Pdi_{tw} between 35 and 39 cmH_2O are recorded in normal subjects, whereas values below 10 cmH_2O are obtained in WF patients [14].

Another important task of the ventilator pump is the ability to endure, that is, to avoid muscle fatigue. The fatigue threshold of the diaphragm can be quantified by the *tension-time index of the diaphragm* (*TTIdi*), derived by the formula $TTIdi = (Pdi/Pdi_{max}) \times (Ti/Ttot)$, where Pdi is the tidal transdiaphragmatic pressure, Pdi_{max} is the maximum transdiaphragmatic pressure, Ti is the inspiratory time, and Ttot is the total breath duration. This equation demonstrates the importance of both the pressure-generating effort of the diaphragm and the relative duration of inspiration as determinants of diaphragmatic fatigue. Diminishing diaphragm strength results in decreased Pdi_{max}, whereas reduced compliance increases Pdi. Similarly, tachypnea increases the Ti/Ttot index, thus promoting muscle fatigue.

In one study, it was reported that the majority of ICU patients had diaphragm muscle weakness at the beginning of mechanical ventilation associated with sepsis and disease severity [24]. The ability of the diaphragm to generate force was assessed by recording occluded twitch tracheal pressure during twitch magnetic stimulation of bilateral phrenic nerves. The twitch tracheal pressure ($Ptaw_{tw}$), measured at the proximal end of the endotracheal tube, was used as a surrogate of transdiaphragmatic pressure independent of patient effort and cooperation. More specifically, 64 % of patients had a $Ptaw_{tw}$ less than 11 cmH_2O, a value that indicates diaphragm muscle weakness.

Hypercapnia is often considered an indirect sign of respiratory muscle fatigue, but one must be careful to take into account other mechanisms leading to it. Nevertheless, it is probably safe to conclude that lack of hypercapnia, combined with absence of acid–base disturbances, practically rules out the possibility of fatigue as a cause for weaning failure.

It has been suggested that the f/V_T ratio gives an estimate of the capability of sustaining unsupported breathing and could be a surrogate of the most-difficult to measure *TTIdi or* Pdi/Pdi_{max}.

For the first time, Jubran et al. [7] showed that, in patients with COPD, the major determinant between a successful and failed weaning trial was a change in the breathing pattern rather than an intrinsic derangement of pulmonary mechanics. In another study, Vassilakopoulos et al. [9] reported that, compared with WS patients, WF patients had greater total resistance, intrinsic PEEP, dynamic hyperinflation, ratio of mean to maximum inspiratory pressure, less MIP, and a breathing pattern that was more rapid and shallow. They also found that TTI and f/V_T were the only significant parameters that predicted weaning success. Finally, in a study by Capdevila et al. [15], the WF was associated with high breathing frequency, increased $P_{0.1}$, minute ventilation, intrinsic PEEP, and persistent hypercapnia.

Although *TTI and* $Pdi/Pdi_{max.}$ are too difficult to measure in everyday practice, they seem to be more accurate in determining the potential reserve of the patients during the weaning trial. On the other hand, the f/V_T ratio may not give a thorough insight into the weaning capabilities of ventilator-dependent patients because it could be affected either by their psychological burden resulting in tachypnea or by their tendency not to increase f to avoid dynamic hyperinflation [16].

Carlucci et al. [16], by recording active respiratory mechanics in true ventilator-dependent patients with multiple weaning failures in the past, showed that the major determinant of WS was associated with the significant improvement of diaphragmatic inotropism at the time of gaining liberation from the ventilator, as expressed by increased Pdi_{max}. They also found that these patients on PMV have increased

mechanical load/capacity balance, predominantly because of reduced Pdi_{max} rather than excessive load, so that once they are on unassisted breathing, they breathe above the threshold of diaphragmatic fatigue. In both the WF and WS patients, a tension-time index (TTI) above the fatigue threshold was noted at the first attempt of weaning trial.

Specifically, in PMV patients, the recovery of an inadequate respiratory muscle force could be the major determinant of late weaning success, because this factor allows them to breathe far below the diaphragm fatigue threshold. Many factors contribute to the reduced Pdi_{max} in ventilator-dependent patients (e.g., age, hypercapnia, hypoxia, malnutrition, inactivity, mechanical ventilation–induced atrophy, sepsis, prolonged use of corticosteroids, and cardiovascular compromise). Purro et al. [11] showed that the patients who could not be weaned had small tidal volume, high neuromuscular drive, abnormal lung mechanics, and reduced inspiratory muscle strength as soon as they resumed spontaneous breathing.

For many years, electromyography (EMG) of the diaphragm has been a useful research tool in evaluating respiratory muscle dysfunction. It can be obtained in ICU patients using a special esophageal catheter with multiple electrodes [27]. The signal that is taken is referred as the *electrical activity of the diaphragm* (*EAdi*) and it is considered as a direct measure of neural respiratory drive. Thus, it is considered the gold standard to detect the onset and duration of neural inspiration and expiration and thus patient-ventilator asynchronies [27].

The V_T/EAdi ratio represents the *neuroventilatory efficiency* (*NVE*) of the diaphragm. An improved NVE indicates the capability of the patient to generate the same VT with lower Eadi [27]. It was suggested as an index to discriminate between extubation success and failure in patients weaning from the ventilator. Another index is the *neuro-mechanical efficiency* (*NME*), indicated by the ratio Pdi/EAdi; a gradual decrease in NME suggests the development of diaphragmatic weakness [27]. Although EMG of the diaphragm has some limitations, it seems to be a reasonable method for monitoring respiratory muscles during the course of a weaning trial in PMV patients.

Ultrasonography has been used to investigate diaphragmatic atrophy or dysfunction in critical care settings. By using B-mode ultrasonography with a linear array transducer, the diaphragm thickness at the zone of apposition could be precisely and reproducibly measured in spontaneously breathing patients during a weaning trial [28]. Kim et al. [29] evaluated diaphragmatic dysfunction during a SBT after patients had been ventilated for more than 48 h. They found diaphragmatic dysfunction (defined as <10 mm vertical excursion) in 29 % of patients, and there was a correlation with longer mechanical ventilation and WF. Moreover, this ultrasonographic criterion to predict WF was similar to the rapid shallow breathing index.

1.3 Cardiac Determinants

The transition from the positive pressure ventilation to spontaneous breathing exerts an additional load on the cardiovascular system and can impose or unmask cardiac dysfunction, either systolic or diastolic. These factors may thus be causes of unsuccessful weaning. The heart-lung interactions during the weaning procedure are complex. Spontaneous breathing raises WOB and oxygen consumption by the respiratory muscles and promotes adrenergic stress and negative swings in the

intrathoracic pressure. These alterations lead to increases in both preload and afterload of right and left ventricles through the augmented venous return, resulting in weaning-induced cardiac dysfunction.

At the end of a weaning trial, oxygen consumption is equivalent in WS and WF patients [17]. However, the response of the cardiovascular system to the oxygen demand differs in the two groups. In WS patients, oxygen demand is met by the augmented oxygen delivery mediated through the expected increase in cardiac output on release of positive pressure ventilation [17]. In WF patients, because they have relatively decreased oxygen delivery, oxygen demand is met by the increase in oxygen extraction. Under these circumstances, the greater oxygen extraction results in a significant decrease in SvO_2, contributing to hypoxemia [17].

In 2015, it was reported that, in acute critically ill patients, it was found that a negative passive leg-raising test performed before SBT, suggesting preload independence, was associated with weaning-induced cardiac dysfunction [23].

Diastolic dysfunction is a common and underdiagnosed entity. More than 60 % of people over 65 years of age experience diastolic dysfunction, and in more than 50 % of patients with heart failure, it is of the diastolic type. Moreover, differentiation between systolic and diastolic cardiac failure is clinically important because of distinct therapeutic approaches [21]. Diastolic dysfunction with relaxation impairment has been found to predict weaning failure. The principal feature of LV diastolic failure is reduced compliance of the ventricle due to various causes (e.g., coronary artery disease, myocardial hypertrophy and fibrosis, infiltrative diseases, hypoxia, or acidosis).

There is growing evidence to advocate that transthoracic echocardiography (TTE) plays a key role in the evaluation of patients who are difficult to wean due to cardiac origin. However, it is not possible to use it in every critically ill patient because of certain limitations (e.g., excessive pulmonary emphysema, or thoracic trauma). In tissue Doppler imaging TTE, the ratio of mitral Doppler inflow E velocity to annular tissue Doppler Ea wave velocity (E/Ea) provides an accurate estimate of the degree of diastolic dysfunction. Moreover, these echocardiographic measurements can also be performed on patients with atrial fibrillation with reasonable sensitivity and specificity.

In 2010, Gaille et al. [20], in an unselected cohort of patients, found that weaning failure occurred more often in patients with systolic heart failure. More precisely, in patients with ejection fraction (EF) <50 % they found signs of diastolic dysfunction (decreased E/A and depressed acceleration time of E wave) during a SBT. Moreover, Moscietto et al. [18] showed that in 68 patients with sinus rhythm and atrial fibrillation on mechanical ventilation more than 48 h, the measurement of E/Ea with Doppler tissue imaging TTE could predict weaning failure as early as 10 min after the beginning of the SBT. They also suggested that diastolic dysfunction with relaxation impairment was strongly associated with weaning failure. Conversely, in the same study, the systolic dysfunction was not associated with weaning outcome. In another study with similar findings [19], the authors suggested that an E/Ea >7.8 may indentify patients at high risk of WF.

In conclusion, diastolic dysfunction of the left ventricle seems to be important in the evolution of WF. By measuring E and Ea waves even in patients with atrial fibrillation, TTE with Doppler tissue imaging measuring is a key examination that

can be easily applied before and after the weaning trial. It has also been demonstrated that the transition from mechanical ventilation to sustained breathing could lead to myocardial ischemia in patients with coronary artery disease. Ischemia can be detected by electrocardiogram before and at the end of the SBT and the significance of anemia as a precipitating factor should not be underestimated.

Mixed venous oxygen saturation (SvO_2) can be used as a marker of cardiac performance, with *superior vena cava oxygen saturation* ($ScvO_2$) serving as a reasonable surrogate. In difficult-to-wean patients, a decrease in SvO_2 during the weaning procedure should raise the suspicion about the presence of inadequate cardiac output. Patients with cardiac dysfunction largely rely on increasing the oxygen extraction ratio instead of raising the cardiac output, resulting in SvO_2 reduction as demonstrated by Jubran et al. [17] in a study comparing 8 patients who failed at SBT with 11 patients who successfully completed the SBT. The decrease in SvO_2 was related to the inability to improve cardiac output and consequently oxygen transport. Increased afterloads of the right and left ventricle were found in these patients.

It is imperative to note that reduction in $ScvO_2$ is the normal response to increased loading. In normal subjects on moderate exercise, it was found that $ScvO_2$ decreases below 50 %. Therefore, a reduction in $ScvO_2$ should not necessarily be interpreted as a marker of heart failure. Accordingly, in WF patients, without a reduction in $ScvO_2$, heart dysfunction is highly unlikely [21]. Conversely, in those patients who failed a weaning trial and had reduced $ScvO_2$, heart dysfunction could be a limiting factor and further investigation with echocardiography and/or insertion of a Swan-Ganz catheter is warranted [21].

Brain natriuretic peptide (BNP) is a neurohormone synthesized in the cardiac ventricles and released from the myocardium upon stretch. It is released by the myocytes as pre-proBNP that is cleaved into *proBNP* and finally into BNP and the inactive N terminal proBNP peptide (*NT-proBNP*). Its release into the circulation is directly proportional to the ventricle expansion and volume overload of the ventricles. Thus, it serves as a marker of the systolic and diastolic left ventricular dysfunction. The value of BNP or NT-proBNP as a predictor of weaning failure due to cardiovascular reasons seems to be well established in the literature. Nevertheless, the accepted cut-off values pose a clinical challenge for data interpretation.

A study by Grasso et al. [22] demonstrated that serial measurements of NT-proBNP could detect acute cardiac dysfunction during an unsuccessful weaning trial in difficult-to-wean patients with COPD. Baseline NT-proBNP levels were significantly higher (median, 5,000; interquartile range, 4,218 pg/mL) in patients with cardiac dysfunction. It was also shown that levels of NT-proBNP increased significantly at the end of the spontaneous breathing trial only in patients with acute cardiac dysfunction (median, 12,733; interquartile range, 16,456 pg/mL).

Conclusions

The ultimate goal for CCIS patients on PMV is liberation from the ventilator. Repeated weaning failure has been associated with an imbalance between increased load and reduced capacity of the respiratory muscles or, to a lesser extent, with the cardiovascular impairment. A systematic approach to the problem

Increased motor drive (very common)

rapid shallow breathing index (f/VT)

Resistive loads

Lung Elastic loads

elastance, resistance, PEEPi

Chest wall elastic loads

Cardiovascular impairment

systolic dysfunction

diastolic dysfunction (E<A, E/Ea >11)

passive leg raising test

pro-BNP or NT-proBNP

SvO2 or ScvO2

Impaired motor drive (uncommon)

Impaired neurotransmission (uncommon)

Inspiratory muscle weakness or dysfunction (very common)

rapid shallow breathing index (f/VT)

maximal inspiratory pressure (MIP)

transdiaphragmatic pressure (Pdi)

Tension time index (TIdi)

Pressure time product of the diaphragm

Pdi/Pdimax

electrical activity of the diaphragm (Eadi)

neuroventilatory efficiency (NVE) of the diaphragm

B-mode ultrasonographic evaluation of diaphragm thickening

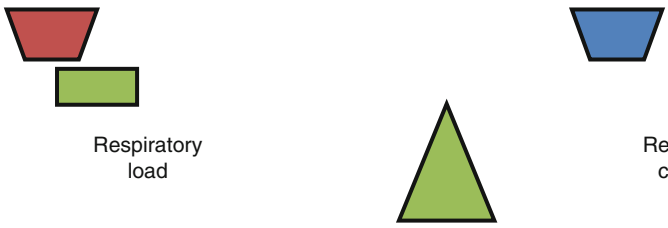

Fig. 1.1 Balance between load (↑motor drive, ↑resistive, ↑elastic, cardiovascular impairment) and capacity (↓motor drive, ↓neurotransmission, inspiratory muscle weakness) determines the ability to sustain spontaneous ventilation

Key Points

- In PMV and unweanable patients, the imbalance between inspiratory muscle work load and inspiratory muscle capacity is of paramount importance.
- The rapid shallow breathing pattern is the hallmark of weaning failure.
- In PMV patients, the major determinant of prolonged weaning is inspiratory muscle weakness or dysfunction, as expressed by TTIdi that is above the fatigue threshold.
- During the course of a weaning trial, most WF patients significantly increase respiratory load as a result of severe worsening of respiratory mechanics (e.g., resistance, elastance, or PEEPi).
- In PMV patients, the recovery of inadequate inspiratory muscle force seems to be the major determinant of WS allowing them to breathe below the diaphragmatic fatigue threshold.
- A less common cause of WF is impairment of cardiovascular performance.

of difficult-to-wean and unweanable patients is to understand in-depth the physiologic determinants characterizing the two sides of the balance (Fig. 1.1). This approach may help identify the factors that play a role in the specific patient so that appropriate therapeutic strategies can be applied.

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Prolonged Weaning from Mechanical Ventilation: Pathophysiology and Weaning Strategies, Key Major Recommendations

2

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Abbreviations

APACHE	acute physiology and health evaluation
ARDS	acute respiratory distress syndrome
BNP	B-type natriuretic peptide
CCI	chronically critically ill
CCIS	chronic critical illness syndrome
CINM	critical illness neuromyopathy
COPD	chronic obstructive pulmonary disease
ICU	intensive care unit
GCS	Glasgow coma scale
LTAC	long-term acute care
MV	mechanical ventilation
NAMDRC	National Association for Medical Direction of Respiratory Care
NIV	noninvasive ventilation
PMV	prolonged mechanical ventilation
PSV	pressure support ventilation
RCT	randomized controlled trial
RSBI	rapid shallow breathing index
SBT	spontaneous breathing trial
SWU	specialized weaning units

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2.1 Introduction

Advances in the management of critically ill patients in intensive care unit (ICU) have improved mortality and morbidity as well as reduced length of stay and, subsequently, cost of treatment. However, despite improvements in short-term mortality and stabilization of acute organ dysfunction, a small but substantial population of critically ill patients who survive the initial critical illness continue to suffer from prolonged dependence on life support or to need long-term therapeutic interventions. These patients have been grouped under the classification of chronically critically ill (CCI) patients. Such a group is characterized by heterogeneity, prolonged need for high-cost interventions, and high long-term (around 1 year) mortality rate [1]. The best characterized component of the CCI population is patients on prolonged mechanical ventilation (PMV). In 2005, the National Association for Medical Direction of Respiratory Care (NAMDRC) defined PMV as the need for ≥21 consecutive days of mechanical ventilation (MV) for ≥6 h/day [2]. According to the European Respiratory Society Task Force, these patients constitute a particular group needing prolonged weaning from the ventilator, defined as more than three spontaneous breathing trials (SBTs), or more than 7 days from the first unsuccessful SBT [3]. Nevertheless, other investigators have favored Medicare's definition of MV >96 h, with tracheostomy as the marker of PMV [2].

Patients requiring PMV have clearly different needs and resource consumption patterns in relation with patients during the acute phase of critical illness. Moreover, these patients may represent as many as 14 % of patients admitted to the ICU for intubation and MV, whereas it is estimated that they account for 37 % of all ICU costs and are associated with in-hospital mortality up to 32 % [4, 5]. Finally, available data suggest that the global prevalence of PMV in Europe ranges from 2 to 30 per 100,000 population according to different countries [6], whereas different studies have demonstrated that as many as 20 % of medical ICU patients remained dependent on ventilator support after 21 days [3].

2.2 Discontinuation of PMV

2.2.1 Pathophysiology of Weaning Failure

The successful weaning process from PMV is based on the understanding of the complexity of different causes associated with the need for prolonged ventilatory support. In this respect, it has been suggested that the major mechanisms of weaning failure in this group of patients include either an isolated failure of the respiratory system or respiratory failure occurring within the context of chronic critical illness syndrome (CCIS) [2, 3, 7].

It is estimated that pulmonary disease accounts for approximately 50 % of causes for PMV, associated with inspiratory muscle weakness, increased work of breathing, and reduced respiratory drive [2, 7]. Pulmonary disease results in reduced lung

compliance and increased load upon respiratory muscles. In this respect, ventilator-associated pneumonia and acute respiratory distress syndrome (ARDS) are considered the main pulmonary pathologies leading to prolonged weaning from the ventilator. Airway disease in patients with chronic obstructive pulmonary disease (COPD) may also increase work of breathing through air-flow limitation, dynamic hyperinflation, and auto-positive end-expiratory pressure (PEEP). Furthermore, congestive heart disease has been reported in up to 26 % of patients hospitalized in long-term acute care (LTAC) hospitals in the United States [8]. Such cardiac dysfunction can be uncovered during SBTs due to increased venous return, end-diastolic volume augmentation, and increased metabolic demands. In these cases, performance of cardiac echocardiography and determination of B-type natriuretic peptide (BNP) serum levels during SBTs can be of significant value for early diagnosis and prompt treatment of possible myocardial dysfunction and/or hypervolemia [7–9].

Critical illness neuromyopathy (CINM) can manifest itself as ICU-acquired weakness and subsequent PMV, usually associated with multiple organ failure, muscle inactivity, hyperglycemia, or use of corticosteroids and neuromuscular blockers. As a result, early mobilization, minimizing the use of deep sedation and steroids, and avoidance of hyperglycemia have been advocated as effective preventive strategies during the acute phase of critical illness [7, 10]. Ventilator-induced diaphragm dysfunction constitutes a rapid form of skeletal muscle injury that may occur within only 18 h of MV [7, 11]. Age, malnutrition, and continuous mandatory ventilation have been found to promote such muscle weakness, whereas pressure support ventilation (PSV) seems to minimize diaphragmatic ventilator-induced injury [11]. In addition, optimal patient-ventilator synchrony through properly adjusted ventilator settings, psychotropic medications, and delirium management seems to reduce work of breathing and further promote earlier weaning from ventilatory support [7].

Finally, managing PMV patients requires careful consideration and management of all issues related to CCIS, such as severe nutritional deficits, endocrine dysfunction, including loss of glycemic control and hypothyroidism, bone loss, and immune and autonomic nervous system dysfunction, that usually arise between 7 and 14 days post acute illness, if the patients do not fully recover from the acute episode [1].

2.2.2 Weaning Strategies in PMV Patients

Weaning rates in PMV patients vary significantly, ranging from 42 to 83 % across different studies, due to the heterogeneity of the population requiring prolonged MV [2, 3]. A prospective observational cohort study that was carried out in 23 LTACs in the United States and included 1,419 patients remains the main source of weaning data in patients with PMV [8]. In this study, 20 % failed to wean from PMV. From this group, 80 % required full-time PMV, 18 % part-time, and 2 % were managed with noninvasive ventilation (NIV). More than half of ventilator-dependent survivors from critical illness were successfully separated from prolonged mechanical ventilation [8].

According to the 2005 NAMDRC report, successful weaning in PMV patients was defined as breathing unassisted for 7 days [2]. In this respect, the recommendations included weaning the PMV patient to about 50 % of ventilator requirements using PSV mode (10–15 cmH_2O) and, subsequently, respiratory therapist-driven SBTs of increasing duration using tracheostomy or T-piece. Moreover, a rapid shallow breathing index (RSBI) of up to 97 was found to correlate with successful 1-hour tolerance of SBT in these patients, shortening the time to weaning by approximately 12 days [2].

What have we learned since the NAMDRC report? It seems that different protocols combining gradual decrease of pressure support ventilation, SBTs in a stepwise manner, daily RSBI measurements, and capping of the tracheostomy tube with NIV could be effective in reducing weaning time in PMV patients [7]. In this respect, a bundle of weaning approaches has also been suggested in the acute care setting for reducing length of MV in survivors of critical illness [12]. Thus, the "ABCDE" bundle, which includes daily *A*wakening, spontaneous *B*reathing trials, sedation *C*hoice, *D*elirium monitoring, and *E*xercise/early mobility, has been proposed in patients with prolonged weaning. Recently, a randomized controlled trial (RCT) that was conducted among 316 PMV patients in a single LTAC facility found that unassisted breathing through a tracheostomy (trash collar) compared with PSV resulted in shorter median weaning time, although weaning mode had no effect on survival at 6 and 12 months [13].

In addition, increased age, severity of illness estimated with Acute Physiology and Chronic Health Evaluation (APACHE) II score, elevated body-mass index and blood urea nitrogen levels, lower Glasgow Coma Scale (GCS), serum albumin, and maximal inspiratory pressure have been associated with failure to wean from PMV [2, 7]. As a result, better identification of different groups of patients requiring prolonged MV is needed for individualizing different weaning strategies. Moreover, the "3 M approach," including *minimizing* sedation, *maintaining* nutrition, and *maximizing* mobility, has been proposed as a simple approach to treating such a complex medical condition [7]. Such efforts should take place in long-term acute care hospitals and specialized weaning units (SWUs), reducing cost of treatment and providing at the same time a multidisciplinary approach of early rehabilitation. These units with specialized teams, including nurses, physiotherapists, and nutritionists, might be an appropriate "bridge-to-home" environment for PMV patients [3]. It has been suggested that SWUs could be of two types: (1) step-down or noninvasive respiratory units within acute care hospitals and (2) regional weaning centers separate from hospitals, where different studies have demonstrated that 34–60 % of patients can be successfully weaned from ventilatory support [3, 8].

Another subset of patients includes those who remain ventilator dependent, requiring long-term ventilator support, which could be provided as NIV in the home setting. Thus, different studies in various groups of PMV patients have shown that approximately 9 % were discharged home with partial ventilator support, with 1 % using NIV and 8 % requiring MV via the tracheostomy [3, 14].

Conclusions

The NAMDRC report included 12 recommendations regarding early identification, management, and research priorities for patients requiring PMV [2]. Such patients by definition have failed multiple SBTs and usually require the placement of a tracheostomy tube. The first priority for the management of this subgroup of critically ill patients is the optimization of any reversible factor contributing to PMV. Thus, early mobilization, discontinuation of high doses of narcotics and benzodiazepines, early recognition, and management of mental disorders, such as delirium, are a few actions that can accelerate the weaning process, in association with treatment of underlying causes of respiratory failure. Moreover, weekly monitoring of proteins and albumin levels should be part of the plan to make sure nutrition goals are met. Ensuring adequate nutrition in CCI patients improves immune function and muscle strength, preventing excess breakdown of lean body mass. Furthermore, a multidisciplinary rehabilitation program must be implemented on an individualized basis, either in the acute care hospital, or to a specialized weaning center, where a team of physiotherapists and nutritionists could manage or even restore muscle weakness and atrophy. Such therapies apart from muscle strengthening can also facilitate the resolution of inflammation, turn off catabolic stimuli, and restore glycemic control [3, 15]. Another important issue is the transition from PMV to long-term MV. It seems that patients with COPD and neuromuscular diseases are more amenable to long-term MV, with 3-year mortality more than 50 % [14]. Furthermore, patients with age >65 with sacral ulcers and abnormal renal function constitute the group with the worse prognosis [14, 15]. In such cases, better communication between caregivers, patients, and families and resetting of expectations regarding weaning failure can facilitate the management of such patients in different settings more effectively.

Key Major Recommendations

- Patients who need ventilatory support for more than 21 days, have failed at least 3 SBTs, or require mechanical ventilation for more than 7 days since the first unsuccessful SBT and have a tracheostomy tube have been categorized in the group needing prolonged mechanical ventilation.
- Such patients are usually chronically critically ill patients with many endocrine, metabolic, neuromuscular, and immunological disorders because the self-adaptation to acute stress has been transformed to a self-defense response, preventing restoration of normal physiology, despite apparent resolution of the causes of acute illness.
- The process of liberating these patients from the ventilator demands, first, the treatment of underlying disease and, second, a multidisciplinary approach, where a group of health-care professionals, such as physiotherapists and nutritionists, apply early mobilization and nutritional support to restore neuromuscular, metabolic, and immunological functions toward "normalcy."

- Weaning protocols may accelerate the weaning process in the acute care setting, however, the heterogeneity of PMV patients limits their diagnostic accuracy, prompting an individualized approach, usually in specialized weaning centers, separate from the acute care hospitals.
- The better communication between caregivers, patients, and families, along with an advanced palliative care system, will restore confidence between health-care professionals and relatives, resetting possibly unrealistic expectations for those patients needing long-term ventilation, usually with NIV even at home.

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Automated Weaning Modes

3

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Mechanical ventilatory support (MV) management of critically ill patients has undergone profound changes over the past 10 years. This practice has evolved from deep sedation associated with a totally controlled ventilation mode for prolong periods to minimal sedation and the corollary use of spontaneous ventilation modes. By reducing the duration and the depth of the sedation, the duration of invasive mechanical ventilation in intensive care units (ICUs) has been significantly shortened. Evidence showing the benefit of such strategies is now clearly demonstrated [1–3].

Weaning is the process of decreasing ventilator support and allowing patients to assume a progressively increasing part of their work of breathing or proportion of their ventilation. It is essential and represents nearly 40 % of the total duration of mechanical ventilation [4]. It has been shown in clinical practice that the use of protocols or algorithms was safe and effective in reducing the time spent on MV [4]. The first step consists of assessing the "readiness to wean," using objective criteria screened daily by nurses or ventilatory therapists to look for contraindications to spontaneous breathing (absence of vasopressors, patient awake, and ad hoc ventilation parameters) [5]. When they are present, a spontaneous breathing trial (SBT) is made, after which the practitioner decides whether to extubate the patient [6]. There is, therefore, a scientific, economic, and human rationale to reduce the duration of ventilation (and sedation).

In fact, a prolonged duration of mechanical ventilation has a cost [7]. In the next decade, the need for ventilation will increase, both because of the aging of patients admitted to the ICU and global population growth [8, 9]. The availability of medical and paramedical personnel will decrease, with a risk of burnout among caregivers resulting from an increased workload [10–13].

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