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Effect of Inspiratory Muscle Training on Weaning Success in Critically Ill Intubated Patients

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1. Introduction

Approximately 50% of patients hospitalized in an intensive therapy center (ITC) rely on mechanical ventilation (MV) for an indeterminate time, depending on the reason for intubation. Once the triggering cause of acute respiratory failure (ARF) is stabilized, patients are weaned from mechanical ventilation, which is followed by extubation (Scheinhorn, Chao, & Stearn-Hassenpflug, 2000).

Weaning from MV is the gradual removal of the ventilatory prosthesis, a different process to extubation, which involves removing the endotracheal tube (MacIntyre et al., 2001). The best moment to start weaning depends on multiple factors. Premature extubation puts the patient at risk of reintubation and airway trauma. However, prolonged weaning exposes individuals to risk of hospital infection and increases hospitalization costs (C. D. Epstein et al., 2002).

Although there are a wide range of weaning protocols, there is still no consensus on which one is the best (Butler et al., 1999). In addition to this diversity, there are also so-called predictors for ventilatory interruption and successful extubation (Chatila et al., 1996; Khamiees et al., 2001; Meade et al., 2001). Among these markers are: (a) rapid shallow breathing index or Tobin index (Li et al., 2000; Meade, et al., 2001) (b) maximal inspiratory pressure (MIP) (A. Bruton, 2002; El-Khatib et al., 1996); and (c) maximal expiratory pressure (MEP) (Salam et al., 2004) .

The Tobin index is the most commonly used predictor to indicate the possibility of weaning success (Frutos-Vivar et al., 2006; Meade, et al., 2001; Stroetz & Hubmayr, 1995), although some studies did not find a significant response with respect to effective extubation (Conti et al., 2004).

Even though these and other predictors are well controlled, during the weaning process some individuals experience spontaneous breathing difficulties, thereby prolonging dependence on the mechanical ventilator (Chao & Scheinhorn, 1998).

One of the main causes of unsuccessful weaning and extubation is failure of the respiratory muscle pump, mainly inspiratory muscles (diaphragm, external intercostals, scalene,

pectoral and sterno-cleido-occipito-mastoid) (X. Capdevila et al., 1998; Cluzel et al., 2000; A. D. Martin et al., 2002; S T Venkataraman et al., 2000), which leads to prolonged dependence on MV (Vassilakopoulos & Petrof, 2004). Muscle failure is due to two factors: (1) increase in workload to which the muscle is submitted owing to carbon dioxide (CO₂) accumulation; greater airway resistance; reduced pulmonary compliance; increase in dead space or in respiratory stimulus; (2) lower neuromuscular capacity caused by phrenic nerve dysfunction; neuromuscular disorder due to dependence on MV and reduced respiratory muscle strength or endurance (Chao & Scheinhorn, 1998; Meade, et al., 2001; Spitzer et al., 1992)

This reduction in muscle strength results from systemic problems, such as kidney failure, poor glycemic control, history of prolonged corticosteroid use, sepsis, electrolytic and mineral abnormalities, in addition to the use of neuromuscular blockers, widely administered in cases of patients who are not successfully sedated (A. A. Bruton et al., 2002). To delimit this muscle weakness, the most observed symptom is dyspnea (McConnell & Romer, 2004; Nield, 1999), and the most reliable parameter for measuring this variable (inspiratory muscle strength) is MIP (Clanton et al., 2002; Green et al., 2002).

To prevent dyspnea from interfering in the weaning process and successful extubation, some studies suggest the need to increase inspiratory muscle strength (Caruso et al., 2005; Chang et al., 2005).

The effectiveness of inspiratory muscle strengthening has been reported in apparently healthy elderly (S A Cader et al., 2007), athletes (Inbar et al., 2000) and diverse pathologies, such as: restrictive pulmonary disease (Chatham, 2000); myotonic muscular dystrophy (de Freitas Fregonezi et al., 2006); Duchenne muscular dystrophy (Topin et al., 2002); cystic fibrosis (Enright et al., 2004), chronic obstructive pulmonary disease (COPD) (Sturdy et al., 2003), congestive heart failure (CAD) (Laoutaris et al., 2004), asthma (Weiner et al., 2002), sarcoidosis (Brancaleone et al., 2004), cancer (Bruera et al., 2000), spinal cord injury (SCI) (Liaw et al., 2000), tetraplegia (Uijl et al., 1999), ankylosing spondylitis (Van der Esch et al., 2004), osteoporosis (Cimen et al., 2003) and multiple sclerosis (Klefbeck et al., 2003). However, there are no studies on inspiratory muscle strengthening in critically ill intubated patients, hospitalized in an ITC.

One of the inspiratory muscle training (IMT) techniques used in intubated patients is a reduction of pressure support mechanical ventilation. However, no studies are available that have investigated on the threshold IMT instrument.

The present study assumes that, when aiming for strength, IMT is suitable for patients with weak inspiratory muscles, since it exhibits the same principles as limb strength training, namely: (1) as with skeletal muscles, respiratory muscle weakness occurs with disuse; (2) with increased demand on the respiratory system, patients are unable to maintain spontaneous breathing for long periods; and (3) perception of respiratory effort is inversely proportional to inspiratory muscle strength, that is, the lower the inspiratory muscle strength, the higher the effort perception (A. A. Bruton, et al., 2002; Campbell et al. Gandevia, Killian et al., 1980; Cluzel, et al., 2000; Yue & Cole, 1992).

Although IMT performed over a short time period is not sufficient to cause significant changes in distribution of muscle fiber types or in the transverse muscle section, a number

of mechanisms are evident: changes in the motor program and excitability of the neuromuscular system; neural adaptation, including a decrease in coactivation in the antagonist muscle group, a synchronic increase in the motor unit and a more efficient motor program (Carolan & Cafarelli, 1992; McComas, 1994; Milner-Brown et al., 1975; Sale, 1988; Yue & Cole, 1992).

Prior explanation is needed regarding mechanical ventilation and its ventilatory modes, weaning and its predictors, as well as inspiratory muscle strengthening itself in order to better understand the proposal of this chapter.

2. Mechanical ventilation

The main reason for patient admittance to the ICU is the need to install MV so as to reduce respiratory work and offer adequate oxygen support (Tobin, 2001). Esteban et al. (Esteban et al., 2000) conducted a study in eight countries with a total of 1638 patients. Indications for MV included the following: acute respiratory failure (ARF – 66%); coma (15%); COPD (13%); and neuromuscular disorders (5%). ARF primarily involved pneumonia, sepsis, surgical complications and trauma.

ARF is defined as the failure of lungs and the respiratory pump to maintain adequate gas exchange between oxygen (O₂) and CO₂. Hypoxemic ARF (type 1) consists of intrapulmonary impairment resulting in alveolar collapse, which leads to intrapulmonary shunting and inadequate arterial oxygenation. Hypercapnic ARF (type 2) is an extrapulmonary injury, resulting in the inability of the respiratory pump to sustain sufficient alveolar ventilation to eliminate metabolically produced CO₂ (Manthous et al., 1998). In this respect, the mechanical ventilator can substitute the respiratory pump until the initial disorder triggering ARF has been suitably reversed (Esteban et al., 1997; S. T. Venkataraman et al., 2000).

Chronic dependence on the mechanical ventilator is not only the main medical problem (N Ambrosino, 2005), but also an extremely uncomfortable state with important social implications (Appendini et al., 1996). Furthermore, MV is associated to greater complications, such as infection, barotrauma, cardiovascular impairment, tracheal lesion, oxygen toxicity and MV-induced pulmonary injury (Vassilakopoulos & Petrof, 2004). Some investigators have sought to determine the pathological mechanisms that lead to unsuccessful weaning in patients chronically dependent on MV (Appendini, et al., 1996).

2.1 Conventional ventilation modes

All patients using ventilatory prostheses are ventilated by the following modes: assist/control (volume or pressure) and synchronized intermittent mandatory ventilation (SIMV) with pure pressure support (PS) or pressure support ventilation (PSV) (Esteban, Anzueto, et al., 2000). Choosing the ideal ventilation mode and adequate parameters is essential to reducing respiratory muscle overload, since there must be patient-ventilator synchronism (Tobin et al., 2001).

In the assist/control mode (the most widely used), the ventilator releases a previously established current volume (volume-controlled ventilation – VCV) or inspiratory pressure

(pressure-controlled volume – PCV) to the patient, in the presence of patient effort (assist mode) or not (control mode) (Rose & Ed, 2006). Even though VCV guarantees adequate current volume, it does not limit peak pressure, which may result in altered compliance and resistance, in addition to provoking barotrauma. However, although PCV does not ensure constant current volume, it reduces the risk of pulmonary injury by determining and maintaining airway pressure and gas exchange, decreasing respiratory work and preventing hyperdistension of healthy alveoles (Esteban, Alia, et al., 2000).

SIMV with PS was introduced to provide a gradual level of assistance. This mode intersperses mandatory cycles, with pre-established respiratory frequency and current volume, as well as spontaneous cycles, in which respiratory frequency and current volume are peculiar to the patients themselves. The spontaneous cycle offers pressure support for the patient to generate adequate current volume (Leung et al. 1997).

PSV and SIMV with PS also offer ventilatory assistance, which is gradually lowered, but consists only of spontaneous cycles. PS is reduced according to the patient's respiratory frequency and current volume (Esteban, Anzueto, et al., 2000). PSV, characterized by low pressure values (7-8 cmH₂O), has been used as a weaning protocol. Kuhlen et al. (Kuhlen et al., 2003) observed that among T-piece, automatic tube compensation and PSV, the latter provided the least ventilatory overload in patients submitted to weaning, about to be disconnected from the ventilatory prosthesis. Similar data were recorded by Ezingear et al. (Ezingear et al., 2006). However, Haberthur et al. (2002) found no significant differences among the 3 protocols.

In a randomized prospective study, Matic et al. (Matic et al., 2007) observed that PSV weaning was more successful than the T-piece device in the extubation process ($p < 0.001$) and the group submitted to T-piece also spent more time on MV and in the ICU ($p < 0.001$), when compared with the PSV group.

New ventilation modes are increasingly being introduced. Recent proposals include increasing respiratory muscle rest; preventing deconditioning; improving gas exchange; avoiding pulmonary lesion and increasing patient-ventilator coordination (Tobin, 2001). Within these modalities are volume assured pressure support (VAPS), bilevel positive airway pressure (BiPAP) and airway pressure release ventilation (APRV) (Rose & Ed, 2006).

3. Weaning and extubation

Difficult-to-wean patients account for 20-25% of those mechanically ventilated (Vassilakopoulos & Petrof, 2004), and an even higher percentage of individuals with COPD (Matic, et al., 2007). Weaning commences with reduced ventilatory support, depending on the degree to which patients can ventilate themselves adequately, culminating with their being withdrawn from MV; extubation consists of removing the endotracheal tube (Meade, et al., 2001; Robriquet et al., 2006).

Weaning failure is a result of complications in the central respiratory stimulus or, more commonly, due to neuromuscular abnormalities, such as muscle fatigue and altered pulmonary mechanisms or gas exchange (Meade, et al., 2001). The ventilatory pattern of

patients that are not successfully weaned is similar to that observed during AFR (Del Rosario et al., 1997). Thus, some investigators recommend inspiratory muscle strengthening to reduce respiratory muscle fatigue (Chang, Boots, Brown, et al., 2005) and facilitate weaning from MV (Del Rosario et al., 1997).

Extubation failure occurs when there is a need to reinstitute ventilatory support between 24 and 48 hours after tube removal. Rothaar & Epstein (Rothaar & Epstein, 2003) summarize the main causes for extubation failure can be seen in chart 1.

Although the ideal weaning procedure has yet to be established, the literature reports three main techniques: SIMV, T-piece and PSV. Other less widely used methods are automatic tube compensation, continuous positive airway pressure (CPAP) and bilevel positive airway pressure - BiPAP (Esteban et al., 1995; Haberthur, et al., 2002).

MV must be interrupted as soon as the patient is capable of adequately sustaining spontaneous breathing and gas exchange (Farias et al., 2002). However, the benefits of rapid release from MV must be weighed against the risks of weaning and premature extubation (Meade, et al., 2001), since these have been associated to increased hospital costs, mortality, length of ICU stay and need for tracheostomy (Esteban, et al., 1997; Rothaar & Epstein, 2003), in addition to generating new trauma in the airways due to reintubation (Noizet et al., 2005).

1- Airway obstruction (granulous tissue, inflammation, ulceration and edema)
2- Excessive respiratory tract secretion
3- Inability to protect airways
4-Heart failure or ischemic heart disease
5- Encephalopathy
6- Respiratory failure (imbalance between respiratory muscle strength and the overload to which it is submitted)
7- Gastrointestinal bleeding
8- Sepsis
9- Need for surgery

Thus, the complex decision to extubate requires an adequate assessment of predictor accuracy in this process (S. K. Epstein, 2002).

Chart 1. Main causes of extubation failure.

3.1 Weaning and extubation predictors (Tobin index, MIP and MEP)

Several indices have been proposed to predict weaning from MV. These reflect respiratory mechanics, pulmonary gas exchange, respiratory control and respirastory muscle function (chart 2) (Alvisi et al., 2000; Hoo & Park, 2002; Meade, et al., 2001; Rothaar & Epstein, 2003). Despite the wide range of predictors, Vallverdú et al. (Vallverdu et al., 1998) considered the Tobin index, MIP and MEP as good predictors.

1- Minute ventilation (MV)
2- Vital capacity (VC)
3- Current volume (CV)
4- Respiratory rate (RR)
5- Negative inspiratory force (NIF)
6- Tobin index (RR/CV)
7- MIP and Pimax
8- Respiratory work (Wob)
9- Dead space
10- Central respiratory stimulus (P0.1)
11- CROP index (compliance, rate, oxygenation and MIP)
12- Pressure-time index
13- P0.1/Pimax ratio
14- Dynamic compliance
15- MIP/Pimax

Chart 2. Diversity of weaning and extubation predictors.

A common finding in patients who were not successfully weaned is a high Tobin index score (RR/CV, in liters). It is measured with the patient disconnected from MV, where minute volume is verified during a 1-minute period (Goldstone, 2002). Yang and Tobin (Yang & Tobin, 1991) observed that this index has a sensitivity of 0.97 and a specificity of 0.64.

Thus, the Tobin index is the most widely accepted of the weaning indices. However, it does not consider ventilatory force or endurance, important requirements for successful weaning (Vassilakopoulos & Petrof, 2004).

Although the cause of weak respiratory muscles is not entirely known, it is suggested that electrolytic disorders (hypophosphatemia and hypomagnesemia), the use of certain medications (curarizing agents) and malnutrition, combined with muscle disuse, have contributed to the occurrence of this phenomenon. The atrophy process is rapid, occurring from 7 to 10 days after a reduction in maximum diaphragmatic pressure of 50% (Anzueto et al., 1997).

MIP is the index used to measure inspiratory muscle strength (Volianitis et al., 2001). Although there is a conventional way to check this variable (Green, et al., 2002), Marini, Smith and Lamb (Marini et al., 1986) suggest a protocol that does not require patient collaboration. Some studies applying it as a predictor observed a significant difference ($p<0.05$) between patients who were successfully weaned (higher values) and those who were not (A. Bruton, 2002; C. D. Epstein, et al., 2002). Using a cutoff point of -17 cmH₂O, Conti et al. (Conti, et al., 2004) found a sensitivity of 0.95, specificity of 0.42 and accuracy of 0.71.

For an effective cough, deep inspiration and expiratory force are important, since they generate high thoracic-abdominal pressure. Based on this cough mechanism, several assisted cough techniques were developed for individuals with respiratory muscle

weakness. Although expiratory muscle strength is essential for an effective cough, inspiratory muscle strength is equally important in the inspiratory phase of a cough, capable of generating an ideal pulmonary volume (Jaeger et al., 1993). Nevertheless, there are few studies that correlate cough capacity with inspiratory muscle strength (Kang et al., 2006).

Chart 3 demonstrates the variability of MIP and Tobin index thresholds, as predictors of successful weaning and/or extubation.

PREDICTORS	VALUES	REFERENCES
Tobin index	60 ipm/l	X. J. Capdevila et al.(1995)
	96 ipm/l	Gandia & Blanco (1992)
	100 ipm/l	Vallverdu et al.(1998)
	100 ipm/l	Yang (1993)
	100 ipm/l	S. K. Epstein & Ciubotaru (1996)
	100 ipm/l	S. K. Epstein (1995)
	100 ipm/l	Afessa et al. (1999)
	100 ipm/l	Del Rosario et al.(1997)
	100 ipm/l	Dojat et al. (1996)
	100 ipm/l	Chatila et al. (1996)
	100 ipm/l	Leitch et al.(1996)
	105 ipm/l	Krieger et al. (1997)
	105 ipm/l	Mergoni et al. (1996)
	105 ipm/l	Lee, Hui, & Chang (1994)
	105 ipm/l	Yang & Tobin (1991)
MIP	15 cmH2O	Yang (Yang, 1993)
	20 cmH2O	Leitch et al. (1996)
	20 cmH2O	Del Rosario et al.(1997)
	20 cmH2O	Krieger et al.(1997)
	20 cmH2O	Afessa et al.(1999)
	25 cmH2O	Manthous et al.(1998)
	25 cmH2O	Goldwasser (1998)
	30 cmH2O	Mergoni et al. (1996)

Chart 3. Variability of MIP and Tobin index values for successful weaning and/or extubation.

4. Respiratory muscle training

Malnutrition, detraining and disuse due to prolonged bedrest may induce severe skeletal muscle dysfunction. Combined with other factors, such as reduced oxygen support, metabolic acidosis, electrolytic alterations and endocrine disorders, muscle disuse may lead to muscle atrophy (N Ambrosino, 2005; A. A. Bruton, et al., 2002). Within these factors there is predominance of isolated corticosteriod action, potentialized by neuromuscular blocking agents, immobility and sepsis (Larsson et al., 2000).

Respiratory muscles are skeletal muscles and, during prolonged mechanical ventilation (more than 48 hours), (A. Bruton, 2002; Chang, Boots, Brown, et al., 2005; Chelluri et al. 2002; Douglas et al. 2002) – inspiratory muscles move passively, favoring atrophy from disuse (Angus et al., 2005; A. A. Bruton, et al., 2002). The level of this strength reduction is related to duration of MV (Chang, Boots, Brown, et al., 2005; Powers et al., 2002; Sassoon, Caiozzo et al., 2002; Sprague & Hopkins, 2003) and has been reported as one of the major determinants of weaning failure in patients receiving MV (N Ambrosino, 2005; Gayan-Ramirez & Decramer, 2002). Anzueto et al. (Anzueto, et al., 1997) observed a transdiaphragmatic pressure reduction of 45% in rats after 11 days of controlled MV.

Thus, some studies described using IMT to increase inspiratory muscle effectiveness, in order to assist with difficult weaning. The main training techniques are: (a) endurance training using eucapnic hyperventilation (Aldrich & Karpel, 1985; Aldrich et al., 1989; Aldrich & Uhrlass, 1987; Belman, 1981); (b) threshold IMT strength training (A. D. Martin, et al., 2002; U. J. Martin et al., 2005; Sprague & Hopkins, 2003) and (c) resistance training by reducing ventilatory prosthesis sensitivity (Caruso, et al., 2005). The literature also cites other less widely used IMT methods, such as the solenoid device and the weighted plunger (Bardsley et al., 1993; Johnson et al., 1996).

Inspiratory muscle endurance can be defined as sustained MIP (SMIP), which is the ability to maintain a simple contraction over a prolonged period of time. It is a dynamic maneuver, whereas MIP is considered static or quasi-static (A. A. Bruton, et al., 2002).

SMIP is measured from residual volume and the orifice must be occluded immediately at the onset of inspiration so as to generate negative intrathoracic pressure, which is verified on the gauge (Green, et al., 2002). Inspiration must last at least 3 seconds, with maximum force and time possible. This procedure is repeated three times, and the best result is used (Cimen, et al., 2003). However, it is not easy to reproduce in mechanically ventilated patients due to lack of understanding or even interaction with the examiner. As such, an alternative for this problem is the use of an expiratory unidirectional valve since it permits free expiration, progressively reaching a volume close to the residual value, whereas for inspiration the patient must exert an effort to generate sufficient negative pressure (Caruso et al., 1999; Marini, et al., 1986).

Several factors indicate an individual should be submitted to IMT, the primary one being a simple decrease in MIP. After MIP is measured, inspiratory muscle strengthening and conditioning can be initiated (S A Cader, 2006). However, a significant problem for the inspiratory muscle training program is the protocol used. Chart 4 illustrates the range of protocols, none of which are standardized because of different medical conditions.

Although IMT of patients under MV is of short duration, some studies report that muscle strength gain is due to neural adaptation (A. D. Martin, et al., 2002; Sprague & Hopkins, 2003) rather than muscle hypertrophy. The following mechanisms emerge as a result of this adaptation: increased maximum voluntary contraction ability; decreased antagonist muscle coactivation; greater motor unit synchrony or a more efficient motor program (Carolan & Cafarelli, 1992; McComas, 1994; Milner-Brown, et al., 1975; Sale, 1988; Yue & Cole, 1992). However, further studies are needed given that the exact mechanism cannot be determined from these findings.

PROTOCOL	REFERENCES	PATHOLOGY
8W/ 3X/ 10' / 20-60%	Fregonezi et al.(Fregonezi, Resqueti, Guell, Pradas, & Casan, 2005)	Miasthenia gravis
8W/ 3X / 45'* / 80%	Enright et al.(Enright, et al., 2004)	Cystic Fibrosis
10W/ 3X/ time?/ 60%	Laoutaris et al.(Laoutaris, et al., 2004)	CHF
10W/ INT (2)/ 3s-10x/ 40-60%	Klefbeck & Hamrah Nedjad (Klefbeck & Hamrah Nedjad, 2003)	Multiple Sclerosis
8W/ 3X/ 20'-2' + 1' / 70%	Sturdy et al.(Sturdy, et al., 2003)	COPD
5W/ 5X/ 30' / 40-50%	Ramirez-Sarmiento et al. (Ramirez-Sarmiento et al., 2002)	COPD
12W/ 3X(2)/ 5' / 5-7% de increase every 2 S	de Freitas Fregonezi, Resqueti, & Casan (de Freitas Fregonezi, et al., 2006)	Myotonic muscular dystrophy
24W/ 6X/ 30' / 60-70%	Sanchez Riera et al. (Sanchez Riera et al., 2001)	COPD
6W/7x(2)/10'/30%	Topin et al. (Topin, et al., 2002)	Duchenne muscular dystrophy
16W/ 7X/ 30' / 30-60%	Covey et al. (Covey et al., 2001)	COPD
6W/ 5X/ 20' / > 40%	de Jong et al. (de Jong, van Aalderen, Kraan, Koeter, & van der Schans, 2001)	Cystic fibrosis
6W/ 7X (2)/ 15-20' / ?	Liaw et al.(Liaw, et al., 2000)	SCI
6W/ 7X (2)/ 15' / 70%	Uijl et al. (Uijl, et al., 1999)	Tetraplegia
60W/ 7X (2)/ 15' / 15%	Sudo et al.(Sudo et al., 1997)	COPD
10W/ 6X/ 30' / 30%	Lisboa et al.(Lisboa et al., 1997)	Chronic airflow limitation
8W/ 7X (3)/ 5-15' / 20%	Cahalin, Semigran, & Dee (Cahalin, Semigran, & Dee, 1997),	CHF (indication for transplantation)

W: weeks; X: times per week; (2): twice a day; (3): three times a day; %: percentage of MIP.

Chart 4. Diversity of inspiratory training protocols.

4.1 Effects of inspiratory muscle training on the weaning process in critically ill tracheostomized patients – Results of previous studies

Imbalance between increased ventilatory overload and reduced ventilatory endurance is an important determinant of MV dependence. Thus, respiratory muscle weakness is nearly always present as a contributing factor for weaning failure. Even so, there is little information on the use of the respiratory muscle training program for difficult-to-wean patients (Caruso, et al., 2005; A. D. Martin, et al., 2002; U. J. Martin, et al., 2005). Although some studies recommend IMT during MV (Chang, Boots, Brown, et al., 2005; Del Rosario, et al., 1997; Sprague & Hopkins, 2003), Chang et al. (2005) observed a post-weaning reduction in inspiratory muscle strength in patients submitted to long-term MV.

Hawkes et al. (2007) used electromyographic analysis to examine the effect of submaximal inspiratory overload on MIP and diaphragm and intercostal muscles. Results obtained 15 minutes after the test revealed an increase in MIP ($p=0.032$). During MIP, an increase was recorded in the range of diaphragm ($p=0.032$) and intercostal ($p=0.016$) muscle action ($p=0.039$). These data support the hypothesis of greater muscle strength after submaximal inspiratory effort or IMT, resulting in a reduction in perception of respiratory effort (dyspnea) (McConnell & Romer, 2004; Volianitis, et al., 2001).

A.D. Martin et al. (2002) carried out a study in which IMT was performed using threshold IMT in 10 patients (tracheostomized) dependent on MV (34 ± 33 days), in order to facilitate weaning from the ventilator by improving inspiratory muscle strength. In addition to a significant increase in strengthening workload used (7 ± 3 cmH₂O for 18 ± 7 cmH₂O, $p<0.05$) in threshold IMT, after 44 ± 43 days of IMT, 9 of 10 patients submitted to training were successfully weaned.

The effect of IMT was observed in a randomized prospective study, evidenced by reduced ventilator sensitivity and shorter weaning and reintubation time in critically ill patients (Caruso, et al., 2005). These authors observed no significant difference between training and control groups with respect to weaning time ($p=0.24$), reintubation frequency ($p=0.39$) and final MIP value ($p=0.34$). However, it is important to underscore that, in addition to the small sample size, the fact that some patients did not have an ideal level of consciousness to determine MIP may have caused its underestimation. Consequently, the load applied might have been too low. This fact, along with short training time, could have influenced the final results.

Sprague & Hopkins (2003) aimed at weaning tracheostomized MV-dependent patients by increasing inspiratory muscle strength using threshold IMT. The protocol consisted of: 6-7 days per week, once a day, 4 series of 6-8 repetitions, with a 5-10-minute rest period (under MV) between series. Training load (approximately 50% of MIP) was modified according to Borg's scale. After IMT, patients remained under spontaneous breathing via T-piece, with a gradual increase over time. Results showed a rise in training load (9.3 cmH₂O for 27.5 cmH₂O; 195%); increase in MIP (22.0 to 54.0 cmH₂O; 140%); and successful weaning in all patients over a period of 9 to 28 days.

Frutos-Vivar et al. (2006) observed that pneumonia, as the initial trigger of AFR (with consequent intubation), is one of the primary causes of intubation. Based on this information, the study conducted by Kang et al. (2006) is very important to the present investigation, since these authors aimed to assess the relationship between inspiratory muscle strength and cough capacity, but in patients with SCI. They obtained intermediate correlation between VC and voluntary cough capacity ($r=0.583$; $p<0.001$), intermediate-high between VC and MIP ($r=0.749$; $p<0.001$) and intermediate between VC and MEP ($r=0.438$; $p<0.001$); MIP also exhibited more significant correlations ($p<0.001$, with intermediate to intermediate-high correlations) with peak cough flow compared to MEP ($p<0.05$, with low-intermediate and intermediate correlations).

Another investigation, conducted in patients with spinal cord injury, underscored the importance of respiratory muscle training, increasing endurance and strength, in order to wean these individuals (Gutierrez et al., 2003). Comparative pre- and post-training data

demonstrated a significant rise in MIP ($p<0.001$; mean of -18 cmH₂O), MEP ($p<0.001$; mean of 21.6 cmH₂O) and VC ($p<0.001$; mean of 340 cc) in patients with lower lesions, favoring their weaning.

Few studies have assessed the magnitude and extent of generalized muscle weakness in patients with prolonged MV dependence. In a retrospective analysis of 49 patients submitted to whole-body rehabilitation (with strength and resistance training of upper (UULL) and lower limbs (LLLL), inspiratory muscles with threshold IMT, as well as the torso and gait), U. J. Martin et al. (2005) recorded a significant response ($p<0.001$) in functional status and inspiratory muscle, UULL and LLLL strength, in addition to a significant ($r=0.720$; $p<0.001$) and inverse correlation between UULL strength at admission and weaning time.

Additional research, conducted in patients with no artificial airway, is also relevant for the present study (Baker et al., 2003; Mathers-Schmidt & Brilla, 2005; Sapienza et al., 1999). Some post-extubation impairments occur in the upper airways (UAW), with only glottic edema and vocal cord lesions causing stridor. These alterations restrict air flow in the upper airways, resulting in dyspnea and eventual reintubation (Esteban, et al., 1997). Baker et al. (Baker, et al., 2003) studied in patients with upper airway limitation (glottis) resulting from bilateral vocal cord paralysis (abductors). The authors aimed to determine whether inspiratory muscle strengthening increases inspiratory muscle ability, generating greater MIP and reducing dyspnea sensation during exercise and speech. Threshold IMT was used for training, with the following protocol: load of 75% of MIP, 5 times per week, 5 series of 5 repetitions, for 5 weeks. Results revealed an increase of 47% in MIP, reduced dyspnea during exercise and speech of 2-4 points on Borg's scale and improved airway opening (glottis), visualized by endoscopy.

In a study performed with healthy individuals, nuclear magnetic resonance showed acute and chronic responses of the UAWs to ITM (How et al., 2007). Results demonstrated significant alterations in the experimental group, as follows: increase in UAW dilator muscles (genioglossus and geniohyoid) ($p<0.001$); increase in transverse section area ($p=0.025$) and lateral diameter ($p=0.003$) of the UAWs; increase in nasopharynx ($p<0.001$) and laryngopharynx ($p=0.031$).

Another advantage of IMT was the reduction in central respiratory stimulus ($P_{0.1}$) (Huang et al., 2003). In this investigation, the intervention was performed in 23 apparently healthy individuals (28.8 ± 2.3 years). The protocol used an increasing inspiratory load of 30% to 75% of MIP; 5 days a week; 4 series of 6 repetitions; for 4 weeks. The significant increase in MIP ($p<0.001$) had a significant negative correlation ($p<0.01$) with the significant reduction in $P_{0.1}$ ($p<0.001$). These data suggest that once $P_{0.1}$ is reduced, the occurrence of dyspnea decreases (N. Ambrosino et al., 1994; Ferrari et al., 1997).

4.2 Effects of inspiratory muscle training on the weaning process of critically ill intubated patients – Results of a pioneering study

Cader et al. (S. A. Cader et al., 2010) investigated elderly patients to determine whether inspiratory muscle training improved maximal inspiratory pressure, breathing pattern and length of weaning time from mechanical ventilation. Of the 198 patients screened during the recruitment, the 67 eligible individuals were monitored daily to assess readiness to start

weaning. Twenty of the subjects were tracheostomized, 5 died and 1 was transferred to another center before the start of weaning. The remaining 41 were randomized: 21 to the experimental group and 20 to the control group. Four participants in each group died before extubation. Three participants in the experimental group and two in the control group were tracheostomized before extubation.

Group data for all outcomes at the start of weaning and at extubation for the experimental and control groups are presented in Table 1. Maximal inspiratory pressure increased significantly more in the treatment group than in the control group (MD 7 cmH₂O, 95% CI 4 to 10). The Tobin index decreased significantly more in the treatment group (73.6 br/min/l to 81.5 br/min/l) than among controls (81.5 br/min/l to 95.9 br/min/l). In those who did not die or receive a tracheostomy, time to weaning was significantly shorter in the treatment group than in the control group (MD 2 days, 95% CI 0 to 3).

Among uncensored participants, inspiratory muscle training significantly reduced the spontaneous ventilation (ie, weaning) period. Inspiratory muscle training shortened weaning time by 1.7 days (95% CI 0.4 to 3.0), as shown in Table 2.

Outcome	Groups				Within-group difference		Between-group difference
	Pre-test		Post-test		Post-test minus pre-test		Post-test minus pre-test
	Exp (n = 14)	Con (n = 14)	Exp (n = 14)	Con (n = 14)	Exp	Con	Exp minus Con
MIP (cmH ₂ O)	15.1 (2.6)	15.3 (2.2)	25.0 (3.9)	17.6 (1.9)	9.9 (2.5)	2.3 (2.1)	7.6 (5.8 to 9.4)
TI (br/min/L)	73.6 (8.8)	81.5 (6.9)	79.7 (11.2)	95.9 (12.4)	6.1 (3.6)	14.4 (9.1)	-8.3 (-13.7 to -2.9)

TI = Tobin index (ipm/l); MIP = maximal inspiratory pressure (cmH₂O); Exp = experimental group; Con = control

Table 1. Mean (SD) outcomes for each group, mean (SD) difference within groups and mean difference (95% CI) between groups.

Outcome	Groups		Mean inter-group difference
	Exp (n=14)	Con (n=14)	Exp minus Con
Total ventilation period	10.4 (4.0)	11.0 (3.5)	0.6 (-3.5 to 2.4)
Spontaneous ventilation period	3.6 (1.5)	5.3 (1.9)	1.7 (0.4 to 3.0)

Exp = experimental group, Con = control group

Table 2. Mean (SD) duration (days) of total ventilation and spontaneous ventilation periods among uncensored participants.

The receiver operating characteristic curve (sensitivity and specificity) was used to evaluate weaning predictors and extubation success. Beginning with the Tobin index, the receiver operating characteristic curve reveals an area below the curve of 0.877 (SD 0.06). Sensitivity and specificity of the Tobin index was 1.00 and 0.36, respectively. Maximum inspiratory pressure was 0.86 for sensitivity and 0.72 for specificity. The area below the receiver operating characteristic curve shows a value of 0.845 (SD 0.07).

Thus, it was concluded that inspiratory muscle training in the experimental group contributed to a significant increase in maximum inspiratory pressure (between and within groups) and a reduction in the Tobin index (within groups). These are considered good weaning predictors, which is consistent with our finding that inspiratory muscle training significantly reduces the weaning period in patients who did not die or receive a tracheostomy.

5. Conclusions

This chapter presented an important alternative for inspiratory muscle training in critically ill patients. The technique aims at shortening weaning time and increasing extubation success rates.

IMT was found to improve some weaning predictors such as TI, MIP and MEP, favoring a reduction in weaning time. This outcome is essential in the ICU, since it decreases physiological alterations and complications from ventilator use, such as barotrauma, tracheal lesion, sarcopenia, repeated infections, scabs and surgical invasions. These findings are relevant because they contribute to shorter hospital stays and lower costs, resulting in fewer social repercussions and enhanced pre-and post-hospitalization quality of life.

Recommendations of the present study are divided into two categories: applicability and continuity.

With respect to continuity, the authors recommend that randomized controlled studies be carried out to compare IMT using threshold IMT with other interventions, such as reduced sensitivity and mechanical ventilator support pressure in intubated subjects. We further suggest that prospective studies be applied to assess the effects of training and its continued use in hospital patients, following up these individuals after discharge from the intensive therapy center (ITC) to a semi-intensive unit, infirmary or room.

In relation to the applicability of the present study, it is hoped that the knowledge acquired will be made available to physical therapists so they may intervene as early as possible, thereby avoiding future complications previously experienced in the ICU, mainly when elderly patients are involved. It is suggested that this intervention (IMT) be implemented in ITCs in order to reduce the number of tracheostomies, increase extubation success rates and decrease hospital stays, all of which will improve patient well-being.

6. References

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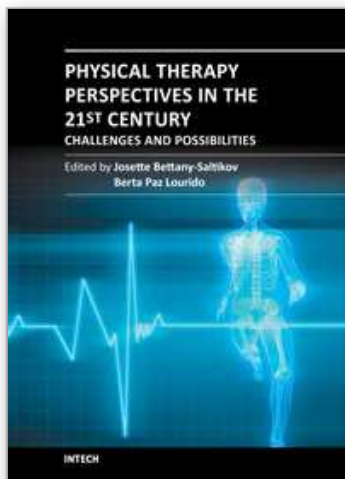
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