



Perspectives on prolonged nocturnal invasive mechanical ventilation

Robert L Vender

DOI: <https://doi.org/10.53097/JMV10126>

Cite: Vender RL. Perspectives on prolonged nocturnal invasive mechanical ventilation. J Mech Vent 2025; 6(2):80-86.

Abstract

The utilization of invasive mechanical ventilation (IMV) has become common practice for a large and expanding number of patients with chronic respiratory failure resultant from several medical diseases and disorders. Although protocols and guidelines for the acute management of respiratory failure exist with expansive evidence-based data, the chronic management of similar patients outside the Intensive Care Unit (ICU) setting requiring extended and prolonged durations on invasive mechanical ventilation (IMV) has been much less clearly defined. In addition, the vast majority of such care is administered outside the acute hospital environment. Most adults requiring chronic mechanical ventilatory support include two general populations: 1) patients with severe intrinsic lung disease who have experienced acute respiratory failure initially managed in ICU settings and 2) patients with progressive neuromuscular diseases. When attempts at weaning, ventilator liberation, and achievement of successful full-time continuous spontaneous ventilation fail, mechanical ventilation management often reverts to part-time (predominately nighttime) nocturnal invasive mechanical ventilation (NIMV) with periods of spontaneous breathing duration awake daytime hours. Such a practice pattern has been advocated by peer-reviewed published expert opinions. Yet such a pattern would appear to potentially exclude many patients from eventual total weaning, ventilator liberation and even decannulation and progression to independent sustained life-long spontaneous ventilation. In addition, such a ventilator management program may not even be indicated for patients with progressive neuromuscular disorders in the absence of an acute concomitant lung disease. The predominate physiological factors prohibiting sustained spontaneous ventilation would appear to be 1) inspiratory muscle (predominantly diaphragmatic) fatigue, which per definition should be correctable with appropriate management including the concept of ventilatory muscle rest (VMR) and/or 2) inspiratory muscle weakness resultant from a pathological disease entity. However, the elusive nature of defining, appropriately monitoring, and subsequent therapy of diaphragmatic fatigue clearly hinders the practical management of this large volume of patents thus rendering them “Ventilator Dependent”. In addition, the absence of disease modifying therapies for chronic progressive neuromuscular disorders contributes to the eventual outcome of fulminant respiratory failure. The purpose of this narrative review is not to challenge but rather to attempt to validate or not validate the concept that nocturnal invasive mechanical ventilation (NIMV) is a “valid” goal of IMV on a chronic long-term basis.

Keywords: Prolonged mechanical ventilation, Nocturnal mechanical ventilation

Authors

1. MD, Professor of Medicine. Division of Pulmonary, Allergy, and Critical Care Medicine. Penn State Health Milton S, Hershey Medical Center. Hershey, Pennsylvania, USA.

Corresponding author: rvender@pennstatehealth.psu.edu

Conflict of interest/Disclosures: None

Introduction

The application and utilization of various modalities of assisted ventilation on a chronic basis (outside their application during intensive care unit (ICU) and inpatient hospital care) has rapidly expanded over the last decades. Ventilatory assist devices are now routinely utilized in the care of a wide variety of chronic patient diseases and disorders including: 1) intrinsic lung diseases such as cystic fibrosis (CF), chronic obstructive pulmonary disease (COPD), interstitial lung diseases (ILDs), 2) neuromuscular disorders such as muscular dystrophy (MD), amyotrophic lateral sclerosis (ALS), multiple sclerosis (MS), or myasthenia gravis (MG), 3) abnormalities of the chest wall such as kyphoscoliosis (KS) or ankylosing spondylitis, 4) diseases causing diaphragm dysfunction such as cervical spinal cord injury, diaphragmatic paralysis due to phrenic nerve injury, 5) diseases of central respiratory drive most notable for central congenital hypoventilation syndrome (CCHS) or obesity hypoventilation syndrome (OHS), 6) a variety of diseases that alter overall mentation, alertness, functionality, and mobility such as cerebral palsy (CP), traumatic brain injury (TBI), stroke or intracerebral hemorrhage (ICH), “locked-in syndrome”, and 7) disease causing general loss of muscle function, strength and endurance such as terminal cancer, cachexia, emaciation, and critical illness weakness.

The various chronic long-term ventilatory assist modalities are generally simplified under two classifications: 1) non-invasive ventilation (NIV) using nasal, mouth or combination interfaces and 2) invasive mechanical ventilation (IMV) via tracheostomy interface. The more intensive requiring modalities of respiratory support can now be delivered in a variety of medical settings outside of the acute care hospitals including specialized weaning units, long term acute care hospitals, specialized nursing facilities, or often at home. Patients requiring these services can range in age from newborns to the elderly. Ventilatory assistance can range from hours to full-time continuous ventilatory support depending upon the uniqueness of each disease and each individual patient. In relation to the ventilator management of acute hypoxemic respiratory failure and the non-invasive ventilatory support for chronic neuromuscular diseases, various guidelines exist in attempts to provide consensus and standardization to clinical care practices, but individualized care requires the knowledge and expertise of care providers who can identify and adapt to these various clinical situations in multiple clinical care settings on a regular and timely basis.¹⁻⁴ In general, it appears that for clinical scenarios of chronic long-term non-invasive ventilation (NIV) that the preferred first option is early

initiation prophylactically prior to hypercapnia or overt respiratory failure and usually initiating non-invasive ventilation (NIV) only at nighttime (nocturnal) support with the latter transition to invasive mechanical ventilation (IMV) as per disease course and progression.^{3,4} Yet, there does not exist the same consensus as to the strategy and duration of invasive mechanical ventilation (IMV) via surgically inserted tracheostomy tube interface for chronic prolonged ventilatory support especially during the night (nocturnal), regardless of disease entity or clinical scenario.

In the following narrative review the primary focus will be a discussion of the scientific and clinical data to validate current clinical practice patterns for patients receiving invasive mechanical ventilation (IMV) on an intermittent basis and predominately at night (nocturnal) in adults, i.e. nocturnal invasive mechanical ventilation (NIMV) on a chronic long-term basis and not as therapy for acute onset respiratory diseases. The purpose of this review is not to challenge but rather to attempt to validate or not validate the concept that nocturnal invasive mechanical ventilation (NIMV) is indeed a “valid” goal of IMV on a chronic long-term basis which by definition would then seem to potentially exclude a significant number of patients from efforts of full -time liberation and possible de-cannulation. Acknowledging consensus conference statements, even the concept of nocturnal invasive mechanical ventilation (NIMV) as a transition to potential full liberation seems to lack objective evidence-based support.^{5,6}

This review is not intended to discuss various preventive strategies and/or potentially efficacious therapies and/or interventions to assist in the successful weaning and subsequent liberation from invasive mechanical ventilation (IMV) for patients requiring prolonged mechanical ventilation such as 1) concept of lung-and-diaphragm protective ventilation, 2) mobilization and physical therapy, 3) inspiratory muscle training, or 4) phrenic nerve or diaphragmatic pacing.⁷⁻¹¹

One keynote is the awareness of the differences of the timing of and/or pathway to the initiation of mechanical modalities between nocturnal support for non-invasive (NNIV) and nocturnal support for invasive ventilation (NIMV) that being: 1) in general non-invasive ventilation (NIV) is initiated when ventilatory capacity is compromised but still adequate (often associated with normocapnia or mild hypercapnia) for spontaneous ventilation but not failing so as to result in overt respiratory failure and 2) the transition to nocturnal invasive mechanical ventilation (NIMV) is often in the reverse direction in comparison to guidelines for NIV. Nocturnal invasive mechanical ventilation (NIMV) is

usually initiated after temporary full-time invasive mechanical ventilation (IMV) has been first started as therapy for diseases such as acute respiratory distress syndrome (ARDS) or acute exacerbation of chronic obstructive pulmonary disease (COPD) with intent to successfully treat the primary underlying disease process and eventually achieving full-time liberation from invasive mechanical ventilation (IMV).

Review of the medical literature would appear to stratify the potential justification and clinical indications of nocturnal-only invasive mechanical ventilation (NIMV) into two distinct disease populations: 1) intrinsic lung diseases of high-level severity and 2) diseases affecting the respiratory muscles predominately the diaphragm.¹²⁻¹⁵ A second keynote is the recognition that the mechanisms of chronic respiratory failure between these two disease categories is vastly different, i.e. inspiratory muscle fatigue due to work overload contrasted with diaphragmatic weakness related to pathological disease processes. Thus, it is irrational to think that respiratory systems management would be similar in these two distinct populations of patients requiring some modality and some duration of chronic mechanical ventilatory support.

Prevalence Chronic Nocturnal Invasive Mechanical Ventilation

In a review of 212 eligible patients requiring prolonged mechanical ventilation (PMV) related solely to intrinsic lung diseases without any concomitant neuromuscular disorders: 1) 127 (60%) achieved eventual complete ventilator independence, 2) 13 (6%) achieved nocturnal ventilation only via tracheostomy, 3) 2 (1%) transitioned to non-invasive nocturnal ventilatory support only, 4) 4 (2%) required full nocturnal and partial daytime daily invasive mechanical ventilation, 5) 28 (13%) continued to require full time 24 hours per day invasive mechanical ventilatory dependency, and 6) 38 (18%) died during the study period.¹²

A second review of 259 patients with prolonged mechanical ventilation (PMV) attributed solely to COPD noted that only 24% were able to return to spontaneous ventilation but the majority (76%) required nocturnal invasive mechanical ventilation (NIMV) as the predominant or even exclusive form of ventilation support with nighttime durations measured as 9.9 ± 2.5 hours and 10.7 ± 1.4 hours.¹³ A third review of a series 1414 non-neuromuscular diseased patients with prolonged mechanical ventilation (PMV) reported: 1) 766 (54%) were successfully weaned to full-time spontaneous ventilation, 2) 353 (25%) died and 3) 295 (21%)

continued be ventilator dependent; of these 295 ventilator dependent subjects 232 (16%) required full time invasive mechanical ventilation (IMV) with tracheostomy, 53 (4%) part-time or nocturnal IMV with tracheostomy, and 10 (< 1%) received non-invasive pressure support).¹⁴ Review of these three studies in cohorts of patients with chronic respiratory failure secondary to intrinsic lung disease reported wide variation as to rates of continuous long-term nocturnal invasive mechanical ventilation (NIMV); 21%-76% requiring some duration of nocturnal invasive mechanical ventilation (NIMV) and a significant proportion requiring NIMV only as the predominate chronic ventilatory assist modality. Similarly, in a cohort of 19 adult patients with a variety of neuromuscular diseases results demonstrated that 1) 9 required part-time nocturnal non-invasive ventilation (NNIV) but 2) 10 required invasive mechanical ventilation (IMV) via tracheostomy with 6 part-time nocturnal and 4 full time.¹⁵

These reports clearly demonstrate the routine standard practice that in many cases, providers revert to only nocturnal nighttime invasive mechanical ventilation (NIMV) as the sole mechanical ventilatory support modality and have led to multiple expert opinions or organizational statements to make the recommendations: 1) nocturnal ventilation (invasive or noninvasive) is a “modified goal” distinct from successful liberation⁵ and “weaning to just nocturnal ventilation should also be considered”.⁶ However, review of the literature does not provide any consensus nor standardization as to 1) indicators for nocturnal invasive mechanical ventilation (NIMV) compared to other modalities, 2) optimal duration of invasive nocturnal ventilation, 3) objective goals or outcomes to support part-time ventilatory support modalities, nor 4) importantly specific patient characteristics to justify and validate these judgments. Furthermore, these recommendations would appear to deny the possibility of potential full-time liberation from any sort of ventilatory support in a large number of patients (i.e. intrinsic lung disease and/or chronic respiratory muscle fatigue) and also to risk the development of potentially abrupt and fatal respiratory failure in patients with clearly evident progressive pathological diaphragmatic failure.

Potential Benefits of Nocturnal Invasive Mechanical Ventilation (NIMV)

Acknowledging the life-sustaining benefit of any modality of ventilatory support for a defined period of time, this therapy should not in itself deny patients the potential for full weanability and liberation from invasive mechanical ventilation and even the possibility of decannulation without

defined objective supporting clinical and scientific data. The purported benefits of both nocturnal non-invasive ventilation (NNIV) and/or nocturnal invasive mechanical ventilation (NIMV) are multiple and include: 1) respiratory ventilatory muscle rest (VMR), 2) resetting and/or maintaining the carbon dioxide (CO₂) sensitivity in central nervous system breathing centers closer to normal values, and 3) improvement in lung mechanics and overall gas exchange.¹⁶

Concept of Ventilatory Muscle Rest (VMR)

The utilization of nocturnal invasive mechanical ventilation (NIMV) uniquely mandates full mechanical ventilatory support as the primary predominate clinical benefit with correction of all three factors. However, the first noted clinical benefit would appear to be most tenable in the achievement of the goal of eventual full-time liberation from invasive mechanical ventilation (IMV). The rationale supporting the potential benefit of nocturnal invasive mechanical ventilation (NIMV) for patients with intrinsic lung disease appears to be based upon the concept of prevention of inspiratory muscle fatigue, specifically the diaphragm, given elevations in work of breathing and strain overload. Skeletal muscle fatigue is defined as the loss in muscle capacity to develop force or shorten resulting from muscle fiber activity under a load that is reversible at rest.¹⁷

Muscle fatigue can be defined as the loss of contractile function-force, velocity, power, or work caused by prolonged exercise and/or excessive loads and reversible by rest.¹⁷ Thus, given the definition of skeletal muscle fatigue that is correctable by rest, then theoretically “resting” the diaphragm might assist in allowing more efficient daytime spontaneous ventilation without overt muscle fatigue and resultant respiratory failure, i.e. concept of ventilatory muscle rest (VMR). If respiratory muscle fatigue is indeed of clinical importance, ventilatory muscle rest therapy should be beneficial to patients not only intermittently but also chronically to achieve full-time continuous spontaneous ventilation. Patients at high risk for respiratory muscle fatigue include: 1) patients with chronic respiratory loads such as severe COPD, and 2) patients facing a sudden increase in respiratory load such as acute asthma or acute respiratory distress syndrome (ARDS). Yet multiple reviews have termed the evaluation and identification of respiratory muscle fatigue as elusive and problematic.

The concept of correctable ventilatory muscle fatigue has not been proven in clinical practice during periods with stable but severe lung disease even in relation to patients with

chronic hypercapnia. In addition, studies designed to directly test the hypothesis of “ventilatory muscle rest” (VMR) have failed to identify improvements in clinical or laboratory parameters. One study that evaluated 5 male subjects with severe COPD (FEV₁ = 0.68 ± 0.08L [24 ± 2% predicted]) utilized 4 weeks of daily “in-hospital-supervised” negative pressure ventilation (4 hours per day) and demonstrated a 16 ± 4% increase in maximal sustainable ventilation over a 12 minute testing period compared to pre-intervention values (4.0 ± 1.3 L/min [P < 0.05]).¹⁸ However negative pressure ventilation under these specific study applications elicited no improvement in any clinical or laboratory parameters. Investigators at that time identified potential limitations to the interpretation of their study that remain relevant even today including; 1) the absence of definitive criteria to establish that a ventilatory muscle is indeed at rest, 2) accurate clinical definition and measurement of fatigue, 3) correlation with clinical outcomes, and 4) duration, extent and modalities to achieve and sustain ventilatory muscle rest (VMR) that would eventually result in sustained prolonged spontaneous ventilation. Limitations in the current state of knowledge in relation to the definition, diagnosis, objective measurement, monitoring and treatment and/or resolution of inspiratory ventilatory muscle fatigue remain major barriers to the successful weaning and liberation for patients requiring any modality of chronic invasive mechanical ventilatory support, including nocturnal invasive mechanical ventilation (NIMV).

Conversely, three decades later one of these same investigators conclusively demonstrated that total diaphragmatic inactivity (i.e. paralysis) during acute illness results in diaphragmatic myofibril damage and disuse atrophy even after relatively short periods of absent neurological activity (18 - 69 hours).¹⁹ Such disuse atrophy of the diaphragm and other skeletal muscles during periods of acute catabolic illness and prolonged immobilization have been previously reported and purported as a major limitation to successful liberation from invasive mechanical ventilation (IMV).⁵ In fact, recent studies including a randomized clinical trial attempted to assess the potential clinical benefit of continuous diaphragm neural stimulation at basal levels to maintain some degree of muscle tone without the development of dis-use atrophy.^{10,20} These studies of acute critically ill patients although not analogous to chronic stable outpatients again raise the issue of ventilatory muscle rest [VMR] (dependent upon the degree of ventilatory support) potentially worsening diaphragm strength and endurance which is exactly opposite to the goal of nocturnal nighttime invasive ventilation (NIMV).

In contrast to the hypothetical association of ventilatory muscle rest (VMR) to assist in minimization of fatigue in states of diaphragm work overload in patients with severe lung diseases (most commonly COPD), this same rationale or hypothesis would appear less tenable in patients with defined pathological neuromuscular diseases where the actual muscle dysfunction is resultant from weakness (per definition not recoverable by rest) and not fatigue. Skeletal muscle weakness is defined as the impairment in the capacity of a fully rested muscle to generate force.^{21,22} Muscle fatigue tends to result from stress, overuse, or metabolic factors whereas muscle weakness tends to result from pathological neuromuscular diseases. In fact, there does not appear to be any reported randomized controlled clinical trial (RCT) to date that conclusively demonstrated improved respiratory muscle function in patients with progressive neuromuscular disease once progressed to tracheostomy and initiation of invasive mechanical ventilation. Thus, the goal of initiation of invasive mechanical ventilation (IMV) with only nocturnal support in patients with progressively worsening neuromuscular diseases and respiratory muscle weakness such as muscular dystrophy (MD) and amyotrophic lateral sclerosis (ALS) would not appear to have any evidence-based nor theoretical disease-based justification; acknowledging reports of successful liberation of invasive mechanical ventilation (IMV) with decannulation in patients with chronic and long-term cervical cord traumatic injury.²³

Concept of Central Nervous System Carbon Dioxide (CO₂) Chemosensitivity

In relation to the potential benefit of nocturnal invasive mechanical ventilation (NIMV) as a mechanism to preserve central CO₂ chemosensitivity to respiratory drive, note that central respiratory control and rhythmicity are regulated by neurons in the pons and medulla. During wakefulness there are additional inputs to breathing control but during sleep these compensatory wakefulness mechanisms are either reduced or absent which predisposes to control of breathing problems during sleep. Thus, during sleep chemical control of breathing predominates as the sole regulatory mechanism.

Typically, during sleep minute ventilation is reduced and minor elevations in arterial partial pressure of carbon dioxide (PaCO₂) can occur when compared to wakefulness.²⁴ For individuals receiving nocturnal invasive nighttime mechanical ventilation (NIMV) there does exist the potential for reduced sensitivity or increased CO₂ thresholds for breathing regulation as contributing factors to hypercapnia and potential failure to liberate from nighttime support. In addition, there exists the potential for mechanical ventilation preservation of normal levels of PaCO₂ will then translate

into improved wakefulness and preserved sensitivity to CO₂ to maintain normocapnia. However, in patients with severe COPD, multiple studies have documented increased central respiratory drive and not suppression in response to the mechanically induced increased work of breathing related to airflow obstruction, lung air-trapping and chest wall hyperinflation with any elevations in PaCO₂ even during wakefulness attributed to increased lung dead space fraction and not reductions in minute ventilation nor tidal volume.^{25,26}

However, similar to the sparse information in relation to inspiratory muscle fatigue, from a clinical perspective validation and proof of this hypothetical concept of central nervous system preserved chemosensitivity also remains absent and elusive. Thus, in the absence of concomitant diagnoses of obstructive sleep apnea, obesity hypoventilation syndrome, or drug induced sedation the beneficial effects of nocturnal invasive mechanical ventilation (NIMV) as a mechanism to preserve central respiratory drive would not be validated, again supporting the primary clinical and physiological benefit of NIMV being inspiratory ventilatory muscle rest (VMR).

Conclusion

In specific relation of individualized care, each patient must first be assessed as to the primary focus necessitating the requirement for invasive mechanical ventilation (IMV) and just as importantly per the specific goal of this therapy. For awake, alert, daytime ambulatory patients with severe intrinsic pulmonary airway or parenchymal lung disease such as COPD, they should be involved intricately in terms of their individual goals which may include lifetime continuation of nocturnal invasive mechanical ventilation (NIMV) or conversely their desire for full liberation. This same goal may not be applicable to patients with severe neurological impairment such as cerebral palsy (CP) with reduced cognitive function/awareness and no locomotor ambulation whereby the goals may be more directed to family members, care provides, resources, or even financial. However, regardless of specific disease, regardless of clinical and social circumstances, regardless of each individual patient uniqueness, the expectation is that all decisions are based upon objective validated information without decisions being made based upon personal judgments or even biases. Acknowledging the potential utility of electromyographic power spectrum analysis of the diaphragm and twitch transdiaphragmatic pressure measurements through phrenic nerve stimulation to diagnoses inspiratory muscle fatigue, the requisite high level technology and expertise limits these assessments to a very

few selective academic medical centers which are well beyond the capabilities of most practitioners and medical facilities where the vast majority of individuals requiring prolonged invasive mechanical ventilation actually receive their care, at times including the home environment.²⁷⁻²⁹

Thus, from all practical perspectives, respiratory muscle fatigue cannot be adequately diagnosed by muscle directed studies in routine clinical practice, the clinical development of “task failure” could represent a potential surrogate indicator. Indeed, task failure is often defined clinically by criteria such as increased respiratory rate, decreased oxygen saturation, perception of increased dyspnea or perception of increased work of breathing but specifically do not reflect direct measurements of respiratory muscle function per se and thus remain questionable indicators of failure to wean; nor does task failure provide any insight scientifically, clinically or therapeutically as to specific mechanism accounting for respiratory failure.

With the anticipation of the continued expansion and increased numbers of patients being prescribed some modality of mechanically assisted ventilation, efforts to better define scientifically respiratory muscle fatigue, to better diagnose respiratory muscle fatigue clinically, to develop actual specific therapies and treatments to correct and improve respiratory muscle fatigue would seem paramount rather than relegating such important patient impacting decisions to subjective speculation or with reliance upon indirect assessments, often impacted by non-patient specific directed factors. Thus based upon currently available up-to-date clinical and scientific information, there does not appear to be any direct support for 1) relegation of patients with severe intrinsic lung diseases to life-long chronic nocturnal invasive mechanical ventilation (NIMV) without daily efforts towards the goal of full liberation and potential decannulation, and also 2) the initiation of part-time nocturnal invasive mechanical ventilation (NIMV) for any patients with neuromuscular weakness in deference to the acceptance of chronic long-term full invasive mechanical ventilation (IVM).

References

1. Fan E, Del Sorbo L, Goligher EC, et al. An official American Thoracic Society/European Society of Intensive Care/Society of Critical Care Medicine Clinical Practice Guideline: Mechanical Ventilation in Adult Patients with Acute Respiratory Distress Syndrome. *Am J Respir Crit Care Med* 2017; 195(6):1253-1263.
2. Lee KG, Roca O, Casey JD, et al. When to intubate in acute hypoxaemic respiratory failure? options and opportunities for evidence-informed decision making in the intensive care unit. *Lancet Respir Med* 2024; 12:642-654.
3. Miller RG, Jackson CE, Kasarskis EJ, et al. Quality standards subcommittee of the American College of Neurology. Practice parameter update: The care of the patient with amyotrophic lateral sclerosis, drug, nutritional, and respiratory therapies (an evidence-based review): Report of the quality standards subcommittee of the American College of Neurology. *Neurology* 2009; 73(15):1218-1226.
4. Hansen-Flaschen J, Ackrivo J. Practical guide to management of long-term noninvasive ventilation for adults with chronic neuromuscular disease. *Respir Care* 2023; 68(8):1123-1157.
5. MacIntyre NR, Epstein SK, Carson S, et al. Management of patients requiring prolonged mechanical ventilation. report of a NAMDRC consensus conference. *Chest* 2005; 128:3937-3954.
6. Make BJ, Hill NS, Goldberg AI, et al. Mechanical ventilation beyond the intensive care unit: Report of a consensus conference of the American College of Chest Physicians. *Chest* 1998; 113 Supplement 5:289S-344S.
7. Goligher EC, Dres M, Patel BK. Lung- and diaphragm - protective ventilation. *Am J Respir Crit Care Med* 2020; 202(7):950- 961.
8. Vender RL. The Diaphragm: The forgotten factor in ventilator weaning. *The Journal of Lancaster General Hospital* 2021; 12:13-16.
9. Vorona S, Sabatini U, Al-Maqbali S, et al. Inspiratory muscle rehabilitation in critically ill adults: A systematic review and meta-analysis. *Ann Am Thorac Soc* 2018; 15(6):735-744.
10. Dres M, Gama de Abreu M, Merdji H, et al. Randomized clinical study of temporary transvenous phrenic nerve stimulation in difficult-to-wean patients. *Am J Respir Crit Care Med* 2022; 205(10):1169-1178.
11. Van Hollebeke M, Poddighe D, Hoffman M, et al. Similar weaning success rate with high-intensity and sham inspiratory muscle training. *Am J Respir Crit Care Med* 2025; 211(3):381-390.

12. Dasgupta A, Rice R, Mascha E, et al. Four-year experience with a unit for long-term ventilation (Respiratory Special Care Unit) at the Cleveland Clinic Foundation. *Chest* 1999; 116:447-455.
13. Muir J-F, Girault C, Cardinaud J-P, et al, and the French Cooperative Study Group. Survival and long-term follow-up of tracheostomized patients with COPD Treated by Home Mechanical Ventilation. A multicenter french study in 259 patients. *Chest* 1994; 106:201-209.
14. Scheinhorn DJ, Hassenpflug MS, Votto JJ, et al. Post-ICU mechanical ventilation at 23 long-term care hospitals: A multicenter outcomes study. *Chest* 2007; 131:85-93.
15. Dale CM, King J, Nonoyama M, et al. Transitions to home mechanical ventilation: The experience of Canadian ventilator assisted adults and their family caregivers. *Ann Am Thorac Soc* 2018; 15:357-364.
16. Windisch W. Home mechanical ventilation. In Martin J. Tobin, editor. *Principles and Practice of Mechanical ventilation- Third Edition*, New York: McGraw-Hill Medical 2013:683-698.
17. Respiratory muscle fatigue. Report of the Respiratory Muscle Fatigue Workshop Group. *Am Rev Respir Dis* 1990; 142:474-480.
18. Levine S, Levy SF, Henson DJ. Effect of negative pressure ventilation on ventilatory muscle endurance in patients with severe chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1992; 146:722-729.
19. Levine S, Nguyen T, Taylor N, et al. Rapid disuse atrophy of diaphragm fibers in mechanically ventilated humans. *N Engl J Med* 2008; 358:1327-1335.
20. Reynolds SC, Meyyappan R, Thakkar V, et al. Mitigation of ventilator-induced diaphragm atrophy by transvenous phrenic nerve stimulation. *Am J Respir Crit Care Med* 2017; 195:339-346.
21. Kelsen SG, Criner, G.J. Pump failure: The pathogenesis of hypercapnic respiratory failure in patients with lung and chest wall disease. In: AP Fishman, editor. *Fishman's Pulmonary Disease and Disorders*, New York: McGraw-Hill Health Professions Division 1988:2605-2625.
22. Mador JM. Respiratory muscle fatigue and breathing pattern. *Chest* 1991; 100:1430-1435.
23. Bach JR, Saporito LR, Shah HR, et al. Decanulation of patients with severe respiratory muscle insufficiency: Efficacy of mechanical insufflation-exsufflation. *J Rehabil Med* 2014; 46:1037-1041.
24. Eckert DJ, Butler JE. Respiratory Physiology: Understanding the control of Ventilation. In: M. Kryger, T. Roth, WC Dement, editors. *Principles and Practice of Sleep Medicine Sixth Edition*, Philadelphia: Elsevier 2011:167-173.
25. De Troyer A, Leeper JB, McKenzie DK, et al. Neural drive to the diaphragm in patients with severe COPD. *Am J Respir Crit Care Med* 1997; 155:1335-1340.
26. McCartney A, Phillips D, James M, et al. Ventilatory neural drive in chronically hypercapnic patients with COPD: Effects of sleep and nocturnal noninvasive ventilation. *Eur Respir Rev* 2022; 31:220069.
27. Bower JS, Sandercock TG, Rothman E, et al. Time domain analysis of diaphragmatic electromyogram during fatigue in men. *J Appl Physiol: Respirat Environ Exercise Physiol* 1984; 57(3):913-916.
28. Nava S, Zanotti E, Ambrosino N, et al. Evidence of acute diaphragmatic fatigue in a "natural" condition; The diaphragm during labor. *Am Rev Respir Dis* 1992; 146: 1226-1230.
29. Laghi F, Cattapan SE, Jubran A, et al. Is weaning failure caused by low-frequency fatigue of the diaphragm. *Am J Respir Crit Care Med* 2003; 167:120-127.



2025

SOCIETY OF MECHANICAL VENTILATION CONFERENCE



EXPAND YOUR KNOWLEDGE, NETWORK, IMPROVE OUTCOME, HAVE FUN



Speakers

JOHN DOWNS MD
ANDREW FENG MD
BRENT MATSUDA MD
CHERIE CHU PHARM D
DAVID WILLMS MD
KIMIYO YAMASAKI RRT

BENJAMIN DAXON MD
RYOTA SATO MD
SAMUEL EVANS MD
STEPHEN TUNNEL RRT
EHAB DAOUD MD

DATE: JUNE 30 - JULY 1, 2025
7:00 AM TO 12:30PM

LOCATION: KUAKINI MEDICAL CENTER AUDITORIUM
347 NORTH KUAKINI STREET, HONOLULU

////// IN PERSON OR ONLINE
REGISTER TODAY

<https://societymechanicalventilation.org/2025-2/>