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The Clinical Utility of Surface Electromyography in Assessing Respiratory and Accessory Muscles as Predictors of Respiratory Failure: A Comprehensive Review

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Abstract

Surface electromyography (sEMG) has emerged as a non-invasive modality for assessing respiratory muscle activity, including both primary and accessory muscles involved in breathing. Increasing evidence suggests that sEMG-derived indices—such as neural respiratory drive (NRD), muscle recruitment patterns, and fatigue signatures—may serve as biomarkers for respiratory failure, disease severity, and mortality risk across diverse clinical specialties. This review synthesizes current literature on the physiological basis, technical considerations, and clinical applications of sEMG in respiratory medicine, with a particular focus on its prognostic value. We examine its role in acute respiratory failure, chronic obstructive pulmonary disease (COPD), neuromuscular disorders, and critical care settings, including mechanical ventilation and weaning. Particular emphasis is placed on accessory muscle activity as a surrogate for respiratory load-capacity imbalance and its implications for clinical outcomes. Despite promising associations between sEMG signals and outcomes such as dyspnea, ventilatory failure, and mortality, limitations in standardization and interpretation remain barriers to clinical implementation. Future directions include integration with multimodal monitoring and predictive modeling.

Keywords

Surface Electromyography, Respiratory Failure, Non-Invasive, Respiratory Muscles

1. Introduction

Respiratory failure represents a major global health burden and is a leading cause

of ICU admission and mortality [1]-[3]. It arises from diverse etiologies, including acute respiratory distress syndrome (ARDS), COPD exacerbations, pneumonia, and neuromuscular disorders, yet shares a common physiological endpoint: failure of the respiratory system to meet metabolic demands [4]-[6].

Traditional assessments of respiratory muscle function—such as maximal inspiratory pressure (MIP), transdiaphragmatic pressure (Pdi), and spirometry—are limited by invasiveness, reliance on patient effort, and feasibility constraints in critically ill populations [7]. Esophageal pressure monitoring, although considered a gold standard, is technically demanding and not routinely used outside specialized settings [8].

Surface electromyography (sEMG) offers a non-invasive, real-time alternative for evaluating respiratory muscle activity. By detecting electrical potentials generated during muscle contraction, sEMG provides insight into motor unit recruitment and neural respiratory drive [9] [10]. Unlike invasive diaphragm EMG (EMGdi), sEMG enables simultaneous assessment of multiple respiratory and accessory muscles, including the diaphragm, parasternal intercostals, sternocleidomastoid, and scalene muscles [11] [12].

Importantly, respiratory failure reflects not only impaired gas exchange but also a mismatch between respiratory load and muscle capacity [13]. This imbalance results in increased NRD and recruitment of accessory muscles, phenomena that can be quantified using sEMG [14]. Recent studies have demonstrated that these measures correlate with clinically meaningful outcomes, including dyspnea, intubation risk, and mortality [1] [15].

This review provides a comprehensive synthesis of the physiological basis, methodological considerations, and clinical applications of sEMG, with a focus on its emerging role as a prognostic tool in respiratory failure. sEMG-derived measures should be interpreted both as markers of current physiological severity and as potential predictors of outcomes; however, while several studies adjusted for illness severity and gas exchange abnormalities, the independence of sEMG as a prognostic marker remains incompletely established.

2. Physiological Basis of Respiratory Muscle Activity

2.1. Neural Respiratory Drive

Neural respiratory drive (NRD) reflects the output of central respiratory centers in the brainstem to respiratory muscles and is modulated by chemoreceptor input, mechanoreceptor feedback, and higher cortical influences [16] [17]. NRD increases in response to hypoxia, hypercapnia, and increased mechanical load, serving as a compensatory mechanism to maintain ventilation.

Because direct measurement of central respiratory output is not feasible in clinical practice, surrogate measures such as diaphragm EMG are used [12]. sEMG provides an indirect but clinically accessible measure of NRD by quantifying electrical activity in respiratory muscles [1].

Studies have demonstrated strong correlations between sEMG signals from

parasternal intercostals and invasive measures of diaphragm activity, supporting their use as non-invasive surrogates [11] [14]. However, interpretation requires consideration of factors such as electrode placement, tissue impedance, and muscle geometry [9].

2.2. Load-Capacity Imbalance

The balance between respiratory load and muscle capacity is a central determinant of respiratory function [13]. Load includes airway resistance, lung compliance, and metabolic demand, while capacity reflects muscle strength and endurance.

When load exceeds capacity, NRD increases to compensate. This leads to recruitment of accessory muscles and increased work of breathing. Persistent imbalance results in muscle fatigue and eventual ventilatory failure [18].

sEMG provides a direct measure of this imbalance by quantifying both the intensity and distribution of muscle activation. Elevated accessory muscle activity reflects increased load and reduced diaphragm efficiency, making it a potential biomarker of disease severity [3].

2.3. Accessory Muscle Recruitment

Accessory respiratory muscles—including the sternocleidomastoid, scalene, and intercostals—are minimally active during quiet breathing but become progressively engaged as respiratory demand increases [13]. Recruitment follows a hierarchical pattern in which diaphragm activation initially increases, followed by contribution from the intercostal muscles, and ultimately recruitment of accessory neck muscles as demand continues to rise. This sequence reflects an adaptive strategy that prioritizes mechanical efficiency and fatigue resistance. However, increased reliance on accessory muscles is energetically inefficient and is associated with greater oxygen consumption [19].

sEMG enables detailed characterization of these recruitment patterns, providing insight into compensatory mechanisms and facilitating early detection of respiratory distress.

3. Surface EMG Technology and Methodology

3.1. Signal Acquisition

sEMG involves placing electrodes on the skin surface to detect electrical activity generated by muscle fibers. Accurate electrode placement is critical to ensure signal fidelity and minimize contamination [20]. However, signal acquisition is subject to several important limitations, including crosstalk from adjacent muscles, cardiac interference (ECG contamination), attenuation by subcutaneous adipose tissue, and motion artifacts during breathing, all of which can affect signal amplitude and timing [2] [9]. High-density sEMG arrays have been proposed to improve spatial resolution and reduce crosstalk, thereby enhancing signal specificity [21].

3.2. Signal Processing

Raw sEMG signals require processing to extract meaningful information. This typically involves band-pass filtering to remove noise, rectification to convert the signal into a unidirectional waveform, and smoothing to generate a linear envelope that reflects the temporal pattern of muscle activation. More advanced methods, such as wavelet analysis and independent component analysis, can further improve signal quality and enable separation of overlapping physiological signals [2] [10].

Normalization is essential for comparison across subjects; however, commonly used approaches such as maximal voluntary contraction (MVC) are often not feasible in critically ill patients, limiting standardization in this population [1].

3.3. Advanced Analytical Techniques

Machine learning and pattern recognition techniques are increasingly applied to sEMG data. These approaches enable identification of complex patterns associated with fatigue, coordination, and disease progression [10] [22]. Such methods hold promise for improving predictive accuracy but require large datasets and standardization.

3.4. Key sEMG Outputs and Bedside Interpretation

Several quantitative features are commonly derived from sEMG signals, each with distinct physiological meaning. The root mean square (RMS) reflects the amplitude of muscle activation and is proportional to motor unit recruitment and firing frequency; clinically, higher RMS indicates increased respiratory effort and neural drive. The integrated EMG (iEMG) represents total electrical activity over time and provides an estimate of the cumulative work of breathing during a respiratory cycle. In contrast, median frequency, a frequency-domain parameter, decreases with muscle fatigue as a result of slowing muscle fiber conduction velocity. Signal efficiency describes the relationship between electrical activity and mechanical output, with reduced efficiency suggesting neuromechanical uncoupling or developing fatigue.

At present, there are no universally accepted thresholds or reference ranges for these parameters in respiratory applications, and most studies rely on within-subject trends or relative changes rather than absolute cutoffs, limiting standardization in clinical practice.

3.5. Recording Sites and Signal Characteristics

The choice of recording site significantly influences both signal quality and clinical interpretation. The parasternal intercostals provide a reliable surrogate of diaphragm activity, with relatively low crosstalk and a strong correlation with neural respiratory drive, making them the most well-supported site for monitoring NRD. Surface recordings over the diaphragm region offer direct physiological relevance but are technically challenging due to muscle depth and interference from cardiac

signals. The sternocleidomastoid is easily accessible and generates strong, high-amplitude signals, which makes it useful for detecting increased respiratory effort, although it is more susceptible to activation from non-respiratory factors. The scalene muscles reflect early accessory muscle recruitment and are sensitive indicators of increased respiratory load, but they are also prone to crosstalk and variability in electrode placement.

Overall, parasternal recordings are preferred for estimating neural respiratory drive, whereas sternocleidomastoid and scalene activity are most informative for identifying accessory muscle recruitment and clinical deterioration.

4. Clinical Assessment of Accessory Respiratory Muscles

The clinical assessment of accessory respiratory muscle use has traditionally relied on qualitative bedside observation, including visible contraction of the sternocleidomastoid or supraclavicular retractions. While useful, these observations lack sensitivity and are highly operator-dependent [3]. Surface electromyography (sEMG) transforms this subjective assessment into a quantitative, reproducible metric capable of capturing subtle changes in muscle recruitment that may precede overt clinical deterioration [1] [14].

4.1. Quantification of Accessory Muscle Recruitment

sEMG allows precise measurement of activation amplitude in accessory muscles such as the sternocleidomastoid and scalene muscles. Increased amplitude reflects greater motor unit recruitment and firing frequency, which are indicative of elevated neural respiratory drive (NRD) [9] [12]. Importantly, these changes can occur even when traditional clinical parameters such as respiratory rate remain within normal ranges, highlighting the sensitivity of sEMG as an early biomarker.

Quantitative indices such as RMS amplitude and integrated EMG (iEMG) have been used to characterize muscle activity. Studies have demonstrated that RMS values of accessory muscles increase proportionally with inspiratory load, suggesting a direct relationship between sEMG amplitude and respiratory effort [10] [19].

4.2. Temporal Coordination and Synchrony

Beyond amplitude, the timing of muscle activation provides critical insight into respiratory mechanics. In healthy individuals, accessory muscles are minimally active during quiet breathing and are recruited later in the inspiratory cycle when demand increases. However, in pathological states, early activation of these muscles is often observed [14].

This early recruitment reflects a shift in the load-sharing strategy of the respiratory system and may indicate reduced diaphragm efficiency. sEMG studies have shown that patients with COPD or acute respiratory failure exhibit altered timing patterns, including prolonged activation and reduced synchrony between muscle groups [3] [15].

Asynchrony between respiratory muscles can also reflect neuromechanical un-

coupling, a condition in which neural drive is not effectively translated into mechanical ventilation. This phenomenon is particularly relevant in mechanically ventilated patients and is associated with increased work of breathing and poor outcomes [8].

4.3. Clinical Correlation and Outcome-Specific Interpretation

sEMG-derived measures of accessory muscle activity consistently map to specific clinical outcomes rather than serving as nonspecific markers. Increased sternocleidomastoid and scalene activation correlates with dyspnea severity and reduced exercise tolerance in COPD, reflecting heightened neural respiratory drive (NRD) and load-capacity imbalance [3] [14]. In acute and critical care settings, persistently elevated accessory muscle activity—particularly when sustained despite support—is associated with non-invasive ventilation (NIV) failure and progression toward intubation [15].

In mechanically ventilated patients, elevated baseline NRD and early accessory muscle recruitment during spontaneous breathing trials predict weaning failure, indicating insufficient respiratory reserve [1] [15]. Across ICU and chronic disease populations, sustained high sEMG amplitude—reflecting unresolved physiological stress—has been associated with increased mortality, particularly when combined with evidence of fatigue or neuromechanical uncoupling [1] [15].

4.4. Non-Respiratory Determinants of sEMG Signal Amplitude

Interpretation of elevated accessory muscle sEMG requires consideration of non-respiratory factors that can independently increase signal amplitude. Postural changes, particularly upright positioning or forward leaning, can increase baseline muscle activation. Speech and voluntary movements also transiently elevate activity in cervical and thoracic muscles. Psychological factors such as anxiety can increase respiratory drive independent of physiological load, while pain may alter breathing patterns and muscle recruitment. Obesity and increased subcutaneous tissue can both attenuate and distort signals, complicating amplitude interpretation. Additionally, variability in electrode placement and inter-electrode distance can significantly affect recorded activity. These factors should be accounted for to avoid overestimating respiratory distress based solely on sEMG amplitude.

5. sEMG in Acute Respiratory Failure

In acute respiratory failure, sEMG primarily reflects escalating respiratory load and failing compensatory mechanisms. Increased diaphragm and accessory muscle amplitude, prolonged activation, and reduced coordination represent rising NRD and declining efficiency [2].

Clinically, these patterns are linked to specific outcomes. Early increases in accessory muscle activity correlate with worsening dyspnea, often preceding abnormalities in oxygenation or blood gases [1]. Persistently elevated sEMG signals during NIV identify patients at high risk of NIV failure, reflecting inadequate unload-

ing of respiratory muscles [15]. When high NRD and accessory recruitment remain sustained despite support, this pattern predicts progression to endotracheal intubation, indicating that compensatory mechanisms are exhausted [15].

At later stages, sustained high NRD combined with signs of fatigue is associated with increased mortality, as it reflects irreversible load-capacity mismatch and impending ventilatory collapse [1] [23].

5.1. Pathophysiological Mechanisms

The pathophysiology of acute respiratory failure involves a combination of increased airway resistance, reduced lung compliance, and impaired gas exchange. These factors increase the work of breathing and necessitate compensatory increases in NRD [6].

As NRD increases, the diaphragm initially augments its activity. However, when the diaphragm reaches its functional limits, accessory muscles are recruited to sustain ventilation. This compensatory mechanism is reflected in increased sEMG amplitude across multiple muscle groups [13].

5.2. sEMG Signatures of Deterioration

sEMG studies have identified characteristic patterns associated with acute respiratory failure, including increased amplitude of diaphragm and accessory muscle signals, prolonged activation duration, and reduced coordination between muscle groups. Together, these changes reflect both increased respiratory load and reduced efficiency of the respiratory system [2].

Importantly, these sEMG signatures often precede changes in traditional clinical parameters such as oxygen saturation or arterial blood gases. This temporal advantage suggests that sEMG could serve as an early warning tool for impending respiratory failure [1].

5.3. Prognostic Implications

Elevated NRD and accessory muscle activity have been associated with increased risk of intubation and mortality. Patients who fail non-invasive ventilation (NIV) often exhibit persistently high sEMG activity despite support, indicating ongoing respiratory distress [15].

From a mechanistic perspective, sustained high NRD reflects a failing compensatory response. As respiratory muscles fatigue, the system becomes unable to maintain ventilation, leading to rapid clinical deterioration [23].

6. sEMG in Mechanical Ventilation and Weaning

sEMG provides a direct measure of patient effort during mechanical ventilation and allows identification of clinically meaningful outcomes related to patient-ventilator interaction. Abnormal timing between sEMG activity and ventilator cycles reflects patient-ventilator asynchrony, which increases work of breathing and contributes to prolonged ventilation [8] [24]. Excessively low sEMG activity indicates

over-assistance and risk of diaphragm disuse, whereas persistently high activity reflects under-assistance and excessive load, both of which are associated with worse outcomes [5].

During weaning, specific sEMG patterns predict weaning failure. Elevated baseline NRD, early recruitment of accessory muscles, and sustained high amplitude during spontaneous breathing trials indicate inadequate respiratory capacity and a high likelihood of failure [1] [15]. Conversely, a reduction in accessory muscle reliance and normalization of activation patterns are associated with successful weaning.

6.1. Patient-Ventilator Interaction

Patient-ventilator asynchrony occurs when there is a mismatch between patient effort and ventilator support. This mismatch can lead to increased work of breathing, discomfort, and prolonged ventilation [24]. sEMG provides a direct measure of patient effort, allowing detection of asynchrony that may not be apparent from ventilator waveforms alone. For example, delayed or ineffective triggering can be identified by comparing sEMG activity with ventilator cycles [8].

6.2. Monitoring Respiratory Muscle Load

Mechanical ventilation can lead to both underloading and overloading of respiratory muscles. Underloading may result in muscle atrophy, while overloading can lead to fatigue [5].

sEMG enables real-time monitoring of muscle activity, allowing clinicians to titrate ventilator support to maintain an optimal level of muscle engagement. This approach aligns with the concept of "lung and diaphragm-protective ventilation" [1].

6.3. Weaning and Failure Prediction

Weaning failure is a complex process involving multiple factors, including respiratory muscle weakness, excessive load, and impaired coordination [4]. sEMG studies indicate that failure is associated with elevated baseline neural respiratory drive, early recruitment of accessory muscles, and persistently high signal amplitude during spontaneous breathing trials, all of which reflect insufficient respiratory capacity to sustain independent ventilation [1] [15]. These findings suggest that sEMG could be incorporated into weaning protocols to improve prediction accuracy and guide clinical decision-making.

7. sEMG in Chronic Respiratory and Neuromuscular Disease

In chronic respiratory disease, sEMG reflects persistent physiological stress and adaptation to long-term load-capacity imbalance. In COPD, elevated resting NRD and increased accessory muscle activity correlate strongly with dyspnea severity and reduced exercise capacity [3] [14]. Longitudinally, sustained high NRD is associated with increased risk of hospitalization and mortality, reflecting chronic

respiratory muscle overload [3].

In neuromuscular disorders, declining sEMG amplitude and altered timing reflect progressive muscle weakness and are associated with impending ventilatory failure [25]. Changes in recruitment patterns may precede overt clinical deterioration, highlighting the role of sEMG as an early prognostic marker.

7.1. COPD

COPD is characterized by airflow limitation and hyperinflation, which impair diaphragm function and increase reliance on accessory muscles [18].

sEMG studies have demonstrated that patients with COPD exhibit elevated NRD even at rest, reflecting chronic respiratory stress [14]. This persistent elevation is associated with increased dyspnea, reduced exercise capacity, and higher mortality [3].

7.2. Neuromuscular Disorders

In neuromuscular diseases such as ALS, progressive weakness of respiratory muscles leads to ventilatory failure [25], sEMG can detect early changes in muscle activation patterns, including reduced amplitude and altered timing. These changes may precede clinical symptoms, suggesting a role for sEMG in early diagnosis and monitoring.

7.3. Longitudinal Monitoring and Prognosis

The ability of sEMG to provide continuous, non-invasive monitoring makes it particularly valuable in chronic disease. Longitudinal changes in NRD and muscle recruitment patterns may provide insight into disease progression and prognosis.

8. Respiratory Muscle Fatigue

Respiratory muscle fatigue is a key factor in the development of respiratory failure. It involves both central and peripheral mechanisms, including reduced motor neuron output and impaired muscle contractility [23]. Surface electromyography (sEMG) markers of fatigue include decreased median frequency, increased amplitude variability, and reduced signal efficiency. However, interpretation is complicated by changes in muscle recruitment patterns and signal noise [10]. Despite these challenges, sEMG remains one of the few tools capable of assessing fatigue in real time, making it a valuable component of respiratory monitoring.

9. Prognostic Value of sEMG for Mortality

Elevated sEMG activity—particularly when reflecting persistently high NRD—functions both as a marker of current disease severity and a predictor of adverse outcomes. Across ICU and chronic disease populations, higher sEMG amplitudes and sustained accessory muscle recruitment have been associated with increased mortality [1] [15].

However, most existing studies are observational, and the extent to which

sEMG independently predicts mortality beyond established severity indices remains uncertain. Several studies adjusted for markers of illness severity, such as gas exchange abnormalities and clinical scores, and still found significant associations between elevated NRD and outcomes, suggesting that sEMG may provide incremental prognostic information [15]. Nonetheless, residual confounding cannot be excluded, and further prospective validation is required.

9.1. Mechanistic Basis

Elevated NRD reflects severe physiological stress and an inability of the respiratory system to meet metabolic demands. Persistent high NRD is associated with muscle fatigue, ventilatory failure, and death [16].

9.2. Evidence across Clinical Populations

Studies have demonstrated associations between sEMG-derived measures and mortality in ICU patients, COPD populations, and neuromuscular disease [1] [15].

9.3. Integration into Clinical Practice

sEMG could be incorporated into risk stratification models to identify high-risk patients and guide early intervention. However, further validation is needed.

10. Limitations and Challenges

Despite the growing body of evidence supporting the clinical utility of surface electromyography (sEMG) in respiratory monitoring, several methodological, technical, and translational limitations continue to hinder its widespread adoption. One of the most significant challenges is the lack of standardized acquisition protocols. Variability in electrode placement, skin preparation, inter-electrode distance, and signal filtering methods can substantially influence signal quality and reproducibility [9] [20].

Signal contamination represents another critical limitation. Crosstalk from adjacent muscles, particularly in the thoracic region where multiple respiratory and non-respiratory muscles overlap, can obscure the specificity of recorded signals [21]. Additionally, electrocardiographic (ECG) interference is a persistent issue when recording from chest wall muscles, requiring advanced filtering techniques that may inadvertently distort the underlying EMG signal [10].

Inter-individual variability further complicates interpretation. Factors such as age, sex, body composition, and disease state influence sEMG amplitude and frequency characteristics [2]. Moreover, normalization techniques such as maximal voluntary contraction (MVC) are often not feasible in critically ill patients, limiting the ability to standardize measurements [1].

From a clinical perspective, interpretation of sEMG signals requires specialized expertise. Unlike more established monitoring modalities, sEMG lacks widely accepted clinical guidelines, posing a barrier to routine implementation [3]. Finally,

although observational studies demonstrate associations between sEMG and outcomes, large-scale prospective trials are lacking [15].

11. Future Directions

Future research should focus on addressing the current limitations of sEMG while expanding its clinical applicability. A primary priority is the development of standardized protocols for signal acquisition, processing, and interpretation. International consensus guidelines, similar to those established for other physiological monitoring techniques, would facilitate reproducibility and enable broader adoption [20].

Advances in hardware technology are likely to play a critical role in the evolution of sEMG. High-density electrode arrays and wearable sensor systems offer improved spatial resolution and the potential for continuous, ambulatory monitoring [21]. These innovations could enable real-time assessment of respiratory muscle activity in both hospital and outpatient settings, providing a more comprehensive understanding of disease progression.

Integration with multimodal monitoring systems represents another promising avenue. Combining sEMG with physiological parameters such as respiratory rate, tidal volume, and gas exchange indices could enhance the accuracy of predictive models for respiratory failure [5]. Such multimodal approaches may capture the complex interplay between neural drive, mechanical function, and metabolic demand.

Machine learning and artificial intelligence (AI) have the potential to transform sEMG analysis. Advanced algorithms can identify complex patterns in high-dimensional data, enabling automated detection of respiratory distress, fatigue, and patient-ventilator asynchrony [10] [22]. These approaches may also facilitate the development of predictive models for clinical outcomes, including mortality.

Importantly, future studies should focus on large-scale, prospective validation of sEMG as a prognostic tool. Randomized controlled trials evaluating the impact of sEMG-guided interventions on patient outcomes are needed to establish their clinical value. Such studies should include diverse patient populations to ensure generalizability.

12. Conclusions

Surface electromyography represents a promising and increasingly sophisticated tool for assessing respiratory muscle function in both acute and chronic disease states. By providing a non-invasive, real-time measure of neural respiratory drive and muscle recruitment patterns, sEMG offers unique insight into the physiological mechanisms underlying respiratory failure [1] [2].

A key strength of sEMG lies in its ability to quantify accessory muscle activity, which serves as a surrogate marker of respiratory load-capacity imbalance. This capability enables early detection of respiratory distress and provides a functional biomarker that complements traditional measures such as spirometry and blood

gas analysis [3].

Emerging evidence suggests that sEMG-derived indices are associated with clinically meaningful outcomes, including dyspnea severity, weaning success, and mortality [15]. These findings highlight the potential of sEMG to serve not only as a monitoring tool but also as a prognostic modality capable of guiding clinical decision-making.

However, significant challenges remain, including a lack of standardization, signal variability, and limited validation in large-scale studies. Addressing these limitations will require coordinated efforts across research, clinical, and engineering disciplines.

In conclusion, sEMG has the potential to transform the assessment of respiratory function by providing a dynamic, integrative measure of physiological stress. With continued technological and methodological advancements, it may become an essential component of respiratory monitoring and prognostication in modern practice.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Jonkman, A.H., Warnaar, R.S.P., Baccinelli, W., Carbon, N.M., D'Cruz, R.F., Doorduin, J., *et al.* (2024) Analysis and Applications of Respiratory Surface EMG: Report of a Round Table Meeting. *Critical Care*, **28**, Article 2. https://doi.org/10.1186/s13054-023-04779-x

[2] Silva Junior, E.F.F.d., Campos, S.L., Leite, W.S., Melo, P.V.d.S., Lins, R.A.C., Araújo, M.d.G.R.d., *et al.* (2023) Surface Electromyography Signal Processing and Evaluation on Respiratory Muscles of Critically Ill Patients: A Systematic Review. *PLOS ONE*, **18**, e0284911. https://doi.org/10.1371/journal.pone.0284911

[3] Laveneziana, P., Albuquerque, A., Aliverti, A., Babb, T., Barreiro, E., Dres, M., *et al.* (2019) ERS Statement on Respiratory Muscle Testing at Rest and during Exercise. *European Respiratory Journal*, **53**, Article ID: 1801214. https://doi.org/10.1183/13993003.01214-2018

[4] Tobin, M.J. (2006) Principles of Mechanical Ventilation. 2nd Edition, McGraw-Hill. https://accessmedicine.mhmedical.com/book.aspx?bookID=520

[5] Dres, M. and Demoule, A. (2020) Monitoring Diaphragm Function in the ICU. *Current Opinion in Critical Care*, **26**, 18-25. https://doi.org/10.1097/MCC.0000000000000682

[6] Fan, E., Brodie, D. and Slutsky, A.S. (2018) Acute Respiratory Distress Syndrome. *JAMA*, **319**, 698-710. https://doi.org/10.1001/jama.2017.21907

[7] American Thoracic Society/European Respiratory Society (2002) ATS/ERS Statement on Respiratory Muscle Testing. *American Journal of Respiratory and Critical Care Medicine*, **166**, 518-624.

[8] Akoumianaki, E., Maggiore, S.M., Valenza, F., Bellani, G., Jubran, A., Loring, S.H., *et al.* (2014) The Application of Esophageal Pressure Measurement in Patients with Respiratory Failure. *American Journal of Respiratory and Critical Care Medicine*, **189**, 520-531. https://doi.org/10.1164/rccm.201312-2193ci

[9] De Luca, C.J. (1997) The Use of Surface Electromyography in Biomechanics. *Journal of Applied Biomechanics*, **13**, 135-163. https://doi.org/10.1123/jab.13.2.135

[10] Phinyomark, A., Phukpattaranont, P. and Limsakul, C. (2012) Feature Reduction and Selection for EMG Signal Classification. *Expert Systems with Applications*, **39**, 7420-7431. https://doi.org/10.1016/j.eswa.2012.01.102

[11] Hudson, A.L., Joulia, F., Butler, A.A., Fitzpatrick, R.C., Gandevia, S.C. and Butler, J.E. (2016) Activation of Human Inspiratory Muscles in an Upside-Down Posture. *Respiratory Physiology & Neurobiology*, **226**, 152-159. https://doi.org/10.1016/j.resp.2015.05.012

[12] De Troyer, A. and Estenne, M. (1988) Functional Anatomy of the Respiratory Muscles. *Clinics in Chest Medicine*, **9**, 175-193. https://doi.org/10.1016/s0272-5231(21)00498-6

[13] De Troyer, A., Kirkwood, P.A. and Wilson, T.A. (2005) Respiratory Action of the Intercostal Muscles. *Physiological Reviews*, **85**, 717-756. https://doi.org/10.1152/physrev.00007.2004

[14] Jolley, C.J., Luo, Y., Steier, J., Reilly, C., Seymour, J., Lunt, A., *et al.* (2008) Neural Respiratory Drive in Healthy Subjects and in Copd. *European Respiratory Journal*, **33**, 289-297. https://doi.org/10.1183/09031936.00093408

[15] Suh, E., Mandal, S., Harding, R., Ramsay, M., Kamalanathan, M., Henderson, K., *et al.* (2015) Neural Respiratory Drive Predicts Clinical Deterioration and Safe Discharge in Exacerbations of Copd. *Thorax*, **70**, 1123-1130. https://doi.org/10.1136/thoraxjnl-2015-207188

[16] Sinderby, C., Navalesi, P., Beck, J., Skrobik, Y., Comtois, N., Friberg, S., *et al.* (1999) Neural Control of Mechanical Ventilation in Respiratory Failure. *Nature Medicine*, **5**, 1433-1436. https://doi.org/10.1038/71012

[17] Dempsey, J.A., Veasey, S.C., Morgan, B.J. and O'Donnell, C.P. (2010) Pathophysiology of Sleep Apnea. *Physiological Reviews*, **90**, 47-112. https://doi.org/10.1152/physrev.00043.2008

[18] O'Donnell, D.E., Banzett, R.B., Carrieri-Kohlman, V., Casaburi, R., Davenport, P.W., Gandevia, S.C., *et al.* (2007) Pathophysiology of Dyspnea in Chronic Obstructive Pulmonary Disease: A Roundtable. *Proceedings of the American Thoracic Society*, **4**, 145-168. https://doi.org/10.1513/pats.200611-159cc

[19] Aliverti, A., Cala, S.J., Duranti, R., Ferrigno, G., Kenyon, C.M., Pedotti, A., *et al.* (1997) Human Respiratory Muscle Actions and Control during Exercise. *Journal of Applied Physiology*, **83**, 1256-1269. https://doi.org/10.1152/jappl.1997.83.4.1256

[20] Hermens, H.J., Freriks, B., Disselhorst-Klug, C. and Rau, G. (2000) Development of Recommendations for SEMG Sensors and Sensor Placement Procedures. *Journal of Electromyography and Kinesiology*, **10**, 361-374. https://doi.org/10.1016/s1050-6411(00)00027-4

[21] Farina, D., Merletti, R. and Enoka, R.M. (2004) The Extraction of Neural Strategies from the Surface EMG. *Journal of Applied Physiology*, **96**, 1486-1495. https://doi.org/10.1152/japplphysiol.01070.2003

[22] Clancy, E.A., Morin, E.L. and Merletti, R. (2002) Sampling, Noise-Reduction and Amplitude Estimation Issues in Surface Electromyography. *Journal of Electromyography and Kinesiology*, **12**, 1-16. https://doi.org/10.1016/s1050-6411(01)00033-5

[23] Gandevia, S.C. (2001) Spinal and Supraspinal Factors in Human Muscle Fatigue. *Physiological Reviews*, **81**, 1725-1789. https://doi.org/10.1152/physrev.2001.81.4.1725

[24] Colombo, D., Cammarota, G., Alemani, M., Carenzo, L., Barra, F.L., Vaschetto, R., *et al.* (2011) Efficacy of Ventilator Waveforms Observation in Detecting Patient-Ventilator Asynchrony. *Critical Care Medicine*, **39**, 2452-2457. https://doi.org/10.1097/ccm.0b013e318225753c

[25] Lechtzin, N., Wiener, C.M., Shade, D.M., Clawson, L. and Diette, G.B. (2002) Spirometry in the Supine Position Improves the Detection of Diaphragmatic Weakness in Patients with Amyotrophic Lateral Sclerosis. *Chest*, **121**, 436-442. https://doi.org/10.1378/chest.121.2.436