Respiratory muscle dysfunction in acute and chronic respiratory failure: how to diagnose and how to treat?

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Shareable abstract (@ERSpublications)
Respiratory muscle dysfunction can be both a cause and consequence of respiratory failure. It is often reversible and should be diagnosed and treated. This review covers available assessment and treatment options in acute and chronic respiratory failure. https://bit.ly/3XZBeVa

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Abstract

Assessing and treating respiratory muscle dysfunction is crucial for patients with both acute and chronic respiratory failure. Respiratory muscle dysfunction can contribute to the onset of respiratory failure and may also worsen due to interventions aimed at treatment. Evaluating respiratory muscle function is particularly valuable for diagnosing, phenotyping and assessing treatment efficacy in these patients. This review outlines established methods, such as measuring respiratory pressures, and explores novel techniques, including respiratory muscle neurophysiology assessments using electromyography and imaging with ultrasound.

Additionally, we review various treatment strategies designed to support and alleviate the burden on overworked respiratory muscles or to enhance their capacity through training interventions. These strategies range from invasive and noninvasive mechanical ventilation approaches to specialised respiratory muscle training programmes. By summarising both established techniques and recent methodological advancements, this review aims to provide a comprehensive overview of the tools available in clinical practice for evaluating and treating respiratory muscle dysfunction. Our goal is to present a clear understanding of the current capabilities and limitations of these diagnostic and therapeutic approaches. Integrating advanced diagnostic methods and innovative treatment strategies should help improve patient management and outcomes. This comprehensive review serves as a resource for clinicians, equipping them with the necessary knowledge to effectively diagnose and treat respiratory muscle dysfunction in both acute and chronic respiratory failure scenarios.

Introduction

Respiratory muscle dysfunction can contribute to the onset of respiratory failure and may worsen with interventions to unload the respiratory muscles and treat underlying conditions [1]. The respiratory system consists of two main components: lungs, the gas-exchanging organ, and the pump for lung ventilation. The pump includes the chest wall, respiratory muscles, central nervous system (CNS) controllers and pathways connecting these controllers to the respiratory muscles, such as spinal and peripheral nerves. Acute respiratory failure (ARF) is characterised by a sudden and significant impairment in the respiratory

system's ability to maintain adequate gas exchange, developing within minutes or hours. In contrast, chronic respiratory failure (CRF) develops gradually over weeks, months or years.

Lung failure, caused by conditions such as pneumonia, emphysema or interstitial lung disease, leads to hypoxaemia with normocapnia or hypocapnia (hypoxaemic or type I respiratory failure) (figure 1) [1]. Acute lung failure can occur during asthma or COPD exacerbations, pneumonia, acute respiratory distress syndrome (ARDS) or pulmonary oedema. Chronic lung failure can develop in chronic obstructive and restrictive lung diseases (*i.e.* COPD and interstitial lung disease).

Pump failure mainly results in hypercapnia (hypercapnic or type II respiratory failure) (figure 1) due to alveolar hypoventilation, although hypoxaemia can coexist [1]. Acute pump failure can occur in neuromuscular disorders (*e.g.*, myasthenia gravis or Guillain–Barré syndrome), chest wall abnormalities (*e.g.*, flail chest), increased abdominal pressure (*e.g.*, pancreatitis) or CNS depression (*e.g.*, drug overdose and brainstem injury). Chronic pump failure develops from progressive respiratory muscle dysfunction in conditions, such as amyotrophic lateral sclerosis (ALS) or muscular dystrophies, and in chronic chest wall disorders, such as kyphoscoliosis or severe obesity (obesity hypoventilation syndrome) that restrict lung expansion. The balance between restrictive forces on lung expansion and respiratory muscle capacity determines the likelihood of a respiratory muscle contribution to pump failure [2–4].

Both types of respiratory failure can coexist in the same patient. For example, severe COPD patients and chronic type I respiratory failure may develop additional ventilatory failure due to worsening of respiratory muscle dysfunction, causing muscle fatigue and carbon dioxide retention [3, 5]. Similarly, severe pulmonary oedema or asthmatic crises may initially cause hypoxaemia, followed by hypercapnia as the condition persists [1]. Prolonged mechanical ventilation for severe lung failure can also gradually lead to or exacerbate pre-existing respiratory muscle dysfunction, prolonging mechanical ventilation and complicating weaning [6].

Assessing respiratory muscle function is therefore essential for diagnosing and phenotyping patients, evaluating treatment efficacy and follow-up [7]. Recent advances in respiratory muscle neurophysiology and imaging, such as surface electromyography and respiratory muscle ultrasound, are increasingly used in clinical settings [8–10]. This review will discuss the potential of these novel methods alongside established techniques for assessing respiratory muscle function, including voluntary and evoked manoeuvres to measure airway opening, oesophageal and gastric pressures [7].

We will review established and emerging techniques for assessing respiratory muscle function in patients with ARF and CRF. Additionally, we will present current practices and evidence on treatment strategies aimed at supporting overworked respiratory muscles or enhancing their capacity through training and evaluate their impact on clinical outcomes. The presented information is not based on a systematic search of the literature including search terms and inclusion criteria. Instead, we performed a subjective selection

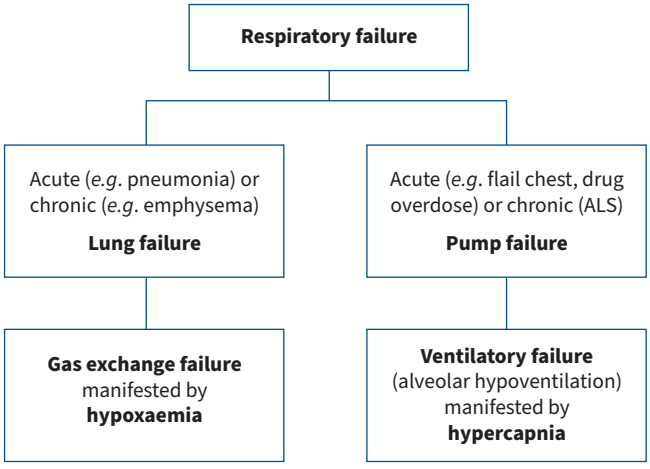

FIGURE 1 Types of respiratory failure. The respiratory system can be considered as consisting of two parts: 1) the lung and 2) the pump, including the chest wall, respiratory muscles, and central/peripheral nervous system. Adapted from Roussos and Koutsoukou [1].

of the literature that we deemed most relevant. To do this, we based our approach on recent systematic reviews [10–15] and task force statements [7, 16], and performed cited reference searches on these papers using Web of Science to find more recent original articles.

Assessment of respiratory muscle dysfunction in ARF

ARF can be the initial presentation of respiratory muscle dysfunction, potentially complicating treatment and affecting underlying diseases. The diaphragm, the main inspiratory muscle, is often affected in such cases.

Techniques that do not depend on patient cooperation

Evoked manoeuvres for the assessment of airway opening, oesophageal pressure (P_{es}) and gastric pressure (P_{ga})

Bilateral anterior magnetic phrenic nerve stimulation uses two figure-of-eight magnetic coils, positioned at the posterior border of the sternocleidomastoid muscle, to generate a supramaximal phrenic nerve stimulation. This triggers diaphragm contraction, measured as twitch transdiaphragmatic pressure ($P_{di,twitch}$). $P_{di,twitch}$ is the difference between P_{ga} and P_{es} assessed with balloons positioned in the stomach and oesophagus and has been successfully used in critically ill patients [17]. $P_{di,twitch}$ is the gold standard for assessing diaphragm function in noncooperative patients. Increased duration of mechanical ventilation and infection are linked with reduced $P_{di,twitch}$ [18, 19]. $P_{di,twitch}$ <20 cmH_2O is typically used to confirm a diagnosis of diaphragm weakness (figure 2). $P_{di,twitch}$ <10 cmH_2O was associated with prolonged mechanical ventilation and increased mortality [19]. An alternative measure, twitch tracheal pressure (Tw,tr), which does not require balloon catheters, defines diaphragm weakness (Tw,tr <11 cmH_2O) and occurs twice as often as peripheral muscle weakness at the time of weaning [20]. However, this technique is expensive and only available in selected centres worldwide.

Respiratory muscle ultrasound

Muscoskeletal ultrasound (MUS) as a part of point-of-care ultrasound is a safe, cost-effective and repeatable test to assess diaphragmatic dysfunction in acute and resource-limited settings [21, 22]. MUS is

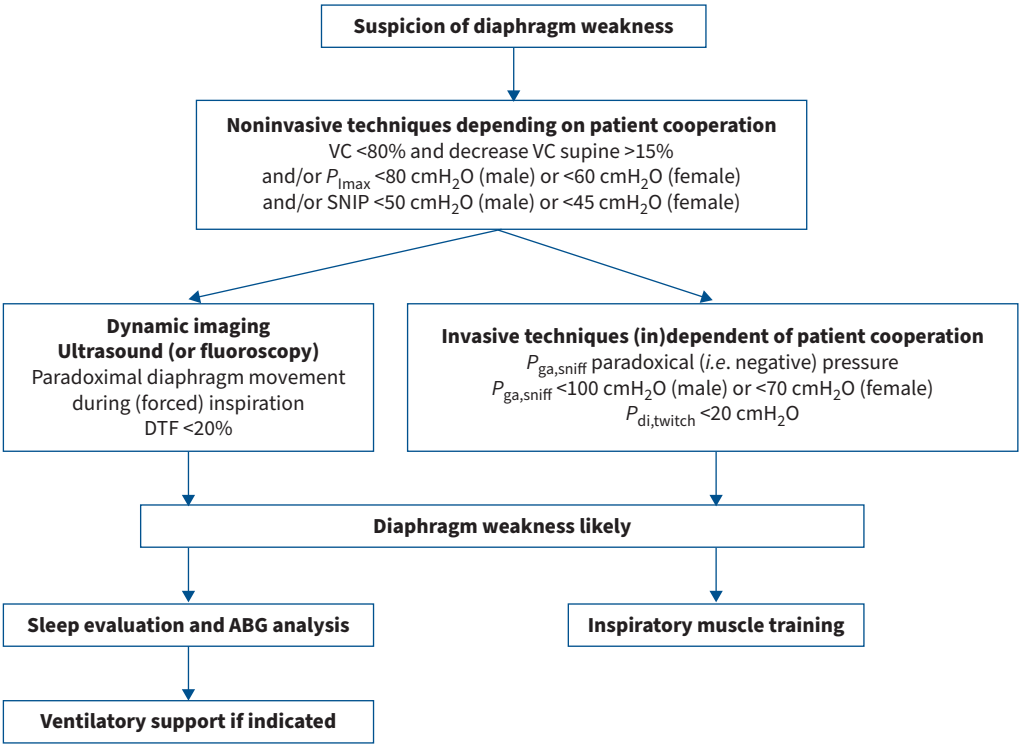

FIGURE 2 Diagnosis and treatment of diaphragm weakness. ABG: arterial blood gas; DTF: thickening fraction of the diaphragm; P_{Imax}: maximal inspiratory pressure; $P_{di,sniff}$: sniff transdiaphragmatic pressure; $P_{di,twitch}$: twitch transdiaphragmatic pressure; $P_{ga,sniff}$: sniff gastric pressure; SNIP: sniff nasal inspiratory pressure; VC: vital capacity.

increasingly used in research and clinical settings to evaluate multiple aspects of respiratory muscle function, including diaphragm and extradiaphragmatic muscle thickness, thickening fraction and diaphragm excursion (DE). In healthy subjects, normal values of diaphragm thickness measured at a functional residual capacity (FRC) range from 1.8 mm (lower limit of normal (LLN): 1.6 mm) in females to 2.2 mm (LLN: 1.9 mm) in males [23]. During quiet breathing, diaphragm thickening fraction (DTF) ranges typically from 25 to 40% [24, 25] and DE from 1.5 cm in females to 1.7 cm in males (LLN: 1.3 cm in females and 1.5 cm in males) [23]. A DTF of <20% is suggested as a cut-off to establish a diagnosis of diaphragm dysfunction (figure 2) [7, 26]. The diaphragm can be acutely affected by several neuromuscular entities and present with diaphragmatic paralysis leading to acute pump failure [27, 28]. Diaphragmatic ultrasound (DUS) aids in timely diagnosing of these emergencies. In the emergency department, DUS may reliably predict respiratory failure in community-acquired pneumonia patients by assessing DTF [29]. In coronavirus disease 2019 (COVID-19) patients, trends in DTF correlate with deteriorating respiratory status and the need for intubation [30]. Moreover, a systematic review has highlighted the importance of lung and diaphragm ultrasound to predict noninvasive ventilation (NIV) failure, indicating the need for intensive care unit (ICU) care [11]. Recommendations for diaphragm ultrasonography methodology in the ICU are available and the technique is frequently applied in clinical practice [16]. In the ICU setting, DUS can be used to monitor the evolution of diaphragm function [31]. A recent meta-analysis found DTF to have the highest accuracy among various respiratory muscle assessments for predicting weaning outcomes, with DTF cut-offs between 25 and 33% [10]. DE has also high accuracy to predict weaning success with cut-offs between 1.0 and 1.4 cm [10]. MUS of extradiaphragmatic inspiratory muscles, particularly parasternal intercostal muscles, is also clinically feasible [32]. Measuring the thickening fraction of parasternal intercostal muscle (PTF) may help assess extradiaphragmatic respiratory effort when the DTF is low [33]. Moreover, higher PTF values during a spontaneous breathing trial (SBT) are observed in patients failing an SBT compared to those who successfully passed it [34]. Similarly, patients who failed weaning have significantly higher PTF values than successfully weaned patients [35, 36].

Airway occlusion pressure at 100 ms ($P_{0.1}$) and occluded inspiratory airway pressure (P_{occ})

In mechanically ventilated patients breathing spontaneously, two noninvasive assessments based on brief occlusion manoeuvres can evaluate respiratory neural drive and effort. Airway occlusion pressure at 100 ms ($P_{0.1}$) can be rapidly measured with modern mechanical ventilators [37]. $P_{0.1}$ reflects respiratory drive and effort [7, 37–40]. P_{occ}, an emerging assessment of respiratory effort, measures airway pressure deflection during a single-breath inspiratory occlusion [41]. $P_{0.1}$ accurately identifies low diaphragmatic effort, while P_{occ} showed high accuracy for identifying low and high diaphragmatic effort [42]. However, data on their accuracy to predict weaning outcomes are limited [10].

Rapid shallow breathing index (RSBI)

RSBI is the ratio of respiratory frequency to tidal volume during spontaneous breathing [43]. Traditionally, RSBI is measured with a handheld spirometer when patients are disconnected from mechanical ventilation [43]. However, RSBI is now available on modern ventilators and can be assessed before, during and after different types of SBT [12]. These assessments should be performed without pressure support. Regardless of the technique, RSBI as a standalone test has moderate sensitivity and poor specificity to predict extubation success [12]. Combining RSBI with DTF assessments can improve prediction of successful weaning compared to using RSBI alone [44, 45].

Respiratory muscle electromyography

Surface electromyography (sEMG) involves placing electrodes on the skin over respiratory muscles such as the sternocleidomastoid, scalenes, intercostals and abdominals [8, 46, 47]. sEMG is particularly advantageous in clinical settings due to its simplicity, minimal patient discomfort and can be useful when patient cooperation is limited [46]. Analysing sEMG signal amplitude (*e.g.*, root mean square (RMS)) indicates muscle activation levels, while frequency analyses provide insights into muscle fatigue [46, 48]. Timing among the activation of several respiratory muscles facilitates understanding muscle coordination during breathing [46, 49]. Unfortunately, there are no normal values of respiratory muscle sEMG. Additionally, sEMG mainly reflects superficial muscle activity, limiting its effectiveness in assessing deeper muscles such as the diaphragm [46]. By quantifying inspiratory activity and contributions of various respiratory muscles, sEMG can help to titrate optimal ventilatory support level [46, 50–53]. During SBTs, sternocleidomastoid EMG activity was present in 73% of patients who failed an SBT, compared to 13% those who succeeded [51]. Sternocleidomastoid sEMG RMS doubled in patients who failed the trial but remained unchanged in those successfully weaned [51]. sEMG is also sensitive in detecting patient–ventilator asynchrony [46, 50–53]. In patients with weaning difficulties receiving inspiratory muscle training (IMT), sEMG activity of extradiaphragmatic inspiratory muscles better correlates with total inspiratory effort (measured by P_{es} swings) than with external IMT load, reflected by changes in airway

pressure swings [54]. Therefore, sEMG activity could help to better titrate training load imposed during IMT [54].

Diaphragm electromyography *via* oesophageal catheter (EA_{di}) provides a precise measurement of diaphragmatic activity and closely reflects brain motor output at the bedside [55]. This technique involves inserting a catheter with embedded electrodes into the oesophagus, near the diaphragm to detect diaphragmatic electrical activity accurately and provide detailed information about its function [56]. In mechanically ventilated patients, average EA_{di} values range between 5 and 20 µv [39]. Despite its advantages, EA_{di} is more invasive than sEMG, requiring specialised equipment and expertise. Patient discomfort is similar to standard feeding tubes and minimal potential complications from catheter placement in patients without formal contraindications. EA_{di} can guide ventilatory support adjustments (*e.g.*, neurally adjusted ventilatory assist) [56].

Oesophageal and gastric pressure swings during spontaneous breathing

P_{es} swings (ΔP_{es}) are the most accessible parameter to estimate breathing effort at the bedside [39]. ΔP_{es} is measured as the difference between end-expiratory P_{es} and the most negative P_{es} during inspiration [7]. The work of breathing (WOB) quantifies energy expenditure required for breathing when volume is generated [7]. While pressure–time product (PTP) reflects the cumulative pressure exerted by respiratory muscles over time, regardless of volume displacement [7]. Respiratory muscle pressure (P_{mus}) represents the pressure generated by the respiratory muscles during spontaneous breathing [57]. Normal values in healthy people for ΔP_{es} are between 3 and 15 cmH_2O, for WOB between 2.4 and 4.0 $J \cdot min^{-1}$ (0.35–0.70 $J \cdot L^{-1}$), for PTP between 50 and 150 $cmH_2O \cdot s^{-1} \cdot min^{-1}$, and for P_{mus} between 5 and 15 cmH_2O [39, 58]. In ARF, these indices help detect respiratory effort levels, guiding interventions [58, 59]. Low inspiratory effort indicates over-assistance while high effort indicates under-assistance with both being associated with the development of diaphragm dysfunction [58, 59]. ΔP_{es} monitoring provides an accurate quantification of total training load during IMT in acute settings. Exploratory data showed that total inspiratory effort during IMT (as quantified by ΔP_{es}) can be three to four times larger than the external IMT load (as assessed by changes in airway pressure swings) [54].

Techniques (partly) depending on patient cooperation

Voluntary manoeuvres for the assessment of airway opening pressures

Maximum static inspiratory (P_{Imax}) or expiratory (P_{Emax}) pressures at the mouth allow a simple volitional assessment of global respiratory muscle strength in a clinical setting that depend on full patient cooperation [7]. P_{Imax} is usually measured at residual volume and P_{Emax} at total lung capacity, with the maximum value of three manoeuvres recorded when they vary <10% [7]. Recently, objective cut-off points for weakness were defined based on T-scores ⩾2.5 SD below the peak mean P_{Imax} value at a young age as 83 cmH_2O for males and 62 cmH_2O for females [60]. Updated P_{Emax} cut-offs for diagnosing expiratory muscle weakness are also available (table 1). MARINI *et al*. [61] developed a reliable method to estimate respiratory muscle strength in uncooperative, mechanically ventilated patients [61, 62]. Patients breathe through a unidirectional valve for 25 s while being encouraged to inhale or exhale forcefully [61]. This method provides estimates of the respiratory muscle strength even without patient cooperation. P_{Imax} is often used to determine the external load during IMT [63]. Some studies showed that a low P_{Imax} is an independent risk factor for prolonged mechanical ventilation [64] and 1-year mortality [65], while P_{Emax} may be related to extubation failure [66] and mechanical ventilation duration [67]. However, a recent systematic review found P_{Imax} less reliable in predicting successful weaning from mechanical ventilation compared to other respiratory muscle assessments, such as DTF and DE [10].

Treatment of respiratory muscle dysfunction in ARF

Unloading respiratory muscles

In acute exacerbations of obstructive diseases there is an improvement in mechanical lung emptying and increased functional strength of the inspiratory muscles through pharmacological deflation of the lung with bronchodilators and correction of intrinsic positive end-expiratory pressure (PEEP) [68, 69]. Short-term steroid treatment does not seem to reduce respiratory muscle strength and has been shown to even improve some lung function results, such as P_{Imax}, P_{Emax} and of sniff nasal inspiratory pressure (SNIP) in COPD [70]. In patients with an acute exacerbation of COPD, oxygen supplementation can be implemented to improve pulmonary haemodynamics and respiratory muscle function, but a conservative approach and titration is advised [71]. High-flow noninvasive oxygen is able to unload the respiratory muscles by diminishing the inspiratory effort in both acute hypoxaemic and hypercapnic respiratory failure; however, the first-line treatment for acute hypercapnic COPD patients still remains NIV [72].

TABLE 1 Cut-off values for diagnosing weakness for each respiratory muscle test, test characteristics, practical applications and costs

Test	Cut-off values for diagnosing weakness	Respiratory muscle group assessed	Level of invasiveness and difficulty	Practical applications	Cost
P_{Imax}	M: 80 cmH_2O F: 60 cmH_2O	Diaphragm and extradiaphragmatic inspiratory muscles	Not invasive Simple	Assessment of inspiratory muscle function before and after interventions aimed at unloading or improving respiratory muscle function Determination of the external load before starting inspiratory muscle training	Low
SNIP	M: 50 cmH_2O F: 45 cmH_2O	Diaphragm and extradiaphragmatic inspiratory muscles	Not invasive Simple	Assessment of inspiratory muscle function before and after interventions aimed at unloading or improving respiratory muscle function in patients unable to perform P_{Imax} manoeuvre reliably	Low
P_{Emax}	M: 110 cmH_2O F: 80 cmH_2O	Expiratory muscles	Not invasive Simple	Assessment of expiratory muscle function before and after interventions aimed at unloading or improving respiratory muscle function	Low
PCF	M and F: 270 $L{\cdot}min^{-1}$	Expiratory muscles	Not invasive Simple	Estimation of airway clearance effectiveness in patients with NMDs Estimation of expiratory muscle function in patients with NMDs	Low
DTF	M and F: 20%	Diaphragm	Not invasive Medium	Evaluation of diaphragmatic function in the intensive care unit Confirmation or refinement of the diagnosis of (hemi-) diaphragmatic dysfunction Prediction of weaning outcomes in mechanically ventilated patients	Medium
$P_{es,sniff}$	M: 55 cmH_2O F: 50 cmH_2O	Diaphragm and extradiaphragmatic inspiratory muscles	Invasive Simple	Confirmation or refinement of the diagnosis of respiratory muscle weakness when noninvasive assessments provide equivocal results	Medium
$P_{di,sniff}$	M: 100 cmH_2O F: 70 cmH_2O	Diaphragm and extradiaphragmatic inspiratory muscles	Invasive Simple	Confirmation or refinement of the diagnosis of respiratory muscle weakness when non-invasive assessments provide equivocal results P_{ga}/P_{es} ratio can provide information about diaphragm function	Medium
$P_{di,twitch}$	M and F: 20 cmH_2O	Diaphragm	Invasive Complex	Confirmation or refinement of the diagnosis of diaphragmatic weakness Prediction of prolonged mechanical ventilation and mortality in the intensive care unit	High

Cut-off values are based on Steier *et al*. [102], Lista-Paz *et al*. [60] and Laveneziana *et al*. [7]. M: male; F: female; NMD: neuromuscular disease; PCF: peak cough flow; $P_{di,sniff}$: sniff transdiaphragmatic pressure; $P_{di,twitch}$: twitch transdiaphragmatic pressure; P_{Emax}: maximal expiratory pressure; P_{es}: oesophageal pressure; $P_{es,sniff}$: sniff oesophageal pressure; P_{ga}: gastric pressure; P_{Imax}: maximal inspiratory pressure; SNIP: sniff nasal inspiratory pressure; DTF: diaphragm thickness fraction.

NIV can, through external pressure support, counteract the dynamic intrinsic PEEP associated with lung hyperinflation and help in the unloading of the respiratory muscles [73]. The application of NIV can improve the fatigue of respiratory muscles during an exacerbation of obstructive airway disorders such as COPD and asthma by diminishing their workload and help avoid invasive ventilation [74]. NIV is also recommended for managing acute pulmonary oedema and acute or acute-on-chronic hypercapnic respiratory failure caused by respiratory muscle weakness in patients with neuromuscular disease (NMD) [75]. Supplemental oxygen therapy is recommended for ARF in acute and subacute neurological and muscular conditions producing respiratory muscle weakness [76].

When noninvasive strategies are not indicated or fail, invasive mechanical ventilation is applied. Since both excessive and insufficient unloading of the diaphragm are linked with diaphragm dysfunction and prolonged mechanical ventilation, adjusting ventilator support within the physiological range appears to be a logical approach. This strategy is known as “diaphragm protective ventilation” [77]. Maintaining diaphragm activity, however, cannot be uncoupled from avoiding lung injury by vigorous spontaneous efforts during the acute phase of respiratory failure, the so-called “self-inflicted lung injury” [78]. Limiting tidal volume as part of lung-protective ventilation in ARDS is standard of care and improves outcomes [79]. Hence, a conceptual framework of diaphragm and lung-protective ventilation has been proposed, which in theory should optimally protect both the lungs and the diaphragm. This involves measuring

inspiratory effort as well as lung stress and strain and titrating ventilator support, sedation and possibly extracorporeal gas exchanges techniques to optimise these factors. Although optimal effort remains to be established, some targets have been suggested, such as $P_{0.1}$ 1–4 cmH_2O, ΔP_{occ} 8–20 cmH_2O, ΔP_{es} 3–15 cmH_2O and tidal change in transdiaphragmatic pressure 5–15 cmH_2O, while maintaining safe tidal volume (4–8 $mL \cdot kg^{-1}$), driving pressure <15 cmH_2O or dynamic transpulmonary driving pressure <15–20 cmH_2O [80]. Weaning should be attempted as soon as possible [77].

Respiratory muscle training

IMT follows the same principles as training other skeletal muscles [81, 82]. IMT aims to improve strength, contraction velocity and endurance of the inspiratory muscles. IMT shows promise in enhancing or restoring respiratory muscle function in patients recovering from ARF. It can improve P_{Imax} and clinical outcomes, such as dyspnoea, physical functioning and lung function, and benefits patients with difficult weaning from mechanical ventilators, post-weaning and COVID-19 recovery, and those with NMDs [63, 83–89]. Common IMT protocols applied in patients with weaning difficulties vary in intensity, duration and frequency. Mechanical pressure threshold loading (TL) is the most commonly used technique, using a spring-loaded valve to provide a constant and flow-independent external load during inspiration [63]. Patients are typically instructed to exhale to residual volume and then perform inspirations against a load of 30–50% P_{Imax} [63, 90]. A novel alternative, tapered flow resistive loading (TFRL), has gained popularity as it shows promise in improving patient feedback and IMT efficacy [81, 91–96]. TFRL gradually decreases the external load during inspiration, allowing nearly constant intermediate flow rates over a larger range of lung volumes than TL [81]. A recent study in patients with weaning difficulties reported that high intensity IMT (~30% P_{Imax}) and (sham) low-intensity IMT (<10% P_{Imax}) were perceived as equally demanding by participants (self-reported respiratory effort) and were equally effective in terms of improvements in P_{Imax} and weaning outcomes [93]. These findings are in contrast with prior data from MARTIN *et al*. [97], who observed greater benefits on P_{Imax} and clinical outcomes with high-intensity IMT compared to sham low-intensity IMT. Differences may stem from differences in breathing instructions that were provided in the sham low-intensity IMT groups. MARTIN *et al*. [97] instructed patients to perform long and slow inspirations, whereas a recent randomised control trial (RCT) instructed participants to perform fast and deep inspirations. Additional assessments of ΔP_{es} and respiratory muscle sEMG during different IMT modalities revealed that performing slow inspirations against a low external load (comparable to instructions provided in sham control group from study of MARTIN *et al*. [97] resulted in relatively modest total inspiratory effort (ΔP_{es}) and respiratory muscle activation (sEMG), while performing inspirations fast and deep resulted in significantly higher total inspiratory effort and respiratory muscle activation regardless of the external load applied (high or low) [54, 98]. These findings indicate that the external load (as determined by the load set on the training device and quantified by airway pressure swings) might not be the main determinant of the total load imposed on the respiratory muscles of these patients during training. It also highlights that additional assessments of ΔP_{es} and sEMG can reveal important additional information about the actual workload that is imposed on the respiratory muscles during different breathing conditions in these patients.

Diaphragm neurostimulation-assisted ventilation in critically ill patients

Recently, combining positive pressure ventilation with simultaneous stimulation of the phrenic nerves has been proposed as a potential strategy to treat or prevent ventilator-induced diaphragm dysfunction, with possible additional benefits on lung-protective ventilation, haemodynamics and lung–brain interaction [99]. Transvenous stimulation of the phrenic nerves *via* the subclavian vein during mechanical ventilation was successfully and safely applied in a randomised trial of prolonged weaning patients as a method aimed to facilitate weaning from mechanical ventilation [100]. Although weaning success rates were not affected, P_{Imax} improved, likely reflecting improvement of diaphragm function [100]. Further data are awaited from a larger RCT applying this technology in failed-to-wean patients (NCT03783884). Additionally, the technique is promising to prevent diaphragm atrophy in the early stage of critical illness, where respiratory drive needs to be suppressed due to severity of illness and to allow lung-protective ventilation. Preliminary data showed that on-demand stimulation by the transvenous approach in this setting can reduce the time during which the diaphragm is inactive [101]. Hence this technology holds promise to simultaneously allow lung-protective ventilation and prevent diaphragm atrophy due to inactivity. Further studies are needed to confirm clinical benefits.

Assessment of respiratory muscle dysfunction in CRF

Techniques that do not depend on patient cooperation

Evoked manoeuvres for the assessment of airway opening, P_{es} and P_{ga}

Nonvolitional evaluation of diaphragm function and fatigue (*i.e.* a reduction in the ability to produce force/pressure following contractile activity) can be performed by supramaximal magnetic phrenic nerve

stimulation [102]. The resulting diaphragm contraction causes a sudden and short-lasting fall in P_{es} and a rise in P_{ga}, the difference providing $P_{di,twitch}$ [7]. Abdominal muscles can be evaluated by stimulation of supramaximal magnetic stimulation of thoracic nerve roots with measurement of gastric pressure $P_{ga,twitch}$ [103]. For technical considerations during these assessments, we refer the reader to the latest European Respiratory Society (ERS) statement on respiratory muscle testing [7]. A noninvasive estimate of $P_{di,twitch}$ can be obtained by measuring pressure changes in the upper airway or the mouth ($P_{mo,twitch}$) [104]. Resting values of $P_{ga,twitch}$ have a slightly higher variability (coefficient of variation 9–10%) than $P_{di,twitch}$ (6%) [7]. Age- and sex-specific normal values for adults are lacking, but a cut-off for $P_{di,twitch}$ of 20 cmH_2O has been suggested for diagnosis of diaphragm weakness (table 1) [102]. Studies involving nonvolitional evaluation of diaphragm and abdominal muscle function in patients with CRF have shown that these measurements are valuable for 1) diagnosing respiratory muscle weakness or paralysis [29, 103, 105], 2) evaluating the effectiveness of interventions aimed at improving respiratory muscle function [106, 107], 3) predicting the need for and response to mechanical ventilation [103], and 4) can even serve as a predictive biomarker for survival in patients with ALS [100]. The predictive power of SNIP as a less invasive measure was, however, also excellent in this setting [108].

Respiratory muscle ultrasound

Signs of chronic wear of the diaphragm, common in ventilated patients, can be assessed through DUS. Ultrasound presents good reproducibility in detecting loss of muscle mass and is easy to perform at the bedside [109]. Besides chronic anatomic changes, DUS could identify chronic dysfunction in the form of diaphragmatic fatiguability in myasthenia gravis patients [110]. In ALS/motor neuron disease, DUS has been studied as a promising alternative to pulmonary function tests for assessing respiratory function [111]. Moreover, DUS may be used to diagnose and assess for potential functional recovery from diaphragm weakness or diaphragmatic paralysis (figure 2) [26, 112].

Respiratory muscle EMG

In CRF (*e.g.*, COPD and interstitial lung disease), EMG analysis has elucidated mechanisms underlying breathlessness. Increased diaphragm EMG RMS is strongly associated with dyspnoea intensity in several patient populations [113, 114]. sEMG has also shown promise in predicting changes in clinical conditions, serving as a prognostic tool for both recovery and deterioration, as well as post-discharge outcomes [115, 116]. For instance, in the context of acute exacerbations of COPD, changes parasternal intercostal EMG RMS after discharged was independently associated with mortality and long-term oxygen use [115, 116]. Furthermore, EMG can help assessing the effectiveness of interventions, for instance with the use of IMT or NIV to reduce dyspnoea and neural drive/muscle activity [9, 46, 117].

Oesophageal and gastric pressure swings during spontaneous breathing and activities

ΔP_{es} and gastric pressure swings (ΔP_{ga}) help in identifying the degree of respiratory muscle dysfunction and like the EMG evaluating the effectiveness of interventions such as NIV and IMT [7]. ΔP_{es} quantifies the effort required by all inspiratory muscles to achieve a certain ventilation. Increased ΔP_{es} during inspiration may indicate increased respiratory muscle workload, which is common in respiratory muscle dysfunction [7]. However, increased or normal ΔP_{es} accompanied by low transdiaphragmatic pressure swings (ΔP_{di}) or an abnormal ($\geqslant$1) ratio of $\Delta P_{es}/\Delta P_{ga}$ (*e.g.* during $P_{di,sniff}$; table 1) can be indicative of specific diaphragmatic dysfunction or fatigue (*e.g.*, due to COPD or diaphragm paresis/paralysis) [7, 106, 118–121]. Increased ΔP_{di} after an intervention (*e.g.*, IMT) indicates improved diaphragm function [117]. Furthermore, an increase in the maximum ΔP_{es} value a patient can generate indicates an enhanced capacity of the respiratory muscles [7]. This can lead to a relative reduction in $\Delta P_{es}/\Delta P_{esmax}$ for a given activity, which is associated with greater exercise tolerance and reduced dyspnoea intensity [7, 117].

Techniques depending on patient cooperation

Voluntary manoeuvres for the assessment of airway opening, P_{es} and P_{ga}

As an alternative to P_{Imax}, the maximal SNIP can be recorded by a pressure transducer connected to a catheter placed in the nostril [7]. The test is performed at FRC. Peak cough flow (PCF) can help to estimate the effectiveness of mucus clearance and expiratory muscle function in neuromuscular disorders [7, 122]. PCF can help to inform the need to start manual/mechanical cough-assistance therapy with PCF <270 $L \cdot min^{-1}$ being associated with a higher likelihood of pulmonary complications in neuromuscular disorders [7, 123]. Healthy (children) adults achieve PCF of approximately 470–600 $L \cdot min^{-1}$ [7, 124]. PCF <160 $L \cdot min^{-1}$ is associated with a higher likelihood of extubation/weaning failure in neuromuscular disorders [7, 125]. Measurements of ΔP_{es} and ΔP_{ga} while sniffing and coughing are useful when noninvasive measures of in- and expiratory muscle function (*i.e.* P_{Imax}, P_{Emax}, SNIP and PCF) provide equivocal information [7]. These tests can help to confirm or refine diagnoses [7, 102]. Cut-off values for the diagnosis of weakness based on these assessments are available (table 1).

Spirometry

Pulmonary function tests, especially measurements of upright and supine vital capacity (VC) are noninvasive and readily available measurements contributing to the evaluation of respiratory muscle function, especially the diaphragm [17, 105, 125]. Unilateral diaphragm weakness is usually associated with a modest decrease in VC, to approximately 75% of predicted, with a further 10–20% decrease in the supine position [7, 26, 126–128]. Up to 15% reduction is typically considered the lower limit of normal (figure 2) [7, 26, 126–128]. In severe bilateral diaphragm weakness, VC is usually 50% predicted and can further decrease by 30% or more when supine [7, 26, 126–128]. A normal supine VC makes the presence of clinically significant diaphragmatic weakness unlikely [7]. A decrease in forced vital capacity has been shown to predict respiratory failure several days before intubation is needed in patients with Guillain–Barré syndrome [129].

Treatment of respiratory muscle dysfunction in CRF

Unloading respiratory muscles

Bronchodilator drugs are a cornerstone of the management of patients with chronic obstructive pulmonary disease, as they can reduce the WOB [68, 69]. Nevertheless, the treatment regimen should be individualised and guided by severity of symptoms and risk of exacerbations [130]. Long-term oxygen therapy improves survival in severe daytime hypoxaemia in COPD patients [131]. Supplemental oxygen during exercise for patients with COPD lowers the need for pulmonary ventilation, the strain on the respiratory muscles, the feeling of dyspnoea and the contraction of the pulmonary arteries [132–135]. This improvement of pulmonary mechanics through lifting of vasoconstriction will lead to increased systemic oxygen delivery and subsequently improve respiratory muscle function [136]. Oxygen therapy significantly improved respiratory muscle dysfunction in spinal cord injury patients [137].

In a recent study with stable COPD patients, high-flow nasal canula (HFNC) not only reduced the workload of primary exercise muscles but also indicated a reduction in oxygen consumption in the respiratory muscles [138]. The positive pressure delivered by HFNC could also help to unload the respiratory muscle pump by counterbalancing intrinsic PEEP induced by dynamic hyperinflation in COPD [139].

In patients with CRF, NIV can unload the respiratory muscles by reducing the number of required patient efforts and reducing the muscle load, for a given tidal volume, during an interactive assisted breath [140]. It should be noted that isolated nocturnal use of NIV in patients with stable CRF could lead to improvement of daytime respiratory failure [141]. Possible explanations include an improvement of the CO_2 sensitivity of the respiratory centres leading to an increased ventilatory drive [142], improvement of ventilation/perfusion ratio mismatch and recruitment of atelectatic lung parts [143], and improved respiratory muscle function by offsetting the direct influence of hypercapnia and hypoxia on muscle force and possible reduction of chronic respiratory muscle fatigue [144]. In COPD patients, a reduction of daytime hyperinflation has also been shown after nocturnal NIV use [145].

Although possible pathophysiological mechanisms are well described, the majority of studies evaluated clinical outcomes and quality of life. Based on different RCTs demonstrating decreased mortality and prevention of rehospitalisation, an ERS guideline conditionally recommend using long-term home NIV for patients with chronic stable hypercapnic COPD [146]. The intervention is also recommended for patients with COPD following a life-threatening episode of acute hypercapnic respiratory failure requiring acute NIV, if hypercapnia persists following the episode [146]. With regards to neuromuscular disorders, the use of long-term home NIV has been well established in NMDs for several decades [147]. Survival benefit was demonstrated in patients with ALS using NIV [148].

The load on the respiratory muscles can be lowered by reducing the ventilatory requirements. In addition to improving arterial oxygenation and lung mechanics by lowering airway resistance (bronchodilator treatment and airway clearance) and improving lung compliance (resolving atelectasis), ventilatory requirements can also be diminished by reducing the metabolic requirements during physical activity. Anaerobic metabolism and a rise in lactate concentration occurs during higher exercise intensity, especially in deconditioned people. This leads to an exponential rise in minute ventilation as a respiratory compensation mechanism [149]. Exercise training has been shown to improve muscle aerobic metabolism and reduce lactate accumulation and, consequently, minute ventilation [150]. This reduction is sensed as less demanding for the respiratory muscles and associated with reduced shortness of breath or dyspnoea [13]. This reduction in ventilatory requirement also enables patients to breathe at a slower pace, which is particularly beneficial for those with dynamic hyperinflation. It allows these patients to breath at a lower end-expiratory lung volume (EELV) and thus unload the respiratory muscles [151]. In addition, isolated lower limb resistance training reduces EELV, breathing frequency and dyspnoea [152]. More specific

breathing strategies, such as pursed lips breathing or slow and deep breathing, also reduced minute ventilation, respiratory rate [14] and inspiratory muscle activation in patients with COPD [153].

Respiratory muscle training

IMT can improve respiratory muscle function, symptoms of exertional dyspnoea and exercise capacity in patients with COPD [15]. In patients with stroke and spinal cord injury, training of both inspiratory and expiratory muscles can improve respiratory muscle function and symptoms, as well as helping to reduce the incidence of respiratory complications [154–157]. Even in patients with persisting diaphragm dysfunction, IMT has been show to result in improvements in inspiratory muscle function, exertional dyspnoea and exercise capacity, probably by improving (the coordination of the (partly) dysfunctional diaphragm with) extradiaphragmatic inspiratory muscles [106]. Similar findings have recently been obtained in long-COVID-19 patients with persisting symptoms and diaphragm dysfunction [107]. Although there are some indications that respiratory muscle training might improve some outcomes in NMDs, the very low certainty of evidence in available studies currently prevents drawing definitive conclusions [158, 159].

Discussion and conclusion

In the previous paragraphs we have provided a comprehensive overview of the clinical tools available for evaluating and treating respiratory muscle dysfunction, summarising both established techniques and recent methodological advancements. Notably, we highlighted the complementary roles of noninvasive assessment methods such as EMG and MUS, alongside established invasive and noninvasive pressure assessment techniques. While MUS is already frequently applied in clinical practice, EMG remains currently underused. The applications of these assessments are diverse, ranging from ARF to patients to those receiving chronic home mechanical ventilation. Currently available tools are useful for evaluating muscle function, titrating ventilatory support and guiding treatment decisions. MUS, in particular, is effective for bedside diagnosis and monitoring of diaphragm dysfunction, and it can predict clinical outcomes in critically ill, mechanically ventilated patients. EMG of the diaphragm and extradiaphragmatic muscles can optimise the dosing of unloading interventions and can help to optimise muscle stimulation during training interventions. Future advancements in information technology, hardware and software are expected to facilitate the broader adoption of these technologies in clinical settings in the years to come. If larger datasets, including results from multiple noninvasive and invasive respiratory muscle function assessments alongside clinical case data, become available, artificial intelligence based data analysis may emerge as a powerful tool in respiratory muscle function test interpretation. This could potentially help clinicians to refine and standardise diagnoses of respiratory muscle dysfunction in analogy with advances that have been made recently in pulmonary function test interpretation [160]. It could also potentially help to improve predictions of weaning and clinical outcomes in both acute and chronic settings by integrating results from multiple (non)invasive respiratory muscle assessments.

Points for clinical practice

- Evaluating respiratory muscle function is valuable for diagnosing, phenotyping and assessing treatment efficacy in patients with ARF and CRF.
- EMG and MUS play an increasingly important role alongside established invasive and noninvasive pressure assessment techniques.
- While MUS is already frequently applied in clinical practice, EMG remains currently underused.
- Both are useful tools for evaluating muscle function, titrating ventilatory support, optimising muscle stimulation during training interventions and guiding treatment decisions.

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