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State-of-the-Art Review

Assessing respiratory muscle effort during noninvasive respiratory support in acute hypoxemic respiratory failure: A State-of-the Art Review from physiology to bedside

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ABSTRACT

Objectives: Respiratory muscle effort is an important determinant of pathophysiology and clinical outcomes in patients with acute hypoxemic respiratory failure receiving noninvasive respiratory support (NIRS). Excessive effort reflects an imbalance between ventilatory load and muscle capacity and has been associated with patient self-inflicted lung injury, treatment failure, and worse outcomes, particularly when escalation to invasive ventilation is delayed. This review provides a clinically focused synthesis of the physiological determinants, bedside assessment, and practical implications of respiratory muscle effort during NIRS.

Methods: We included physiological, translational and clinical studies evaluating the assessment and clinical implications of respiratory muscle effort in patients with AHRF receiving NIRS. Evidence was selected to emphasize clinical applicability and bedside relevance.

Results: Clinical signs and simple indices remain essential for early detection of increased respiratory effort but lack specificity. They are widely available, scalable, and low-cost, although they require clinical experience for accurate interpretation. Advanced monitoring—including oesophageal pressure measurement, respiratory

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muscle ultrasound, and electrical impedance tomography—provides physiological insight but is variably available, more resource-intensive, and requires expertise.
A tiered approach to monitoring, integrating scalable bedside assessment with selective use of advanced tools, may represent a pragmatic framework for clinical implementation. NIRS modalities—including high-flow nasal oxygen, continuous positive airway pressure, and bilevel ventilation—affect respiratory effort through distinct mechanisms and may fail to adequately unload respiratory muscles if not carefully titrated. Persistently elevated effort may occur despite acceptable oxygenation and is associated with clinical deterioration.
Conclusions: Respiratory muscle effort should be considered a key component of patient assessment during NIRS. Structured and repeated evaluation using a combination of clinical signs and, when available, advanced monitoring, may help identify harmful breathing patterns and support timely escalation of care.
Future research should prioritise pragmatic monitoring strategies, actionable thresholds, and integration into routine practice across diverse healthcare settings.
Implications for clinical practice: Routine assessment of respiratory effort, integrating clinical observation with available monitoring tools, may improve titration of respiratory support, reduce the risk of delayed intubation, and support more individualized care in patients with AHRF. Embedding a tiered, feasibility-informed approach to respiratory effort monitoring may enhance translation from physiological insight to routine bedside care.

Introduction

Acute hypoxemic respiratory failure (AHRF) is a leading cause of intensive care unit (ICU) admission and a major contributor to morbidity, mortality, and healthcare burden [1,2]. It arises from a range of pulmonary and extrapulmonary conditions, including pneumonia and sepsis, and may progress to acute respiratory distress syndrome (ARDS) in the presence of bilateral lung infiltrates. Despite advances in supportive care, mortality in ARDS remains high, reaching up to 40%, and survivors often experience long-term functional impairment and reduced quality of life [1,3,4].

Noninvasive respiratory support (NIRS), including high-flow nasal oxygen (HFNO), continuous positive airway pressure (CPAP), and bilevel positive airway pressure (BiPAP), has emerged as a cornerstone of AHRF management. These strategies can reduce the need for invasive ventilation and its associated complications and, in selected patients, improve outcomes [5,6]. Their widespread use during the coronavirus disease 2019 (COVID-19) pandemic further underscored their clinical value [7,8]. However, failure rates remain substantial, particularly in de novo AHRF, affecting up to 30–50% of patients [9].

A key determinant of NIRS success is the balance between ventilatory load and respiratory muscle capacity. Disruption of this balance increases respiratory muscle effort, resulting in elevated work of breathing, dyspnoea, and, in some cases, patient self-inflicted lung injury (P-SILI) and patient–ventilator asynchrony [10–13]. Notably, excessive effort may persist despite apparently adequate oxygenation, potentially delaying recognition of worsening clinical status and contributing to adverse outcomes.

National investigations, including reports from the National Confidential Enquiry into Patient Outcome and Death [14] and the Health Services Safety Investigations Body (HSSIB) [15], have identified recurrent gaps in care, including delayed recognition of deterioration, inadequate monitoring of respiratory effort, lack of standardized escalation plans, variable staff competencies, and delayed referral to the ICU. These findings highlight a persistent gap between physiological understanding and clinical implementation.

This narrative review addresses this gap by focusing on three areas: (1) the physiological and clinical significance of respiratory muscle effort during NIRS; (2) methods for bedside assessment; and (3) integration into clinical decision-making to guide NIRS titration and timely escalation of care.

Physiological bases of respiratory muscle effort

Respiratory muscle effort reflects the energy expenditure and mechanical work required to sustain ventilation under an imposed load. It results from the interaction between three principal load components, resistive, elastic, and threshold, and the capacity of the respiratory muscles to overcome them. In AHRF, increased respiratory drive is driven not only by hypoxaemia and altered lung mechanics but also by systemic and pulmonary inflammation, including cytokine-mediated pathways and vagal afferent activation (**Fig. 1**). Patients compensate through increased inspiratory muscle recruitment to maintain ventilation.

Although initially adaptive, sustained high inspiratory effort increases energy expenditure and accelerates the development of respiratory muscle fatigue. This burden can be quantified by the work of breathing, defined as the integral of muscular pressure over volume, and by the pressure–time product, defined as the integral of muscular pressure over time. Together, these metrics reflect the mechanical and temporal components of respiratory effort. When mechanical load exceeds respiratory muscle capacity, compensatory changes in breathing pattern, such as increased respiratory rate and enhanced diaphragmatic recruitment, occur to delay fatigue.

Load components

Respiratory effort is determined by the mechanical loads that inspiratory muscles must overcome to generate airflow. In AHRF, these loads are substantially altered by underlying lung pathology and involve both the lung and chest wall. The three principal components are elastic, resistive, and threshold loads. Although these are described separately, they frequently coexist and interact dynamically with respiratory muscle capacity, neural drive, and the response to NIRS.

Elastic load

Elastic load represents the pressure required to expand the respiratory system against its recoil forces and include both lung and chest wall components. In AHRF the lung component is often dominant because alveolar oedema, consolidation, interstitial inflammation [10,16–19], and heterogeneous atelectasis reduce lung compliance and create regional mechanical disparities [17,19,20]. Chest wall mechanics may also contribute, particularly in patients with obesity, abdominal distension, pleural effusions, oedema, or reduced chest wall compliance. Increased chest wall elastance raises the pressure required to expand the respiratory system and may increase inspiratory muscle effort even when lung aeration appears only moderately impaired. Loss of functional residual capacity shifts ventilation to a less compliant portion of the pressure–volume curve [18]. As a result, greater negative pleural pressure swings are required to generate tidal volume, increasing respiratory rate and work of breathing [21].

Resistive load

Resistive load reflects the pressure required to overcome airway resistance. Although generally less dominant than elastic load in AHRF, it may increase significantly in the presence of tachypnoea, which

elevates inspiratory flow and resistance [22], airway narrowing or fluid accumulation [23], and airflow limitation due to secretions or upper airway narrowing [23]. Because resistive load rises disproportionately with increasing respiratory rate, rapid shallow breathing further amplifies inspiratory effort [13].

Threshold load

Threshold load represents the pressure required to initiate inspiratory flow. Intrinsic positive end-expiratory pressure (iPEEP) may develop in AHRF even in the absence of obstructive disease. It is primarily driven by tachypnoea with shortened expiratory time [24], heterogeneous regional time constants, and small airway closure requiring reopening pressures [24,25].

Inspiratory pressure requirements can be significantly increased by even low levels of iPEEP (2–5 cmH_2O), especially when respiratory rate exceeds 30–35 breaths per minute [26].

In AHRF, elevated elastic load, tachypnoea-related resistive load, and clinically relevant iPEEP act synergistically to increase respiratory muscle pressure generation, creating a high work of breathing state associated with NIRS failure [16,27–29]. When combined with increased neural drive, these factors promote rapid ventilatory demand–capacity mismatch, neuromechanical uncoupling, and respiratory muscle fatigue.

Respiratory muscle capacity

Respiratory muscle capacity refers to the ability to generate and sustain force. In addition to the diaphragm, inspiratory effort involves the coordinated activity of intercostal muscles, while accessory muscles such as the sternocleidomastoids are recruited under conditions of increased demand.

Expiratory muscle activity also contributes to the overall respiratory muscle response. In healthy quiet breathing, expiration is largely passive; however, in AHRF, abdominal and other expiratory muscles may be recruited to increase expiratory flow, reduce end-expiratory lung volume, or assist subsequent inspiration. While this may initially support ventilation, sustained expiratory muscle recruitment increases total respiratory muscle energy expenditure and may indicate severe load–capacity imbalance. It may also contribute to dynamic airway compression, intrinsic PEEP, or thoraco-abdominal asynchrony in selected patients [30].

Capacity depends on several factors. Muscle strength, typically assessed by maximal inspiratory pressure, may be reduced by malnutrition, sepsis, corticosteroid exposure, prolonged inactivity, and neuromuscular blockade [31]. Endurance, the ability to sustain submaximal contractions, is equally important and depends on adequate perfusion and metabolic reserve.

In acute respiratory failure, even when peak strength is preserved, reduced endurance may lead to early fatigue. Diaphragmatic geometry also plays a critical role: hyperinflation flattens the diaphragm and impairs its length–tension relationship, reducing efficiency. Neural drive modulates muscle recruitment through central respiratory centres; however, when mechanical output is impaired, increased drive may become maladaptive [32].

Two related concepts—neuromechanical and neuroventilatory coupling—describe the efficiency with which neural activation is translated into pressure generation and tidal volume, respectively [33–35]. Both may be severely impaired in conditions such as ARDS,

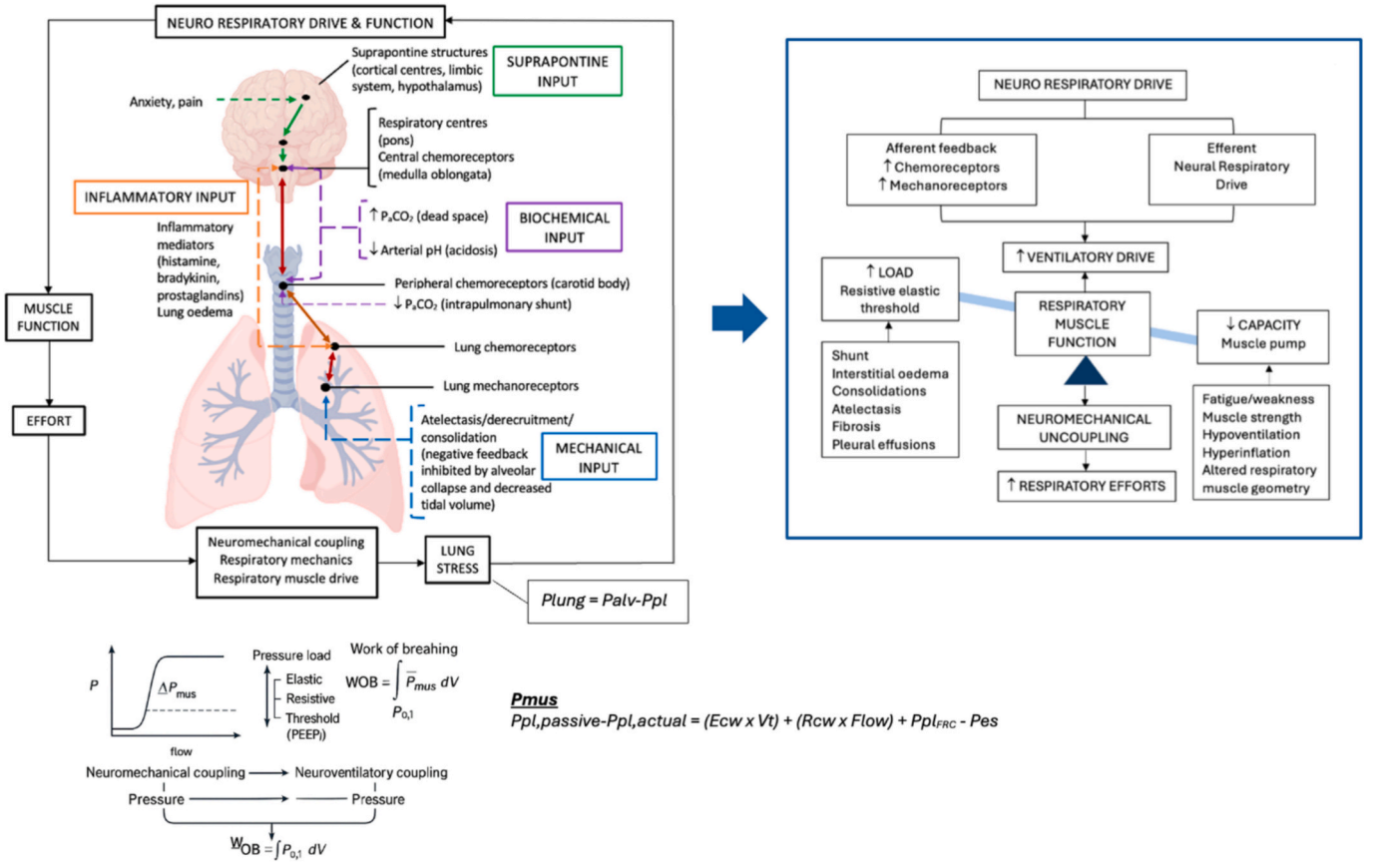


Fig. 1. Pathophysiological model in acute hypoxemic respiratory failure. The model links the pathological processes to physiological factors leading to respiratory muscle load–capacity imbalance and neuromechanical uncoupling. Abbreviations: P: Pressure; Pmus: change in muscle pressure; PEEP: positive end expiratory pressure; WOB: work of breathing; PdV: area under the pressure-volume curve; Ppl: pleural pressure; Ecw: chest wall elastance; Vt: tidal volume; Rcw: resistance of the chest wall; Pes: oesophageal pressure; P0.1: airway occlusion pressure at 0.1 s.

where reduced compliance limits volume generation despite high inspiratory pressures [8].

When respiratory muscle capacity is insufficient relative to load, fatigue ensues. Clinically, this is reflected by tachypnoea, shallow breathing, paradoxical respiratory movements, and accessory muscle recruitment (Fig. 2).

Respiratory effort and pressure–time product

Respiratory effort refers to the pressure generated by inspiratory muscles to overcome respiratory system loads [36,37]. At the bedside, it can be assessed using oesophageal pressure swings (ΔPes), transdiaphragmatic pressure, or validated surrogates such as airway occlusion pressure swing derived estimates of inspiratory muscles pressure and the pressure muscle index [25,38–43].

The pressure generated by respiratory muscles is clinically important not only as a marker of effort but also because it influences transpulmonary pressure. During spontaneous breathing, high inspiratory Pmus produces large negative pleural pressure swings. When these swings are transmitted unevenly across an injured, heterogeneous lung, they may increase regional transpulmonary pressure, promote pendelluft, and amplify local stress and strain (See Fig. 3). This provides the physiological link between excessive respiratory effort and effort-related lung injury, including P-SILI [44].

The energetic cost of breathing can be described by work of breathing, defined as the integral of pressure over volume ($\int P \times dV$), and by pressure–time product, defined as the integral of pressure over time ($\int P \times dt$). Work of breathing increases with reduced compliance, increased resistance, or larger tidal volumes, whereas pressure–time product reflects the cumulative inspiratory load and correlates with oxygen consumption and fatigue risk.

Although related, these metrics are not interchangeable: high inspiratory effort may occur with low tidal volumes (low work of breathing), whereas large tidal volumes may increase WOB despite modest pressure generation [36,45].

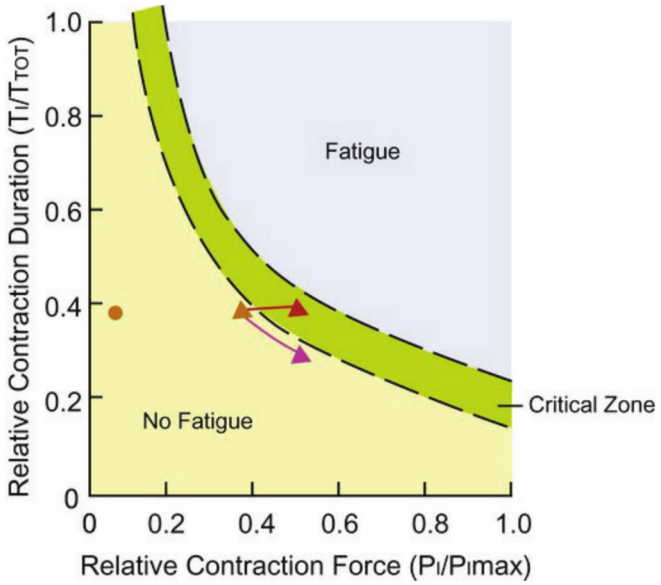


Fig. 2. The relationship between relative contraction force (PI/PImax) and relative contraction duration (TI/TTOT). The hatched area represents the critical zone in which fatigue develops. With progressive mechanical loading (e.g., exercise and acute exacerbation), the patient needs to increase PI and, therefore, might fall into the fatiguing zone (dashed arrow). This is generally avoided by a concomitant reduction in the duration of inspiration, TI/TTOT (solid arrow). This results in a decrease in tidal volume and a consequent increase in respiratory rate to maintain constant minute ventilation. Interventions aimed at reducing the load on the inspiratory muscles (PI) or increasing the maximum capacity of the inspiratory muscles (PImax) reduce the relative contraction force of the inspiratory muscles. This prevents muscle fatigue and, thus, respiratory insufficiency. (Modified from Bellemare F, Grassino A: Effect of pressure and timing of contraction on human diaphragm fatigue, J Applied Physiol 53:1190–1195, 1982 (32)).

Neural drive and neuroventilatory uncoupling

Neural drive refers to central respiratory activation originating from brainstem respiratory centres [16,46,47]. In AHRF, it is typically elevated due to hypoxaemia, hypercapnia, and metabolic acidosis. When neural drive cannot be effectively translated into mechanical or ventilatory output, uncoupling occurs.

Neuromechanical uncoupling describes a state in which neural drive is high, but pressure generation is inadequate, as seen in diaphragm dysfunction or hyperinflation [29]. Neuroventilatory uncoupling occurs when both neural drive and pressure are high, yet tidal volume remains low, as in severe ARDS with reduced compliance [16].

A third form, mechanical–stress uncoupling, arises when muscular effort generates disproportionate or heterogeneous transpulmonary stress due to regional differences in compliance, overdistension, and regional ventilation heterogeneity. In all cases, patients may exhibit marked respiratory distress despite limited effective ventilation. Persistent uncoupling during NIRS increases the risk of adverse outcomes, including respiratory muscle exhaustion and delayed intubation [48].

These mechanisms manifest differently depending on the underlying condition. In ARDS, reduced compliance leads to high elastic load and large pleural pressure swings, promoting *pendelluft* and regional overdistension, thereby increasing the risk of P-SILI [44,49]. In acute exacerbations of chronic obstructive pulmonary disease, threshold load due to iPEEP predominates and may be alleviated by appropriate expiratory pressure during bilevel ventilation. In sepsis or critical illness myopathy, reduced muscle capacity renders even moderate loads unsustainable, necessitating strategies that minimize effort and support recovery.

NIRS can modify these mechanisms by reducing one or more components of the load–capacity imbalance. HFNO may reduce resistive load through dead-space washout and flow matching; CPAP may reduce elastic and threshold load by increasing end-expiratory lung volume and counterbalancing airway closure or intrinsic PEEP; and BiPAP may provide direct inspiratory muscle unloading through pressure support. However, these effects are highly dependent on patient phenotype, interface performance, settings, tolerance, and synchrony [50]. Therefore, NIRS may either reduce effort or mask persistently excessive effort if monitoring is limited to oxygenation alone. These modality-specific mechanisms are discussed further in Section 4.

Assessment of respiratory muscle effort at the bedside

Understanding respiratory muscle effort is essential for interpreting patient response to NIRS. An increase in respiratory effort, whether identified through clinical assessment, simple indices, or advanced monitoring, signals failure of the balance between ventilatory demand and muscle capacity. Accurate evaluation can guide optimisation of NIRS and support timely management decisions [51].

Although advanced physiological tools provide quantitative measurements, bedside assessment remains fundamental. Given the heterogeneity of lung injury in AHRF, both insufficient and excessive inspiratory effort may indicate impending NIRS failure; however, they carry distinct risks. Excessive effort is the primary driver of P-SILI, through high negative pleural pressure swings, pendelluft, and regional overdistension. Insufficient effort — whether due to over-assistance, diaphragm weakness, or excessive sedation — carries separate risks including diaphragmatic atrophy, hypoventilation, atelectasis, and prolonged respiratory muscle weakness (Fig. 3). Assessment of respiratory effort therefore plays a central role in guiding NIRS titration (e.g.,

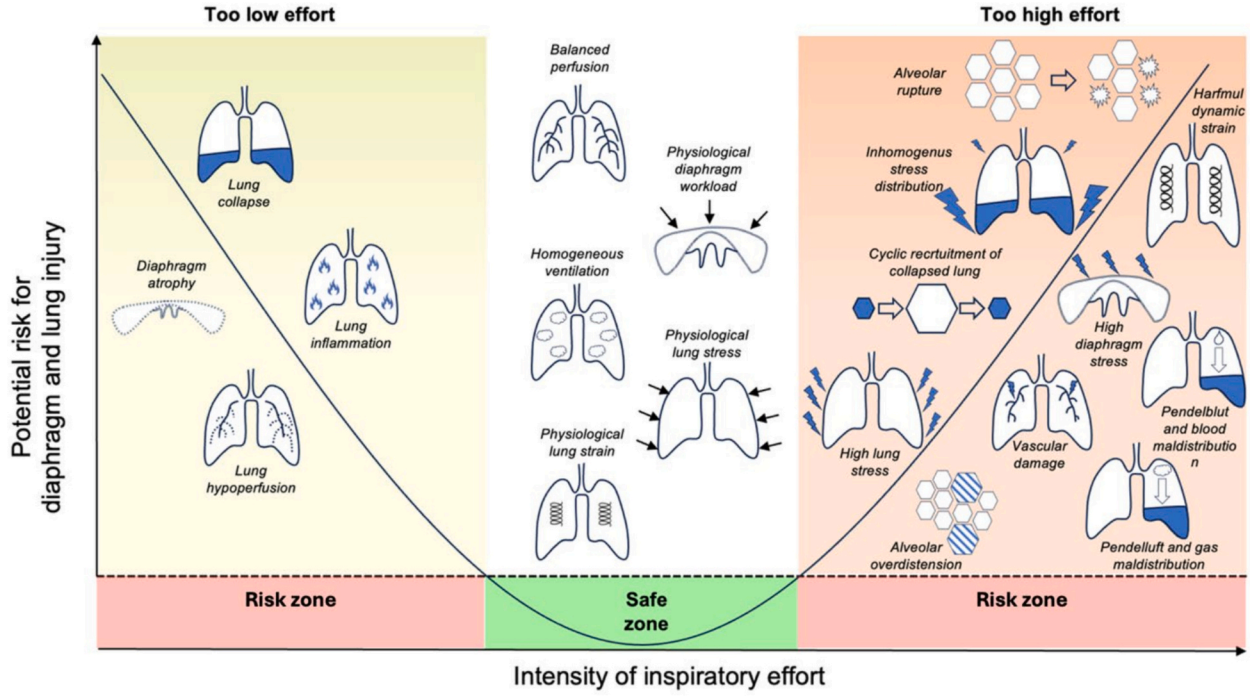


Fig. 3. Implication of intensity of inspiratory effort (from too low to too high effort) including potential risks for diaphragm and lung injury. Pendelluft refers to intraparenchymal gas redistribution during spontaneous inspiration, in which gas moves from non-dependent (faster-emptying) to dependent (slower-emptying) lung regions without entering the airways. In the setting of high inspiratory effort and heterogeneous lung injury, pendelluft can markedly amplify local transpulmonary stress in dependent regions, contributing to effort-related lung injury even when global tidal volume appears acceptable [Modified from Yoshida T, Torsani V, Gomes S, De Santis RR, Beraldo MA, Costa EL, Tucci MR, Zin WA, Kavanagh BP, Amato MB. Spontaneous effort causes occult pendelluft during mechanical ventilation. Am J Respir Crit Care Med. 2013 Dec 15;188(12):1420–7. doi:https://doi.org/10.1164/rccm.201303-0539OC].

interface selection, flow, and pressure settings), identifying early clinical worsening, and prompting escalation to invasive ventilation when required [39].

Bedside clinical evaluation and simple indices

Clinical evaluation remains the cornerstone of respiratory effort assessment and often provides the earliest indication of load–capacity imbalance. Tachypnoea (>30–35 breaths·min^{-1}) is a sensitive but non-specific marker of increased effort. Abnormal breathing patterns, including an elevated rapid shallow breathing index, reflect disproportionate effort relative to tidal volume [52]. Thoraco-abdominal asynchrony and abdominal paradox suggest diaphragmatic dysfunction or fatigue, while visible recruitment of accessory muscles indicates increased neural drive [16,53].

Subjective dyspnoea correlates with rising effort, and worsening breathlessness despite stable gas exchange may indicate neuromechanical uncoupling [53]. Altered mental status, including agitation or reduced alertness, may reflect respiratory exhaustion or gas exchange failure and is strongly associated with NIRS failure [54].

Simple indices complement clinical evaluation. The ROX index $(SpO_2/FiO_2)/RR$, validated in patients receiving HFNO, predicts failure when <3.85 at 12 h after initiation of HFNO (9). Maximal inspiratory and expiratory pressures provide estimates of respiratory muscle strength [55,56]. However, their reliance on patient cooperation limits their utility in acute settings. They are therefore better interpreted as measures of respiratory muscle capacity rather than real-time effort [55,56].

Additional physiological signals, including heart rate, dyspnoea scores, comfort, and interface tolerance, offer indirect insight into physiological reserve. Capnography and transcutaneous CO_2 monitoring can identify inadequate ventilation, particularly in patients with increased respiratory drive [57]. Importantly, the predictive value of these variables improves when assessed serially rather than in isolation.

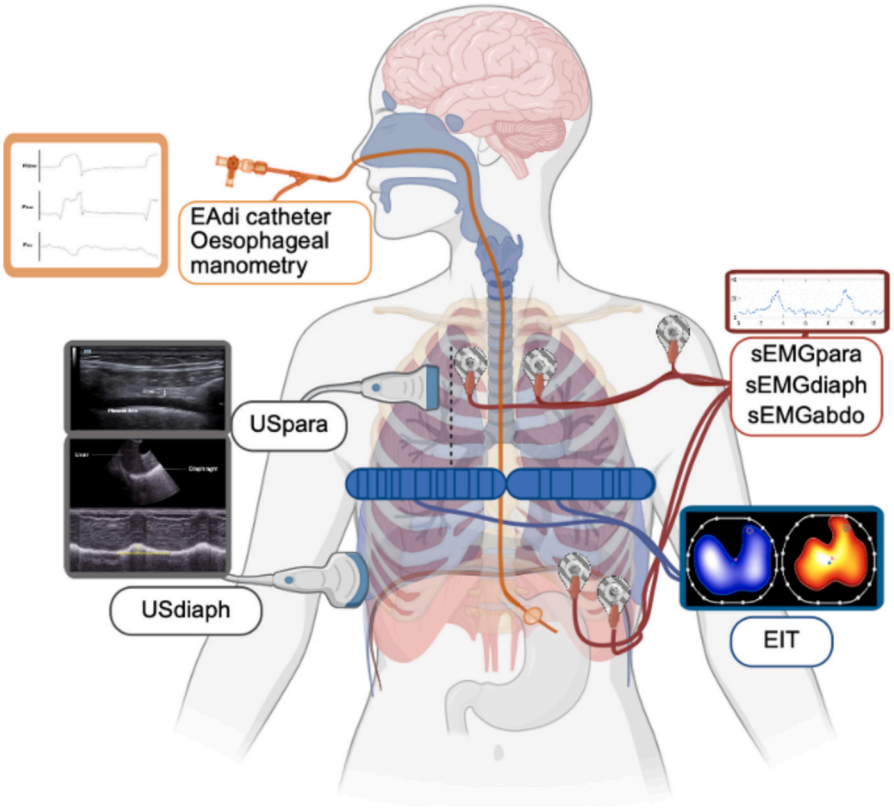
Composite indices may further refine bedside assessment. The ROX index integrates oxygenation with tidal volume to identify patients maintaining oxygenation at the cost of excessive Vt [58]. BREF models (base excess, respiratory rate, and FiO_2) have also been proposed to estimate inspiratory effort when invasive monitoring is unavailable (e.g., ΔPes) [59]. Regardless of the index used, tidal volume must be interpreted within the context of patient–ventilator interaction: persistently elevated Vt (e.g., >9 mL/kg predicted body weight), high inspiratory flow demand, or frequent asynchronies suggest excessive effort and should prompt rapid reassessment. Subjective dyspnoea should be explicitly and serially assessed using validated scales (e.g., visual analogue scale or numeric rating scale) [60], as worsening breathlessness despite stable gas exchange may indicate neuromechanical uncoupling and often precedes objective deterioration [61]. Additional physiological signals including heart rate, comfort, and interface tolerance offer further indirect insight into physiological reserve.

Advanced monitoring

Advanced monitoring techniques, although not universally available, allow quantitative assessment of respiratory effort and provide mechanistic insight into underlying physiological imbalance. They may facilitate earlier recognition of deterioration and guide individualized management. Fig. 4 summarises key modalities, and Table 1 provides relevant threshold values.

Oesophageal manometry

Oesophageal pressure measurement is commonly considered the physiological reference technique for quantifying inspiratory effort because it provides a direct estimate of pleural pressure swings and allows calculation of pressure–time product. Swings in ΔPes >10–15 cmH_2O or persistently elevated pressure–time product (>200 cmH_2O·s/



Monitoring instrument	OESOPHAGEAL MANOMETRY	EAdi CATHETER/ DIAPHRAGMATIC EMG	SURFACE EMG	US	EIT
Measure	Oesophageal and gastric pressure Transpulmonary/ transdiaphragmatic pressure	Diaphragmatic activation Neural timing Respiratory cycle	Parasternal activation Diaphragmatic activation Abdominal muscle activation	Muscular mass and movements Lung aeration and consolidations	Ventilation distribution Compliance/Homogeneity Ventilation-perfusion (V/Q) matching
Measurements	$P_{0.1}$ $P_L/\Delta P_L$ $P_{es}/\Delta P_{es}$ $PTP_{es}/\Delta PDI$ P_{gas}	ΔEadi ΔPdi/ΔEadi Pei index (Pmus/Eadi) Cdyn Asynchrony index	$EMG_{para}/\Delta EMG_{para}$ $EMG_{abdo}/\Delta EMG_{abdo}$ Asynchrony index	Parasternal thickness/ thickening fraction Diaphragmatic thickness/ thickening fraction Diaphragmatic excursion	Tidal volume EELI/ΔEELI GI index CoV%
Characteristics & settings	Invasive Disposable catheter Continuous/intermittent Used in ICU	Invasive Disposable catheter Continuous/intermittent N/A on HFNO Used in ICU	Non-invasive Disposable electrodes Continuous/intermittent Used in ICU & ward	Non-invasive Portable Reusable device/probe Intermittent Used in ICU & ward	Non-invasive Portable Reusable device/belt Continuous/intermittent Used in ICU & ward
Use	Evaluate respiratory drive/ effort Detect asynchronies	Evaluate respiratory effort/ drive Detect asynchronies Synchronise ventilation	Evaluate respiratory drive and fatigue Detect asynchronies	Evaluate lung aeration and oedema Assess muscular work	Assess lung volume & compliance Dynamic evaluation of global/regional ventilation and perfusion

Fig. 4. Characteristics of advanced monitoring instruments available at the bedside. Abbreviations: AI, asynchrony index; Cdyn, dynamic compliance; CoV%, center of ventilation (percent); ΔEAdi, change in electrical activity of the diaphragm; ΔEMGabd, change in abdominal surface electromyography; ΔEMGpara, change in parasternal surface electromyography; ΔEELI, change in end-expiratory lung impedance; ΔPdi, change in transdiaphragmatic pressure; ΔPes, change in oesophageal pressure; ΔPL, change in transpulmonary pressure; EAdi, electrical activity of the diaphragm; EELI, end-expiratory lung impedance; EMG, electromyography; EMGabd, abdominal surface electromyography; EMGpara, parasternal surface electromyography; EIT, electrical impedance tomography; EX, diaphragmatic excursion; GI index, global inhomogeneity index; HFNO, high-flow nasal oxygen; ICU, intensive care unit; P0.1, airway occlusion pressure at 0.1 s; Pdi, transdiaphragmatic pressure; Pes, oesophageal pressure; Pgas, gastric pressure; PL, transpulmonary pressure; PTPes, oesophageal pressure–time product; TF, thickening fraction; US, ultrasound; V/Q, ventilation–perfusion; Vt, tidal volume.

Table 1
Summary of bedside assessment methods and reported/proposed threshold ranges for inspiratory effort in AHRF during NIRS.

Methods	Threshold for low effort/over assistance	Threshold for high effort/under assistance
Gold standard reference methods		
Pes	< 3–5 cmH_2O (1,2)	>14–18 cmH_2O [25,41]
EAdi	EAdi-derived ΔPes < 5 cmH_2O (3)	EAdi-derived ΔPes > 10 cm H_2O [42]
Methods to assess respiratory drive		
sEMG	sEMG-derived ΔPes < 5 cmH_2O (3–5)	sEMG-derived ΔPes > 10 cmH_2O [42,43,64]
Methods to assess muscle effort		
ΔPnose	Not defined	> 5.1 cmH_2O [75]
BREF models	Not defined	Estimated ΔPes > 10 cmH_2O [59]
USdi	TFdi < 15% (8,9)	TFdi > 30–40% [96,97,116]
ΔCVP	Not defined	> 10–15 cmH_2O [72,73,115]
Vt	Not defined	> 9.0–9.5 ml/kg of predicted body weight [70]
Clinical assessment	Not defined	Dyspnoea-VAS > 3 and Dyspnoea-NRS ≥ 4 are associated with high P0.1 [60, 61]

Abbreviations: Pes, oesophageal pressure; EAdi, electrical activity of the diaphragm; ΔPes, change in oesophageal pressure; sEMG, surface electromyography; ΔPnose, nasal pressure swing (change in nasal pressure); BREF, breathing-effort model (breathing-effort estimate/model); USdi, diaphragm ultrasound; TFdi, diaphragm thickening fraction; ΔCVP, change in central venous pressure; CVP, central venous pressure; Vt, tidal volume; VAS, visual analogue scale; NRS, numerical rating scale; P0.1, airway occlusion pressure at 0.1 s.

min) despite optimisation indicate excessive and potentially harmful effort [29]. Although invasive, this technique provides detailed physiological information, particularly in complex cases. It should be noted that the threshold values cited above were primarily established in studies of invasively mechanically ventilated patients; direct prospective validation of these thresholds in spontaneously breathing patients receiving NIRS remains limited, and caution is warranted when extrapolating them to this population.

When Pes monitoring is unavailable, surrogate measures can be used, such as P0.1 (airway occlusion pressure at 100 ms) and ΔPocc (airway occlusion pressure swing), which provides an estimate of inspiratory effort [11,62].

However, despite its physiological validity, the practical application of oesophageal manometry in spontaneously breathing patients receiving NIRS is substantially more challenging than in invasively ventilated patients. Therefore, should not be interpreted as a simple bedside standard. Accurate measurement requires correct catheter positioning, balloon calibration, and validation of the signal. During non-invasive support, air leaks around the interface — particularly with helmet or mask CPAP/BiPAP — can alter intrathoracic pressure swings and confound the interpretation of ΔPes. Balloon placement and validation (e.g., by occlusion pressure or the cardiac oscillation method) may be difficult in awake, dyspnoeic patients, and patient discomfort or intolerance may impede catheter insertion or sustained monitoring. Upper airway contributions, and variable transmission of pressure may impair signal quality and introduce measurement artefacts. Motion artefacts related to coughing, swallowing, or positional changes further limit signal quality. Accurate interpretation requires expertise particularly in patients with complex respiratory mechanics, vigorous inspiratory effort, or patient–ventilator asynchrony. Such expertise and knowledge may not be widely available outside specialist centres. Solid-state oesophageal catheters are under active development and may mitigate some of these limitations in the future by improving signal fidelity and reducing placement artefact. They may reduce some technical limitations in the future, but their performance during non-invasive respiratory support requires further evaluation. These considerations mean that, while oesophageal manometry remains the reference standard from a physiological standpoint, its application may be confined to selected patients, specialist centres and research settings.

Electromyography and diaphragm electrical activity monitoring

Surface electromyography of parasternal intercostals and diaphragm electrical activity reflect neural respiratory drive. Increased electrical activity in the presence of low tidal volume is indicative of neuromechanical uncoupling [16,63,64] and identifies patients at risk of NIRS failure despite preserved oxygenation.

Neurally Adjusted Ventilatory Assist delivered non-invasively (NIV-NAVA) uses the EAdi signal as both the trigger and the proportional controller of pressure support, thereby coupling ventilator assistance directly to the patient's neural respiratory output [65]. This mechanism may offer the theoretical advantage of superior patient–ventilator synchrony compared with conventional pressure-triggered BiPAP, particularly in patients with high and variable neural drive [65–68]. Available evidence suggests that NIV-NAVA reduces the incidence of asynchrony events and may improve patient comfort in acute respiratory failure [65–68]. However, its clinical adoption remains limited by the requirement for a dedicated nasogastric catheter for EAdi signal acquisition, the technical expertise needed for catheter placement and signal optimisation, and restricted device availability outside specialist centres. Whether the synchrony benefits of NIV-NAVA translate into improved clinical outcomes in AHRF requires further prospective investigation.

In addition to EAdi monitoring via nasogastric catheter, surface electromyography (sEMG) applied to the chest wall or abdomen offers a fully non-invasive means of assessing respiratory muscle activation (Fig. 4). sEMG of the parasternal intercostals can quantify neural drive and has been used to assess neuro-mechanical and neuro-ventilatory coupling during both invasive and non-invasive ventilation [33,64]. sEMG of the abdominal muscles provides information on expiratory muscle recruitment, which, when present, indicates severe load–capacity imbalance [64]. Although sEMG signals require careful acquisition, filtering, and normalisation, and are susceptible to noise and crosstalk, they represent an emerging non-invasive modality that may complement EAdi-based monitoring in patients receiving NIRS.

Ultrasound of the diaphragm and respiratory muscles

Bedside ultrasound is a valuable, non-invasive tool for assessing respiratory muscle function. Diaphragmatic excursion correlates with inspiratory effort, while the diaphragm thickening fraction (DTF) primarily reflects contractility and the degree of muscle shortening during inspiration rather than the pressure generated against a pressure load. Although DTF does not directly quantify inspiratory effort in the way pressure-based measures do, high DTF values reflect intense diaphragmatic activation and, at the extremes, can serve as a clinically useful indicator of potentially excessive effort or, conversely, of insufficient muscle activation. Diaphragm thickening fraction >40% suggests high diaphragmatic activation consistent with elevated effort, whereas values <15–20% indicate weakness or over-assistance [36,63]. Importantly, the relationship between DTF and the actual pressure generated is influenced by respiratory system compliance: in patients with low compliance, high DTF may coexist with modest pressure swings and limited tidal volume, and DTF thresholds should therefore be interpreted alongside other effort indices. These thresholds for diaphragm thickening fraction (>40% for high activation; <15–20% for weakness or over-assistance) were established largely in mechanically ventilated patients, and their direct validation in patients receiving non-invasive support is limited; they should be interpreted with this caveat in clinical practice. Serial measurements can enhance clinical utility [69].

Ultrasound of parasternal intercostal muscles has been proposed an additional non-invasive surrogate of inspiratory effort [69], particularly when diaphragmatic imaging is limited. Several studies have reported correlations between parasternal intercostal muscle thickening ans

ΔPes, suggesting that it may reflect load–capacity balance and identify accessory muscle recruitment in a feasible and reproducible fashion [70,71]. However, the evidence base for parasternal intercostal ultrasound as a reliable and validated surrogate of inspiratory effort remains less established than for diaphragm ultrasound. Hoermann et al. [72] recently challenged the robustness of this correlation, reporting important limitations in reproducibility and criterion validity across patient populations. The approach requires standardized technique and operator training, and threshold values have not been prospectively validated in patients receiving NIRS. The use of parasternal intercostal ultrasound as a surrogate of inspiratory effort should therefore be considered exploratory, and its findings interpreted alongside other clinical and physiological parameters.

Electrical impedance tomography

Electrical impedance tomography (EIT) enables real-time, non-invasive assessment of regional ventilation. It can detect *pendelluft* and guide optimisation of ventilatory settings, including PEEP (See Fig. 3). Increased ventilation heterogeneity on EIT has been associated with worse outcomes [73,74].

Patient–ventilator interaction

Patient–ventilator asynchrony is both a consequence of high inspiratory effort — where neural demand outstrips the ventilator response — and a mechanism that further increases effort and neural drive, creating a vicious cycle of worsening load–capacity imbalance. Asynchronies, such as ineffective triggering, double triggering, and premature cycling, increase respiratory effort and discomfort [49]. This is particularly important in patients receiving non-invasive ventilation as synchrony between patient effort and ventilator support is critical. These events occur in up to 25–40% of breaths [75,76] and can be identified through waveform analysis combined with clinical observation. Optimisation of trigger sensitivity, rise time, and cycling criteria can reduce effort and improve comfort [49].

Central venous pressure swing

Central venous pressure swings may reflect pleural pressure changes and correlate with inspiratory effort [77]. In patients with central venous access, this signal may serve as a practical surrogate for ΔPes, although validation in AHRF remains limited [77,78].

Airway occlusion–derived indices

Indices such as P0.1 provide an estimate of respiratory drive, whereas ΔPocc offers a dynamic estimate of inspiratory muscle pressure (Pmus ≈ 0.75 × ΔPocc) [79]. It is important to note that P0.1 and ΔPocc require brief airway occlusion manoeuvres and are therefore measurable only in patients connected to a ventilator circuit — most practically in those receiving BiPAP or invasive mechanical ventilation. They are not routinely obtainable in patients receiving HFNO, where the open-circuit nature of the delivery system precludes occlusion without dedicated additional equipment.

However, current evidence is largely derived from bench and simulation studies, and from invasively ventilated patients; accuracy during non-invasive support may be further affected by leaks and interface characteristics [62,79]. Clinical validation of these indices specifically in patients receiving NIRS remains limited and is an important priority for future research.

Nasal pressure swing

Nasal pressure swing has emerged as a fully non-invasive surrogate of inspiratory effort. It reflects upper airway pressure changes and correlates with ΔPes and respiratory muscle activation [80,81]. Early clinical studies suggest good accuracy in detecting increased inspiratory effort, supporting its potential use in settings where invasive monitoring is not feasible [80,81].

An important limitation to note is that the strength of evidence differs substantially across monitoring techniques and clinical settings. Some approaches, such as oesophageal pressure measurement, nasal pressure swing, and selected physiological studies of high-flow nasal oxygen, continuous positive airway pressure, or non-invasive ventilation, have been evaluated directly in patients receiving non-invasive respiratory support. By contrast, ultrasound, electrical activity of the diaphragm, electromyography, and airway occlusion-derived indices have been developed or validated primarily in invasively ventilated or weaning populations. Their application during non-invasive respiratory support is therefore physiologically plausible but not always directly validated, and interpretation should account for interface-related leaks, patient tolerance, upper airway effects, and the specific mode of support.

Effort during non-invasive respiratory support

NIRS modulates respiratory effort through modality-specific mechanisms that act on different components of the load–capacity balance. Understanding these effects is essential, as suboptimal application may mask ongoing excessive inspiratory effort and increase the risk of P-SILI or delayed intubation (Table 2).

High-flow nasal oxygen

HFNO reduces respiratory effort through several mechanisms, including anatomical dead space washout, generation of low-level expiratory positive airway pressure (≈2–5 cmH_2O), improved matching of inspiratory flow demand, and enhanced airway conditioning through humidification [9,10,16,59,82].

In the FLORALI trial, the primary intubation outcome did not differ significantly between groups; however, HFNO was associated with improved outcomes in patients with $PaO_2/FiO_2 \leq 200$ mmHg and lower 90-day mortality in secondary analyses [10]. More recently, the SOHO trial found no significant reduction in 28-day mortality with first-line HFNO compared with standard oxygen therapy in unselected AHRF [83].

Physiological studies indicate that HFNO may provide insufficient reduction in inspiratory muscle load in ARDS patients [27,82]. In this setting, persistent accessory muscle use or paradoxical breathing suggests unsustainable effort and should prompt reassessment. Moreover, high inspiratory effort during HFNO may generate excessive lung strain, contributing to lung injury and clinical deterioration.

Because HFNO can improve oxygenation and comfort despite ongoing high inspiratory effort, reliance on oxygenation alone may delay recognition of treatment failure and appropriate escalation [84].

Continuous positive airway pressure

CPAP primarily reduces elastic and threshold loads by increasing functional residual capacity, promoting alveolar recruitment, and improving respiratory system compliance [85,86]. In AHRF and early ARDS, this may translate into reduced inspiratory effort and improved oxygenation. Importantly, CPAP also reduces the threshold inspiratory load imposed by iPEEP by providing a continuous positive baseline pressure that the inspiratory muscles no longer need to overcome before flow begins. This mechanism is clinically relevant even in patients without overt obstructive airway disease, as tachypnoea-related dynamic hyperinflation and small airway closure can generate significant iPEEP in AHRF. The combination of these two mechanisms — compliance improvement and threshold load reduction — means that CPAP can reduce inspiratory effort even when oxygenation and recruitment appear modest.

The physiological response to CPAP is highly dependent on interface, flow delivery, pressure stability, and patient tolerance [87,88]. When appropriately applied, CPAP reduces inspiratory effort by improving lung mechanics without promoting carbon dioxide (CO_2) rebreathing

Table 2
Impact of NIRS modalities on respiratory effort. The relative contribution of each mechanism depends on patient phenotype; threshold load reduction may be clinically significant even when recruitment effects are modest.

Modality	High-flow nasal cannula (HFNC)	Continuous positive airway pressure (CPAP)	Bilevel positive airway pressure (BiPAP)
Primary physiological mechanisms	Dead space washout Low-level positive airway pressure (≈2–5 cmH_2O) Flow matching to inspiratory demand	Constant positive airway pressure Increases FRC and recruits alveoli Offers intrinsic PEEP (PEEPi)	EPAP offers PEEPi and improves oxygenation IPAP provides pressure support
Load components influenced	↓ Resistive load ↓ Threshold load (mild)	↓ Elastic load ↓ Threshold load	↓ Threshold load ↓ Elastic load ↓ Direct inspiratory effort
Key clinical effects	Improves comfort and oxygenation Reduces inspiratory resistance and dyspnoea	Improves compliance, oxygenation, and reduces inspiratory threshold burden	Most effective for unloading respiratory muscles; reduces $PaCO_2$ in hypercapnic failure (e.g. COPD)
Limitations/ caveats	Limited unloading in high elastic load (e.g. ARDS); failure may be delayed without close monitoring	Reduce inspiratory effort via: 1- alveolar recruitment and improved respiratory system compliance, reducing elastic load 2- counterbalancing of intrinsic PEEP (iPEEP)	Patient–ventilator asynchrony frequent (25–40% of breaths); risk of gastric insufflation at high IPAP; requires close monitoring

Abbreviations: cmH2O: centimetres of water; ARDS; acute respiratory distress syndrome; FRC: functional residual capacity; PEEP: positive end expiratory pressure; EPAP: Expiratory Positive Airway Pressure; IPAP: **Inspiratory Positive Airway Pressure;** COPD: Chronic Obstructive Pulmonary Disease.

[89].

However, excessive PEEP may lead to alveolar overdistension and haemodynamic compromise through reduced venous return [18]. In addition, poor interface tolerance or air leaks may increase neural drive and inspiratory effort by impairing patient–device interaction [29].

Bilevel positive airway pressure

BiPAP provides the most direct reduction in inspiratory effort through inspiratory positive airway pressure (IPAP), while expiratory positive airway pressure (EPAP) improves oxygenation and counterbalances intrinsic PEEP. Its effectiveness depends on careful adjustment of ventilatory settings and optimisation of patient–ventilator interaction. Benefit is most likely when pressure support effectively unloads inspiratory muscles and synchrony is preserved. In contrast, ongoing high inspiratory effort despite non-invasive ventilation is strongly associated with treatment failure [90].

Physiological data indicate that BiPAP reduces respiratory effort only when synchrony is optimised. Asynchronies may instead increase respiratory effort and discomfort [49]. Persistent asynchrony should prompt adjustment of ventilator settings or consideration of escalation to invasive ventilation.

Integrating NIRS into effort-based management

Although NIRS can reduce respiratory effort, it may also permit prolonged exposure to excessive inspiratory effort when escalation is delayed. In AHRF, patients may maintain adequate arterial oxygenation through vigorous and sustained inspiratory effort, even in the presence of high respiratory load, impaired lung mechanics, and ongoing ventilatory demand–capacity imbalance. This scenario carries two major risks: respiratory muscle fatigue with delayed intubation, and effort-related lung injury and clinical deterioration. Sustained excessive effort can lead to muscle exhaustion, and delayed intubation in this context has been associated with worse clinical outcomes [38,69].

No NIRS modality eliminates the need for close and repeated reassessment. Structured evaluation windows (e.g., 1–2 h after initiation and at regular intervals thereafter) are essential to limit prolonged exposure to high effort. Integrating oxygenation indices (SpO_2/FiO_2, ROX), gas exchange, and direct markers of respiratory effort provides a more reliable basis for therapeutic decision-making [91].

Each modality affects respiratory effort through distinct physiological mechanisms: HFNO primarily reduces resistive load through dead-space washout and improved flow matching; CPAP reduces elastic and threshold loads through lung recruitment and counterbalancing of iPEEP; and BiPAP provides direct inspiratory muscle unloading via pressure support. However, all modalities may mask ongoing excessive effort, highlighting the critical importance of integrating serial effort assessment — rather than oxygenation monitoring alone — into routine clinical care.

Management strategies to optimise NIRS

Interface selection and fit

Interface selection is a critical determinant of NIRS performance [92,93]. Poorly fitted interfaces increase resistance, promote leaks, and exacerbate patient–ventilator asynchrony. Oronasal or full-face masks

generally provide more effective pressure delivery for both CPAP and BiPAP but may impair secretion clearance and reduce tolerance [94]. Helmet interfaces offer important advantages for CPAP delivery — including improved comfort, reduced leak, and better patient tolerance — and have demonstrated physiological benefits in AHRF and COVID-19 [8,80]. Helmet interfaces are most used for continuous positive airway pressure, although pressure-support ventilation through a helmet has been reported in selected settings. However, the high internal compliance of the helmet circuit substantially increases triggering delay during pressure-supported BiPAP, impairing patient–ventilator synchrony and limiting its use in this mode outside specialist centres with specific expertise and adapted ventilator algorithms [89]. The discussion of triggering dynamics and synchrony optimisation in therefore applies principally to oronasal or full-face mask BiPAP interfaces. Continuous monitoring for discomfort and skin injury is required.

Humidification

Dry gas delivery increases airway resistance and impairs secretion clearance. Active humidification, inherent to HFNO, improves comfort and reduces resistive load [95]. Heated humidification should also be considered during CPAP and BiPAP, particularly at higher pressures or during prolonged use [89,96].

Flow and pressure titration

Appropriate titration of flow and pressure is central to reducing inspiratory effort and preventing harmful breathing patterns.

For HFNO, flows of 40–50 L/min are commonly used and adjusted according to patient comfort and reduction in respiratory effort [97]. Higher flows enhance dead-space washout and generate low-level positive airway pressure but may cause discomfort or gastric distension. While HFNO can improve breathing pattern, insufficient unloading, particularly in conditions with high elastic load, may allow excessive effort to persist [10,85].

For CPAP, initial pressures of 5–8 cmH_2O are typically applied and titrated to improve oxygenation, functional residual capacity, and effort. Excessive pressures (>12–15 cmH_2O) may result in overdistension and haemodynamic compromise [98].

For BiPAP, inspiratory positive airway pressure (IPAP) usually starts at 10–12 cmH_2O and is increased incrementally to reduce accessory muscle activity and ΔPes, while avoiding excessive pressures (>20–24 cmH_2O) that may cause discomfort or gastric insufflation [76]. Adequate pressure support improves neuromechanical coupling and reduces pressure–time product, whereas suboptimal settings may increase respiratory effort [99,100].

Across all modalities, optimisation of patient–ventilator synchrony is essential. Asynchronies increase respiratory effort and neural drive and should be promptly corrected through adjustment of trigger sensitivity, rise time, and cycling criteria. Persistent asynchrony should prompt reassessment and consideration of escalation.

Strategies to reduce the risk of P-SILI include ensuring adequate PEEP/EPAP to offset threshold load and prevent derecruitment; limiting excessive tidal volumes (>9 $mL \cdot kg^{-1}$ per body weight); minimising leaks and asynchrony; using advanced monitoring tools such as ultrasound or EIT to detect regional overdistension or pendelluft [101,102]; and proceeding to invasive ventilation when excessive effort persists despite optimisation [13].

Supportive strategies

Positioning

Upright or semi-recumbent positioning improves diaphragmatic mechanics, reduces abdominal pressure, and enhances ventilation–perfusion matching. Lateral positioning may benefit patients with unilateral lung disease. Awake prone positioning can improve oxygenation and reduce regional heterogeneity in selected patients [103], although current evidence outside the COVID-19 population remains limited [104].

Secretion clearance

Retained secretions increase resistive load and impair synchrony. Strategies to enhance clearance include physiotherapy techniques, mobilisation, humidification, and airway suctioning when indicated [105]. Pharmacological adjuncts, such as bronchodilators, mucolytics, or hypertonic saline, may be considered in selected patients [106,107].

Adjunctive pharmacological therapies

Pharmacological interventions may reduce respiratory load or improve tolerance of NIRS. Bronchodilators decrease airway resistance in obstructive disease [108–110], while diuretics reduce pulmonary congestion and elastic load in cardiogenic oedema [111]. Conservative fluid management improves lung function in ARDS [111,112]. Carefully titrated anxiolysis or light sedation (e.g., dexmedetomidine) may enhance tolerance without suppressing protective respiratory drive [29].

Criteria to consider for escalation to invasive mechanical ventilation

Despite optimal non-invasive support, some patients continue to exhibit excessive respiratory effort. Early recognition of NIRS failure is therefore essential to avoid delays in escalation and reduce the risk of adverse outcomes. Indications for escalation to invasive mechanical ventilation include persistent tachypnoea (>35 $breaths \cdot min^{-1}$) with low tidal volume; marked accessory muscle recruitment or signs of respiratory muscle exhaustion; worsening hypoxaemia (PaO_2/FiO_2 <150 despite maximal support) or rising $PaCO_2$ with acidosis; deterioration in mental status or loss of airway protective reflexes; and sustained high inspiratory effort, as reflected by diaphragmatic thickening fraction >40–50%, ΔPes >15 cmH_2O, or elevated electrical diaphragmatic activity [29,113,114].

Delayed intubation following prolonged, unsuccessful NIRS is consistently associated with increased mortality and should be avoided [115].

Implications for clinical practice: A Stepwise Algorithm for Monitoring Respiratory Effort

Effective management of AHRF with NIRS requires a structured, stepwise approach to the assessment of respiratory effort, integrating bedside evaluation with progressively advanced monitoring according to clinical risk (**Fig. 5**).

Assessment should begin with systematic bedside observation, which represents the minimum standard for all patients. This includes serial evaluation of respiratory rate, accessory muscle activity, thoracoabdominal asynchrony, abdominal paradox, abdominal muscle recruitment, and dyspnoea. Visible retractions (intercostal, supraclavicular, and infraclavicular) further indicate increased pleural pressure and work of breathing. These findings provide early evidence of a mismatch between ventilatory load and muscle capacity and should be documented over time.

When abnormalities are detected or when HFNO is initiated [116,117], simple objective indices should be incorporated. The ROX index, applied serially, helps track trajectory, with values <3.85 suggesting a high risk of failure. Additional markers—including heart rate, patient comfort, and CO_2 monitoring—provide accessible indicators of rising respiratory drive and effort [118]. Interpretation should focus on trends over time rather than isolated values.

If clinical assessment and simple indices remain abnormal or inconclusive, intermediate physiological tools may be used to refine evaluation. Diaphragm ultrasound provides a direct estimate of

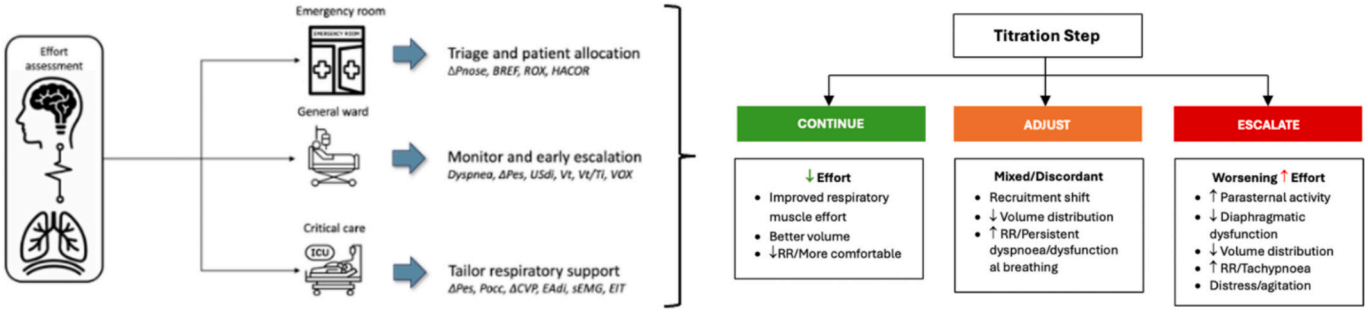


Fig. 5. A stepwise algorithm for monitoring respiratory muscle effort in patients with acute hypoxaemic respiratory failure requiring non-invasive respiratory supports. Abbreviations: Pnose: changes in nasal pressure; BREF: B (base excess), REspiratory rate, and FiO2 (fraction of inspired oxygen); ROX: the ratio of oxygen saturation as measured by pulse oximetry/FiO2 to respiratory rate; HACOR: Heart rate, Acidosis, Consciousness, Oxygenation, Respiratory rate; Pes: changes in oesophageal pressure; USdi: ultrasound of the diaphragm; Vt: tidal volume; Vt/Ti: tidal volume/inspiratory time; VOX: volume-oxygenation index; PoCC: occlusion pressure; CVP: changes in central venous pressure; EAdi: electrical activity of the diaphragm; sEMG: surface electromyography; EIT: electrical impedance tomography.

muscular effort, with diaphragmatic thickening fraction >40% indicating excessive effort and values <15–20% suggesting weakness or over-assistance [101,119]. Parasternal intercostal ultrasound and Pnose offer additional non-invasive surrogates of inspiratory effort.

In patients at high risk of deterioration or when escalation is being considered, advanced monitoring allows mechanistic assessment. Oesophageal manometry remains the reference standard, with ΔPes >10–15 cmH_2O or pressure–time product >200 cmH_2O·s/min indicating potentially injurious effort [22,56,62]. Central venous pressure swing may provide complementary information when central access is available [77,120].

Across all levels of monitoring, predefined thresholds for escalation are essential. Persistent high effort, manifested by tachypnoea, elevated diaphragmatic thickening fraction, increased ΔPes, declining ROX index, paradoxical breathing, or altered mental status, should prompt immediate reassessment and consideration of invasive ventilation, even when oxygenation appears preserved.

Finally, monitoring results should guide selection and adjustment of NIRS modality. HFNO is most appropriate for improving comfort and reducing resistive load; CPAP for promoting alveolar recruitment and reducing elastic load; and BiPAP when direct inspiratory muscle unloading is required. Aligning monitoring intensity with clinical risk and linking physiological targets to modality selection supports timely escalation, reduces delayed intubation, and ensures that NIRS is applied as a targeted, protective strategy rather than solely as an oxygenation tool.

Future directions and research priorities

Although the physiological determinants of respiratory muscle effort are increasingly well described, their translation into routine clinical practice remains incomplete. Future work should therefore move beyond demonstrating that respiratory effort matters, towards defining how it can be assessed reliably, affordably, and at scale. A pragmatic research agenda should consider both feasibility and clinical impact, recognising that not all monitoring approaches are equally available across healthcare systems.

Standardisation of respiratory muscle effort thresholds

There is currently no consensus on actionable cut-offs for indices such as diaphragm or intercostal thickening fraction [121], oesophageal pressure swings, or pressure–time product. Establishing pragmatic thresholds that correlate with clinically meaningful outcomes is essential. Prospective multicentre studies are needed to validate effort-based criteria for NIRS escalation.

Multimodal monitoring approaches

Emerging evidence suggests that inspiratory effort is best assessed using combined modalities rather than isolated measurements. Integration of clinical evaluation with ultrasound, EIT, or EMG may provide a more comprehensive assessment of the load–capacity balance.

A deeper understanding of the heterogeneity of lung injury [113] and morphological subphenotypes [114,122] in AHRF is also required. During spontaneous breathing, these factors likely influence the interaction between inspiratory effort and the response to different NIRS modalities. Future studies should determine whether multimodal "monitoring bundles" improve prediction of NIRS success and reduce delayed intubation.

Technology and automation

Wearable sensors and automated waveform analysis offer the potential for continuous, non-invasive monitoring of respiratory effort. Machine learning applied to ventilator waveforms or physiological signals may enable early detection of excessive effort and generation of actionable alerts. The accuracy, feasibility, and clinical impact of these approaches require prospective evaluation.

Implementation

Translation into practice will require structured education and system-level adaptation.

A tiered approach may help guide implementation. *Level 1 tools* include clinical observation, respiratory rate, accessory muscle use, thoraco-abdominal asynchrony, dyspnoea, ROX index, gas exchange, and simple physiological indices such as P0.1 where available [69]. These are low-cost, repeatable, scalable, and require training rather than major infrastructure. Future studies should prioritise standardising these bedside assessments, embedding them into observation charts and escalation pathways, and testing whether structured serial assessment improves recognition of NIRS failure.

Level 2 tools include point-of-care modalities such as diaphragm and parasternal intercostal ultrasound, and potentially surface electromyography. These require training and competency development but are increasingly feasible in many ICUs. Research should focus on reproducibility, minimum training standards, clinically meaningful thresholds, and integration with bedside signs rather than use as isolated measurements.

Level 3 tools include oesophageal manometry, electrical impedance tomography, continuous waveform analysis, wearable sensors, and AI-supported monitoring. These provide deeper physiological insight but remain limited by cost, expertise, availability, and technical complexity.

Their immediate role may be greatest in specialist centres, research settings, and high-risk patients where escalation decisions are uncertain.

Over the next five years, the most achievable priorities are likely to include: development of pragmatic bedside effort-monitoring bundles; validation of simple escalation thresholds; training programmes for respiratory effort assessment; and multicentre studies evaluating whether structured monitoring reduces delayed intubation. Longer-term priorities include continuous multimodal monitoring, automated detection of excessive effort or asynchrony, AI-supported decision aids, and integration of effort monitoring into personalised NIRS selection.

Future studies should also address implementation across different clinical environments. A monitoring strategy that is effective only in highly specialised ICUs may have limited global impact. The greatest clinical benefit may come from combining scalable bedside assessment with selective use of advanced tools, allowing respiratory effort to be recognised early while reserving complex monitoring for patients in whom the physiological picture remains uncertain.

By aligning physiology, feasibility, training, and implementation science, respiratory effort assessment can evolve from a specialist measurement into a practical component of routine NIRS care. This tiered approach may support safer escalation decisions, reduce delayed recognition of treatment failure, and promote more individualized respiratory support for patients with acute hypoxemic respiratory failure.

Conclusion

Respiratory muscle effort is a central determinant of outcomes in patients with AHRF receiving non-invasive respiratory support. The balance between resistive, elastic, and threshold loads and respiratory muscle capacity determines whether spontaneous breathing remains sustainable or progresses to fatigue and decompensation. Recognition of excessive effort is critical not only for predicting NIRS failure but also for preventing effort-related lung injury and associated adverse outcomes, in which vigorous inspiratory effort exacerbates lung injury and contributes to delayed, high-risk intubation.

Current evidence indicates that oxygenation indices alone are insufficient for monitoring. Bedside clinical assessment, complemented by advanced tools such as diaphragm or intercostal ultrasound, oesophageal manometry, and electrical impedance tomography, provides a more comprehensive evaluation of whether respiratory support is adequately unloading inspiratory effort.

Future research should prioritise standardisation of effort thresholds, validation of multimodal monitoring strategies, and integration of respiratory effort assessment into routine clinical practice.

Use of Artificial Intelligence

The authors declare that artificial intelligence tools were used to support figure preparation and language editing. BioRender was used to support the creation of figures. A generative artificial intelligence language model, ChatGPT by OpenAI, was used only for grammar refinement, language polishing, and improving clarity of expression. The authors reviewed, edited, and approved all AI-assisted text and take full responsibility for the scientific content, accuracy, interpretation, and conclusions of the manuscript.

CRediT authorship contribution statement

Brigitta Fazzini: Writing – review & editing, Writing – original draft, Visualization, Methodology, Conceptualization. **Roberto Tonelli:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Conceptualization. **Rik Gosselink:** Writing – review & editing, Writing – original draft, Visualization. **Alberto Lucchini:** Writing – review & editing, Writing – original draft, Visualization, Validation. **Alessandro Galazzi:** Writing – review & editing, Writing – original draft, Visualization. **Ema Swingwood:** Writing – review & editing, Writing – original draft, Visualization. **Dani Marti:** Writing – review & editing, Writing – original draft, Visualization. **Emanuele Rezoagli:** Writing – review & editing, Writing – original draft, Visualization, Methodology. **Frederique Paulus:** Writing – review & editing, Writing – original draft, Visualization. **Marcus J. Schultz:** Writing – review & editing, Writing – original draft, Visualization. **Patricia R.M. Rocco:** Writing – review & editing, Writing – original draft, Visualization. **Denise Battaglini:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Conceptualization.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: DB received lecturer fees from ESTOR, not related to this work. Given his role as Associate Editor, Alberto Lucchini was not involved in the peer review of this article and had no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to another journal editor. BF hold a Doctoral Research Fellowship grant awarded by the UK National Institute of health and Research (NIHR). However, this study was not funded by this grant. Additionally, the NIHR had no role in the design, collection, analysis or interpretation of the data, nor in writing or decision to submit the manuscript. All other authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

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