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Chronic obstructive pulmonary disease and dysphagia: how are they connected, and what is the role of physical and rehabilitation medicine? a narrative review

Abstract

Chronic obstructive pulmonary disease (COPD) is a leading cause of morbidity and mortality worldwide, and its economic and social burden is projected to keep rising over the next decades. Beyond airflow limitation, COPD is increasingly recognized to affect swallowing, and oropharyngeal dysphagia is now understood as an underdiagnosed comorbidity that may itself contribute to disease exacerbations. This narrative review summarizes current evidence on the pathophysiological mechanisms linking dysphagia and COPD, the prevalence of swallowing impairment across disease stages, available screening and diagnostic tools, and the impact of dysphagia-focused rehabilitation. A literature search of MEDLINE/PubMed, Scopus, and Embase was conducted for studies published through June 2026. The evidence indicates that impaired breathing–swallowing coordination, reduced laryngopharyngeal sensitivity, and COPD-related biomechanical changes of the thorax and upper airway increase the risk of penetration and aspiration, and that dysphagia is associated with a higher frequency of COPD exacerbations; prospective cohort data indicate that this relationship is not merely cross-sectional, with detectable aspiration predicting subsequent severe exacerbations. Only a minority of patients are formally screened for dysphagia, despite the availability of validated, low-cost tools. Emerging intervention studies suggest that structured dysphagia education and rehabilitation, integrated into pulmonary rehabilitation programs, can improve swallowing-related quality of life and functional status, although the evidence base for specific therapeutic techniques remains limited. Given its expertise in functional rehabilitation, physical and rehabilitation medicine (PRM) is well positioned to lead the integration of dysphagia screening and management into multidisciplinary COPD care.

Keywords: COPD, chronic obstructive pulmonary disease, dysphagia, deglutition, deglutition disorders, exacerbations, aspiration, rehabilitation

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Marta Moreira da Silva, João Machado, Maria Bonito, Ana Afonso, José Couto, Filipa Pereira
Department of Physical and Rehabilitation Medicine, Santo António University Hospital Center, Portugal

Correspondence: Marta Moreira da Silva, Department of Physical and Rehabilitation Medicine, Santo António University Hospital Center, Largo Professor Abel Salazar, Porto, Portugal

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Abbreviations: COPD, chronic obstructive pulmonary disease; PRM, physical and rehabilitation medicine; AECOPD, acute exacerbation of chronic obstructive pulmonary disease; GOLD, Global Initiative for Chronic Obstructive Lung Disease; VFSS, videofluoroscopic swallow study; FEES, fibre-optic endoscopic evaluation of swallowing; EAT-10, Eating Assessment Tool-10; CAT, COPD Assessment Test; FOIS, Functional Oral Intake Scale; SWAL-QoL, Swallowing Quality of Life questionnaire; mMRC, modified Medical Research Council dyspnea scale; NIV, non-invasive ventilation; BiPAP, bilevel positive airway pressure; EMST, expiratory muscle strength training; ESE, expiration-swallow-expiration pattern; IFC-TESS, interferential current transcutaneous electrical sensory stimulation; PCO2, partial pressure of carbon dioxide; EMG, electromyography; ASPEKT, Analysis of Swallowing Physiology: Events, Kinematics, and Timing method

Introduction

Chronic obstructive pulmonary disease (COPD) is a leading cause of global morbidity and mortality, characterized by chronic, largely irreversible airflow limitation driven mainly by tobacco smoking, occupational and environmental exposures, and adverse socioeconomic conditions.[1] It presents with cough and dyspnea and is diagnosed by post-bronchodilator spirometry showing an FEV1/FVC ratio below the lower limit of normal.[1] A less widely recognized but clinically important consequence of COPD is its effect on deglutition, affecting the dynamics and coordination of swallowing.[2] COPD prevalence is estimated at 10.3% worldwide[3] and ranks among the top three causes of global mortality, with most deaths occurring in low- and middle-income countries.[1] Its burden is projected to keep rising, with the number of people living with COPD growing by roughly 23% by 2050, global costs exceeding US$1.3 trillion annually by 2025, and annual exacerbations rising from about 456 million in 2025 to nearly 568 million by 2050;[4] exacerbations are therefore a central target for disease management. Dysphagia is itself common in the general population, affecting roughly one in six adults.[5] Swallowing is a complex, largely involuntary process occurring up to 600 times a day and involving more than 30 muscles,[6] tightly coordinated with breathing: breathing pauses briefly at the moment of swallowing and resumes in the expiratory phase, protecting the lower airway from aspiration.[6–8] Loss of this coordination is associated with laryngeal penetration (entry of material above the vocal cords) and/or aspiration (entry below the vocal cords), with risk of aspiration pneumonia.[7] Patients with COPD are particularly prone to this desynchrony because of ventilatory dysfunction and altered thoracoabdominal biomechanics, often showing oral and pharyngeal stasis, delayed swallowing reflex, decreased laryngeal elevation, and greater risk of penetration and aspiration.[2,7,9,10] Laryngeal aspiration during swallowing is thought to contribute to exacerbation of COPD symptoms,[11–15] and swallowing abnormalities are linked with more frequent exacerbations,[2,10,16] although whether they are already

present in mild disease remains unclear.[17] Despite this, only 4-5% of COPD patients are referred for formal swallowing evaluation,[20,21] and screening for dysphagia is recommended to identify patients at higher exacerbation risk.[19] Recent studies have refined prevalence estimates, clarified underlying mechanisms, and reported the first randomized evidence on dysphagia-focused rehabilitation in COPD. This narrative review synthesizes current evidence on the pathophysiology, prevalence, screening/diagnostic tools, and rehabilitation impact of dysphagia in COPD, informing the role of physical and rehabilitation medicine (PRM) in the multidisciplinary management of these patients.

Methods

This narrative review was chosen to allow a broader, clinically oriented synthesis across pathophysiology, prevalence, screening, and rehabilitation. A literature search was performed in MEDLINE/PubMed, Scopus, and Embase, combining the terms ("Deglutition Disorders"[Mesh] OR "Deglutition"[Mesh] OR swallowing OR dysphagia) AND ("Pulmonary Disease, Chronic Obstructive"[Mesh] OR pulmonary emphysema OR lung emphysema OR COPD exacerbations), covering studies published over the last 35 years through June 2026, with no language restriction; reference lists of retrieved articles were also hand-searched. Studies were included if they screened for or assessed oropharyngeal dysphagia or aspiration pneumonia in adults with COPD, or evaluated the relationship between dysphagia and COPD exacerbations, its mechanisms, or its rehabilitation. Studies addressing exclusively esophageal dysphagia, gastroesophageal reflux without oropharyngeal involvement, dysphagia attributable to an independent neurological disease, head and neck cancer, tracheostomy or upper airway surgery, pediatric populations, and opinion pieces or case reports without extractable data were excluded from the primary evidence base (though a few are cited elsewhere for context). Methodological features of each study (sample size, comparison group, assessment method) are described narratively in the text or, where not covered there, in Table 1.

Table 1 Summary of selected studies evaluating swallowing function in COPD

Study	Population (age/etiology/groups)	COPD definition and diagnostic technique	Swallowing assessment	Main conclusion
de Deus Chaves et al.[40]	Stable COPD (N=20; M=10/F=10; mean age 59, SD 4.1) vs healthy controls (N=20; M=10/F=10; mean age 59, SD 4.4)	GOLD criteria; spirometry; body plethysmography	Videofluoroscopic swallow study (VFSS): 3, 5, 10 mL liquid; 7 mL paste; solid dipped in 2.5 mL paste	Stable COPD patients may show physiological adaptations (protective swallowing maneuvers) to avoid aspiration; valleculae residue alone is not a reliable isolated marker in this population
Cassiani et al.[27]	COPD (N=16; mean age 68) vs 15 nonsmoking healthy controls (mean age 65)	Clinical manifestations and pulmonary function tests	Clinical and videofluoroscopic evaluation; duplicate swallows of 5 and 10 mL liquid, paste, and solid boluses	COPD patients have a longer pharyngeal swallowing phase than controls, associated with a reduced difference between maximal laryngeal elevation duration and pharyngeal transit duration
Mancopes and Steele[41]	Stable COPD (N=28; M=18/F=10; mean age 65) vs healthy age/sex-matched controls	GOLD criteria	Videofluoroscopy of thin, mildly, moderately, and extremely thick boluses; parameters per the ASPEKT method	COPD patients showed measurable differences in swallowing physiology relative to healthy aging controls across bolus consistencies
Eichhorn et al.[42]	Moderate-to-severe COPD (N=25; all male; mean age 69, SD 8) vs healthy controls (N=25; M=12/F=13; mean age 64, SD 9)	Spirometry	Simultaneous respiratory and surface EMG monitoring (mylohyoid, anterior belly of digastric, geniohyoid)	COPD patients more often swallowed solids during inhalation and semisolids at low tidal volumes, with shortened deglutitive apnea and subsequent inhalation after swallowing
Prestes et al.[43]	Clinically stable COPD in pulmonary rehabilitation (N=23; mean age 60.4, SD 9.9); mild (N=4), moderate (N=8), severe (N=8), very severe (N=3)	GOLD criteria	Eating Assessment Tool (EAT-10); COPD Assessment Test (CAT); peak flow	EAT-10 showed a positive, moderate relationship with health status measured by CAT: better health status was associated with lower likelihood of altered swallowing
Shaker et al.[35]	N=31 in 3 groups: COPD during exacerbation (1A) and stable state (1B) (N=10, age 51-75); healthy elderly (N=11, age 63-83); healthy young controls (N=10, age 18-34)	Not specified	Submental surface EMG with concurrent respirography	Group 1A swallowed more frequently during inspiration and resumed respiration more often on inspiration than 1B and group 2; the deglutition/respiration index was significantly smaller in 1A than 1B
Macri et al.[44]	COPD patients (sample recruited from a pulmonary outpatient clinic)	Clinical diagnosis	Clinical swallowing evaluation and fibre-optic endoscopic evaluation of swallowing (FEES)	Clinical and endoscopic assessment identified swallowing alterations in COPD patients, supporting instrumental evaluation as more sensitive than clinical judgment alone

VFSS, videofluoroscopic swallow study; EMG, electromyography; ASPEKT, Analysis of Swallowing Physiology: Events, Kinematics, and Timing method; EAT-10, Eating Assessment Tool-10; CAT, COPD Assessment Test; FEES, fibre-optic endoscopic evaluation of swallowing.

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Prevalence of dysphagia in COPD

Reported prevalence of dysphagia in COPD ranges widely (17-78%), depending on population, disease stage, and assessment method (self-reported versus instrumental).[22] A 2022 meta-analysis estimated pooled prevalence of oropharyngeal dysphagia in COPD at 32.7% (95% CI 30.1-35.4%, I^2=91.5%), identifying dyspnea, gastroesophageal reflux disease, xerostomia, reduced physical capacity, and elevated inflammatory markers as risk factors.[23] A 2026 meta-analysis of 18 studies (4608 patients) estimated pooled prevalence at 38.2% (95% CI 29.8-46.6%, I^2=97.3%), identifying older age, higher mMRC/CAT scores, missing teeth, malnutrition, smoking history, exacerbation stage, and gastroesophageal reflux as risk factors, while non-invasive ventilation use was associated with lower odds.[22] This wide range and high heterogeneity indicate that true prevalence remains uncertain and highly method-dependent. Dysphagia symptoms may already be present in mild COPD, although swallowing difficulties are not always recognized or self-reported, so aspiration should be monitored from an early disease stage.[17] Clinical observation may miss dysphagia in the absence of overt signs; videofluoroscopy is sometimes required to detect silent aspiration.[2]

Good-Fratturelli et al. found that of 1996 COPD patients, only 4% had been referred for videofluoroscopic swallow study (VFSS), of whom 85% had swallowing alterations, although 23% had a history of stroke, a potential confounder.[20] Ghannouchi et al. reported 17-42% of COPD patients had aspiration episodes, highest in those with cerebrovascular disease.[16,20] Gonzalez Lindh et al, using a questionnaire and water-and-biscuit test, found 78% had swallowing dysfunction, more pronounced with moderate-to-severe airflow limitation.[8] Drozdz et al., using VFSS in patients with chronic cough, found 20% with mild and 6.7% with mild-to-moderate dysphagia.[24] More recent data confirm that swallowing impairment remains common even after clinical stabilization: a 2026 video fluoroscopic cohort study of patients recovering from acute respiratory hospitalizations found aspiration in about one-quarter overall, and severe airway invasion (Penetration-Aspiration Scale 6-8) in nearly half of the COPD subgroup, with semisolid-consistency aspiration and vallecular residue as the strongest predictors.[25] Some earlier studies found no aspiration at all, but these generally used smaller bolus volumes and compensatory maneuvers (e.g., prolonged breath-holding), suggesting that contrast volume, concentration, and viscosity influence detection.[20,26,27] Taken together, the evidence supports dysphagia as a common, likely underestimated comorbidity of COPD, though larger, standardized studies are needed to establish its true prevalence.

Underlying pathophysiological mechanisms

Several, likely interacting, mechanisms may predispose patients with COPD to penetration and aspiration.[28] **Reduced laryngopharyngeal sensitivity.** Cvejic et al. found that COPD patients present pharyngeal stasis and greater risk of penetration and aspiration with larger liquid volumes (100 mL) on videofluoroscopy.[9] Clayton et al. demonstrated significantly reduced laryngopharyngeal mechanosensitivity in COPD versus healthy controls, independent of disease severity, proposing inhaled corticosteroids and smoking-related laryngeal edema as contributors,[29] and subsequently linked this to delayed detection of pharyngeal residue and increased aspiration risk during oral intake.[30] A 2026 fiberoptic endoscopic study in 29 patients with severe COPD found deficits in at least one swallowing mechanism (most often delayed pharyngeal reflex triggering) in over half, with penetration/aspiration in 4/29 (silent in 3); dysphonia and dysphagia were markedly underestimated by self-report versus objective assessment, reinforcing the case for systematic instrumental screening even without complaints.[31]

Disrupted breathing-swallowing coordination. COPD can alter breathing-swallowing coordination through dyspnea and altered thoracoabdominal biomechanics, producing tachypnea, an increased tendency to swallow during inspiration, reduced deglutitive apnea duration, and altered swallowing mechanics.[6,32] In healthy individuals, an expiration-swallow-expiration (ESE) pattern predominates, exhaling after swallowing to clear pharyngeal residue and providing maximal airway protection;[33,34] this pattern may be disrupted when respiratory muscles are overloaded or PCO2 rises, as in neuromuscular disorders or COPD. Both stable and exacerbated COPD show increased respiratory rate versus healthy adults,[35] with more pre-swallow inspirations as rate rises;[9,35] higher respiratory rate and lower baseline oxygen saturation are predictors of aspiration.[9] A 2024 study in an anesthetized rat model of COPD found increased respiratory duty cycle and inspiratory swallow-reflex frequency, with altered digastric/thyrohyoid muscle activity but no visible tissue damage, suggesting functional rather than structural changes underlie swallowing difficulty.[36] A 2025 study in patients with severe-to-very-severe stable COPD found the ESE pattern still predominant (≈80% of 400 swallows across 21 patients) but with significantly prolonged respiratory cycle times, suggesting ventilatory adaptation rather than pattern substitution even in advanced disease.[16,37]

Anatomical and biomechanical changes. Emphysema causes air trapping and lung hyperinflation, while chronic bronchitis-related inflammation destroys parenchyma and decreases elastic recoil, contributing to airway obstruction. Hyperinflation flattens the diaphragm, restricts its movement, alters thoracoabdominal biomechanics, and requires recruitment of accessory inspiratory muscles, which overload rapidly; these changes affect laryngeal and pharyngeal position, with potential functional consequences for swallowing.[2] Experimentally, acutely increasing lung volume in healthy volunteers prolongs swallow latency and reduces swallowing frequency during continuous pharyngeal water infusion, indicating that hyperinflation can modulate the swallowing reflex independently of structural airway pathology.[38] Pulmonary hyperinflation also raises intra-abdominal pressure, altering the diaphragm-lower esophageal sphincter relationship - a plausible mechanism for the increased gastroesophageal reflux disease, and consequent esophageal dysphagia, seen in COPD.[2]

Motor deficits. Motor impairments described include reduced tongue control and strength, delayed pharyngeal swallow, reduced tongue base retraction and laryngeal elevation, slowed vestibule closure, abnormal swallow reflex, penetration/aspiration, and cricopharyngeal dysfunction.[32] However, one study found no difference in isometric tongue strength between COPD patients and controls, but a delayed inhibitory reflex response of inspiratory muscles to airway occlusion specifically in patients with video fluoroscopic airway invasion, suggesting impaired neural/reflex timing, rather than primary muscle weakness, may better explain some deficits.[39]

Screening and diagnostic assessment tools

Dysphagia in COPD is frequently under-recognized, and several low-cost, non-instrumental screening tools exist. The Eating Assessment Tool-10 (EAT-10) correlates with health status as measured by the COPD Assessment Test (CAT),[43,45,46] and a Portuguese-language adaptation, alongside the Functional Oral Intake Scale (FOIS), is available for Portuguese-speaking populations.[47] Water swallow tests and bedside protocols combining cookie and

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water swallows help identify patients at high exacerbation risk warranting further evaluation.[19,48,49] In hospitalized AECOPD patients, a predictive model combining age ≥74 years, mMRC ≥2, length of stay ≥7 days, and BiPAP use achieved good discrimination for swallowing risk.[50] A 2026 explainable machine-learning model, validated in 710 COPD patients (208 with dysphagia), identified disease duration, body mass index, tracheal intubation history, muscle strength, and mMRC score as key predictors (AUC 0.921), yielding a web-based calculator for early screening.[51]

When screening is positive, or silent aspiration is suspected despite normal clinical signs, instrumental assessment is warranted. Video fluoroscopy (VFSS) remains the reference standard, visualizing bolus transit, penetration, and aspiration across consistencies/volumes and grading airway invasion via the Penetration-Aspiration Scale.[9,52] Fibre-optic endoscopic evaluation of swallowing (FEES) and laryngopharyngeal sensory discrimination testing have also characterized sensory deficits,[30,44,53] and radionuclide salivagram SPECT/CT has been explored as a complementary, quantifiable alternative for visualizing salivary aspiration during exacerbations.[54] Despite these tools, only a small minority of COPD patients are formally screened or referred for instrumental assessment.[10,20] The stakes of missed dysphagia are considerable: in a broader dysphagia population, both aspiration severity and silent aspiration independently predicted subsequent aspiration pneumonia and reduced survival, reinforcing the case for proactive rather than symptom-triggered screening.[55]

Dysphagia and acute exacerbations in COPD

Discoordination of the ventilation-swallowing pattern increases ventilatory muscle overload, creating a vicious cycle.[10,42,56] COPD patients swallow less frequently using the protective ESE pattern,[16] requiring longer pauses and shortened apnea time to maintain breathing rhythm, compromising swallowing efficacy.[57] Nagami et al. found that swallowing during inspiration (I-SW) and inspiration immediately after swallowing (SW-I) indicate breathing-swallowing discoordination and are strongly associated with exacerbation frequency, though these patterns can rarely occur in healthy individuals too.[18] Steidl et al. highlighted this relationship:[2] patients with an abnormal swallow reflex present significantly more exacerbations, suggesting dysphagia is both a risk factor for exacerbations and worsened by them, in a bidirectional relationship.[32] The strongest evidence for a genuinely predictive, not merely cross-sectional, relationship comes from a prospective cohort of 151 patients with stable COPD, who underwent videofluoroscopic assessment during 100 mL cup drinking and were followed for 12 months. Detectable aspiration, present in roughly one-fifth, was associated with a markedly higher rate of severe exacerbations over the following year (50% vs 18.2% in non-aspirators; OR 4.5, 95% CI 1.9-10.5); silent aspiration carried a smaller but significant risk (OR 2.6, 95% CI 1.1-6.2). These findings support aspiration as a predictor of severe exacerbations, though the authors caution that causality still needs testing.[58]

A 2026 meta-analysis pooling seven cohort studies (669 patients) quantified this association directly: dysphagia was associated with more than double the risk of AECOPD (pooled rate ratio 2.37, 95% CI 1.75-3.20; p<0.00001) and a higher annual exacerbation frequency (mean difference 1.13 episodes/year, 95% CI 0.88-1.38). The effect was attenuated but remained significant after trim-and-fill adjustment for publication bias (rate ratio 1.85, 95% CI 1.35-2.53), and results were robust across subgroup analyses. This corroborates the prospective signal above and strengthens the case for dysphagia as a modifiable, screenable risk factor for AECOPD.[59] More recent hospital-based evidence reinforces this link: a predictive model incorporating age, dyspnea severity, length of stay, and BiPAP use achieved AUC 0.909 for high swallowing risk in AECOPD patients, supporting systematic bedside screening.[50] Gonzalez Lindh et al. found markedly higher self-reported and clinically detected swallowing dysfunction in AECOPD patients versus hospitalized cardiac controls, with swallowing speed roughly three times slower, further supporting nurse-led screening during acute admissions.[48] Feeding a patient with respiratory failure is delicate, given the precarious balance between swallowing apnea and dyspnea; this is more complex with non-invasive ventilation (NIV), since many patients cannot tolerate mask removal long enough to eat, and a conventional ventilator can deliver an unexpected insufflation at the end of the pharyngeal swallow phase. Many such patients ultimately require nasogastric tube feeding, which brings its own discomfort and complications and makes NIV mask sealing harder. Terzi et al. showed that maintaining NIV via nasal mask during oral feeding, using a ventilator adaptation that deactivates swallow-triggered insufflations, improved ventilation-swallowing dynamics: besides reducing hypercapnia, increased tidal volume and maximal expiratory pressure raised subglottic pressure, improving swallowing efficacy and safety. This study used water only, so whether NIV improves solid food intake during exacerbations remains undemonstrated.[56]

Impact of dysphagia rehabilitation in COPD

McKinstry et al. divided 632 COPD patients into three groups: group 1 (n=253) received education on breathing-swallowing function, dysphagia symptoms, and management strategies; group 2 (n=383) underwent dysphagia screening (104/383, 27%, had symptoms/signs of dysphagia); and group 3 (n=55) underwent dysphagia intervention, completing the SWAL-QoL questionnaire. Integrating education, screening, and intervention improved swallowing-related quality of life, with significant gains in the 'Burden of Dysphagia' (p<0.009), 'Physical Problems of Dysphagia' (p<0.012), and 'Managing Diet Options/Food Selection' (p<0.016) subscales.[10] For patients with ventilation-swallowing incoordination, training toward the protective ESE pattern may be useful.[18,42] Though not directly tested in COPD, Martin-Harris et al. showed ventilation-swallow coordination could be improved using biofeedback (visual stimuli encouraging protective-pattern swallows with controlled volume) in patients with chronic dysphagia after head and neck cancer surgery, significantly increasing optimal-pattern swallows - suggesting such training could plausibly reduce COPD exacerbations, though this remains untested in COPD populations.[60] More recent evidence begins to fill this gap: a 2026 randomized controlled trial allocated 90 elderly COPD patients with dysphagia to usual care or a 12-day teach-back-based health education program covering swallowing organ, ingestion, and respiratory function training. Swallowing symptom frequency itself did not differ significantly between groups, but the intervention group showed significantly better health status (CAT 19.6 vs 23.4), daily living ability (Barthel Index 82.9 vs 68.8), and swallowing-related quality of life. This is among the first randomized evidence for structured dysphagia education in COPD; its quality-of-life benefits echo McKinstry et al.'s observational findings, but the lack of symptom-frequency improvement suggests education alone may not suffice to modify underlying impairment, warranting further trials targeting that outcome directly.

Expiratory muscle strength training (EMST) targets expiratory muscle weakness that may impair airway protection during swallowing. However, a 2026 survey of speech-language pathologists found near-universal awareness of EMST (99%) but implementation

by only about half (53%), with just 20% using objective measures (e.g., manometry) to guide therapy; most who used EMST followed an informally-adopted 'rule of fives' protocol (5×5 repetitions, 5 days/week, 5 weeks, at 75% maximal expiratory pressure), and fewer than a third had formal training. This gap, attributed to limited training and inconsistent manometry access, illustrates the broader challenge of translating promising techniques into standardized practice.[62] A more mechanistic proposal comes from Oku, who suggested that non-invasive physiological approaches — low-pressure continuous positive airway pressure and transcutaneous electrical sensory stimulation (IFC-TESS) — may counteract the airway-protection deficits underlying breathing-swallowing discoordination [63]. This is grounded in physiological reasoning rather than controlled COPD trial data and remains untested in this population, but outlines a distinct, non-behavioural avenue complementing the education- and exercise-based approaches above.

Discussion

The prevalence of dysphagia in COPD varies widely (17-78%) depending on population and methodology, with pooled meta-analytic estimates near 32.7-38.2%;[22,23] true prevalence remains uncertain given the substantial heterogeneity and methodological limitations of available studies. Patients with swallowing disorders may be entirely asymptomatic; altered laryngopharyngeal sensitivity may explain their lack of awareness of dysphagia's presence and severity [64]. Coughing, a main warning sign of aspiration, is also a frequent COPD symptom itself, so patients may not attribute value to it, potentially masking aspiration [8]. Even on clinical evaluation, patients may show no signs and still have silent microaspiration detectable only by videofluoroscopy, the reference standard.[8] Given the association between dysphagia and increased exacerbation frequency — now supported by prospective evidence that aspiration predicts subsequent severe exacerbations and by pooled evidence of more than doubled AECOPD risk[58,59] — early detection appears likely to help reduce exacerbations and mortality.[2,32] Given COPD's high prevalence and resource constraints, universal screening may not be practical; patients most likely to benefit include those with frequent exacerbations (≥3/year), aspiration pneumonia, weight loss, malnutrition/cachexia, feeding-related warning signs, or self-reported swallowing changes, who should be referred for videofluoroscopy.[19,20] Identifying this 'exacerbator phenotype' may allow more targeted intervention, interrupting the vicious cycle between dysphagia and exacerbations.

Given this clinically meaningful relationship, it seems pertinent to include dysphagia rehabilitation within respiratory rehabilitation programs to reduce microaspiration-related exacerbations. Maneuvers taught to patients include bolus volume restriction, dry swallows after eating, diet modification, supraglottic swallow, and postural changes.[20,21] Recent randomized evidence supports that structured, multimodal dysphagia education improves health status and swallowing-related quality of life in COPD;[61] however, the literature remains too limited and heterogeneous in technique and outcome measures to identify which specific treatment most reduces exacerbation risk.[19,62] This is precisely the gap where physical and rehabilitation medicine (PRM) has a natural role. The European PRM specialty body (UEMS PRM Section) has formally recommended that PRM physicians lead multidisciplinary pulmonary rehabilitation and that biopsychosocial assessment specifically include swallowing function, listing dysphagia interventions among the 'add-on' PRM interventions for pulmonary rehabilitation.[65] Extending this competency to systematic screening and to prescribing/supervising swallowing-focused rehabilitation (with speech-language pathology) would integrate this under-addressed comorbidity into standard COPD care, rather than leaving it to opportunistic, symptom-triggered referral.

As with any narrative review, this synthesis carries its own limitations: the literature search and selection were conducted by a single reviewer without independent dual screening, and no formal risk-of-bias or quality-appraisal tool was applied, consistent with the narrative design chosen. This cannot exclude selection bias in which studies were identified or emphasized, and findings from methodologically stronger and weaker studies were not weighted differently.

Conclusion

COPD is a growing public health problem, with tobacco smoking as its leading cause, and cigarette consumption frequency associated with greater airflow limitation.[2] Important limitations remain across the reviewed literature: heterogeneous diagnostic and assessment criteria, heterogeneous samples, and small sample sizes — consistent with a broader pattern, since geriatric studies link oropharyngeal dysphagia to multimorbidity and polypharmacy independent of any single respiratory diagnosis, complicating attribution to COPD alone.[66] Despite these limitations, the evidence fairly consistently demonstrates an association between dysphagia and COPD exacerbations, and recent studies (2021-2026) have reinforced and refined, rather than overturned, this picture. Dysphagia diagnosis requires a high level of clinical suspicion in COPD to enable timely rehabilitation. While the most effective treatment approach for reducing exacerbation risk remains unclear, both ventilation-swallowing coordination training and structured multimodal dysphagia education show promise. Recognizing this association underscores the importance of early detection, both to identify the 'exacerbator phenotype' and to reduce patient and healthcare burden. Further research should standardize methodology and determine the real impact of dysphagia-focused intervention on COPD outcomes, and whether dysphagia severity correlates with COPD severity, clarifying which patients would most benefit from early intervention. This gap is increasingly recognized elsewhere: a registered systematic review protocol on dysphagia risk factors in elderly COPD patients was published in 2025.[67] Given its central role in pulmonary rehabilitation, PRM is well placed to lead this integration in routine practice.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

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