AAVBC

American Academy of Value Based Care

Chronic Respiratory Failure

Quick Coding Guide

AAVBC Chronic Respiratory Failure - Quick Reference Guide

Table of Contents

1. CLINICAL SNAPSHOT

Definition: Chronic Respiratory Failure is clinically defined as a long-standing, persistent inability of the respiratory system to maintain adequate gas exchange, resulting in chronically low arterial oxygen (hypoxemia) or high carbon dioxide (hypercapnia) levels. Unlike the acute form, which is a sudden medical emergency, the chronic definition requires that the condition has developed over a period of days, weeks, or longer, typically as a result of underlying diseases like COPD, neuromuscular disorders, or interstitial lung disease.[1,2]

ICD-10 Codes and HCC/RAF V28: Chronic respiratory failure is coded by subtype: J96.10 (unspecified), J96.11 (with hypoxia), J96.12 (with hypercapnia), all mapping to HCC 283, RAF 0.282. Acute-on-chronic respiratory failure (J96.20–J96.22) maps to HCC 282, RAF 0.783, reflecting the added clinical complexity and resource intensity. A critical requirement for capturing these chronic codes is the clinical documentation of long-term oxygen or ventilator dependence, ensuring the medical record reflects the patient's ongoing need for life-sustaining respiratory support.

ICD-10 CODE	HCC CATEGORY	RAF WEIGHT	DOCUMENTATION REQ.
J96.10 (Chronic respiratory failure, unspecified)	HCC 283 — Chronic Respiratory Failure, Chronic	0.282	**Avoid unspecified when gas exchange type is identifiable.** Document hypoxia (SpO2 ≤88% on RA) or hypercapnia (PaCO2 ≥45 mmHg) when present.
J96.11 (Chronic hypoxemic respiratory failure — Type I)	HCC 283 — Chronic Respiratory Failure, Chronic	0.282	Document supplemental O2 use or formal requirement (e.g., home oxygen order). Confirm chronicity — not an acute-only event.
J96.12 (Chronic hypercapnic respiratory failure — Type II)	HCC 283 — Chronic Respiratory Failure, Chronic	0.282	Document CO2 retention (elevated PaCO2 on ABG) or BiPAP/ventilator dependence. Confirm chronicity — not an acute episode alone.
J96.21 (Acute-on-chronic hypoxemic respiratory failure)	HCC 282 — Acute-on-Chronic Respiratory Failure	0.783	Use during acute inpatient or emergency exacerbation in a patient with established chronic respiratory failure. Sequence the underlying chronic condition as an additional code.
J96.22 (Acute-on-chronic hypercapnic respiratory failure)	HCC 282 — Acute-on-Chronic Respiratory Failure	0.783	Common in COPD exacerbations and obesity hypoventilation syndrome (OHS). Confirm acute worsening of established chronic hypercapnic failure. Sequence underlying chronic code additionally.

Prevalence: Chronic respiratory failure, frequently driven by progressive diseases like COPD, represents a significant public health challenge in the United States. As of 2017, the incidence rate reached approximately 1,275 cases per 100,000 adults, reflecting the widespread prevalence of the condition. Mortality rates for adults aged 45 and older have seen a sharp upward trend, climbing from 3.71 per 100,000 in 1999 to 10.50 per 100,000 in 2023. As of 2023, this mortality risk remains slightly more pronounced in men (11.14 per 100,000) compared to women (9.94 per 100,000).[3,4]

2. RECOGNITION AND DIAGNOSIS

Medicare Screening/Diagnostic Tools and CPT Reference

Screening Tools[5–8]

1. Symptom-Based Screening Tools

Because chronic respiratory failure is often a "silent" progression of an underlying disease, these tools help clinicians identify subtle signs of respiratory muscle fatigue or poor gas exchange. **See appendix links to tools.**

Tool	Description	Primary CPT Code	Medicare Coverage
CAT	A 10-item questionnaire that measures the impact of symptoms like cough, phlegm, and chest tightness on a patient's life. A high score (typically >10) suggests a higher risk of complications	96160	Covered (often part of the Annual Wellness Visit or a Chronic Care Management plan)
mMRC	A simple 0–4 scale used to grade the severity of breathlessness. Higher grades correlate with an increased risk of respiratory failure	96160	Covered (often part of the Annual Wellness Visit or a Chronic Care Management plan)
CAPTURE Tool	Designed specifically for primary care, this tool combines a 5-item questionnaire with **Peak Expiratory Flow (PEF)** measurements to identify patients with undiagnosed, clinically significant COPD	96160 + 94150	Covered as a screening service in primary care for at-risk patients
STOP-BANG	While primarily for Obstructive Sleep Apnea (OSA), it is frequently used to screen for nocturnal hypoventilation, a precursor to chronic hypercapnic respiratory failure	96160	Covered; used as the clinical justification for a subsequent sleep study (PSG)

Abbreviations: CAT, COPD Assessment Test; CPT, Current Procedural Terminology; mMRC, Modified Medical Research Council; NIV, Non-Invasive Ventilation; PSG, Polysomnography; STOP-BANG, Snoring, Tiredness, Observed apnea, high Blood pressure, BMI, Age, Neck circumference, Gender

2. Clinical "Red Flag" Screening (Physical Signs)

For patients with known chronic conditions, clinicians screen for these specific clinical markers during routine exams:

- **Morning Headaches:** A classic indicator of nocturnal CO_2 retention (hypercapnia)
- **Orthopnea:** Difficulty breathing when lying flat, which may signal diaphragm weakness
- **Paradoxical Breathing:** The abdomen moving inward rather than outward during inhalation, indicating respiratory muscle exhaustion

3. Objective Physiological Screening

If a patient is identified as high-risk via questionnaires, the following tests act as the "second tier" of screening:

Tool	Screening Purpose	Significance
Pulse Oximetry (SpO2)	Detects chronic hypoxia	A resting SpO2 ≤88% often triggers the need for a full ABG
Forced Vital Capacity (FVC)	Measures lung volume	In neuromuscular patients, a 20% drop in FVC when moving from sitting to supine is a strong predictor of impending failure
Maximal Inspiratory Pressure (MIP)	Assesses muscle strength	Values less than 60 cm H2O indicate significant respiratory muscle weakness

Abbreviations: FVC, Forced Vital Capacity; MIP, Maximal Inspiratory Pressure; SpO2, Peripheral Capillary Oxygen Saturation; H20, water/hydrogen dioxide

Core Diagnostic Testing[9,10]

Medicare recognizes specific tests to validate the diagnosis of chronic respiratory failure (hypoxic or hypercapnic) These are the key diagnostic tests:

CPT Reference

Diagnostic Tool (CPT Code)	Description	Clinical Thresholds (Medical Necessity)	2026 Medicare Coverage
Arterial Blood Gas (ABG) (82803)	The "gold standard." To qualify for many treatments	PaCO2 >45 mmHg (Hypercapnia) or PaO2<55 mmHg (Hypoxia). Must be in a stable, non-acute state	Covered. The "Gold Standard" required for initial oxygen and BiPAP qualification
Pulse Oximetry (SpO2) (94760/94761)	Measures oxygen saturation in the blood	SpO2 <88% at rest, during sleep, or with exertion. Cannot be during acute illness or steroid taper	Covered. Often required as part of a 6-minute walk test (94618) to justify LTOT
Pulmonary Function Tests (PFTs) (94010/ 94060)	Spirometry is a non-invasive breathing test that measures lung function—specifically the amount and speed of air inhaled and exhaled	Evaluation of FEV1/FVC ratio and severity of obstructive or restrictive defects	Covered. Used to stage the underlying disease (e.g., GOLD Stage IV COPD) for NIV eligibility
Plethysmography (94726)	Generally ordered when more common tests, such as spirometry, are insufficient to determine the cause of breathing issues or to map out the	Measures Residual Volume (RV); used to document hyperinflation/air trapping	Covered. Supports the clinical need for lung volume reduction or specific NIV settings

Diagnostic Tool (CPT Code)	Description	Clinical Thresholds (Medical Necessity)	2026 Medicare Coverage
	total volume of air in the lungs		
DLCO (94729)	Measures how well the lungs transfer oxygen from inhaled air into the bloodstream. It is a crucial diagnostic tool for evaluating the integrity of the alveolar-capillary membrane	Assesses gas exchange efficiency at the alveolar-capillary membrane	Covered. Crucial for differentiating between "Pump" and "Lung" failure

Abbreviations: ABG, Arterial Blood Gas; BiPAP, Bilevel Positive Airway Pressure; COPD, Chronic Obstructive Pulmonary Disease; CRF, Chronic Respiratory Failure; DLCO, Diffusing Capacity of the Lungs for Carbon Monoxide; FEV1, Forced Expiratory Volume in 1 Second; FVC, Forced Vital Capacity; GOLD, Global Initiative for Chronic Obstructive Lung Disease; LTOT, Long-Term Oxygen Therapy; NIV, Non-Invasive Ventilation; PaCO2, Partial Pressure of Carbon Dioxide; PaO2, Partial Pressure of Oxygen; PFT, Pulmonary Function Test; RV, Residual Volume; SpO2, Peripheral Capillary Oxygen Saturation

Spirometry Parameters

Measurement	Obstructive Pattern (e.g., COPD)	Restrictive Pattern (e.g., OHS/NMD/ILD)
Forced Vital Capacity (FVC)	Decreased or Normal	Decreased (Primary Marker)
Forced Expiratory Vol. 1s (FEV1)	Decreased (Primary Marker)	Decreased or Normal
FEV1/FVC Ratio	Decreased (<0.70)	Normal or Increased
Total Lung Capacity (TLC)	Normal or Increased (Air Trapping)	Decreased
Common CRF Mechanism	Expiratory resistance and dynamic hyperinflation	Mechanical "bellows" failure or "stiff" lungs

Abbreviations: COPD, Chronic Obstructive Pulmonary Disease; CRF, Chronic Respiratory Failure; FEV1, Forced Expiratory Volume in 1 Second; FVC, Forced Vital Capacity; ILD, Interstitial Lung Disease; NMD, Neuromuscular Disease; OHS, Obesity Hypoventilation Syndrome; PFT, Pulmonary Function Test; TLC, Total Lung Capacity

Subtle Early Signs[3]

1. The "Nagging" Morning Cough: Instead of a chest-wracking fit, early COPD often manifests as a "smoker's cough" or a persistent need to clear the throat first thing in the morning

2. "Lifestyle Creep" (Activity Avoidance): This is the most common subtle sign. Patients unconsciously modify their lives to avoid breathlessness

3. Frequent "Chest Colds": A hallmark of early COPD is increased vulnerability to respiratory infections

4. Chest Tightness and "Heavy" Breathing: Rather than gasping for air, early-stage patients often describe a vague sensation of chest tightness or feeling like it takes more *effort* to breathe

5. Subtle Audible Clues: A slight whistling sound/wheeze that only occurs during deep physical exertion or when exhaling forcefully

Risk Factors[11,12]

Risk Factor	Risk Signal	Evidence Summary	Clinical Implication
Active Smoking	HR: 2.3 – 3.5	Dose-Response: Clear correlation between pack-years and FEV1 decline; cessation is the only intervention proven to slow lung function loss	Current smokers have over double the hazard for COPD and CRF compared to never-smokers
Occupational Dust/Fumes	OR: 1.5 – 2.0	Synergistic Effect: Occupational exposure combined with smoking creates a multiplicative risk for accelerated parenchymal destruction	Exposure to silica, coal, or vapours significantly increases the odds of fixed airflow obstruction
Severe Obesity (BMI ≥40)	OR: 2.8 – 3.2	Mechanical Load: High BMI increases intra-abdominal pressure, reducing FRC and leading to atelectasis and CO2 retention	Strongly associated with Obesity Hypoventilation Syndrome (OHS) and Type II failure
History of Severe Infection	RR: 2.2	Lung Development: Early insults prevent reaching "peak lung function," leading to a lower threshold for failure later in life	Childhood pneumonia or severe viral insult (e.g., RSV) increases the relative risk of adult chronic impairment
Air Pollution (PM2.5)	HR: 1.15 per 10 µg/m3	Inflammatory Trigger: Fine particulate matter (<2.5 µm) bypasses upper airway filters to cause systemic inflammation and alveolar stress	Incremental increases in fine particulate matter correlate with a 15% rise in respiratory-related mortality
Alpha-1 Antitrypsin Deficiency	OR: 20.0+	Inflammatory Trigger: Fine particulate matter (<2.5 µm) bypasses upper airway filters to cause systemic inflammation and alveolar stress	For the PiZZ phenotype, the odds of developing early-onset emphysema are extremely high
Frequent Exacerbations	HR: 1.25 per event	Step-Wise Decline: Lung function (FEV1) often fails to return to the pre-event baseline, creating a "step-down" trajectory	Each severe exacerbation carries a cumulative hazard for permanent lung function decline

Abbreviations: AATD, Alpha-1 Antitrypsin Deficiency; BMI, Body Mass Index; COPD, Chronic Obstructive Pulmonary Disease; CRF, Chronic Respiratory Failure; FEV1, Forced Expiratory Volume in 1 Second; HR, Hazard Ratio; OHS, Obesity Hypoventilation Syndrome; OR, Odds Ratio; PM2.5, Fine Particulate Matter (<2.5 micrometers); RR, Relative Risk; RSV, Respiratory Syncytial Virus

Geriatric Risk Factors[13]

Factor	Risk Signal (Metric)	Evidence Summary	Clinical Implication
Senile Emphysema	AR: 0.1–0.2 L/decade (VC Loss)	Age-related alveolar duct ectasia and loss of elastic recoil (deterioration of VA/Q matching)	Increases Residual Volume (RV); reduces ventilatory reserve during acute infection

Factor	Risk Signal (Metric)	Evidence Summary	Clinical Implication
Chest Wall Calcification	RR: 1.4–1.6 (Work of Breathing)	Calcification of costal cartilages and increased kyphosis reduce thoracic compliance	Significant increase in the mechanical cost of breathing; rapid fatigue during exacerbations
Sarcopenia of the Diaphragm	OR: 2.5 (for Type II Failure)	Loss of Type IIa fast-twitch fibers in the diaphragm reduces Maximal Inspiratory Pressure (MIP)	High risk for Chronic Hypercapnia (J96.12) as the "pump" lacks the power to clear CO2
Blunted Chemoreceptor Sensitivity	↓ 40–50% (Hypoxic Response)	Attenuation of the peripheral and central ventilatory response to O2 and CO2 changes	Patients may be in profound respiratory acidemia without reporting significant dyspnea
Polypharmacy (Sedatives/Opioids)	OR: 2.1 – 5.1 (Respiratory Depression)	Standard doses of benzodiazepines or opioids significantly depress the central respiratory drive	Leading iatrogenic trigger for Acute-on-Chronic Ventilatory Failure in home settings
Silent Aspiration	HR: 1.8 – 2.4 (Pneumonia Risk)	Oropharyngeal dysphagia in neurodegenerative states leads to recurrent chemical pneumonitis	Accelerates Parenchymal Fibrosis and serves as a primary trigger for infectious decompensation

Abbreviations: AR, Annual Rate; COPD, Chronic Obstructive Pulmonary Disease; CRF, Chronic Respiratory Failure; HR, Hazard Ratio; MIP, Maximal Inspiratory Pressure; OR, Odds Ratio; RR, Relative Risk; RV, Residual Volume; VA/Q, Alveolar Ventilation-Perfusion Ratio; WOB, Work of Breathing

RED FLAGS - URGENT ACTION[4,12]

System	Sign/Symptom	Clinical Significance	Urgent Action
Neurological	Asterixis ("Flapping" tremor)	Pathognomonic for severe hypercapnia (CO2 retention) and respiratory acidosis	STAT ABG; initiate NIPPV (BiPAP)
Neurological	Altered Mental Status/ Somnolence	"CO2 Narcosis"; Indicates life-threatening hypercapnia and imminent respiratory arrest	Intubation/Mechanical Ventilation likely required
Respiratory	Paradoxical Breathing	The abdomen moves in while the chest moves out; indicates diaphragmatic fatigue	Immediate ventilatory support; high risk for sudden arrest
Respiratory	Silent Chest on Auscultation	No air movement heard despite visible effort; suggests total airway obstruction or collapse	Emergent bronchodilation and possible airway management
Cardiovascular	New-Onset Peripheral Edema/JVD	Signs of Cor Pulmonale (Right Heart Failure) due to severe pulmonary hypertension	Diuresis and optimization of long-term oxygen therapy (LTOT)

System	Sign/Symptom	Clinical Significance	Urgent Action
Vital Signs	SpO2 <85% on Baseline Oxygen	Reflects a profound V/Q mismatch or shunting that cannot be overcome by standard flow	Escalation to high-flow nasal cannula or invasive support

Abbreviations: ABG, Arterial Blood Gas; AMS, Altered Mental Status; CRF, Chronic Respiratory Failure; JVD, Jugular Venous Distension; LTOT, Long-Term Oxygen Therapy; NIPPV, Non-Invasive Positive Pressure Ventilation (BiPAP); SpO2, Peripheral Capillary Oxygen Saturation; STAT, Latin *statim* (Immediately); V/Q, Ventilation-Perfusion Ratio

Clues to Dig Deeper[4,14,15]

1. The External Signs of Internal Failure

When performing a visual exam, check for evidence of either Parenchymal Destruction or Circulatory Failure.

- **Clubbing of the Fingers:** This indicates chronic, significant hypoxia or systemic inflammation. If this is a new finding in a patient with a smoking history → Necessitates a **high-resolution CT to rule out malignancy**
- **Jugular Venous Distension (JVD) and Peripheral Edema:** These are the cardinal signs of Cor Pulmonale. When the lungs offer too much resistance, the right ventricle dilates → **Transthoracic Echocardiogram** to assess RV function and Pulmonary Artery (PA) pressure
- **Asterixis (Hand Flap):** This is a critical neurologic sign of Hypercapnic Encephalopathy. It suggests CO2 narcosis → **Requires an immediate Arterial Blood Gas (ABG)** to check for acute-on-chronic respiratory acidosis

2. Nocturnal Clues: Testing Respiratory Reserve

Sleep is the "stress test" for the respiratory pump. During REM sleep, accessory muscle activity is naturally suppressed; if the diaphragm is weak, gas exchange fails.

- **Morning Headaches:** These are caused by cerebral vasodilation in response to nocturnal hypercapnia. → It is a key indicator for initiating **Non-Invasive Ventilation (NIV)**
- **Orthopnea and PND:** These suggest that the "respiratory" problem may actually be Congestive Heart Failure (CHF) or a significant cardiac overlap → Warrants a **BNP level and a transthoracic echocardiogram**

3. Diagnostic "Gaps:" When the Numbers Mislead

A "disconnect" between spirometry and patient symptoms often points to the pulmonary vasculature or the "pump" itself.

- **Disproportionate Desaturation:** If the FEV1 is 60% but the patient desaturates to 84% during a 6-minute walk test (6MWT), the issue is likely V/Q mismatching or Pulmonary Hypertension, not just airflow obstruction → **DLCO and 6MWT** to evaluate gas exchange and exercise-induced hypoxemia
- **Unexplained Polycythemia:** If the Hematocrit is >55%, the patient is likely spending significant portions of their day (or night) in a severely hypoxic state that a single "spot-check" pulse oximetry

reading in the clinic has missed → **Nocturnal Oximetry** to identify "silent" desaturation during sleep or exertion

4. Systemic Markers: The "Silent" Predictors of Mortality

Chronic respiratory failure is a systemic disease. The lungs are the primary site, but the "end-organ damage" occurs in the muscles and metabolic stores.

- **Pulmonary Cachexia:** Unintentional weight loss in CRF signifies a massive increase in the Work of Breathing (WOB) → **Medical Nutrition Therapy:** High-protein, low-RQ diet
- **Muscle Weakness:** Grip strength and quadriceps atrophy correlate more closely with 30-day readmission rates than FEV1 does → **Pulmonary Rehabilitation**

Common Oversights[3,14]

1. The "Steroid-Induced" Oversight: Bone Density

In the management of Chronic Obstructive Pulmonary Disease (COPD) and CRF, **DEXA scan** interpretation is frequently a point of diagnostic inertia.

- **Oversight/Diagnostic Gap:** A T-score of -2.5 is often reflexively categorized as age-related or post-menopausal osteoporosis. In the CRF population, however, this often represents **Drug-Induced Osteoporosis (M81.6)** secondary to frequent systemic corticosteroid bursts (GLIO—Glucocorticoid-Induced Osteoporosis)
- **The Mechanical Risk:** Vertebral compression fractures in CRF are not merely orthopedic events; they are respiratory threats. Resultant **Thoracic Kyphosis** leads to a significant reduction in thoracic cage volume and compliance, precipitating or exacerbating **Restrictive Ventilatory Failure**
- **The Tipping Point:** As the chest wall becomes more restrictive, the work of breathing (WOB) increases. For a patient already at their physiological ceiling, this loss of mechanical advantage often triggers the transition from compensated to uncompensated **Hypercapnic Respiratory Failure (J96.12)**

2. The "Metabolic" Oversight: Secondary Polycythemia

Many clinicians ignore an elevated Hemoglobin/Hematocrit in a smoker or COPD patient, assuming it is "normal for them."

- **The Oversight:** Failing to diagnose **Secondary Polycythemia (D75.1)**
- **The Clinical Risk:** This is a red flag for **Refractory Hypoxemia**. The body is overproducing red blood cells to compensate for low oxygen. This increases blood viscosity, raising the risk of **Pulmonary Thromboembolism** and stroke
- **The Solution:** An Hct >55% is a primary criterion to initiate **Long-Term Oxygen Therapy (LTOT)**, even if the patient's resting O2 sat is slightly above the 88% cutoff

3. The "Cardiac" Oversight: Right Ventricular Strain

Right heart failure is the "silent partner" of chronic lung disease. It is often missed until the patient presents with frank anasarca (total body swelling).

- **The Oversight:** Overlooking subtle signs of **Cor Pulmonale (I27.9)** on an Echocardiogram, such as a dilated Right Ventricle or a high Pulmonary Artery Systolic Pressure (PASP)
- **The Clinical Risk:** Only treating the "shortness of breath" with only inhalers but ignore the right heart strain, the patient will continue to decline due to low cardiac output
- **The Solution:** Document the link between lung disease and heart failure. **Cor Pulmonale** justifies the use of diuretics and specialized vasodilators

4. The "Neurological" Oversight: CO2 Narcosis

- **The Oversight:** Failing to screen for **Chronic Hypercapnia (J96.12)** in a patient with a high BMI or severe COPD
- **The Clinical Risk:** This is a "pump" failure. High CO2 levels lead to cognitive decline, morning headaches, and eventually respiratory arrest
- **The Solution:** If a patient is sleepy during the day, don't just order a sleep study for OSA; get an **Arterial Blood Gas (ABG)** to check for a high Bicarbonate (HCO3), which proves chronic CO2 retention

Key Differentials[3,14,16]

Category	Primary Mechanism	Key Differential Diagnoses	Clinical "Red Flags"
Obstructive Airway Disease	V/Q mismatch and air trapping leading to dead space	COPD/Emphysema, Chronic Bronchitis, Cystic Fibrosis, Severe persistent Asthma	Hyperinflation on CXR, decreased FEV1/FVC ratio
Restrictive Lung Disease	Impaired gas exchange due to parenchymal scarring	Idiopathic Pulmonary Fibrosis (IPF), Sarcoidosis, Asbestosis, Hypersensitivity Pneumonitis	Fine "Velcro" crackles, honeycombing on HRCT
Neuromuscular Disorders	Failure of the "pump" (respiratory muscles)	Amyotrophic Lateral Sclerosis (ALS), Myasthenia Gravis, Muscular Dystrophy, Diaphragmatic Paralysis	Orthopnea, weak cough, decreased Maximal Inspiratory Pressure (MIP)
Chest Wall and Pleural Disease	Mechanical restriction of lung expansion	Obesity Hypoventilation Syndrome (OHS), Kyphoscoliosis, Chronic Pleural Effusion/Fibrothorax	Elevated BMI (>30), daytime somnolence, restrictive PFT pattern
Central Drive Disorders	Reduced signal from the brain to breathe	Central Sleep Apnea, Chronic Opioid Use, Brainstem injury/stroke, Obesity (Blunted Chemoreceptor Sensitivity)	Apneic episodes without respiratory effort, bradypnea
Pulmonary Vascular Disease	Perfusion defect limiting oxygen uptake	Chronic Thromboembolic Pulmonary Hypertension (CTEPH), Group 1 Pulmonary Arterial Hypertension	Right heart failure (edema, JVD), loud S2 on auscultation

Abbreviations: ALS, Amyotrophic Lateral Sclerosis; BMI, Body Mass Index; COPD, Chronic Obstructive Pulmonary Disease; CSA, Central Sleep Apnea; CXR, Chest X-ray; CTEPH, Chronic Thromboembolic Pulmonary Hypertension; FEV1, Forced Expiratory Volume in 1 Second; FVC, Forced Vital Capacity; HRCT, High-Resolution Computed Tomography; IPF, Idiopathic Pulmonary

Fibrosis; JVD, Jugular Venous Distension; MIP, Maximal Inspiratory Pressure; OHS, Obesity Hypoventilation Syndrome; PAH, Pulmonary Arterial Hypertension; PFT, Pulmonary Function Test; V/Q, Ventilation-Perfusion Ratio

Comorbidity Screening[17–19]

System and Category	Specific Condition	Est. Prevalence in CRF/Advanced COPD	Clinical Impact in CRF	Screening and Diagnostic Tools
Cardiovascular	PH and Right Heart Failure	40% – 70%	Chronic hypoxia triggers pulmonary vasoconstriction and RV strain (Cor Pulmonale)	Echocardiogram (Check RV function and PA pressure)
	Coronary Artery Disease	20% – 35%	High prevalence due to systemic inflammation; drives exercise intolerance	ECG or Stress Testing
	Atrial Fibrillation	15% – 25%	Triggered by hypoxia and structural right atrial stretch	ECG
Metabolic	OSA/Overlap Syndrome	10% – 20%	Co-exists with COPD or OHS, significantly worsening nocturnal failure.	Polysomnography or Nocturnal Oximetry
	Osteoporosis	30% – 60%	Driven by systemic inflammation, inactivity, and chronic corticosteroid use	DEXA Scan
	Metabolic Syndrome	30% – 50%	Steroid use and inflammation impair glucose metabolism and vascular health	A1c, Fasting Glucose, Lipid Panel, BMI, BP
Nutritional	Skeletal Muscle Dysfunction	25% – 40%	Leads to Pulmonary Cachexia and diaphragmatic wasting	BMI and FFMI (Fat-Free Mass Index)
	Anemia of Chronic Disease	15% – 30%	Reduces O2 carrying capacity, severely worsening dyspnea	CBC and Iron Studies
Neuro-Psych	Anxiety and Depression	35% – 50%	Major trigger for the "Panic-Dyspnea" loop and treatment non-adherence	HADS or PHQ-9
	Cognitive Impairment	20% – 30%	Chronic CO2 retention leads to "brain fog" and executive dysfunction	MoCA (Montreal Cognitive Assessment)

Abbreviations: A1c, Hemoglobin A1c; BMI, Body Mass Index; BP, Blood Pressure; CAD, Coronary Artery Disease; CBC, Complete Blood Count; CO2, Carbon Dioxide; COPD, Chronic Obstructive Pulmonary Disease; CRF, Chronic Respiratory Failure; DEXA, Dual-Energy X-ray Absorptiometry; ECG, Electrocardiogram; FFMI, Fat-Free Mass Index; HADS, Hospital Anxiety and Depression Scale; MoCA, Montreal Cognitive Assessment; O2, Oxygen; OHS, Obesity Hypoventilation Syndrome; OSA, Obstructive Sleep Apnea; PA, Pulmonary Artery; PH, Pulmonary Hypertension; PHQ-9, Patient Health Questionnaire-9; RV, Right Ventricular

Staging/Severity Matrix[17,20–22]

Staging chronic respiratory failure (CRF) is more complex than staging standard COPD or Asthma because it focuses on the **failure of the respiratory pump** (the brain, nerves, and muscles) and **gas exchange** (the lungs) rather than just airflow.

A comprehensive staging and severity matrix evaluates three pillars: **Physiological (Gases)**, **Functional (Capacity)**, and **Clinical (Complications)**. However, **Arterial Blood Gas (ABG)** is the only "gold standard" for diagnosing respiratory failure.

Stage	Classification	Arterial Blood Gas (ABG)	Functional Status	Clinical Markers
Stage I	Early/Latent	Normal at rest; mild Hypoxia (PaO2 <70 mmHg) during exercise	Dyspnea only on heavy exertion (mMRC 0-1)	Stable; no history of hospitalizations
Stage II	Compensated	PaO2 55–60 mmHg; PaCO2 is normal or slightly elevated (<45mmHg)	Dyspnea walking on level ground (mMRC 2)	Occasional "flares"; may require nocturnal O2
Stage III	Decompensated	PaO2 <55 mmHg; PaCO2 45–55 mmHg (Hypercapnia)	Dyspnea during basic ADLs (mMRC 3)	Signs of Right Heart strain; frequent ER visits
Stage IV	End-Stage	PaO2 <50 mmHg;PaCO2 >55 mmHg; pH <7.35 (Acidosis)	Bedbound or chair-bound (mMRC 4)	Cor Pulmonale; refractory to standard therapy; BMI <18

Abbreviations: ABG, Arterial Blood Gas; ADLs, Activities of Daily Living; BMI, Body Mass Index; ER, Emergency Room; mMRC, Modified Medical Research Council (Dyspnea Scale); PaCO2, Partial Pressure of Carbon Dioxide; PaO2, Partial Pressure of Oxygen; pH, Potential of Hydrogen (Acidity/Alkalinity)

Key Staging Tools to Integrate

To "score" a patient accurately within this matrix, clinicians typically use these three validated tools:

1. The BODE Index (Prognostic Power)

This is the "Gold Standard" for predicting mortality in respiratory failure. It moves beyond FEV1 to look at the whole person. **See appendix for details.**

- **B** – BMI (Is the patient wasting away?)
- **O** – Obstruction (FEV1 % predicted)
- **D** – Dyspnea (mMRC scale)
- **E** – Exercise Capacity (6-minute walk distance)

2. The mMRC (Modified Medical Research Council) Scale

A quick, 0–4 scale used to stage the **functional** impact:

- **Grade 0:** Only breathless with strenuous exercise
- **Grade 1:** Short of breath when hurrying or walking uphill
- **Grade 2:** Walks slower than people of the same age because of breathlessness
- **Grade 3:** Stops for breath after 100 yards/meters or every few minutes
- **Grade 4:** Too breathless to leave the house or breathless when dressing

3. The V/Q Gap (Gas Exchange Severity)

- **Type I (Hypoxemic):** PaO2 is low, but PaCO2 is normal/low (usually lung tissue damage)
- **Type II (Hypercapnic):** Both PaO2 is low and PaCO2 is high (this is **Ventilatory Failure**—the pump has given out)

When to "Up-Stage" a Patient

Regardless of the numbers, a patient should be moved to a higher severity tier if they exhibit:

1. **Frequent Flyer Status:** ≥2 hospitalizations in 12 months for respiratory issues
2. **The "Hand Flap":** Presence of asterixis (indicates severe CO2 narcosis)
3. **Pulmonary Cachexia:** Loss of >5 body weight in 6 months without trying
4. **Secondary Erythrocytosis:** Hematocrit >55%, suggesting the body is in a state of permanent "oxygen panic"

Spirometry and CXR facilitate the diagnostic workup by identifying underlying phenotypes and acute triggers, providing critical context to the physiological data defined by the **Arterial Blood Gas**.

Spirometry (Flow-Volume Mechanics)

Spirometry characterizes the **ventilatory defect** (Obstructive vs. Restrictive) that has led to failure.

When to Order:

- **Baseline Phenotyping:** Every patient with suspected CRF needs a baseline to determine the "why"
  - *Obstructive:* COPD, Bronchiectasis (FEV1/FVC <0.70)
  - *Restrictive:* ILD, Chest wall deformity, or Neuromuscular disease FVC reduced with normal ratio)
- **Annual Surveillance:** To monitor the **FEV1 rate of decline**. A loss of >50 mL/year in COPD is a high-risk marker for frequent exacerbations
- **Response to Therapy:** 4–6 weeks after starting long-term bronchodilators or anti-inflammatory therapy to assess "top-tier" lung function
- **Pre-Surgical Clearance:** Mandatory for any thoracic or upper-abdominal procedure to assess the risk of post-operative mechanical ventilation

When to Defer:

- **During Acute Exacerbations:** Do not perform diagnostic spirometry during an acute flare. The results will be artificially low due to temporary inflammation and muscle fatigue. Wait until 4-6 weeks after the flare/post-discharge

- **Post-Ophthalmic or Abdominal Surgery:** Due to the risk of increased intra-thoracic and intra-ocular pressure

2. Chest X-Ray (Anatomical Screening)

CXR is a structural tool. In CRF, it is primarily used to rule out **acute-on-chronic triggers**.

When to Order:

- **Initial Workup:** To identify gross pathology (e.g., emphysematous bullae, interstitial thickening, or skeletal abnormalities like kyphoscoliosis)
- **Acute "Drop-off" in Status:** If a stable CRF patient suddenly requires more O2 or has increased PaCO2
- **Rule Out "The Big Three" Complications:**

  1. **Pneumonia:** New focal infiltrates
  2. **Pneumothorax:** Especially in patients with bullous emphysema or those on Positive Pressure Ventilation (NIV)
  3. **Congestive Heart Failure:** Pulmonary edema or pleural effusions can mimic a COPD flare

- **Suspected Malignancy:** New-onset hemoptysis or unexplained weight loss

3. MEAT DOCUMENTATION ESSENTIALS

A 68-year-old man with a history of Stage III Chronic Hypercapnic Respiratory Failure presents for a routine follow-up. He has been managed with long-term oxygen therapy (LTOT) for the past two years. Today's labs and diagnostic tests show a PaCO2 of 56 mmHg—up from 46 mmHg eighteen months ago—a 6-minute walk distance of 180 meters, and a Hematocrit of 56%. He is relatively asymptomatic at rest, but the trajectory of his hypercapnia, secondary polycythemia, and declining exercise tolerance warrants treatment escalation to Non-Invasive Ventilation (NIV) and urgent coordination with Pulmonary Rehabilitation.

MONITOR: "ABG (Stable Baseline) shows: pH 7.38, PaCO2 56 mmHg, PaO2 58 mmHg, and HCO3 34 mEq/L (indicating chronic compensated hypercapnia). Current SpO2 is 89% on 2L NC. NIV Data Log reveals an average use of 5.2 hrs/night with a median leak of 12 L/min and an AHI of 4.2. Functional testing via 6MWT covers 180 meters with a nadir SpO2 of 84%. Recent DXA Scan shows a T-score of -2.6 (Thoracic Spine), and CBC reveals a Hct of 56% (Secondary Polycythemia). Weight is 142 lbs, reflecting a loss of 6 lbs in 3 months."

EVALUATE: "Reviewed NIV trends; while adherence meets CMS 90-day criteria, persistent nocturnal desaturation suggests that current **Pressure Support (PS)** may be inadequate to offload the work of breathing (WOB). The **Hct of 56%** confirms sustained tissue hypoxia (**D75.1**) despite current LTOT. Nutritional evaluation shows a **BMI of 19.2** with unintentional weight loss consistent with **Pulmonary Cachexia** (**E64.0**). Skeletal risk is high due to chronic corticosteroid "bursts" and the thoracic T-score, which increases the risk for a restrictive ventilatory defect secondary to vertebral compression."

ASSESS: "The patient is asymptomatic at rest, but the clinical trajectory indicates disease progression. Evidence includes increased oxygen demand, declining exercise tolerance (6MWD <200m), and

inadequate ventilation during sleep despite BiPAP. The case is complicated by Cor Pulmonale (I27.9) (trace peripheral edema noted) and Glucocorticoid-Induced Osteoporosis (M81.6). Functional status is severely limited, with dyspnea occurring during minimal ADLs (mMRC Grade 3)."

TREAT: "**NIV Titration:** Increased **IPAP by 2cm H2O** to enhance CO2 clearance; scheduled a home respiratory therapist visit for mask fitting to reduce leak. **LTOT:** Recertified oxygen at 3L with exertion and transitioned the patient from tanks to a **Portable Oxygen Concentrator (POC)** to support Pulmonary Rehab attendance. **Pharmacology:** Initiated Roflumilast for chronic bronchitis phenotype and ordered Vitamin D/Calcium for bone health. **Referrals:** Issued urgent referrals to **Pulmonary Rehabilitation** and Nutritional Counseling for high-calorie/high-protein supplementation."

Clinical Documentation Elements

Specify the Phenotype and Mechanism: "Chronic Hypercapnic (Type II) Respiratory Failure (J96.12) secondary to Stage IV (GOLD Group E) Very Severe Obstructive Pulmonary Disease (J44.9) with prominent centrilobular emphysema."

Link Secondary Complications to the Primary Insult: "Secondary Polycythemia (D75.1) and Group 3 Pulmonary Hypertension (I27.23) directly due to chronic alveolar hypoxia from underlying COPD; patient currently exhibits peripheral edema and JVD consistent with early Cor Pulmonale (I27.9)."

Include Current Objective Data (Physiological Evidence): "Most recent ABG [Date] on 2L NC: pH 7.38, PaCO2 58 mmHg, PaO2 62mmHg, HCO3 34 mEq/L. High bicarbonate and compensated pH confirm chronic, stable hypercapnia. PFTs [Date] show FEV1 0.85L (28% predicted) and FRC 145% predicted, consistent with severe air trapping and mechanical diaphragm disadvantage."

Specify Staging and Technology Dependence: "Chronic Respiratory Failure with dependence on long-term Supplemental Oxygen (Z99.81) at 3L/min via nasal cannula and nocturnal Non-Invasive Pressure Support Ventilation (Z99.89) via BiPAP (Settings: IPAP 18, EPAP 6, Backup Rate 12) for management of Obesity Hypoventilation Syndrome (E66.2)."

Document Chronicity and Longitudinal Management: "Patient in Month 14 of a long-term Pulmonary Rehabilitation and NIV titration program. Currently managing NIV-induced skin breakdown on the nasal bridge (L89.81) and xerostomia; therapy remains essential to prevent acute-on-chronic decompensation and ICU readmission."

Reframing Common Documentation Shortcuts

Instead of...	Document...	Why this supports clarity
"History of respiratory failure"	"Active Chronic Hypercapnic Respiratory Failure (J96.12) secondary to Stage IV (GOLD Group E) COPD; pH 7.38, PaCO2 56mmHg on current settings"	Clarifies physiologic phenotype. Distinguishes between hypoxemic (Type I) and hypercapnic (Type II) failure and confirms the state is chronic/compensated
"Lungs stable"	"Very Severe Airflow Obstruction (J44.9) with BODE Index of 7; FEV1 26% predicted [Date]; 6MWT [Date] shows	Specifies severity via objective data. Uses validated prognostic scores (BODE) and dated physiological testing to justify the level of care

Instead of...	Document...	Why this supports clarity
	nadir SpO2 84% on RA"	
"Swelling in legs"	"Right Heart Failure/Cor Pulmonale (I27.9) secondary to Group 3 Pulmonary Hypertension (I27.23) from underlying Alveolar Hypoxia"	Links complications to the primary etiology. Directly connects the cardiac manifestation to the respiratory failure for a robust clinical picture
"Uses home oxygen"	"Dependence on supplemental Oxygen (Z99.81) at 3 L/min via NC and Nocturnal NIV (Z99.89) (Settings: IPAP 16, EPAP 5) for Sleep Hypoventilation."	Demonstrates technology dependence. Documents the complexity of the patient's home management and the specific life-sustaining equipment required
"Short of breath"	"Acute-on-Chronic Respiratory Failure (J96.21) triggered by Viral Bronchiolitis; ABG shows acute respiratory acidosis (pH 7.28, PaCO2 72)"	Documents acute trajectory. Distinguishes the baseline chronic state from an acute decompensation, requiring a higher tier of intervention
"Weight loss"	"Pulmonary Cachexia (E64.0) with BMI 17.2 due to increased metabolic work of breathing; FFMI [Date] confirms skeletal muscle wasting"	Specifies metabolic impact. Shows the systemic nature of the respiratory disease and justifies nutritional intervention

Abbreviations: 6MWT, 6-Minute Walk Test; ABG, Arterial Blood Gas; BMI, Body Mass Index; BODE, Body-mass index, Obstruction, Dyspnea, and Exercise capacity index; COPD, Chronic Obstructive Pulmonary Disease; EPAP, Expiratory Positive Airway Pressure; FEV1, Forced Expiratory Volume in 1 Second; FFMI, Fat-Free Mass Index; GOLD, Global Initiative for Chronic Obstructive Lung Disease; IPAP, Inspiratory Positive Airway Pressure; J96.12, ICD-10 for Chronic Hypercapnic Respiratory Failure; NC, Nasal Cannula; NIV, Non-Invasive Ventilation; PH, Pulmonary Hypertension; RA, Room Air; SpO2, Peripheral Capillary Oxygen Saturation; WOB, Work of Breathing.

4. TREATMENT AND REFERRAL QUICK GUIDE

Therapy Escalation Criteria[3,17,23,24]

The goal is to maximize airway patency and minimize systemic inflammation to reduce the frequency of acute-on-chronic exacerbations.

- **Inhaled Triple Therapy (LAMA/LABA/ICS):** The cornerstone for **obstructive phenotypes** (COPD/Bronchiectasis). This reduces gas trapping and improves the mechanical advantage of the diaphragm
  - For COPD patients with chronic respiratory failure, long-acting bronchodilators (LABAs/LAMAs) are preferred over short-acting agents for maintenance therapy. Triple therapy (LABA+LAMA+ICS) may reduce exacerbations and mortality in symptomatic patients with frequent exacerbations. In patients with blood eosinophils ≥300 cells/µL uncontrolled on triple therapy, biologics like dupilumab or mepolizumab reduce exacerbations
- **Phosphodiesterase-4 (PDE4) Inhibitors:** (e.g., Roflumilast) For patients with chronic bronchitis and frequent exacerbations. The transition to a PDE4 inhibitor is typically reserved for a high-risk subset of patients who remain unstable despite maximal inhaled triple therapy. Roflumilast is a

non-steroidal anti-inflammatory agent that specifically targets the intracellular signaling pathways responsible for mucus hypersecretion and airway remodeling

- **Low-dose theophylline:** The American Thoracic Society (ATS) and other major guidelines generally relegate theophylline to third- or fourth-line therapy in Chronic Obstructive Pulmonary Disease (COPD) management, typically considered only when inhaled therapies (LABA, LAMA, ICS) are maxed out, unavailable, or unaffordable. While high-dose theophylline is rarely used due to toxicity, low-dose theophylline (aiming for serum levels <5 mg/L) has been investigated for potential anti-inflammatory benefits and reversing corticosteroid resistance in advanced COPD. In patients with COPD, theophylline reduces the rate of sputum neutrophils and the degree of neutrophilic inflammation in the airways
- **Diuretic Therapy:** Carefully titrated for patients with secondary **Right Heart Failure/Cor Pulmonale**.
  - *Caution:* Avoid over-diuresis, which can cause contraction alkalosis and paradoxically depress the respiratory drive by raising HCO3levels
- **Vaccination:** Highly consider Pneumococcal, Influenza, Tetanus, diphtheria, pertussis (Tdap), Respiratory syncytial virus, and COVID-19 series to prevent infectious triggers
- **Antifibrinolytics:** The use of Antifibrotic Agents represents a significant shift in the management of **Restrictive Chronic Respiratory Failure (CRF)**, particularly in patients with **Idiopathic Pulmonary Fibrosis (IPF)** or progressive fibrosing interstitial lung diseases (ILDs)
  - Unlike traditional bronchodilators that target the "pipes" (airways), these agents target the **parenchyma** (lung tissue) to preserve the remaining "bellows" function of the ventilatory pump

The transition from chronic management to therapy escalation is defined by the failure of **compensatory mechanisms**. Escalation is not merely based on "more symptoms," but on objective evidence of **ventilatory fatigue**, **refractory hypoxemia**, or **hemodynamic strain**.

The following criteria represent the triggers for moving from standard medical therapy to advanced interventions (LTOT, NIV, or Transplant evaluation).

2. Long-Term Oxygen Therapy (LTOT)

Indicated for **Type I (Hypoxemic) Failure** to prevent pulmonary hypertension (grade 3) and cognitive decline. The following criteria represent the triggers for moving from standard medical therapy to LOLT:

1. **Primary Absolute Criteria:** Resting PaO2 $\leq$55 mmHg or SaO2 $\leq$88% on room air (confirmed twice over a 3-week period)
2. **Comorbidity-Driven Criteria:** PaO2 between 56–59 mmHg IF there is evidence of:
   - **Secondary Polycythemia:** Hematocrit >55%
   - **P-pulmonale on ECG:** Peaked P-waves in leads II, III, and aVF
   - **Right Heart Strain:** Clinical edema or Echocardiographic evidence of pulmonary hypertension

Key Targets:

- **Standard Target:** Maintain PaO2 >60 mmHg or SaO2 88–92%
- **Dosing:** Minimum of 15 hours per day (including during sleep and exertion)
- **Evidence:** LTOT is one of the few interventions proven to improve survival in COPD patients with severe resting hypoxemia

Oxygen tanks vs concentrators: Oxygen tanks provide highly pure, stored oxygen **without electricity** but are heavy and require regular, costly refilling. Oxygen concentrators produce unlimited oxygen from ambient air, offering convenience and safety without refills, but require power. Concentrators suit active, home-based, or traveling users, while tanks are best for short-term or backup needs. There are different options for oxygen concentrators, whether it be stationary or portable:

Stationary oxygen concentrators are the most commonly used and least expensive home oxygen delivery system (~$600 to $1,500). They are preferred when:

- Patients require continuous oxygen at home for most of the day (~15 hours)
- Reliable electricity is available
- Flow rates of 1-5 L/min are sufficient

Portable concentrators (cost between $1,495 and $3,495) may not consistently sense patients' inspiratory efforts to trigger pulse-dose delivery, making them potentially inadequate for patients requiring continuous flow >3 L/min (e.g., interstitial lung disease, lung transplant candidates). POC battery life depletes more rapidly with higher settings and respiratory rates.

3. Non-Invasive Ventilation (NIV/BiPAP)

The "Gold Standard" for **Type II (Hypercapnic) Failure** (e.g., OHS, Neuromuscular disease, or GOLD Stage IV COPD). Non-Invasive Ventilation (NIV), commonly delivered via a BiPAP (Bilevel Positive Airway Pressure) machine, is used in both inpatient (hospital) and home settings.

NIV is indicated when the "respiratory pump" (the bellows) can no longer maintain eucapnia despite optimal medical therapy.

- **Stable Hypercapnic Trigger:** Daytime $PaCO_2 \geq 50$ mmHg (reflecting Chronic Type II Failure)
- **Nocturnal Hypoventilation:** O_2 desaturation (SpO_2 <88% for >5 minutes) during sleep that does not respond to supplemental O_2 alone, suggesting a need for pressure support
- **Mechanical Failure (NMD/Chest Wall):** Forced Vital Capacity (FVC) <50% predicted or Maximal Inspiratory Pressure (MIP) <60 cm H20
- **Hospitalization History:** >2 admissions in 12 months for acute hypercapnic respiratory failure requiring NIV

Key Targets:

- **Physiologic Goal:** To augment alveolar ventilation and allow the inspiratory muscles to "rest" nocturnally, which resets the central chemoreceptor sensitivity to CO_2
- **Titration:** Use **Pressure Support (PS)**—the difference between IPAP and EPAP—to clear CO_2
- **High-Intensity NIV:** Aiming for near-normalization of $PaCO_2$ has been shown to reduce mortality and re-hospitalization rates in chronic hypercapnic COPD

Accessibility/affordability:

To qualify for a BiPAP (E0470 or E0471), a patient must meet the criteria for a specific clinical group. If the documentation lacks these specific numbers, the claim is typically denied.

Restrictive Thoracic Disease (e.g., Neuromuscular, Kyphoscoliosis)

- Documentation Required:
  - FVC <50% of predicted
  - OR Maximal Inspiratory Pressure (MIP) ≤60 cm H20
  - OR Chronic Hypercapnia (PaCO2 ≥45 mmHg) while awake

Severe COPD (Chronic Respiratory Failure)

- Documentation Required:
  - PaCO2 ≥52 mmHg while awake and stable
  - OR Sleep Oximetry showing SpO2 ≤88% for at least 5 continuous minutes while on the patient's usual O2 flow
  - Prerequisite: Documentation that the patient has tried and failed optimal medical management (inhalers, smoking cessation, and O2 if applicable)

Accessibility Barriers

Even after a device is delivered, coverage is contingent on adherence.

- The 90-Day Rule: Most insurers (Medicare and Private) require a "Compliance Check" between days 31 and 90
- Adherence Definition: The patient must use the device for at least 4 hours per night for 70% of nights in a consecutive 30-day period
- Consequence of Failure: If compliance is not met, the DME provider is often required to reclaim the machine, and the patient must restart the clinical qualification process from the beginning

4. Pulmonary Rehabilitation (PR)

PR is a high-yield intervention that addresses the **systemic** component of CRF. **See Non-Rx Treatment section for more information.**

- **Components:** Exercise training, nutritional counseling, and energy conservation techniques
- **Outcome:** While PR does not significantly improve FEV1, it dramatically improves **6-Minute Walk Distance (6MWD)** and reduces the "Dyspnea-Anxiety" cycle
- **The "Muscle" Factor:** Targets peripheral muscle dysfunction (sarcopenia) to reduce the total oxygen demand during ADLs

Component	Intervention Type	Primary Objective
Airway	LAMA/LABA/ICS	Decrease FRC/RV (Air Trapping)
Oxygenation	Oxygen Concentrator	Prevent Cor Pulmonale (I27.9)
Ventilation	Nocturnal BiPAP	Treat Chronic Hypercapnia (J96.12)
Metabolic	Nutritional Support	Treat Pulmonary Cachexia (E64.0)
Psychosocial	SSRIs/Cognitive Behavioral	Break the Panic-Dyspnea Loop

Abbreviations: BiPAP, Bilevel Positive Airway Pressure; CBT, Cognitive Behavioral Therapy; FRC, Functional Residual Capacity; ICS, Inhaled Corticosteroid; J96.12, Chronic Hypercapnic Respiratory Failure; LABA, Long-Acting Beta-Agonist; LAMA, Long-Acting Muscarinic Antagonist; RV, Residual Volume; SSRIs, Selective Serotonin Reuptake Inhibitors

5. Surgical and Interventional Options

For refractory cases where medical therapy is maximized:

- **Endobronchial Valves (EBV): For heterogeneous emphysema to achieve lung volume reduction without a thoracotomy**
- **Lung Transplantation: Referral should be initiated when the BODE Index is ≥7 or PaCO2 is rising despite NIV**
    - The BODE Index is a multidimensional scoring system (0–10 points) used to predict survival risk in patients with Chronic Obstructive Pulmonary Disease (COPD). It uses four key factors—Body Mass Index (B), Airflow Obstruction (O), Dyspnea (D), and Exercise capacity (E)—to provide a more accurate prognosis than measuring airflow obstruction alone

See Appendix for link to scoring criteria.

Non-Rx Treatment Documentation[25–27]

1. Pulmonary Rehabilitation (The Gold Standard)

Pulmonary Rehab is the most effective non-pharmacological intervention for improving quality of life.

- **Mechanism:** It treats **Peripheral Muscle Dysfunction**. CRF leads to fiber-type shifting in skeletal muscles, making them less efficient at using oxygen
- **Clinical Goal:** Increase the **6-Minute Walk Distance (6MWD)**. Even if FEV1 remains static, improving muscle efficiency reduces the total VO2 (oxygen demand) required for basic tasks
- **Implementation:** Structured resistance training and interval aerobic exercise under O2 monitoring
- **Importance:**
    - PR significantly reduces 30-day hospital readmission rates for COPD patients by roughly 33% to over 50%, particularly when initiated shortly after discharge. The data confirms that initiating PR within **3 to 4 weeks of discharge** (the "early" window) serves as a critical stabilization point, addressing the rapid skeletal muscle deconditioning and metabolic dysfunction that occurs during a hospitalization. However, it is highly underutilized in the US (See table below)
    - Early PR (within 4 weeks of hospitalization) is associated with a significant reduction in mortality, with some studies showing a relative risk (RR) of 0.58, indicating a roughly 42% lower risk of death at the end of treatment[28]

Pulmonary Rehabilitation utilization in the United States: Not hitting Target

Metric	The "Standard" Data	The "Ideal" Benchmark
Referral Rate	Only 3–16% of eligible patients are referred	100% of hospitalized patients (GOLD Group E)
Initiation Rate	Less than 2% initiate PR within 30 days	Target >50% for maximum readmission impact
Readmission Rate	~19–20% (Standard Care)	<10% (With Early PR/Telehealth bundle)

Metric	The "Standard" Data	The "Ideal" Benchmark

Abbreviations: CRF, Chronic Respiratory Failure; GOLD, Global Initiative for Chronic Obstructive Lung Disease; Group E, High-risk COPD (Exacerbators) regardless of symptoms; PR, Pulmonary Rehabilitation

AAVBC PERSPECTIVE

AAVBC advocates for early Pulmonary Rehabilitation (PR) implementation—within 30 days of discharge—to improve clinical outcomes and reduce the fiscal burden of hospital readmissions. By leveraging evidence-based exercise and education, PR decreases emergency department utilization for complex, high-risk respiratory populations. Crucially, this must be paired with Targeted Nutritional Therapy to manage the hypermetabolic state inherent to Chronic Respiratory Failure (CRF). Because the work of breathing in CRF can consume up to 25% of total caloric intake, AAVBC emphasizes nutritional literacy as a vital component in preserving diaphragmatic strength and supporting overall systemic health.

2. Nutritional Optimization

In pulmonary rehabilitation, a "mucolytic diet" refers to a nutritional strategy aimed at reducing the viscosity (thickness) of respiratory secretions to make them easier to clear. The following dietary principles focus on improving **mucociliary clearance** and reducing the systemic inflammation that triggers hypersecretion.

- **The High-Protein/Low-Carb Paradox:**
  - **High Protein:** Required to prevent diaphragmatic and skeletal muscle wasting (Sarcopenia)
  - **Moderate/Low Carbohydrate:** Excess carbs increase **Respiratory Quotient (RQ)**, which produces more CO2. In hypercapnic patients, a high-carb meal can actually trigger a rise in PaCO2
- **The Hydration Pillar (Critical)**
  - **The Goal:** 1.5 to 2.0 Liters of fluid daily (roughly 6–8 glasses), unless restricted by heart or kidney failure
  - **Mechanism:** Adequate systemic hydration maintains the Periciliary Liquid Layer (PCL)—the watery lubricant that allows cilia to beat and move the mucus "blanket" toward the throat
- **Anti-Inflammatory and "Mucus-Thinning" Foods**
  - Certain foods contain phytochemicals that may help modulate the inflammatory signals that tell goblet cells to overproduce mucus

Food Category	Specific Examples	Physiological Rationale
Alliums	Garlic, Onions, Leeks	Contain Allicin, which has natural anti-inflammatory and mild antimicrobial properties to reduce "phlegm" triggers
Omega-3 Fats	Salmon, Mackerel, Walnuts, Flaxseeds	High Hazard Ratio (HR) benefit; reduces systemic inflammation that causes airway swelling and hypersecretion
Warm Broths	Chicken or Vegetable Soup	Proven to increase "mucus velocity." The combination of heat and salt helps break down the disulfide bonds in mucus
Quercetin-Rich	Apples, Berries, Red Onions	Antioxidants that may inhibit the release of histamines and pro-inflammatory cytokines in the lungs
Bromelain	Fresh Pineapple	An enzyme that has been studied for its ability to break down

Food Category	Specific Examples	Physiological Rationale
		protein complexes in thick secretions

3. Breathing Retraining and Airway Clearance

Mechanical efficiency is the difference between stability and a hospital visit.

- **Pursed-Lip Breathing:** Creates **Intrinsic PEEP** (Positive End-Expiratory Pressure). This keeps the small airways open longer during exhalation, allowing for more complete emptying and reducing air trapping (FRC)
- **Diaphragmatic Breathing:** Focuses on shifting the work from "accessory muscles" (neck and shoulders) back to the primary pump
- **Chest Physiotherapy (CPT)/PEP Devices:** For patients with bronchiectatic phenotypes, using "Acapella" or "Flutter" valves daily is essential to prevent mucus plugging, which leads to V/Q mismatch and infection

4. Environmental and Sleep Hygiene

The "External" triggers must be strictly controlled to prevent acute-on-chronic flares.

- **Temperature/Humidity Control:** Extreme cold triggers bronchospasm; high humidity increases the density of inhaled air, increasing the work of breathing
- **Nocturnal Positioning:** For patients with Obesity Hypoventilation or Heart Failure overlap, sleeping at a **30-45 degree incline** reduces the cephalad pressure of abdominal contents on the diaphragm
- **Pollutant Avoidance:** Strict avoidance of biomass fuels, secondhand smoke, and high-VOC (Volatile Organic Compound) cleaning products

Follow-up Timing

1. Post-Hospitalization (The Vulnerable Window)

The first 30 days after an acute-on-chronic event carry the highest risk of readmission and mortality.

- **Timing**: **3 to 7 days** post-discharge
- **Key Objectives:**
  - Confirm **DME (Durable Medical Equipment)** delivery and setup (O2 concentrators, BiPAP)
  - Medication reconciliation (especially corticosteroid tapers and diuretic dosing)
  - Assess for "Post-ICU Syndrome" (cognitive changes or profound muscle wasting)

2. Routine Surveillance (Stable Phase)

For patients who are "dry and stable" on home therapy, the interval depends on the severity of their hypercapnia or hypoxemia.

- **Stage II/III (Compensated):** Every **3 months at least**
- **Stage IV (End-Stage/NIV Dependent):** Every **1 to 3 months**
- **Key Objectives:**

- Review **NIV Data Logs** (Usage hours, median leak, AHI, and pressure support effectiveness)
- Evaluate nutritional status (BMI/Weight trends)
- Screen for secondary complications (Edema, JVD, or mental status changes)

Patient Education and Adherence[29]

1. Device Mastery: The "Teach-Back" Protocol

Inhaler and NIV techniques often degrade over time. Clinical "Non-Adherence" is frequently just "Technical Error."

- **Inhaler Technique:** 90% of patients fail to coordinate the "press-and-breathe" or lack the inspiratory flow for dry powder inhalers (DPIs)
  - *Education:* Transition to **Valved Holding Chambers (Spacers)** or **Slow-Mist Inhalers** to remove the coordination requirement
- **NIV (BiPAP) Literacy:** The "mask-on" time is the most critical metric for preventing CO2 narcosis
  - *Education:* Address "Mask Leak" and "Pressure Sores" immediately. A patient who feels "suffocated" by the machine is likely experiencing **Patient-Ventilator Asynchrony**
  - Ensure the patient understands the peak flow and pulse oximetry values/changes
    - Safety Education: The "Early Warning System": Patients must be taught that their symptoms often lag behind their physiological numbers. By the time they feel "gasping" for air, they may already be in profound acidemia
      - **The 3% Rule:** Teach patients that a drop of **3% or more** from their stable baseline SpO2 (e.g., from 92% to 89%) is a "Yellow Zone" event, even if they feel okay
      - **The Peak Flow "Bellows" Test:** Explain that Peak Flow measures how well their "bellows" (the lungs and diaphragm) are working. A **15% drop** means the air is getting trapped, which will eventually tire out the heart
      - **Morning Headaches:** Educate them that a headache upon waking is a "Red Flag" that their BiPAP isn't clearing enough CO2 at night

2. Recognizing the "Yellow Zone" (Action Plans)

Patients should be empowered to distinguish between a "bad breathing day" and a "clinical exacerbation."

Zone	Status	Patient Action
Green	Baseline: Usual O2 needs; clear sputum	Continue maintenance medications and NIV
Yellow	Change: Increased cough, change in sputum color, or needing "rescue" inhaler >4 times/day	Initiate Action Plan: Start prescribed "standby" steroids/antibiotics; call the clinic
Red	Crisis: Confusion, blue lips, chest pain, or NIV not relieving breathlessness	Call 911/Emergency Room

Abbreviations: ABG, Arterial Blood Gas; CRF, Chronic Respiratory Failure; ER, Emergency Room; NIV, Non-Invasive Ventilation (e.g., BiPAP); O2, Oxygen; RAP, Respiratory Action Plan; SpO2, Peripheral Capillary Oxygen Saturation

Comorbidity Management[30–32]

In chronic respiratory failure (CRF), the lungs are rarely the only failing system. Because CRF creates a state of **chronic systemic inflammation**, **hypoxia**, and **hemodynamic stress**, comorbidities are the primary drivers of hospital readmissions.

Effective management requires shifting from "treating the lungs" to "managing the multi-organ consequences of respiratory failure."

1. Cardiovascular: The Heart-Lung Axis

Chronic hypoxia and hyperinflation create a high-pressure environment for the heart.

- **Group 3 Pulmonary Hypertension (I27.23):** Chronic alveolar hypoxia triggers pulmonary vasoconstriction
  - **Management:** Optimize O2 and NIV to reduce pulmonary vascular resistance (PVR). **Avoid** systemic vasodilators (e.g., Sildenafil) unless Group 1 PH is also present, as they can worsen V/Q mismatch
- **Cor Pulmonale and Right Heart Failure (I27.9):** * **Management:** Judicious use of diuretics. Monitor for **contraction alkalosis** (high HCO3), which suppresses the respiratory drive and worsens CO2 retention
- **Atrial Fibrillation:** Common due to right atrial stretch and hypoxia
  - **Management:** Rate control is preferred over aggressive rhythm control if hypoxia is the primary trigger

2. Metabolic and Nutritional: The "Pulmonary Cachexia"

CRF is an energy-intensive state where the work of breathing consumes a disproportionate share of caloric intake.

- **Pulmonary Cachexia (E64.0):** Defined by a loss of fat-free mass (muscle) despite a "normal" BMI
  - **Management:** High-protein, calorie-dense nutrition. Monitor **Fat-Free Mass Index (FFMI)** rather than just weight
- **Osteoporosis (M81.0):** High risk due to chronic systemic inflammation and frequent corticosteroid use
  - **Management:** Baseline DEXA scan; Vitamin D and Calcium supplementation; emphasize weight-bearing exercises in Pulmonary Rehab
- **Obesity Hypoventilation Syndrome (OHS):** Excess adipose tissue restricts chest wall expansion.
  - **Management:** Aggressive nocturnal NIV (BiPAP/AVAPS) and a structured weight loss program to improve the "bellows" function

3. Neuropsychiatric: The Anxiety-Dyspnea Loop

Breathlessness is a potent trigger for the amygdala, leading to a cycle of panic and worsening air trapping.

- **Anxiety and Depression (F41.1/F32.9):** Prevalent in up to 40% of CRF patients
  - **Management:** Cognitive Behavioral Therapy (CBT) and SSRIs. Avoid benzodiazepines, as they can cause **respiratory depression** and exacerbate hypercapnia

- **Cognitive Impairment:** Chronic nocturnal hypercapnia and intermittent hypoxia can lead to "brain fog" or executive dysfunction
  - **Management:** Screen with the **MoCA** (Montreal Cognitive Assessment). Improvement in cognition is often a surrogate marker for effective NIV titration. **See appendix for link to MoCA assessment**

4. Sleep and Mechanical: The Nocturnal Challenge

- **Obstructive Sleep Apnea (OSA) Overlap:** When COPD and OSA coexist, desaturations are more profound
  - **Management:** Transition from CPAP to **BiPAP** to provide ventilatory support (pressure support) in addition to stenting the airway
- **GERD (K21.9):** Micro-aspiration of gastric acid triggers bronchospasm and parenchymal inflammation.
  - **Management:** Proton Pump Inhibitors (PPIs) and head-of-bed elevation to 30–45°

Cost-Smart Options[14,17]

1. Physiological Support and Equipment Optimization

The selection of durable medical equipment (DME) should be guided by the patient's clinical phenotype and lifestyle requirements to ensure adherence and safety.

- **Non-Invasive Ventilation (NIV):** Utilizing nocturnal BiPAP or AVAPS serves to offload the inspiratory muscles, allowing for diaphragmatic recovery. Effective ventilation resets central chemoreceptor sensitivity, leading to improved daytime alertness and a reduction in hypercapnic symptoms
  - Provides patient with a home option to avoid hospitalization/institutionalization
- **Oxygen Delivery Systems:** The use of portable oxygen concentrators (POCs) or oxygen-conserving reservoir cannulas facilitates increased mobility. These systems reduce the frequency of tank deliveries and support higher levels of physical activity
  - Provides patient with a home option to avoid hospitalization/institutionalization

2. Proactive Exacerbation Management

Empowering the patient through a structured Action Plan is a primary strategy for reducing emergency department utilization.

- **The "Yellow Zone" Protocol:** Providing patients with "standby" prescriptions for corticosteroids and antibiotics allows for immediate intervention at the onset of increased dyspnea or changes in sputum purulence. Early treatment often halts the progression toward acute-on-chronic respiratory failure
- **Nocturnal Monitoring:** Educating patients on the significance of morning headaches or excessive daytime somnolence allows for early detection of nocturnal hypoventilation, prompting timely adjustments to NIV settings

3. Enhancing Ventilatory Reserve

Interventions aimed at the musculoskeletal system are essential for improving the patient's overall oxygen economy.

- **Pulmonary Rehabilitation:** Structured exercise training addresses peripheral muscle sarcopenia. By increasing the efficiency of oxygen utilization in the skeletal muscles, the total ventilatory demand during activities of daily living (ADLs) is decreased
- **Breathing Mechanics:** Training in pursed-lip breathing provides intrinsic positive end-expiratory pressure (PEEP), which mitigates dynamic hyperinflation and reduces the work of breathing

4. Comprehensive Supportive Care

- **Nutritional Therapy:** Given the high metabolic cost of breathing in CRF, a high-protein, calorie-dense diet is necessary to maintain fat-free mass. Small, frequent meals are recommended to prevent gastric distension from impeding diaphragmatic excursion
- **Psychosocial Support:** Addressing the "Dyspnea-Anxiety-Dyspnea" cycle is critical. Integrating cognitive-behavioral techniques helps manage the psychological distress associated with chronic breathlessness, thereby improving treatment adherence

Comparative Cost and Clinical Efficacy

Intervention	Estimated Monthly Cost	Clinical "Best Use" Case	Primary Outcome
Long-Term Oxygen (LTOT)	Low to Moderate ($150–$400)	Type I Failure ($PaO_2 \leq 55$mmHg)	Prevents Cor Pulmonale; improves survival in COPD
Nocturnal NIV (BiPAP)	Moderate ($200–$600+)	Type II Failure ($PaCO_2 \geq 50$mmHg)	Offloads diaphragm; reduces 30-day readmissions
Triple Inhaler Therapy	Moderate ($300–$600)	Obstructive Phenotype (COPD/Asthma)	Reduces exacerbation frequency and air trapping
Pulmonary Rehab	Moderate ($2,000–$4,000/cycle)	All CRF patients with functional deficit	Increases 6MWD; reduces "panic-dyspnea" cycle
Endobronchial Valves	High ($20,000+ upfront)	Heterogeneous Upper-Lobe Emphysema	Improves FEV1 and quality of life in select patients

Abbreviations: 6MWD, 6-Minute Walk Distance; BiPAP, Bilevel Positive Airway Pressure; COPD, Chronic Obstructive Pulmonary Disease; CRF, Chronic Respiratory Failure; FEV1, Forced Expiratory Volume in 1 Second; LTOT, Long-Term Oxygen Therapy; NIV, Non-Invasive Ventilation; $PaCO_2$, Partial Pressure of Carbon Dioxide; PaO_2, Partial Pressure of Oxygen; QoL, Quality of Life

5. CODING REMINDERS AND CASE EXAMPLES BOX

Annual Clinical Review and Confirmation

Requirement	Chronic Respiratory Failure (CRF) Standards
Documentation Frequency	The active status of the respiratory failure and the compensatory state must be documented annually with MEAT (Monitor, Evaluate, Assess, Treat) by 12/31
Face-to-Face	Required. In-person or synchronized audio-visual telehealth visits are eligible for HCC reconfirmation
Physiologic Precision	Must specify the Type (Hypoxemic vs. Hypercapnic), Acuteness (Chronic vs. Acute-on-Chronic), and Mechanism (e.g., secondary to COPD, OHS, or Neuromuscular disease)
Objective Support	Documentation should include recent ABG values (PaCO2, HCO3, pH) and dependence on life-sustaining technology (e.g., Long-term Oxygen or NIV)
Complication Linkage	Must explicitly link secondary conditions, such as Cor Pulmonale or Secondary Polycythemia, to the underlying chronic respiratory failure

Good Documentation is Comprehensive Coding

Requirement	Chronic Respiratory Failure (CRF) Standards
Documentation Frequency	YES—Active respiratory failure, physiologic type (Hypoxemic/Hypercapnic), and dependence on life-sustaining technology (Oxygen/NIV) must be documented annually with MEAT by 12/31
Face-to-Face	Required (In-person or synchronized audio-visual telehealth visits)
Precision	Must specify Type (Hypoxemic vs. Hypercapnic), Acuteness (Chronic vs. Acute-on-Chronic), and Mechanism (e.g., secondary to COPD, Neuromuscular disease, or Kyphoscoliosis)

EHR Tips

Feature	EHR Configuration Tip	Clinical and Quality Impact
Auto-Classification	ABG Phenotype Mapper: Auto-populates problem lists with specific failure types (e.g., J96.12 Chronic Hypercapnic) based on lab interfaces (PaCO2>45, HCO3>30)	Bridges the gap between lab data and the active Problem List; ensures patients are flagged for appropriate ventilation titration
Alert Systems	Surveillance Alert: Flags if no ABG or nocturnal oximetry has been documented in the last 6–12 months for Stage IV patients	Supports GOLD/ATS guidelines and prevents "silent" decompensation or CO2 narcosis between visits.
BPA (Best Practice Advisory)	Cardiopulmonary Loop: Triggers for an Echocardiogram in patients with PaO2<60 mmHg to screen for Group 3 Pulmonary Hypertension	Closes the gap for Cor Pulmonale screening and ensures timely management of right-heart strain
Problem List Prompts	"Technology Dependence" Hard-Stop:	Stratifies disease severity and

Feature	EHR Configuration Tip	Clinical and Quality Impact
	Mandatory field for Z-codes (Z99.81 Oxygen or Z99.89 NIV) when adding a Chronic Respiratory Failure diagnosis	ensures life-sustaining equipment is tracked across the continuum
Outcome Tracking	ePRO Integration: Automated patient portal prompts for the CAT (COPD Assessment Test) or mMRC Dyspnea Scale prior to visits	Provides objective longitudinal data on functional decline and guides treatment escalation or palliative referral

Abbreviations: ABG, Arterial Blood Gas; ATS, American Thoracic Society; BPA, Best Practice Advisory; CAT, COPD Assessment Test; CRF, Chronic Respiratory Failure; EHR, Electronic Health Record; ePRO, Electronic Patient-Reported Outcomes; GOLD, Global Initiative for Chronic Obstructive Lung Disease; HCO3, Bicarbonate; J96.12, Chronic Hypercapnic Respiratory Failure; mMRC, Modified Medical Research Council (Dyspnea Scale); NIV, Non-Invasive Ventilation; PaCO2, Partial Pressure of Carbon Dioxide; PaO2, Partial Pressure of Oxygen; PH, Pulmonary Hypertension

Brief Case Examples

SUCCESS CASE: Accurate Description of Phenotype and Status

Case: "68yo female with **Chronic Hypercapnic Respiratory Failure (J96.12)** secondary to Stage IV (GOLD Group E) COPD. Latest ABG (02/15/2026) on current settings: pH 7.39, PaCO2 58 mmHg, HCO3 mEq/L, consistent with chronic compensation. Patient is adherent to **nocturnal NIV (Z99.89)** (Settings: IPAP 18, EPAP 6) and **Long-Term Oxygen Therapy (Z99.81)** at 3L NC. Denies morning headaches or increased somnolence."

- **Result:** Appropriately documents the specific failure phenotype (Hypercapnic), chronicity (compensated ABG), and technology dependence (NIV and O2 status codes)

PITFALL CASE: The "Unspecified" Diagnostic Trap

Case: "72yo male here for follow-up of chronic lung disease and respiratory failure. Patient is stable on home oxygen. Refilled inhalers and will continue current home setup. Follow up in 6 months."

- **Result:** Documentation fails to specify the phenotype (Hypoxemic vs. Hypercapnic) and lacks **MEAT** evidence (no specific oxygen flow rates, no ABG review, or mechanical settings cited). Without proper documentation, patient would not receive adequate care

FIX: Re-establishing Specificity and Causal Linkage

Case: "**Chronic Hypoxemic Respiratory Failure (J96.11)** stable; patient currently dependent on **Supplemental Oxygen (Z99.81)** at 4 L/min for all ADLs. SpO2 91% on current flow. Condition is secondary to **Group 3 Pulmonary Hypertension (I27.23)** and underlying ILD. Evaluating for signs of **Cor Pulmonale (I27.9)**; trace pedal edema noted, continue Furosemide 20 mg daily. Patient is in Month 4 of Pulmonary Rehabilitation."

- **Result:** Documents the primary failure code by specifying the type (Hypoxemic), links the causal complication (Pulmonary HTN), and provides objective clinical data (oxygen saturation and physical exam findings). Accurately reflects the patient's true clinical complexity

Appendix

Screening and Staging Tool Links

Tool	Focus and Clinical Significance	Link
COPD Assessment Test (CAT)	Symptom Burden: Measures the systemic impact of cough, phlegm, and sleep quality. A score >10 indicates significant impairment	CAT Tool
mMRC Dyspnea Scale	Functional Limitation: A 0–4 scale grading breathlessness during ADLs. Grade ≥2 is a major predictor of mortality and readmission	MmRC Link
CAPTURE Tool	Early Detection: Combines 5 questions with Peak Flow to find "hidden" COPD. Essential for high-risk primary care screening	CAPTURE Link
STOP-BANG	Nocturnal Risk: Screens for OSA and nocturnal hypoventilation—a critical precursor to Chronic Hypercapnic Failure	STOP-BANK Link

BODE Index for COPD Survival: Link

MoCA (Montreal Cognitive Assessment): Link

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