International Journal of Research and Review
[Indexed, Peer-Reviewed Journal]
Volume 13; Issue: 2; February 2026
Website: www.ijrrjournal.com
Review Paper *E-ISSN: 2349-9788; P-ISSN: 2454-2237*

Role of Incentive Spirometry in Patients with Hypercapnic Respiratory Failure Receiving Non-Invasive Ventilation: A Review of Physiological Rationale and Clinical Evidence

Dr. Sipra Dash (PT)[1], Dr. Asit Kumar Bhoi (PT)[2], Dr. Santosh Kumar Behera (OT)[3], Devranjan Suna[4], Dr. Dwarikanath Rout (PT)[5]

[1]Physiotherapist, KIMS School of Physiotherapy, KIMS, KIIT DU, Bhubaneshwar, Odisha, India
[2]Physiotherapist, KIMS School of Physiotherapy, KIMS, KIIT DU, Bhubaneshwar, Odisha, India
[3]Occupational Therapist, KIMS, KIIT DU, Bhubaneshwar, Odisha, India
[4]Intern, KIMS School of Physiotherapy, KIMS, KIIT DU, Bhubaneshwar, Odisha, India
[5]Assistant Professor, KIMS School of Physiotherapy, KIMS, KIIT DU, Bhubaneshwar, Odisha, India

Corresponding Author: Dr. Dwarikanath Rout (PT)

DOI: https://doi.org/10.52403/ijrr.20260329

ABSTRACT

Background: Hypercapnic respiratory failure (HRF) is characterized by elevated arterial carbon dioxide tension resulting from inadequate alveolar ventilation and is most observed in acute exacerbations of chronic obstructive pulmonary disease (COPD). Non-invasive ventilation (NIV) is the recommended first-line therapy and has been shown to reduce mortality, need for intubation, and hospital length of stay. However, while NIV effectively reduces ventilatory load and corrects respiratory acidosis, it does not directly restore inspiratory muscle strength or enhance lung expansion capacity. Incentive spirometry (IS), a volume-oriented breathing exercise promoting sustained maximal inspiration, may offer adjunctive benefits by stimulating diaphragmatic activation and alveolar recruitment. The potential role of IS in patients with HRF receiving NIV remains insufficiently explored.

Objective: To review and synthesize current evidence regarding the physiological rationale, clinical applicability, and potential benefits of incentive spirometry in patients with hypercapnic respiratory failure undergoing non-invasive ventilation.

Methods: A narrative review was conducted using peer-reviewed literature indexed in major biomedical databases, including studies focusing on hypercapnic respiratory failure, non-invasive ventilation, inspiratory muscle training, and incentive spirometry. Clinical trials, meta-analyses, and guideline documents addressing COPD, ventilatory support, and pulmonary rehabilitation were analyzed. Evidence was synthesized to examine mechanistic plausibility, indirect clinical support, and gaps in current research.

Results: Non-invasive ventilation improves alveolar ventilation, reduces respiratory muscle workload, offsets intrinsic positive end-expiratory pressure, and corrects hypercapnia. However, inspiratory muscle dysfunction often persists despite ventilatory unloading. Evidence from COPD rehabilitation and ventilatory weaning studies suggests that inspiratory muscle activation strategies improve maximal inspiratory pressure, dyspnea, and

functional capacity. Incentive spirometry promotes sustained maximal inspiration, enhances diaphragmatic excursion, and facilitates alveolar recruitment. Although direct randomized controlled trials evaluating IS during active NIV in acute HRF are limited, indirect evidence supports its physiological plausibility as an adjunctive therapy. Potential benefits include improved inspiratory muscle engagement, prevention of atelectasis during NIV breaks, and facilitation of NIV weaning.

Conclusion: Incentive spirometry represents a physiologically rational adjunct to non-invasive ventilation in hypercapnic respiratory failure. While current evidence is largely indirect, combining ventilatory unloading with active inspiratory muscle engagement may enhance recovery and functional outcomes. High-quality randomized controlled trials are required to determine its clinical efficacy and establish standardized implementation protocols.

Keywords: *Hypercapnic respiratory failure; Non-invasive ventilation; Incentive spirometry; Inspiratory muscle training; COPD; Respiratory muscle dysfunction; Pulmonary rehabilitation.*

1. INTRODUCTION

Hypercapnic respiratory failure (HRF), also referred to as Type II respiratory failure, is a critical clinical condition characterized by elevated arterial carbon dioxide tension ($PaCO_2$ > 45 mmHg) resulting from inadequate alveolar ventilation. Unlike hypoxemic respiratory failure, where impaired oxygenation predominates, HRF reflects a primary failure of ventilatory mechanics or respiratory drive. The condition represents a significant cause of morbidity and mortality worldwide, particularly among patients with chronic respiratory disorders.

The most prevalent cause of acute hypercapnic respiratory failure is acute exacerbation of chronic obstructive pulmonary disease (COPD). According to the Global Initiative for Chronic Obstructive Lung Disease, COPD remains a leading cause of global mortality and is projected to rank among the top causes of death worldwide. Acute exacerbations are frequently complicated by alveolar hypoventilation, dynamic hyperinflation, respiratory muscle fatigue, and worsening ventilation-perfusion mismatch, all contributing to CO_2 retention and respiratory acidosis.

In addition to COPD, HRF commonly occurs in:

- Obesity hypoventilation syndrome, where excessive body mass increases respiratory load and reduces chest wall compliance
- Neuromuscular disorders such as myasthenia gravis and motor neuron disease, where respiratory muscle weakness impairs ventilatory capacity
- Severe asthma with airflow limitation
- Thoracic cage deformities such as kyphoscoliosis
- Central hypoventilation syndromes

The pathophysiological hallmark of HRF is reduced alveolar ventilation relative to metabolic carbon dioxide production. In many chronic respiratory disorders, hyperinflation alters diaphragm geometry, shortens muscle fibers, and places the inspiratory muscles at a mechanical disadvantage. Over time, this leads to reduced maximal inspiratory pressure (MIP), increased neural respiratory drive, and ultimately ventilatory pump failure.

1.1 Clinical Burden and Significance

Hypercapnic respiratory failure is associated with:

- Increased risk of intubation
- Higher in-hospital mortality
- Prolonged intensive care unit (ICU) stay
- Greater healthcare utilization
- Reduced long-term functional capacity

Acute hypercapnia can lead to respiratory acidosis, altered mental status, hemodynamic instability, and multi-organ dysfunction if not promptly treated.

Therefore, early recognition and timely ventilatory support are crucial.

1.2 Role of Non-Invasive Ventilation in HRF

Non-invasive ventilation (NIV) has revolutionized the management of acute hypercapnic respiratory failure. Clinical practice guidelines from the European Respiratory Society and the American Thoracic Society strongly recommend NIV as first-line therapy in patients with acute exacerbation of COPD presenting with respiratory acidosis.

Extensive randomized controlled trials and meta-analyses indexed in Scopus and Web of Science demonstrate that NIV:

- Improves alveolar ventilation
- Reduces $PaCO_2$ levels
- Corrects respiratory acidosis
- Decreases the need for invasive mechanical ventilation
- Reduces mortality
- Shortens hospital stay

Mechanistically, NIV provides inspiratory positive airway pressure (IPAP) to augment tidal volume and expiratory positive airway pressure (EPAP) to prevent alveolar collapse. By unloading fatigued respiratory muscles, NIV decreases the work of breathing and improves minute ventilation.

1.3 Persistent Challenges Despite NIV

Although NIV effectively stabilizes gas exchange during acute episodes, several physiological limitations remain:

- Inspiratory Muscle Weakness: NIV reduces respiratory muscle load but does not actively strengthen the diaphragm or accessory muscles. Prolonged ventilatory assistance may even reduce spontaneous inspiratory effort.
- Dynamic Hyperinflation and Reduced Lung Expansion: COPD-related hyperinflation persists even after NIV initiation, limiting inspiratory capacity and tidal volume generation.
- Atelectasis and Micro-Derecruitment: Alveolar collapse may occur during breaks from NIV support, particularly in patients with poor inspiratory effort.
- Delayed Weaning and Ventilatory Dependence: Respiratory muscle fatigue contributes significantly to NIV weaning failure.

These challenges highlight the need for adjunctive interventions that target lung expansion and respiratory muscle conditioning during and after NIV support.

1.4 Rationale for Lung Expansion Strategies in HRF

Pulmonary rehabilitation principles emphasize the importance of inspiratory muscle training and lung expansion exercises in improving respiratory mechanics. Incentive spirometry (IS) is a volume-oriented breathing exercise that promotes sustained maximal inspiration and provides visual feedback to encourage patient participation.

Traditionally used in postoperative care to prevent atelectasis, IS increases transpulmonary pressure, facilitates alveolar recruitment, enhances diaphragmatic excursion, and improves inspiratory capacity. Evidence from COPD rehabilitation studies demonstrates improvements in inspiratory muscle strength, dyspnea scores, and exercise tolerance following structured inspiratory training.

Given that hypercapnic respiratory failure is characterized by- Impaired inspiratory muscle performance, reduced inspiratory capacity, ventilatory inefficiency, the theoretical integration of incentive spirometry with NIV appears physiologically sound.

1.5 Gap in Current Literature

Despite established benefits of NIV and growing evidence supporting inspiratory muscle training in chronic respiratory disease, there is limited direct evidence examining the role of incentive spirometry specifically in patients with hypercapnic respiratory failure receiving NIV.

Most available studies focus on:

- Stable COPD populations
- Postoperative pulmonary complications
- Weaning from invasive mechanical ventilation

There remains a clear gap in high-quality randomized controlled trials evaluating whether incentive spirometry can enhance gas exchange, improve inspiratory muscle strength, facilitate NIV weaning, or reduce hospital stay in hypercapnic patients.

1.6 Aim of the Review

In view of the physiological rationale and existing indirect evidence, this review aims to:

- Examine the pathophysiological basis of hypercapnic respiratory failure.
- Analyze the mechanisms and limitations of non-invasive ventilation.
- Synthesize available evidence regarding incentive spirometry and inspiratory muscle training in COPD and ventilatory support contexts.
- Explore the potential synergistic role of incentive spirometry as an adjunct to NIV.
- Identify gaps in literature and propose future research directions.

By integrating evidence from Scopus- and Web of Science-indexed literature, this review seeks to provide a comprehensive understanding of whether incentive spirometry has a clinically meaningful role in the management of hypercapnic respiratory failure patients receiving non-invasive ventilation.

2. PATHOPHYSIOLOGY OF HYPERCAPNIC RESPIRATORY FAILURE

Hypercapnic respiratory failure (HRF) results from an imbalance between carbon dioxide (CO_2) production and alveolar ventilation. It is fundamentally a disorder of ventilatory pump failure rather than oxygenation impairment alone. The condition develops when minute ventilation becomes insufficient to eliminate metabolically generated CO_2, leading to arterial hypercapnia ($PaCO_2 > 45$ mmHg) and often respiratory acidosis.

Understanding HRF requires integration of respiratory mechanics, gas exchange physiology, neuromuscular function, and ventilatory control mechanisms.

2.1 Alveolar Ventilation and CO_2 Retention

Arterial CO_2 tension is determined by the relationship:

Where: $PaCO_2 \propto VCO_2/V_A$

- VCO_2 = metabolic CO_2 production
- V_A = alveolar ventilation

Hypercapnia occurs when alveolar ventilation declines relative to CO_2 production. In acute hypercapnic states, this imbalance leads to rapid respiratory acidosis due to insufficient renal compensation.

In chronic conditions such as COPD, renal bicarbonate retention partially buffers acidosis, but during acute exacerbations, compensatory mechanisms are overwhelmed.

2.2 Mechanical Load–Capacity Imbalance

- One of the central concepts in HRF is the imbalance between respiratory load and muscle capacity.

2.2.1 Increased Respiratory Load

Patients with obstructive lung disease experience:

- Increased airway resistance
- Dynamic hyperinflation
- Intrinsic positive end-expiratory pressure (PEEPi)
- Increased elastic recoil load

Dynamic hyperinflation shortens inspiratory muscle fibers, particularly the diaphragm, shifting them to a mechanically disadvantaged portion of their length–tension curve. This reduces force-generating capacity and increases the work of breathing.

During acute exacerbations, airway narrowing and mucus plugging further elevate resistive load, dramatically increasing ventilatory demand.

2.2.2 Reduced Muscle Capacity

Respiratory muscle dysfunction plays a pivotal role in HRF development. Several mechanisms contribute:

- Chronic hyperinflation-induced diaphragmatic flattening
- Fiber type transformation from fatigue-resistant type I to type II fibers
- Systemic inflammation and oxidative stress
- Malnutrition and corticosteroid-induced myopathy
- Neuromuscular transmission abnormalities

Studies indexed in respiratory physiology literature demonstrate significant reductions in maximal inspiratory pressure (MIP) in patients with advanced COPD. When the load imposed on the respiratory system exceeds the capacity of inspiratory muscles, ventilatory pump failure ensues.

2.3 Dynamic Hyperinflation and Intrinsic PEEP

Dynamic hyperinflation is a hallmark of obstructive hypercapnic respiratory failure. Incomplete expiration due to airflow limitation causes air trapping, increasing end-expiratory lung volume.

Consequences include:

- Elevated intrinsic PEEP (PEEPi)
- Increased inspiratory threshold load
- Reduced inspiratory capacity
- Increased work of breathing

Before initiating each breath, inspiratory muscles must first overcome PEEPi. This threshold loading further accelerates muscle fatigue.

Clinical guidelines from the American Thoracic Society emphasize that dynamic hyperinflation is a major contributor to acute ventilatory failure in COPD exacerbations.

2.4 Ventilation–Perfusion (V/Q) Mismatch and Dead Space Ventilation

Although HRF is primarily ventilatory in origin, gas exchange abnormalities contribute significantly.

In COPD and other chronic lung diseases:

- Heterogeneous airway obstruction leads to V/Q mismatch
- Areas of high ventilation relative to perfusion increase physiological dead space
- Inefficient CO_2 elimination occurs despite increased respiratory effort

Increased dead space fraction (VD/VT ratio) reduces effective alveolar ventilation, thereby promoting hypercapnia even when minute ventilation appears elevated.

2.5 Central Ventilatory Drive Impairment

In some patients, particularly those with obesity hypoventilation syndrome or chronic hypercapnia, central chemosensitivity to CO_2 becomes blunted.

Chronic exposure to elevated $PaCO_2$ leads to:

- Reduced responsiveness of medullary respiratory centers
- Greater dependence on hypoxic ventilatory drive
- Increased vulnerability during oxygen therapy

The European Respiratory Society has highlighted the importance of cautious oxygen administration in hypercapnic patients to avoid further ventilatory suppression.

2.6 Acute-on-Chronic Hypercapnic Failure

Many patients experience acute-on-chronic respiratory failure. In these cases:

- Baseline $PaCO_2$ is chronically elevated
- Renal compensation maintains near-normal pH
- Superimposed infection, bronchospasm, or cardiac dysfunction triggers acute decompensation

The rapid increase in ventilatory demand overwhelms fatigued respiratory muscles, resulting in acute respiratory acidosis.

Clinically, this manifests as:

- Tachypnea
- Use of accessory muscles
- Paradoxical abdominal breathing
- Altered mental status

- Elevated $PaCO_2$ with low pH

Without ventilatory support, progressive CO_2 narcosis may develop.

2.7 NIV and Load Reduction

Non-invasive ventilation addresses several pathophysiological abnormalities:

- Provides inspiratory pressure support to augment tidal volume
- Reduces respiratory muscle workload
- Offsets intrinsic PEEP with external EPAP
- Improves alveolar ventilation

Guidelines from the Global Initiative for Chronic Obstructive Lung Disease recommend early NIV initiation in acute hypercapnic exacerbations with acidosis.

However, while NIV unloads the respiratory muscles, it does not directly restore inspiratory muscle strength or reverse structural changes associated with chronic hyperinflation.

2.8 Implications for Adjunctive Lung Expansion Therapy

Given that HRF is characterized by:

- Inspiratory muscle weakness
- Reduced inspiratory capacity
- Persistent hyperinflation
- Atelectatic tendencies during NIV breaks

Therapeutic strategies that enhance diaphragmatic excursion and promote sustained maximal inspiration may offer physiological benefit.

Incentive spirometry, by encouraging slow, deep inhalation with visual feedback, increases transpulmonary pressure and recruits collapsed alveoli. It may counteract micro-atelectasis and stimulate inspiratory muscle activation, potentially complementing NIV-induced unloading.

The interaction between ventilatory support (load reduction) and respiratory muscle conditioning (capacity improvement) represents a promising conceptual framework in HRF management.

3. NON-INVASIVE VENTILATION IN HYPERCAPNIC RESPIRATORY FAILURE: MECHANISMS AND CLINICAL EVIDENCE

Non-invasive ventilation (NIV) has become the cornerstone of management in acute hypercapnic respiratory failure (HRF), particularly in acute exacerbations of chronic obstructive pulmonary disease (COPD). Over the past three decades, robust randomized controlled trials and meta-analyses have consistently demonstrated that early initiation of NIV significantly reduces mortality, need for endotracheal intubation, and length of hospital stay.

Clinical practice guidelines from the European Respiratory Society and American Thoracic Society strongly recommend bilevel positive airway pressure as first-line ventilatory support in patients with acute hypercapnic exacerbations associated with respiratory acidosis ($pH \leq 7.35$).

3.1 Physiological Mechanisms of NIV in HRF

- The beneficial effects of NIV are mediated through several interrelated physiological mechanisms.

3.1.1 Augmentation of Alveolar Ventilation

- Inspiratory positive airway pressure (IPAP) increases tidal volume and minute ventilation, thereby improving alveolar ventilation (V_A). Enhanced CO_2 clearance results in a progressive reduction in $PaCO_2$ and correction of respiratory acidosis.
- By directly supporting inspiratory effort, NIV compensates for fatigued respiratory muscles and improves ventilation efficiency.

3.1.2 Respiratory Muscle Unloading

One of the most critical mechanisms of NIV is respiratory muscle unloading. In hypercapnic respiratory failure:

- The diaphragm operates at a mechanical disadvantage due to hyperinflation.
- Accessory muscles are recruited excessively.
- Work of breathing is markedly elevated.

NIV reduces inspiratory effort by decreasing transdiaphragmatic pressure swings and lowering neural respiratory drive. Electromyographic studies have demonstrated significant reductions in diaphragmatic activity during NIV application.

This unloading effect interrupts the cycle of fatigue, preventing progression to ventilatory collapse.

3.1.3 Reduction of Intrinsic PEEP

In obstructive lung disease, intrinsic positive end-expiratory pressure (PEEPi) creates an inspiratory threshold load. Application of expiratory positive airway pressure (EPAP) partially offsets PEEPi, thereby:

- Reducing inspiratory threshold work
- Improving patient–ventilator synchrony
- Enhancing comfort

This mechanism is particularly relevant in COPD-related hypercapnic respiratory failure.

3.1.4 Improved Ventilation–Perfusion Matching

- By increasing tidal volume and promoting alveolar recruitment, NIV reduces atelectasis and improves ventilation distribution. This contributes to better ventilation–perfusion matching and more efficient gas exchange.
- Although NIV primarily addresses ventilatory failure, secondary improvements in oxygenation are frequently observed.

3.2 Clinical Evidence Supporting NIV in HRF

- Multiple high-quality randomized controlled trials indexed in Scopus and Web of Science have established NIV as standard therapy in acute hypercapnic respiratory failure.

3.2.1 Reduction in Intubation Rates

Compared to standard medical therapy alone, NIV significantly reduces the need for invasive mechanical ventilation. Avoiding intubation reduces risks such as:

- Ventilator-associated pneumonia
- Airway trauma
- Sedation-related complications

This has translated into improved survival outcomes in acute exacerbations of COPD.

3.2.2 Mortality Reduction

- Meta-analyses consistently demonstrate that NIV reduces in-hospital mortality in patients with acute hypercapnic respiratory failure due to COPD exacerbation. Early initiation, particularly before severe acidosis develops, yields the most favorable outcomes.
- Guidelines from the Global Initiative for Chronic Obstructive Lung Disease recommend prompt NIV initiation when pH falls below 7.35 in the setting of elevated $PaCO_2$.

3.2.3 Shortened Hospital Stay

- NIV reduces ICU and overall hospital length of stay by accelerating correction of respiratory acidosis and stabilizing clinical status. Earlier mobilization and transition to ward-based care are possible when invasive ventilation is avoided.

3.2.4 Chronic Use of NIV

- In selected patients with chronic hypercapnic COPD, long-term nocturnal NIV has demonstrated improvements in gas exchange and health-related quality of life. High-intensity NIV strategies targeting substantial $PaCO_2$ reduction are increasingly explored in chronic management.
- However, evidence regarding optimal patient selection and long-term adherence remains under investigation.

3.3 Limitations of NIV in Hypercapnic Respiratory Failure

- Despite its proven benefits, NIV has limitations that warrant consideration.

3.3.1 Persistent Inspiratory Muscle Weakness

- NIV primarily unloads respiratory muscles but does not directly strengthen them. Prolonged unloading may potentially contribute to disuse atrophy if spontaneous effort is minimized for extended periods.

- Respiratory muscle dysfunction, therefore, may persist even after acute stabilization.

3.3.2 Incomplete Reversal of Hyperinflation

- Although EPAP reduces inspiratory threshold load, it does not fully reverse structural hyperinflation or restore diaphragmatic curvature. Mechanical disadvantages of the respiratory pump may remain after acute episodes.

3.3.3 NIV Failure and Weaning Challenges

NIV failure rates vary between 10–40% depending on severity and patient selection. Predictors of failure include:

- Severe acidosis
- Persistent tachypnea
- Reduced consciousness
- Excessive secretions

Weaning from NIV can also be challenging in patients with poor inspiratory muscle reserve.

3.3.4 Dependence on Passive Support

- NIV is fundamentally a supportive therapy. While it corrects gas exchange abnormalities, it does not actively rehabilitate respiratory muscle performance or enhance lung expansion capacity.
- This limitation creates a rationale for integrating adjunctive strategies aimed at restoring respiratory muscle function.

3.4 Rationale for Combining NIV with Respiratory Muscle Activation

The physiological framework of hypercapnic respiratory failure suggests two therapeutic targets:

- Load reduction (achieved by NIV)
- Capacity enhancement (potentially achievable through inspiratory muscle training or lung expansion exercises)

Incentive spirometry represents a non-invasive, low-cost intervention that promotes sustained maximal inspiration and active diaphragmatic engagement.

While NIV decreases respiratory workload, incentive spirometry may:

- Stimulate diaphragmatic excursion
- Improve inspiratory muscle recruitment
- Promote alveolar expansion
- Reduce micro-atelectasis

The integration of ventilatory support with active respiratory muscle conditioning could theoretically accelerate recovery and facilitate NIV weaning.

3.5 Clinical Gap

Although NIV is strongly evidence-based in hypercapnic respiratory failure, research exploring adjunctive lung expansion exercises during NIV therapy is sparse. Most existing literature focuses on postoperative pulmonary care or stable COPD rehabilitation rather than acute hypercapnic states.

Thus, the question remains:

Can incentive spirometry provide additive benefits in patients receiving NIV for hypercapnic respiratory failure?

This question forms the basis for further exploration in the subsequent section.

4. INCENTIVE SPIROMETRY AND INSPIRATORY MUSCLE ACTIVATION: MECHANISMS AND CLINICAL EVIDENCE

Incentive spirometry (IS) is a volume-oriented breathing exercise designed to promote sustained maximal inspiration through visual feedback. Although traditionally employed in postoperative pulmonary care to prevent atelectasis, its physiological effects extend to respiratory muscle activation, lung expansion, and enhancement of inspiratory capacity. These properties make IS a potentially relevant adjunctive intervention in patients with hypercapnic respiratory failure (HRF) receiving non-invasive ventilation (NIV).

This section examines the physiological mechanisms, clinical evidence, and theoretical relevance of incentive spirometry in the context of ventilatory support.

4.1 Physiological Basis of Incentive Spirometry

Incentive spirometry functions by encouraging slow, deep inhalation to near-total lung capacity, followed by a short inspiratory hold. The key physiological mechanisms include:

4.1.1 Increased Transpulmonary Pressure

Sustained maximal inspiration increases transpulmonary pressure gradients, facilitating:

- Recruitment of collapsed alveoli
- Re-expansion of dependent lung regions
- Improved lung compliance

This mechanism is particularly relevant in patients with micro-atelectasis or regional hypoventilation.

4.1.2 Diaphragmatic Activation

Unlike rapid shallow breathing patterns observed in respiratory distress, IS promotes controlled diaphragmatic breathing. This results in:

- Greater diaphragmatic excursion
- Reduced accessory muscle dominance
- Improved inspiratory muscle coordination

Repeated maximal inspirations may stimulate neuromuscular recruitment and enhance inspiratory muscle endurance over time.

4.1.3 Improved Inspiratory Capacity

- Chronic obstructive pulmonary disease (COPD) and hyperinflation reduce inspiratory capacity. By encouraging deep inhalation, IS may temporarily increase inspiratory capacity and reduce end-expiratory lung volume.
- Although it does not reverse structural hyperinflation, it may improve dynamic lung expansion during recovery phases.

4.1.4 Reduction of Atelectasis

- Atelectasis contributes to impaired ventilation–perfusion matching and reduced effective alveolar ventilation. IS enhances alveolar ventilation distribution and may reduce physiological dead space.
- In postoperative and ICU populations, incentive spirometry has been associated with improved lung volumes and reduced incidence of radiographic atelectasis.

4.2 Inspiratory Muscle Function in Hypercapnic Respiratory Failure

Hypercapnic respiratory failure is strongly associated with inspiratory muscle dysfunction. Mechanisms include:

- Diaphragmatic flattening due to hyperinflation
- Oxidative stress and systemic inflammation
- Muscle fiber remodeling
- Fatigue from sustained high work of breathing

Studies in COPD populations demonstrate reduced maximal inspiratory pressure (MIP) and decreased endurance capacity.

While NIV effectively unloads respiratory muscles, it does not directly enhance muscle strength. Thus, there is theoretical benefit in combining ventilatory unloading with active inspiratory muscle engagement.

4.3 Evidence from COPD and Chronic Respiratory Disease

Pulmonary rehabilitation literature indexed in Scopus and Web of Science demonstrates that inspiratory muscle training (IMT) improves:

- Maximal inspiratory pressure
- Dyspnea scores
- Exercise tolerance
- Health-related quality of life

Although incentive spirometry differs from threshold-loaded IMT devices, both promote sustained inspiratory effort.

Guidance from the Global Initiative for Chronic Obstructive Lung Disease recognizes inspiratory muscle weakness as a contributor to dyspnea and supports rehabilitation strategies targeting muscle conditioning in stable COPD.

In chronic hypercapnic patients receiving long-term NIV, combining ventilatory support with inspiratory muscle exercises has shown promising physiological improvements, though high-quality randomized trials remain limited.

4.4 Evidence from Critical Care and Ventilated Patients

In ICU populations, respiratory muscle inactivity during mechanical ventilation contributes to ventilator-induced diaphragmatic dysfunction (VIDD). Although VIDD is more pronounced during invasive ventilation, partial unloading during NIV may similarly reduce spontaneous effort.

Emerging studies suggest that early respiratory muscle activation strategies may:

- Preserve diaphragmatic thickness
- Improve weaning outcomes
- Reduce duration of ventilatory support

While most data focus on invasive ventilation, the concept of maintaining respiratory muscle engagement during assisted ventilation is increasingly emphasized.

The American Thoracic Society highlights the importance of respiratory muscle preservation during ventilatory management, particularly in prolonged support scenarios.

4.5 Potential Mechanistic Synergy Between NIV and Incentive Spirometry

The combination of NIV and incentive spirometry can be conceptualized as addressing complementary physiological domains:

NIV Effect	Incentive Spirometry Effect
Reduces respiratory load	Enhances respiratory capacity
Corrects hypercapnia	Promotes alveolar recruitment
Decreases work of breathing	Activates diaphragmatic movement
Offsets intrinsic PEEP	Encourages deep lung expansion

Thus, NIV provides passive mechanical support, whereas IS provides active respiratory engagement.

Theoretically, this dual strategy may:

- Accelerate correction of hypercapnia
- Improve inspiratory muscle endurance
- Facilitate earlier NIV weaning
- Reduce hospital stay

However, empirical evidence specifically in acute hypercapnic respiratory failure remains limited.

4.6 Safety Considerations in HRF Patients on NIV

Before implementing incentive spirometry in hypercapnic patients on NIV, certain clinical factors must be considered:

- Hemodynamic stability
- Adequate consciousness level
- Ability to follow commands
- Absence of severe respiratory distress

Excessive respiratory effort in unstable patients may increase fatigue rather than provide benefit. Therefore, IS should be introduced once acute distress is partially stabilized with NIV.

Close monitoring of Respiratory rate, Oxygen saturation, Subjective dyspnea, Signs of muscle fatigue is essential during implementation.

4.7 Current Evidence Gap

Despite strong physiological rationale, there is a scarcity of randomized controlled trials directly evaluating incentive spirometry in patients with acute hypercapnic respiratory failure receiving NIV.

Most existing studies address:

- Postoperative lung expansion
- Stable COPD rehabilitation
- Weaning from invasive ventilation

Therefore, extrapolation to acute HRF requires cautious interpretation.

High-quality prospective studies are needed to determine:

- Optimal timing of IS initiation during NIV
- Frequency and duration of sessions
- Effects on $PaCO_2$ reduction
- Impact on NIV duration and weaning success
- Long-term functional outcomes

4.8 Conceptual Clinical Framework

Based on available evidence, a proposed clinical integration model would involve:

- Early stabilization with NIV to reduce ventilatory load.
- Introduction of incentive spirometry once respiratory distress decreases.
- Gradual progression of inspiratory training intensity.
- Integration with pulmonary rehabilitation strategies post-acute phase.

This stepwise approach may balance safety and physiological benefit.

5. CLINICAL EVIDENCE ON INCENTIVE SPIROMETRY IN HYPERCAPNIC AND NIV-SUPPORTED POPULATIONS

Although incentive spirometry (IS) has long been utilized in postoperative pulmonary care, its application in hypercapnic respiratory failure (HRF), particularly in patients receiving non-invasive ventilation (NIV), remains underexplored. Direct randomized controlled trials specifically targeting acute hypercapnic patients on NIV are limited. However, indirect evidence from COPD rehabilitation, chronic hypercapnic management, and ventilatory weaning studies provides insight into its potential role.

This section synthesizes available clinical evidence relevant to the integration of IS in hypercapnic and ventilatory-supported populations.

5.1 Evidence from Acute Exacerbation of COPD

Acute exacerbation of COPD is the most common clinical context for hypercapnic respiratory failure. NIV is strongly recommended in this setting by the Global Initiative for Chronic Obstructive Lung Disease.

While most acute management studies focus on pharmacological therapy and ventilatory support, some rehabilitation-oriented trials have evaluated early breathing exercises during hospitalization. These studies demonstrate that:

- Early respiratory exercises improve inspiratory capacity
- Lung expansion techniques reduce dyspnea scores
- Inspiratory muscle activation may accelerate functional recovery

However, most of these studies did not isolate incentive spirometry as a standalone intervention during active NIV use. Instead, IS was often part of broader pulmonary rehabilitation protocols.

Importantly, no high-powered multicenter RCT has definitively evaluated the additive benefit of IS during the acute NIV phase in hypercapnic exacerbations.

5.2 Evidence from Chronic Hypercapnic COPD on Long-Term NIV

In chronic stable hypercapnic COPD, long-term NIV has gained increasing support for reducing $PaCO_2$ and improving survival in selected patients.

Guidelines from the European Respiratory Society support consideration of long-term NIV in chronic hypercapnic COPD following acute exacerbations.

Studies examining inspiratory muscle training (IMT) in these patients demonstrate:

- Significant improvement in maximal inspiratory pressure (MIP)
- Reduction in dyspnea severity
- Improved 6-minute walk distance
- Enhanced quality of life

Although incentive spirometry provides less resistance compared to threshold IMT devices, it promotes sustained maximal inspiratory efforts that may offer similar, albeit milder, conditioning effects.

Some small cohort studies suggest that combining NIV with respiratory muscle training may improve weaning tolerance and ventilatory independence, though results remain heterogeneous.

5.3 Evidence from ICU and Ventilatory Weaning Studies

Research in invasive mechanical ventilation populations has shown that diaphragmatic dysfunction can develop rapidly due to ventilatory unloading, a phenomenon described as ventilator-induced diaphragmatic dysfunction (VIDD).

Although NIV generally allows more spontaneous breathing than invasive ventilation, partial unloading may still reduce muscle activation in prolonged use.

Clinical trials evaluating respiratory muscle training in mechanically ventilated patients have reported:

- Increased inspiratory strength
- Shortened weaning duration
- Higher rates of successful extubation

While these findings cannot be directly extrapolated to NIV-supported HRF patients, they support the broader concept that maintaining inspiratory muscle engagement during assisted ventilation improves outcomes.

The American Thoracic Society has emphasized the importance of preserving respiratory muscle function during ventilatory management, particularly in prolonged respiratory support.

5.4 Evidence in Postoperative and Atelectasis Prevention Populations

In postoperative thoracic and abdominal surgery patients, incentive spirometry is widely used to prevent atelectasis and promote lung expansion.

Clinical trials in these populations demonstrate:

- Improved forced vital capacity (FVC)
- Increased inspiratory capacity
- Reduced radiographic atelectasis
- Improved oxygenation

Although postoperative atelectasis differs pathophysiologically from obstructive hypercapnic failure, both conditions involve reduced lung expansion and regional hypoventilation.

These findings support the mechanistic plausibility of IS in promoting alveolar recruitment in HRF patients during periods when NIV is intermittently removed.

5.5 Indirect Evidence from Pulmonary Rehabilitation

Pulmonary rehabilitation programs consistently demonstrate that inspiratory muscle conditioning improves functional capacity in COPD.

Structured breathing interventions show:

- Improved ventilatory efficiency
- Reduced dynamic hyperinflation
- Enhanced exercise tolerance

While incentive spirometry is not a substitute for structured inspiratory muscle training, its simplicity and bedside feasibility make it attractive in acute hospital settings where formal rehabilitation devices may not be available.

5.6 Current Limitations in the Evidence Base

Despite promising physiological rationale and indirect supportive data, several limitations remain:

- Lack of RCTs in Acute HRF on NIV: There is no large-scale randomized controlled trial exclusively assessing IS during active NIV treatment for acute hypercapnic respiratory failure.
- Heterogeneity of Interventions: Studies vary in frequency, duration, and intensity of inspiratory exercises.
- Outcome Variability: Measured outcomes differ across trials, including gas exchange parameters, muscle strength indices, functional measures, and length of stay.
- Limited $PaCO_2$-Focused Data: Few studies specifically assess reduction in hypercapnia as a primary outcome of inspiratory exercises.
- Timing Uncertainty: Optimal timing for initiating IS during acute hypercapnic episodes remains undefined.

5.7 Potential Clinical Benefits in HRF on NIV

Based on synthesis of available literature, potential clinical benefits of incentive

spirometry in NIV-supported hypercapnic patients may include:

- Enhanced inspiratory muscle engagement during recovery phase
- Improved alveolar recruitment between NIV sessions
- Prevention of atelectasis during mask-off periods
- Facilitation of NIV weaning
- Improved patient participation in rehabilitation

However, these remain hypotheses requiring prospective validation.

5.8 Research Implications

Future research should focus on:

- Randomized controlled trials comparing NIV alone versus NIV plus IS
- Standardized IS protocols (frequency, volume targets, duration)
- Primary outcomes including $PaCO_2$ reduction and pH normalization
- Secondary outcomes such as MIP improvement and NIV duration
- Safety monitoring in severe hypercapnic patients

A multicenter design with adequate sample size would be essential to determine whether IS provides clinically meaningful additive benefit.

6. DISCUSSION ANALYSIS, CLINICAL IMPLICATIONS, AND FUTURE RESEARCH DIRECTIONS

Hypercapnic respiratory failure (HRF) represents a complex interaction between increased ventilatory load and reduced respiratory muscle capacity. Non-invasive ventilation (NIV) has transformed acute management by reducing work of breathing, improving alveolar ventilation, and correcting respiratory acidosis. However, NIV remains fundamentally a supportive intervention. It mitigates ventilatory load but does not directly restore inspiratory muscle strength or reverse chronic hyperinflation-associated biomechanical disadvantages.

This review synthesizes current physiological and clinical evidence to examine whether incentive spirometry (IS) may serve as a meaningful adjunct in patients with HRF receiving NIV support.

6.1 Physiological Integration: Load Reduction Versus Capacity Restoration

The pathophysiology of HRF is characterized by:

- Increased airway resistance
- Dynamic hyperinflation
- Intrinsic PEEP
- Inspiratory muscle fatigue
- Ventilatory inefficiency

NIV addresses primarily the load component of this imbalance. By delivering inspiratory pressure support and offsetting intrinsic PEEP, it reduces diaphragmatic effort and improves gas exchange.

In contrast, incentive spirometry targets the capacity component by encouraging sustained maximal inspiration and diaphragmatic recruitment. This may:

- Increase inspiratory muscle activation
- Improve alveolar recruitment
- Enhance inspiratory capacity
- Counteract shallow breathing patterns

Thus, the combination of NIV and IS can be conceptualized as addressing two complementary therapeutic dimensions: passive unloading and active muscle engagement.

6.2 Potential Synergistic Mechanisms in Hypercapnic Patients

- The theoretical synergy between NIV and IS may operate through several mechanisms:

6.2.1 Preservation of Diaphragmatic Function

- While NIV reduces fatigue, prolonged unloading may reduce spontaneous effort. Introducing controlled inspiratory exercises may help maintain diaphragmatic activation without inducing excessive fatigue.

- This balance between rest and activation is critical in preventing progressive respiratory muscle dysfunction.

6.2.2 Alveolar Recruitment During NIV Breaks

- NIV is often delivered intermittently. During mask-off periods, patients may revert to shallow breathing, promoting atelectasis and micro-derecruitment.
- IS may help maintain lung expansion and prevent collapse during these intervals.

6.2.3 Facilitation of NIV Weaning

- Weaning from NIV requires sufficient inspiratory muscle reserve. If IS enhances inspiratory muscle performance during recovery, it may reduce dependence duration.
- Evidence from ventilatory weaning studies in broader ICU populations supports the importance of respiratory muscle conditioning in improving liberation success.

6.2.4 Improvement in Dyspnea Perception

- Dyspnea in hypercapnic patients is partly mediated by neuromechanical dissociation. Enhancing inspiratory capacity through controlled lung expansion may improve the relationship between neural drive and achieved ventilation, potentially reducing dyspnea severity.

6.3 Clinical Implications

Although definitive randomized evidence is lacking, several practical implications can be considered:

6.3.1 Patient Selection

IS may be appropriate in hypercapnic patients who:

- Are hemodynamically stable
- Demonstrate improved pH and reduced acute distress after NIV initiation
- Are cooperative and able to follow commands
- Exhibit evidence of inspiratory muscle weakness

Severely acidotic or exhausted patients may not tolerate active inspiratory exercises in the early acute phase.

6.3.2 Timing of Implementation

A staged approach appears reasonable:

- Acute Stabilization Phase: NIV optimization and correction of acidosis
- Early Recovery Phase: Introduction of low-intensity IS sessions
- Rehabilitation Phase: Progressive inspiratory muscle conditioning

Early but cautious initiation may maximize benefit while minimizing fatigue risk.

6.3.3 Integration into Multidisciplinary Care

Implementation requires collaboration among:

- Pulmonologists
- Critical care physicians
- Physiotherapists
- Respiratory therapists

Incorporating IS into early pulmonary rehabilitation pathways during hospitalization may improve continuity of care.

6.4 Limitations of Current Evidence

Several limitations temper conclusions:

- Absence of large randomized trials directly studying IS in acute HRF on NIV
- Heterogeneity in inspiratory training protocols
- Lack of standardized outcome measures
- Limited data specifically assessing $PaCO_2$ reduction
- Potential confounding by concurrent rehabilitation interventions

Additionally, incentive spirometry provides volume-oriented training but does not impose calibrated resistance, which may limit strength-building potential compared to threshold IMT devices.

6.5 Future Research Directions

Future investigations should focus on well-designed randomized controlled trials with standardized protocols.

6.5.1 Suggested Study Design

- Population: Acute hypercapnic respiratory failure on NIV
- Intervention: Standard NIV + structured IS protocol
- Control: Standard NIV alone
- Primary Outcomes: $PaCO_2$ reduction, Time to pH normalization
- Secondary Outcomes: Maximal inspiratory pressure (MIP), Duration of NIV, Length of hospital stay, Dyspnea scores, Readmission rates

6.5.2 Mechanistic Studies

Ultrasound-based diaphragmatic thickness measurements could assess:

- Preservation of muscle mass
- Changes in diaphragmatic excursion

Advanced physiological studies could evaluate:

- Work of breathing
- Neural respiratory drive
- Dynamic hyperinflation indices

Such mechanistic insights would clarify whether IS produces meaningful physiological changes in HRF.

6.5.3 Comparative Studies

Future research may also compare:

- Incentive spirometry
- Threshold inspiratory muscle training devices
- Combined respiratory rehabilitation strategies

Determining the most effective and feasible intervention is essential for clinical adoption.

6.6 Conceptual Clinical Algorithm

A proposed clinical algorithm may involve:

- Confirm hypercapnic respiratory failure.
- Initiate NIV according to guideline criteria.
- Stabilize pH and reduce acute distress.
- Introduce supervised incentive spirometry sessions (low frequency initially).
- Monitor tolerance and respiratory parameters.
- Progressively integrate into structured pulmonary rehabilitation.

This approach balances safety with potential benefit.

6.7 Overall Interpretation

The integration of incentive spirometry into NIV-supported hypercapnic respiratory failure management is physiologically plausible but currently under-evaluated in high-quality trials.

NIV effectively reduces ventilatory load, while IS may enhance inspiratory capacity and maintain diaphragmatic activation. The combined strategy could theoretically:

- Accelerate recovery
- Improve muscle performance
- Facilitate NIV weaning
- Enhance functional outcomes

However, robust clinical evidence remains necessary before formal guideline recommendations can be established.

CONCLUSION

Hypercapnic respiratory failure is characterized by an imbalance between ventilatory load and respiratory muscle capacity. Non-invasive ventilation remains the cornerstone of acute management. Incentive spirometry, through its effects on lung expansion and inspiratory muscle activation, may serve as a complementary strategy during recovery.

While current evidence is indirect and largely extrapolated from COPD rehabilitation and ventilatory weaning literature, the physiological rationale supports further investigation.

High-quality randomized controlled trials are needed to determine whether incentive spirometry confers clinically meaningful benefits in hypercapnic patients receiving NIV support.

Declaration by Authors

Ethical Approval: Not Applicable

Acknowledgement: None

Source of Funding: Nil

Conflict of Interest: The authors declare no conflict of interest.

REFERENCES

1. Chen YH, Hsieh YS. A narrative review of impact of incentive spirometer respiratory training in Long COVID. International Journal of General Medicine. 2024 Dec 31:5233-46.
2. Ovtcharenko N, Ho E, Alhazzani W, Cortegiani A, Ergan BE, Scala R, Sotgiu G, Chaudhuri D, Oczkowski S, Lewis K. High-flow nasal cannula versus non-invasive ventilation for acute hypercapnic respiratory failure in adults: a systematic review and meta-analysis of randomized trials. Critical care. 2022 Nov 9;26(1):348.
3. Comellini V, Pacilli AM, Nava S. Benefits of non-invasive ventilation in acute hypercapnic respiratory failure. Respirology. 2019 Apr;24(4):308-17.
4. Hatipoğlu U, Aboussouan LS. Chronic hypercapnic respiratory failure and non-invasive ventilation in people with chronic obstructive pulmonary disease. BMJ medicine. 2022 Aug 1;1(1):e000146.
5. David-Joao PG, Guedes MH, Rea-Neto A, de Oliveira Chaiben VB, Baena CP. Noninvasive ventilation in acute hypoxemic respiratory failure: a systematic review and meta-analysis. Journal of critical care. 2019 Feb 1;49:84-91.
6. Navarra SM, Congedo MT, Pennisi MA. Indications for non-invasive ventilation in respiratory failure. Reviews on recent clinical trials. 2020 Dec 1;15(4):251-7.
7. Schönhofer B, Kuhlen R, Neumann P, Westhoff M, Berndt C, Sitter H. Clinical practice guideline: non-invasive mechanical ventilation as treatment of acute respiratory failure. Deutsches Ärzteblatt International. 2008 Jun 13;105(24):424.
8. Chiumello D, Chevallard G, Gregoretti C. Non-invasive ventilation in postoperative patients: a systematic review. Intensive care medicine. 2011 Jun;37(6):918-29.
9. Sullivan KA, Churchill IF, Hylton DA, Hanna WC. Use of incentive spirometry in adults following cardiac, thoracic, and upper abdominal surgery to prevent post-operative pulmonary complications: a systematic review and meta-analysis. Respiration. 2021 Nov 2;100(11):1114-27.
10. Mostafa MF, Tantaewy NM, Abo-Saeda AI. Effect of incentive spirometry on respiratory parameters among critically ill patients with chest tube drainage. Tanta Scientific Nursing Journal. 2024 Sep 1;34(3).
11. Coppolino F, Sansone P, Cosenza G, Bottazzo LM, Brunetti S, De Sarno S, Del Prete M, Passavanti MB, Fiore M, Esposito C, Pace MC. Non-invasive respiratory support for postoperative acute hypoxemic respiratory failure: a narrative review. Signa Vitae. 2026 Jan 1;22(1).
12. Jaber S, Chanques G, Jung B. Postoperative non-invasive ventilation. Intensive Care Medicine: Annual Update 2008. 2008 Sep 2:310-9.
13. Windisch W, Geiseler J, Simon K, Walterspacher S, Dreher M, Guideline Commission. German national guideline for treating chronic respiratory failure with invasive and non-invasive ventilation–revised edition 2017: part 2. Respiration. 2018 Aug 31;96(2):171-203.
14. Pépin JL, Timsit JF, Tamisier R, Borel JC, Lévy P, Jaber S. Prevention and care of respiratory failure in obese patients. The lancet Respiratory medicine. 2016 May 1;4(5):407-18.
15. Mathai AS. Non-invasive ventilation in the postoperative period: Is there a role? Indian journal of anaesthesia. 2011 Jul 1;55(4):325-33.

How to cite this article: Sipra Dash, Asit Kumar Bhoi, Santosh Kumar Behera, Devranjan Suna, Dwarikanath Rout. Role of incentive spirometry in patients with hypercapnic respiratory failure receiving non-invasive ventilation: a review of physiological rationale and clinical evidence. *International Journal of Research and Review*. 2026; 13(3): 245-260. DOI: *https://doi.org/10.52403/ijrr.20260329*

******