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CURRENT STRATEGIES FOR PREVENTION AND MANAGEMENT OF ICU-ACQUIRED WEAKNESS IN CRITICALLY ILL PATIENTS: EVIDENCE, LIMITATIONS AND CLINICAL APPLICABILITY - A NARRATIVE REVIEW

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ABSTRACT

Intensive care unit-acquired weakness (ICU-AW) is a common complication of critical illness, defined as generalized, symmetrical muscle weakness developing during ICU stay that cannot be explained by pre-existing neuromuscular disorders. It is associated with prolonged mechanical ventilation, delayed recovery, and impaired long-term functional outcomes. The objective of this narrative review was to summarize current evidence on strategies for the prevention and early management of ICU-AW, with a particular focus on their effectiveness, limitations, and clinical applicability.

A structured literature search was conducted using PubMed, MEDLINE, and Google Scholar databases, focusing on randomized controlled trials, systematic reviews, and meta-analyses published between 2018 and 2026. Particular attention was given to early mobilization, nutritional support, neuromuscular electrical stimulation (NMES), sedation strategies, and methods for assessing muscle status.

Current evidence suggests that ICU-AW results from multiple interacting factors, including systemic inflammation, metabolic disturbances, immobilization, and inadequate nutritional support. Early mobilization remains the most consistently supported intervention, while nutritional therapy and NMES may provide additional benefits, particularly in selected patient populations. Monitoring of muscle status may facilitate early detection and guide clinical decision-making.

Individual strategies for the prevention and early management of ICU-AW have important limitations when used in isolation. The available evidence suggests that different approaches may have complementary effects, although their combined impact remains incompletely understood. Further research is needed to better define optimal intervention strategies, including their timing, combination, and applicability in diverse patient populations.

KEYWORDS

Intensive Care Unit-Acquired Weakness; Critical Illness; Early Mobilization; Nutritional Support; Neuromuscular Electrical Stimulation; Muscle Assessment

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1. Introduction

Intensive care unit-acquired weakness (ICU-AW) is a frequent and clinically significant complication of critical illness, particularly in patients requiring prolonged mechanical ventilation, deep sedation, and extended immobilization [1,2]. It is defined as a generalized, symmetrical weakness that develops during the course of critical illness and cannot be explained by pre-existing neuromuscular disorders [1,4]. Beyond a simple reduction in muscle strength, ICU-AW reflects a complex clinical condition involving both muscle atrophy and impaired neuromuscular function, often associated with critical illness polyneuropathy and/or myopathy [1,3,4].

The pathogenesis of ICU-AW is multifactorial and involves a combination of systemic inflammation, metabolic disturbances, and disuse-related muscle atrophy. Critical illness induces a hypercatabolic state characterized by increased protein breakdown and reduced protein synthesis, leading to a rapid decline in muscle mass and function [5,8]. These processes are further exacerbated by prolonged immobilization, deep sedation, and mechanical ventilation, which limit spontaneous muscle activity and accelerate muscle wasting [3,6]. Importantly, muscle loss may begin early during ICU stay, even within the first days of critical illness, before overt clinical weakness becomes apparent [3].

The clinical consequences of ICU-AW are substantial. The condition has been associated with prolonged duration of mechanical ventilation, extended length of hospital stay, delayed functional recovery, and persistent physical impairment after discharge [1,2,6]. Given its impact on both short- and long-term outcomes, ICU-AW represents a major challenge in the prevention and management of critically ill patients.

Despite increasing recognition of this condition, both prevention and early management of ICU-AW remain challenging. The available literature describes a range of strategies aimed at reducing muscle loss and preserving neuromuscular function in critically ill patients, including early mobilization, optimized nutritional support with adequate protein delivery, neuromuscular electrical stimulation (NMES), sedation management, and approaches to monitoring muscle status. Among these, early mobilization is the most consistently supported intervention; however, its implementation is often limited by patient instability, deep sedation, and organizational constraints [11–14]. Similarly, nutritional interventions are essential for maintaining muscle metabolism but have not consistently demonstrated clear benefits when used in isolation [21–24]. NMES may offer an alternative means of muscle activation in patients unable to participate in active rehabilitation, although the available evidence remains heterogeneous and lacks standardization [15–19].

These observations highlight an important limitation of current strategies: when applied individually, each approach addresses only selected aspects of the complex pathophysiology of ICU-AW. As a result, no single intervention appears sufficient to fully prevent or mitigate the development of muscle weakness in critically ill patients.

The aim of this narrative review is to summarize current evidence on strategies for the prevention and early management of ICU-AW in critically ill patients, with particular emphasis on the effectiveness, limitations, and clinical applicability of available interventions.

2. Methodology

This study is a narrative literature review aimed at summarizing current evidence on strategies for preventing and managing intensive care unit-acquired weakness (ICU-AW) in critically ill patients. The review focuses on the effectiveness, limitations, and clinical applicability of commonly used interventions, with particular emphasis on early mobilization, nutritional support, neuromuscular electrical stimulation (NMES), sedation strategies, and different methods for assessing muscle mass and function.

A structured literature search was performed using PubMed, MEDLINE, and Google Scholar electronic databases. The search included articles published in English between 2018 and 2026, with additional inclusion of selected earlier landmark studies relevant to the topic. Keywords used in the search strategy included “intensive care unit-acquired weakness”, “critical illness”, “muscle wasting”, “early mobilization”, “neuromuscular electrical stimulation”, “nutritional support”, “muscle ultrasound”.

Inclusion criteria comprised peer-reviewed randomized controlled trials, systematic literature reviews, meta-analyses addressing the prevention and early management of ICU-acquired weakness or related muscle dysfunction in critically ill patients.

Exclusion criteria comprised case reports and small case series due to their focus on individual clinical observations and limited ability to support evaluation of preventive and early management interventions in a broader clinical context. Non-peer-reviewed publications and studies were also excluded from the search.

Relevant data were extracted and synthesized narratively, with emphasis on study design, patient characteristics, and reported outcomes.

As this study is based on previously published data, no direct patient involvement occurred, and ethical approval was not required. This approach provides a structured overview on current evidence on the prevention and management of ICU-AW, while acknowledging the limitations of the available literature.

3. Factors and mechanisms contributing to ICU-acquired weakness

3.1. Inflammation and metabolic disturbances

During critical illness, the excessive release of pro-inflammatory cytokines including tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β) and interleukin-6 (IL-6) promotes activation of catabolic pathways within muscle tissue [1,5,8]. These mediators stimulate proteolytic mechanisms, particularly the ubiquitin-proteasome system and autophagy-related pathways. Simultaneously, inflammatory signaling impairs anabolic pathways responsible for protein synthesis, resulting in a persistent imbalance between protein degradation and regeneration [5,7]. Furthermore, systemic inflammation and oxidative stress disrupt oxidative phosphorylation and reduce ATP production, leading to impaired cellular energy metabolism and progressive muscle wasting [3,4,8]. In addition, prolonged inflammation may negatively affect satellite cell activity and limit muscle regeneration [3,7].

Metabolic disturbances further exacerbate muscle dysfunction during critical illness. Critically ill patients often develop severe insulin resistance and stress-induced hyperglycemia, both of which disrupt glucose uptake and decrease the anabolic effects of insulin on muscle tissue [5,8]. Impaired insulin signaling

contributes to increased proteolysis and reduced glycogen synthesis, thereby promoting further muscle catabolism [6,8]. Persistent hyperglycemia has also been associated with the development of critical illness polyneuropathy and poorer neuromuscular outcomes [8,12]. Moreover, decreased levels of anabolic hormones combined with elevated cortisol concentrations contribute to a persistent catabolic state during prolonged ICU stay [5,7].

Together, these inflammatory and metabolic alterations create conditions that favor progressive muscle wasting and functional impairment in ICU patients [1,4,7].

3.2. Immobilization

Immobilization represents a major contributor to muscle wasting in critically ill patients and is primarily related to the absence of mechanical loading, which is essential for maintaining muscle mass and function [5,6]. Reduced physical activity, commonly associated with mechanical ventilation, deep sedation and severe illness, leads to rapid onset of disuse-related muscle atrophy [1,5]. At the cellular level, lack of mechanical stimulation results in decreased activation of anabolic signaling pathways. At the same time, proteolytic systems, including the ubiquitin–proteasome pathway remain active, further contributing to negative protein balance [5,8]. These changes may occur early during ICU stay and can progress rapidly, particularly in patients with complete bed rest.

Importantly, immobilization does not act independently but interacts closely with the inflammatory and metabolic processes described in the previous section. Systemic inflammation may enhance proteolysis, while metabolic disturbances impair the capacity for muscle regeneration, thereby amplifying the effects of disuse [3,5,8]. As a result, even relatively short periods of inactivity may lead to significant reductions in muscle mass and strength in critically ill patients.

In addition to structural muscle loss, prolonged immobilization may contribute to neuromuscular dysfunction, including the development of critical illness polyneuropathy and myopathy, which further impair functional recovery [1,4].

3.3. Sedation and mechanical ventilation

Sedation and mechanical ventilation are integral components of intensive care management, however, both significantly contribute to the development and progression of ICU-AW. Deep sedation reduces voluntary muscle activity, limits patients' participation in spontaneous movements and frequently prolongs immobilization during ICU stay [1,4]. Mechanical ventilation further contributes to physical inactivity, particularly in patients who remain fully dependent on ventilatory support for prolonged periods of time. Reduced muscle activation in deeply sedated patients promotes disuse-related atrophy and progressive loss of muscle strength, affecting both peripheral and respiratory muscles [4,5]. Moreover, diaphragmatic inactivity during controlled mechanical ventilation may contribute to ventilator-induced diaphragmatic dysfunction, characterized by diaphragmatic weakness and reduced endurance [1,4].

3.4. Nutritional deficits

Nutritional deficits contribute to the development of ICU-acquired weakness by exacerbating the imbalance between protein synthesis and degradation observed during critical illness. The hypercatabolic state characteristic of ICU patients is associated with increased proteolysis and a reduced capacity for effective muscle protein synthesis, leading to progressive loss of skeletal muscle mass [5,8].

Insufficient availability of amino acids further intensifies this process. Skeletal muscle serves as a key metabolic reservoir during critical illness, and in the absence of adequate nutritional supply, endogenous protein breakdown is upregulated to meet systemic metabolic demands [21]. This results in accelerated depletion of muscle protein and impaired maintenance of muscle structure and function.

In addition, metabolic alterations associated with critical illness, including insulin resistance and hormonal dysregulation, reduce the anabolic response to nutrient availability. Even when substrates are present, impaired signaling pathways limit the efficiency of protein synthesis and favor continued catabolism [5,8]. These mechanisms closely interact with inflammatory processes, further amplifying muscle wasting.

4. Prevention and management strategies

4.1. Early mobilization and physical rehabilitation

Early mobilization is regarded as one of the key strategies for preventing and managing ICU-acquired weakness in critically ill patients. Depending on the patient's clinical condition and level of consciousness, it includes various interventions ranging from passive and active-assisted exercises to more advanced rehabilitation techniques [9,10]. Current rehabilitation protocols in the ICU commonly involve passive range-of-motion exercises, proprioceptive neuromuscular facilitation, in-bed cycling, sitting at the edge of the bed, transfer to a chair, standing and assisted walking when clinically feasible [9,12,13]. In deeply sedated and mechanically ventilated patients who are unable to actively participate, passive mobilization and positioning strategies are used to maintain joint mobility, reduce muscle shortening and limit the consequences of prolonged immobilization [6,12].

The primary aim of early mobilization is to minimize the adverse effects of bed rest and preserve neuromuscular function during critical illness. Evidence suggests that muscle wasting develops rapidly during ICU stay, therefore, initiation of rehabilitation during the early phase of critical illness may help preserve muscle mass, muscle strength and function and improve overall physical condition after ICU discharge [5,8,9]. Early rehabilitation has also been associated with improved functional independence and a lower incidence of ICU-acquired weakness [6,7,11]. In a randomized controlled trial conducted by Schweickert et al., early physical and occupational therapy in mechanically ventilated patients resulted in better functional outcomes and shorter duration of delirium compared with standard care [9].

Several meta-analyses and systematic reviews have additionally demonstrated that early mobilization may shorten the duration of mechanical ventilation and reduce ICU and hospital length of stay [10,13,14]. Benefits of this strategy appear to be more significant when rehabilitation is started within the first days of ICU stay and carried out by a multidisciplinary team [11-14].

Despite the benefits of early mobilization reported in the literature, its implementation in everyday clinical practice remains challenging. Hemodynamic instability, prolonged deep sedation, impaired consciousness and dependence on life-support therapies may limit patients' participation in rehabilitation [6,14]. Organizational barriers, including staffing limitations and safety concerns also continue to affect the consistent application of physical rehabilitation protocols in intensive care units [12,14].

4.2. Nutritional support and adequate protein delivery

Adequate nutritional therapy, particularly sufficient protein delivery, is considered an essential component of ICU care [20,22]. Current evidence suggests that protein intake may influence muscle preservation and clinical recovery in critically ill patients. Higher protein delivery has been associated with improved maintenance of lean body mass, better muscle strength and reduced mortality in some patient populations [21,24].

ESPEN guidelines recommend early initiation of enteral nutrition, preferably within the first 24-48 hours after ICU admission, provided that the patient is hemodynamically stable, with gradual advancement of nutritional therapy during the acute phase of critical illness to avoid overfeeding and metabolic complications. The guidelines also emphasize adequate daily protein delivery, generally in the range of approximately 1.3 g/kg/day to support muscle metabolism and recovery [22]. Some studies have demonstrated potential benefits of even higher protein intake, whereas others have reported limited or inconsistent effects on long-term functional outcomes [21,23]. The effectiveness of nutritional therapy may also be influenced by the severity and phase of critical illness, as excessive protein administration during the acute phase may not always translate into improved muscle function [20,23].

In clinical practice, achieving nutritional targets in ICU patients is often difficult. Enteral nutrition may be limited by gastrointestinal intolerance, delayed gastric emptying, interruptions related to diagnostic or therapeutic procedures and hemodynamic instability [22,23]. As a result, critically ill patients frequently receive less protein and energy than recommended, which may further contribute to muscle wasting and delayed functional recovery [21,23,24].

4.3. Neuromuscular electrical stimulation (NMES)

In critically ill patients who are unable to take part in active rehabilitation, neuromuscular electrical stimulation (NMES) may be used as an additional method to help preserve muscle function and reduce muscle loss [15–17]. The technique involves placing surface electrodes on selected muscle groups, most commonly the quadriceps muscles, to generate electrical impulses that produce involuntary muscle contractions similar to normal muscle activity [15,18]. Through repeated stimulation of the muscles, NMES may help limit the effects of prolonged immobilization and disuse-related muscle atrophy [15,16].

Several studies have investigated the use of NMES in mechanically ventilated and critically ill patients. Available evidence suggests that NMES may help maintain muscle mass and improve muscle strength, particularly in patients requiring prolonged ICU treatment and mechanical ventilation [17,18]. Routsi et al. reported that daily use of NMES was associated with a lower incidence of critical illness polyneuromyopathy compared with standard care [18]. Some studies also suggest that NMES may improve local microcirculation and help maintain muscle fiber structure during critical illness [15,17].

NMES is mainly used in patients who cannot actively participate in rehabilitation because of deep sedation, impaired consciousness, or severe clinical condition [16,19]. It is often applied together with standard physiotherapy and early mobilization strategies [15,17]. However, results from clinical studies remain inconsistent, mainly due to differences in stimulation protocols, treatment duration, and patient characteristics [16,17]. The effectiveness of NMES may also depend on the timing of initiation and the overall condition of the patient during ICU stay [16,18]. The routine implementation of NMES in ICU practice may also be limited by organizational factors, variability in stimulation protocols, and the need for appropriately trained staff [15–17].

4.4. Sedation strategies

Current ICU practice increasingly emphasizes the use of lighter sedation strategies whenever clinically feasible in order to reduce the risk of developing ICU-acquired weakness and to support its management [1,4,6]. Limiting unnecessary sedation may facilitate earlier mobilization, improve patient interaction with rehabilitation staff, and support preservation of neuromuscular function [6,9,13]. Studies evaluating early rehabilitation in mechanically ventilated patients suggest that reduced sedation levels are associated with better participation in physical therapy and improved functional outcomes after ICU discharge [9,13]. Lighter sedation has also been linked to shorter duration of mechanical ventilation and reduced ICU length of stay in some patient populations [10,13].

Despite these potential benefits, implementation of minimal sedation strategies remains challenging in critically ill patients. Sedation often remains necessary to ensure patient comfort, reduce anxiety, maintain ventilator synchrony, and allow safe performance of life-support therapies [1,4]. In patients with severe respiratory failure, hemodynamic instability, or agitation, reducing sedation may not be clinically feasible [6,14]. Excessive reduction of sedation may additionally increase the risk of accidental device removal, patient distress, or ineffective mechanical ventilation.

Another limitation is the lack of a universal sedation protocol suitable for all ICU patients. Sedation requirements may differ depending on disease severity, underlying diagnosis, and the need for invasive therapies [1,6]. For this reason, current recommendations emphasize individualized sedation management, with regular assessment of sedation depth and daily evaluation of the possibility of sedation reduction when clinically appropriate [4,6].

4.5. Monitoring and assessment of muscle mass and function

Assessment of muscle mass and function plays an important role in the management of critically ill patients, particularly for early detection of ICU-acquired weakness and monitoring the progression of muscle wasting during ICU stay [1,4]. Early identification of muscle deterioration may help guide rehabilitation strategies and optimize supportive treatment in critically ill patients [2,26].

The Medical Research Council (MRC) scale is one of the most commonly used clinical tools for evaluating muscle strength in ICU patients [1,4]. The scale is based on manual assessment of muscle strength in six bilateral muscle groups: shoulder abduction, elbow flexion, wrist extension, hip flexion, knee extension, and ankle dorsiflexion. Each muscle group is graded on a 6-point scale from 0 to 5, where 0 indicates no visible muscle contraction and 5 represents normal muscle strength. The total MRC sum score ranges from 0 to 60 points, with a score below 48 commonly used as a diagnostic criterion for ICU-acquired weakness [1,4]. Accurate assessment using the MRC scale requires that the patient is awake, cooperative, and able to follow verbal commands, as the examination depends on active voluntary muscle contraction during manual testing

[1,4,26]. Consequently, the applicability of this method is limited in patients with deep sedation, delirium, impaired consciousness, or severe clinical instability, which are common during the acute phase of critical illness [1,26].

As a result, the use of the MRC scale is often restricted in the early stages of critical illness, which may contribute to delayed recognition of ICU-acquired weakness in mechanically ventilated and deeply sedated patients.

For this reason, increasing attention has been given to objective bedside methods that do not require active patient participation. Muscle ultrasound has emerged as a practical and non-invasive technique for assessing muscle mass and muscle architecture in critically ill patients [5,25]. Ultrasound allows repeated bedside evaluation of muscle thickness, cross-sectional area, and muscle quality during ICU stay [25]. Studies suggest that reductions in muscle thickness detected by ultrasound may occur early during critical illness and correlate with muscle wasting, functional impairment, and clinical outcomes [5,25].

Other assessment methods, including bioelectrical impedance analysis (BIA), have also been investigated as tools for evaluating body composition and muscle mass [26]. However, their accuracy in ICU patients may be limited by fluid overload, rapid fluid shifts, and hemodynamic instability, all of which frequently occur during critical illness [26]. Functional assessment tools additionally remain difficult to apply in patients with impaired consciousness, deep sedation, or severe clinical instability [1,4]. Furthermore, currently available assessment methods vary in sensitivity, standardization, and clinical applicability, which may complicate early diagnosis and monitoring of ICU-acquired weakness in routine ICU practice [25,26].

5. Discussion

The available evidence reviewed in this study indicates that prevention and management of ICU-acquired weakness remains a complex clinical challenge, primarily due to the multifactorial nature of muscle wasting during critical illness [3,5,8]. The strategies described in the literature target different but interrelated mechanisms, including prolonged immobilization, metabolic imbalance, and neuromuscular dysfunction, which may explain the variability in their reported effectiveness.

Among the currently available strategies, early mobilization appears to have the strongest and most consistent support in the literature. Its beneficial effects on functional outcomes, muscle strength, and duration of mechanical ventilation have been demonstrated across several randomized trials and meta-analyses [9–14]. These findings support the growing emphasis on minimizing prolonged immobilization in critically ill patients. However, applicability of early mobilization is often limited in the most critically ill patients, particularly those with hemodynamic instability or requiring deep sedation and ventilatory support. This highlights an important gap between evidence and clinical feasibility, as patients at the highest risk of ICU-AW are often those who cannot fully participate in rehabilitation.

Organizational factors, including the availability of trained staff and structured multidisciplinary care protocols, may further influence the implementation and effectiveness of preventive interventions in ICU settings [11–14,27].

Nutritional support represents another essential component of both prevention and early management strategies, particularly in the context of the hypercatabolic state characteristic of critical illness [5,8]. Although adequate protein delivery is recommended and may support muscle preservation, the available data remain inconsistent with regard to its impact on functional outcomes [20–23]. In addition, achieving recommended nutritional targets in ICU conditions is often challenging due to gastrointestinal intolerance or interruptions in feeding [22,23]. These factors suggest that nutritional therapy alone may not be sufficient to effectively counteract muscle wasting.

Optimized metabolic control, particularly avoidance of severe hyperglycemia, may additionally support preservation of neuromuscular function, given its association with adverse metabolic and neuromuscular outcomes in critically ill patients [5,8].

Sedation practices, including strategies such as daily sedation interruption and regular reassessment of sedation depth, represent an important but complex component of ICU care, as they may both contribute to the development of ICU-acquired weakness and influence the feasibility of preventive and early management strategies. While lighter sedation may facilitate early mobilization and improve participation in rehabilitation [9,13], it is not always feasible in critically ill patients requiring ventilatory support or hemodynamic stabilization [1,4]. This reinforces the need for individualized approaches, as strict application of minimal sedation strategies may not be appropriate in all clinical scenarios.

Similarly, ventilation strategies aimed at preserving diaphragmatic activity may help limit respiratory muscle dysfunction associated with prolonged mechanical ventilation [1,4].

In contrast to early mobilization, neuromuscular electrical stimulation (NMES) may offer a practical alternative in patients who are unable to participate in active rehabilitation. NMES allows for muscle activation independent of voluntary effort and may help reduce disuse-related atrophy [15–17]. However, the evidence remains heterogeneous, with differences in stimulation protocols and patient selection contributing to variability in outcomes [16,17]. This limits the ability to draw definitive conclusions regarding its routine use, although it may be particularly useful in selected patient populations.

The assessment of muscle mass and function remains an important but methodologically challenging aspect of ICU care. While tools such as the MRC scale provide clinically relevant information, their use is limited mostly to cooperative patients [1,4]. Objective methods such as bedside muscle ultrasound offer promising alternatives, allowing early detection of muscle loss. Despite this, challenges related to standardization and interpretation still persist [5,25]. These limitations may delay diagnosis and reduce the effectiveness of preventive and supportive interventions. Early identification of muscle deterioration may allow timely adjustment of preventive and supportive strategies, potentially improving clinical outcomes [25,26].

When considered collectively, the analyzed strategies demonstrate that no single intervention fully addresses the complexity of ICU-acquired weakness. Early mobilization, nutritional support, NMES, and sedation optimization each target specific aspects of muscle deterioration but are associated with distinct limitations. This observation is consistent with the multifactorial pathophysiology of ICU-AW and may explain why isolated interventions often produce variable results across studies [3,5,8].

In this context, combining different preventive and early management strategies may represent a rational approach. Interventions such as early mobilization and rehabilitation, nutritional support with adequate protein delivery, NMES, and individualized sedation management may complement each other by targeting different mechanisms involved in muscle wasting. Emerging evidence suggests that integrated or “bundle” approaches may be associated with improved clinical outcomes, including reduced incidence of ICU-acquired weakness and shorter duration of mechanical ventilation [27]. Although the available data remain limited and heterogeneous, this approach may better reflect the complexity of critically ill patients and the practical limitations of individual interventions.

Overall, the findings of this review suggest that prevention and early management of ICU-acquired weakness require a comprehensive and individualized approach that takes into account both the effectiveness and feasibility of available strategies. Further research is needed to better define the optimal combination, timing, and intensity of preventive interventions, as well as to improve the standardization of assessment methods and outcome measures [7,12].

6. Conclusions

In conclusion, prevention and early management of ICU-acquired weakness appear to involve multiple complementary strategies, including early mobilization and rehabilitation, nutritional support, neuromuscular electrical stimulation, and individualized sedation management. These interventions may contribute to preserving muscle function, reducing the burden of muscle wasting, and supporting recovery in critically ill patients; however, their effectiveness is often influenced by clinical condition and practical limitations within the ICU setting.

The current body of evidence highlights both the potential and the limitations of individual strategies, suggesting that their impact may be enhanced when applied in a coordinated and patient-centered manner. In this context, combining complementary approaches targeting both prevention and early management may provide a more coherent framework for clinical practice.

At the same time, existing research remains heterogeneous, particularly with regard to intervention protocols, patient populations, and outcome measures. This underscores the need for further research aimed at optimizing preventive and early management strategies and improving their implementation in routine clinical practice.

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