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Early Mobilization on Patients with Mechanical Ventilation in the ICU

Tsung-Hsien Wang

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Abstract

Patients in intensive care unit (ICU) usually experienced being immobile and restrained, and these can induce muscle weakness, cognitive impairments, psychological difficulties, difficult weaning, and increased length of stay (LOS) in hospital. However, early mobilization has multiple benefits including improved ventilation, perfusion, muscle strength, and functional capacity.

Keywords: early mobilization, ICU-acquired weakness, intensive care unit, mechanical ventilation, delirium, sedation

1. Introduction

Patients in the intensive care unit are often immobile and restrained, which can induce muscle weakness, cognitive impairment, psychological difficulties, and difficulty weaning from mechanical ventilation (MV) and may increase the overall length of stay (LOS) in the hospital. Long-term complications such as postintensive care syndrome, intensive care unit (ICU)-acquired weakness, physical debility, and neuropsychiatric dysfunction have become a clinical and scientific focus, as their impact on long-term quality of life is becoming increasingly obvious [1].

Early mobilization, however, has multiple benefits, including improvements in ventilation, perfusion, muscle strength, and functional capacity. Early mobilization of ICU patients, defined as mobilization within 72 h of ICU admission, remains uncommon. In critically ill medical and surgical patients, mobilization is well tolerated, even in intubated patients.

The purpose of this chapter is to provide a framework for early mobilization in critically ill patients followed by a review of recent developments in the field.

1.1. Critical care in ICU

Once a patient is admitted to the ICU, successful management should aim at patient survival. While patient with the acute present illness, serious trauma, shock, or progression of chronic illness may require intubation with mechanical ventilation. Intubation is the passage of an artificial airway into the patient's trachea, generally through the mouth (endotracheal tube (ETT) intubation) or through the nose (nasotracheal intubation).

Over the last 50 years, mechanical ventilation has been an important tool for maintaining a patient's breathing and improving patient survival and recovery from life-threatening diseases. As a result of mechanical ventilation, the mortality rate in intensive care units has notably decreased. Recent data show that the mortality rate is 25–30% in patients with acute respiratory failure [2]; the focus now must pivot to the sequela of survivors after critical illness. The sequela still exist, even after patients are discharged from the hospital, and the symptoms include functional limitations, cognitive impairment, and mental disorders in the months or even years after a critical illness.

On the other side, many standard ICU interventions have been shown to have negative effects when used excessively: tidal volumes that are too large [3], too many blood transfusions [4], too much oxygen [5], and too much sedation [6] can all have negative effects on outcomes.

Over the past 10 years, there have been substantial decreases in mortality from such conditions as acute respiratory failure and sepsis. Recent data, however, show that many survivors are sent to rehabilitation centers. There is a high rate of mortality among survivors within the first year after ICU discharge, although it is unknown how many survivors could have returned home to stay with their family. For those who survive, the latest data show that 50–70% of ICU survivors will suffer cognitive impairment and 60–80% of survivors will suffer functional impairments or ICU-acquired weakness (ICU-AW) [7].

2. Muscle injury during mechanical ventilation

After critical illness, muscle weakness emerges not only in the peripheral muscles but also in the respiratory muscles, and it is believed to be a key factor leading to prolonged disability. Limb muscle atrophy and functional limitations persist for months after recovery from a critical illness. Muscle weakness of the diaphragm is a key risk factor for prolonged mechanical ventilation and causes long-term complications and death.

2.1. Intensive care unit-acquired weakness

Intensive care unit-acquired weakness is the most common neuromuscular impairment in critically ill patients. There are several distinguishable types of ICU-AW, including critical illness polyneuropathy, critical illness myopathy, or a combination of the two. Diagnosing ICU-AW is difficult, although most clinicians use the Medical Research Council (MRC) sum score. Handgrip is also often used to evaluate limb weakness and to determine if a patient is ventilator dependent or may have difficulty weaning from a mechanical ventilator. Regardless

Figure 1. Time course of limb and respiratory muscle weakness during mechanical ventilation. Respiratory muscle weakness develops more rapidly than limb muscle weakness. Its influence on outcome is greater at an early stage (data from [9]).

of the type of ICU-AW, clinicians must evaluate the patient's muscle strength and assess the electrophysiological functioning of the peripheral muscles and nerves. ICU-AW is associated with difficulty in weaning from the ventilator, prolonged ICU stay, and higher hospitalization charges and increases long-term morbidity and mortality [8].

Most studies include limited cases of diagnosed ICU-AW because they usually evaluate patient weakness later in the course of disease or near hospital discharge. It is currently unknown whether peripheral muscle weakness causes prolonged ventilator use. Recently, Dres and colleagues revealed that, while limb muscle weakness noted at a patient's first spontaneous breathing trial was not correlated with prolonged mechanical ventilation, diaphragm muscle weakness did correlate. This result suggests that diaphragm muscle weakness induces prolonged mechanical ventilation, which in turn increases the risk of developing sustained limb muscle weakness and associated long-term functional impairment (**Figure 1**).

2.2. Definition

The term ICU-AW does not evaluate the time from admission to the ICU to the development of muscle weakness in a patient. In truth, it more likely describes the final resulting weakness that begins with any severe disease, regardless of the location of care. It is very important to diagnose ICU-AW after the onset of critical illness, which is a major distinguishing factor between ICU-AW and Guillain-Barre syndrome or other neuromuscular diseases that might cause acute respiratory failure after ICU admission (**Figure 2**).

Figure 2. Diagnostic approach to patients developing intensive care unit-acquired weakness. EMG, electromyography; ICU-AW, intensive care unit-acquired weakness; MRC, Medical Research Council; NCS, nerve conduction study; NM, neuromuscular (data from [10]).

2.3. Epidemiology of ICU-acquired weakness

Neuromuscular dysfunction has long been recognized as correlating with critical illness. Sir William Osler proposed a “rapid loss of flesh” in patients with severe sepsis in 1892.

In the 1960s, Mertens reported polyneuropathy in patients in a coma state, and proof of acute myopathy was found in a patient with acute severe asthma. De Jonghe et al. reported that the incidence rate of ICU-AW used by the Medical Research Council is 25.3% of patients receiving mechanical ventilation for 7 days who had clear consciousness and could follow commands. Dhand et al. reported overall, 30–50% of critically ill patients suffer from CIP, CIM, or a combination of them.

2.4. Mechanisms

Patients with systemic inflammatory response syndrome and multiple organ dysfunction experiencing respiratory insufficiency are prone to CIP/CIM. Inflammation, apoptosis, thrombosis, and oxidant injury constitute the basis of both multiple organ failure (MOF) and peripheral neuromuscular abnormalities [11], so neuromuscular weakness because of CIP/CIM is due to neural system participation in MOF [12].

Figure 3. Mechanisms of injury to the limb and respiratory muscles during mechanical ventilation (data from [9]).

According to Bolton et al., microcirculation damage in peripheral nerves and muscles triggered by sepsis has a critical role in the CIP/CIM pathogenesis. A wide range of factors contribute to muscle injury and weakness in the context of critical illness. Schreiber et al. provide a summary of these factors, focusing especially on those mechanisms offering potential targets for intervention (**Figure 3**).

2.5. Critical illness polyneuropathy

Critical illness polyneuropathy is a sensory-motor axonal polyneuropathy. Electrophysiological studies show a reduction in the amplitudes of compound muscle action potentials (CMAPs) and sensory nerve action potentials (SNAPs), with normal or near-normal nerve conduction velocity.

2.6. Critical illness myopathy

Critical illness myopathy is an acute original myopathy and is not correlated with denervation, which differentiates the electrophysiological and morphological reports [10].

These conditions often coexist, and the combination of CIP and CIM—indicated as critical illness myopathy and neuropathy (CRIMYNE) or critical illness polyneuropathy and myopathy (CIPNM)—is the most common overlap syndrome (**Table 1**).

Condition	Definition	Diagnosis
Intensive care unit-acquired weakness	Clinically detected, diffuse, symmetric weakness involving all extremities and respiratory muscles arising after the onset of critical illness	Medical Research Council sum score of less than 48/60 or mean MRC score of 4 in all testable muscle groups Dominant-hand handgrip dynamometry scores of less than 11 kg (interquartile range (IQR) 10–40) in males and less than 7 kg (IQR 0–7.3) in females
Critical illness polyneuropathy (CIP)	An axonal, sensory-motor polyneuropathy with reduced nerve excitability and loss of axons with preserved myelin sheet	Reduced amplitude of compound muscle action potentials and sensory nerve action potentials with normal or mildly reduced nerve conduction velocity on electroneurography
Critical illness myopathy (CIM)	A primary acute myopathy with reduced muscle membrane excitability and loss of myosin filaments, fiber atrophy, and necrosis	Reduced amplitude of compound muscle action potentials and normal sensory nerve action potentials on electroneurography and reduced muscle excitability on direct muscle stimulation and myopathic motor unit potentials on needle electromyography
Combined critical illness polyneuropathy and myopathy	Combined CIP and CIM	Reduced amplitude of compound muscle action potentials and sensory nerve action potentials combined with myopathic features on needle electromyography

Data from [13].

Table 1. Definition and diagnostic criteria of intensive care unit-acquired weakness, critical illness polyneuropathy, critical illness myopathy, and combined critical illness polyneuropathy and myopathy.

2.7. Risk factors

There is an abundance of risk factors related to ICU-AW. In addition to disease severity and ICU length of stay, SIRS, sepsis, multiple organ dysfunction syndrome, female sex, severe asthma attack, electrical and ionic abnormalities, immobilization, malnutrition, central nervous system failure, renal failure, low serum albumin, and vasopressor use have all been correlated with a trend toward more severe neuromuscular dysfunction in critically ill patients.

2.8. Bed rest

Disuse of muscles causes rapid muscle weakness and atrophy. Studies show that following 2 weeks of immobilization, young healthy adults experience a loss of muscle mass of 5–9% in the quadriceps and a decrease of 20–27% in muscle strength. These impacts are often accelerated and more obvious in aging adults, with a 3–6-fold greater rate of muscle wasting [14]. In mechanically ventilated patients, skeletal muscle cross-sectional area can decrease by 12.5% over the first week in the ICU [14].

The harmful impacts of prolonged immobilization on muscle structure and function have long been known. During critical illness, the muscles are always in an unloaded condition due to disease progression, sedation, and mechanical ventilation. This unloaded condition

Skeletal muscle atrophy and weakness
Joint contractures
Thromboembolic disease
Insulin resistance
Microvascular dysfunction
Systemic inflammation
Atelectasis
Pressure ulcers
Data from [17].

Table 2. Complications of bed rest.

impacts not only the limb muscles but also the respiratory muscles, and it leads to muscle atrophy resulting from inactivity and a reduction in myofibril tension [15].

Due to the nature of critical illness, medications (e.g., sedation), and devices (e.g., continuous renal replacement therapy) used in the ICU, patients spend a great amount of time immobilized in bed [16].

However, critically ill patients frequently remain on bed rest for many days to weeks and suffer complications of bed rest (**Tables 2** and **3**). Prolonged bed rest and physical activity restraint can result in ICU-AW, pressure ulcers, lung atelectasis, aspiration pneumonia, bone mineral loss, muscle atrophy, hypotension, tachycardia, and cardiac output decreases, which result in obvious declines in physical function [19].

2.9. Sedation

Many medications used to treat critically ill patients have myopathic effects. Sedatives can exert both direct injurious effects and indirect effects by promoting rest and disuse atrophy. Heavy sedation from some medications such as benzodiazepines, propofol, fentanyl, and morphine is needed in most critical illnesses in order for patients to cooperate with a mechanical ventilator. Sustained neuromuscular blockade should be limited; it is therefore helpful to decrease the dose and duration of NMBD administration, especially in high-risk patients suffering from hepatic or kidney failure. Therapy with NMBDs and steroids for over 48 h increases the risk of acute myopathy, so the duration of their administration should be as short as possible.

Neuromuscular blocking agents and steroids should be managed carefully, and when possible, safe, and clinically sound to do so, a daily interruption of sedation should be cautiously undertaken. Heavy sedation should be adjusted following evidence-based strategies to minimize immobilization. If a patient is agitated and requires a continuous sedative infusion, short-acting titratable agents such as propofol are preferable over benzodiazepines to minimize immobility.

Musculoskeletal
<ul style="list-style-type: none">• Decreased muscle protein synthesis• Muscle atrophy and decrease in lean muscle mass• Decreased muscle strength• Decreased exercise capacity• Connective tissue shortening and joint contractures• Decreased bone density• Pressure ulcers
Pulmonary
<ul style="list-style-type: none">• Atelectasis• Pneumonia• Decreased maximal inspiratory pressure and forced vital capacity
Cardiovascular
<ul style="list-style-type: none">• Decreased total cardiac and left ventricular size• Decreased lower extremity venous compliance• Orthostatic intolerance• Decreased cardiac output, stroke volume, and peripheral vascular resistance• Impaired microvascular function• Decreased cardiac response to carotid sinus stimulation
Endocrine and metabolism
<ul style="list-style-type: none">• Decreased insulin sensitivity• Decreased aldosterone and plasma renin activity• Increased atrial natriuretic peptide

Data from [18].

Table 3. Selected adverse effects of prolonged bed rest.

2.10. Sepsis

Infection is a major risk factor for the development of marked diaphragm weakness in mechanically ventilated patients [20]. In patients with sepsis, diaphragm muscle strength has been shown to decrease by an average of 50%. General muscle weakness in sepsis is mediated by cytokines, oxidative stress, and the activation of proteolytic pathways. Sepsis also weakens the body’s ability to deliver oxygen and impairs its use at the cellular level, disrupting muscle metabolism and contractile function. Therefore, sepsis enhances muscle membrane fragility, increasing its sensitivity to the damaging effects of loading. The bed rest and immobilization associated with sepsis, however, may intensify the effects of the systemic infection [9].

2.11. Delirium and ICU-AW

Delirium is defined as an acute onset mental disturbance including loss of awareness and cognitive impairment that changes over time. Delirium is a major complication in critical illness as it shows a decompensation of brain function, a sudden brain failure, in response to other pathophysiologic stressors [21].

It occurs in 60–80% of critically ill patients who are receiving mechanical ventilation and in 20–50% of critically ill patients who are not receiving mechanical ventilation [22]. Prevalence rates of delirium in ICU-AW range between 20 and 40%. Hypoactive delirium in particular is correlated with progressive clinical outcomes, such as prolonged mechanical ventilation use, longer ICU and hospital length of stay, increased mortality rates, and cognitive impairment for up to 1 year after discharge [23, 24]. However there is some strategy to prevent delirium (**Table 4**).

2.12. Muscle weakness, nutrition, metabolism in ICU patients

A major concern in patients with critical illness is their inability to fulfill their own nutritional needs, especially when using mechanical ventilation. Observational data show that malnutrition is accompanied by poor outcomes in critical illness, and many providers have assumed that this means that the use of artificial nutrition in these patients improves outcomes, regardless of baseline nutritional status.

Nutritional status is associated with weakness: starvation in healthy volunteers causes loss of muscle mass, strength, and function [8].

Although the practice of providing nutrition to these patients is almost universal, the specifics vary widely from one ICU to another and even among providers.

Nutrition in the intensive care unit remains challenging; patients in the ICU are always treated using a multidisciplinary team approach, and care is determined by the patient's condition, the severity of their disease, and what they need.

However, differences in preferences among care providers increase the variations in opinions related to controversial topics, especially in nutritional support. In critically ill patients,

Components of Henderson's theory	Bundle components
Breathe normally	Sedation cessation
Participate in various forms of recreation	Pain management
Communicate with others in expressing emotions, needs, fears, or opinions	Sensory stimulation
Move and maintain desirable positions	Early mobility
Sleep and rest	Sleep promotion
Avoid environmental dangers and injury of others. Data from [22].	

Table 4. Components of Henderson's theory that align with components of the delirium prevention bundle.

nasogastric tube feeding is often used due to its simplicity, relatively low cost, and decrease in risk of catheter-induced complications.

Insufficient nutritional support in enterally fed patients remains a global concern; however, as predicted and evaluated, energy insufficiency has been associated with poor outcomes. For the first week following admission to the ICU, most patients experience energy deficits, and these deficits are even more obvious when enteral nutrition is the only available method. Sustained nothing per os (NPO) status in the ICU and the corresponding loss of lean body mass should be at least as relevant as it is for other hospitalized patients.

Inflammation, infection, immobilization, and fatigue may result in noticeable delays in recovery, but a formal evaluation of the 1-year post-hospitalization impact in a large population of critically ill patients has not been completed, although dysfunctional status was found 5 years after an experience of acute respiratory distress syndrome. Overnutrition is more easily identified since many of its impacts are obvious and measurable, such as difficulty weaning from a ventilator, heart disease, and fatty liver disease; other effects of overnutrition, such as increased susceptibility to infection, are difficult to observe. Overnutrition has more often been achieved with parenteral nutrition rather than enteral nutrition. Additionally, overnutrition combined with bed rest has been found to lead to a greater loss of lean body mass.

A variety of metabolic derangements associated with critical illness can exacerbate muscle weakness. Respiratory acidosis, hypercapnia, and hypokalemia contribute to reversible diaphragm weakness, whereas metabolic acidosis seems to have no effect [25]. Hypercapnia can increase ventilator-induced diaphragmatic dysfunction, and hyperglycemia is a significant risk factor for muscle damage. Long-term malnutrition is likened to ICU-AW as a result of a shortage of protein in muscle tissue.

3. Early mobilization in IC

A lack of muscle loading and no contraction are a major cause of muscle damage and weakness, so patient mobilization initiated as soon as possible is necessary to prevent this damage. Patient performance of early mobilization with mechanical ventilation can be feasible and safe. The advantage of passive range of motion exercises should not be ignored, as they can lengthen the muscles when there is no active muscle contraction, leading to significant relief of muscle atrophy [26].

3.1. Safety and feasibility

Immobilization and restraint always occur during a patient's stay in the ICU, leading to the development of some complications, such as physical functional impairment, cognitive dysfunction, and neuromuscular weakness. Early mobilization has been proven to be a feasible, safe, and important preventive measure. Many studies have shown that early mobilization can be performed safely and without adverse events, even when the patient is receiving mechanical ventilation, using an extracorporeal membrane oxygenation device or receiving continuous venovenous hemofiltration [27].

System	Starting criteria	Stopping criteria
Cardiovascular	Heart rate: 60–130 beats/min Systolic blood pressure, 90–180 mmHg, or mean arterial pressure, 60–100 mmHg	Heart rate: <60 or >130 beats/min Systolic blood pressure, <90 or >180 mmHg, or mean arterial pressure, <60 or >100 mmHg
Respiratory	Respiratory rate: 5–40 breaths/min SpO ₂ ≥ 88% FiO ₂ < 0.6 PEEP <10 Artificial airway is properly secured	Respiratory rate: <5 or >40 breaths/min SpO ₂ < 88% Concerns about the disconnection of the artificial airway
Other	Able to open eyes to voice	Changes in consciousness New/symptomatic arrhythmia Chest pain Ventilator asynchrony Falling down Medical device removal Patient intolerance or refusal

SpO₂, peripheral capillary oxygen saturation; FiO₂, fraction of inspired oxygen; PEEP, positive end-expiratory pressure. Data from [31].

Table 5. Examples of safety criteria for starting and stopping rehabilitation in the intensive care unit.

Early mobilization in the ICU has been advocated as a therapeutic strategy to prevent ICU-AW, reducing the negative effects of immobility on muscles and other organ systems [28]. Mobilization in the ICU is feasible and safe provided that consensus guidelines are followed [29]. The potential incidence of adverse events is low, with a cumulative incidence of 2.6%, a rate of hemodynamic events of 3.8%, and saturation instability of 1.9 per 1000 mobilization sessions. Additionally, medical issues are rare, with one study showing a rate of 0.6% of 14,398 mobilization sessions [30]. Premobilization screening criteria to identify patients for whom rehabilitation is suitable and criteria for discontinuing mobilization should be established for each ICU (Table 5).

3.2. Beneficial effects of early mobilization

The current definition of mobilization is the performance of physical activity sufficient to stimulate an acute physiological impact that improves ventilation, circulation, perfusion, muscle metabolism, and alertness and decreases rates of venous stasis and deep vein thrombosis. The present definition of early mobilization refers to physical activity performed within the first 72 h of a critical illness [32].

The evidence of the impact of early mobilization in the ICU continues to increase. ICU physical therapy has also been shown to have a positive impact on patient outcomes, such as functional ability, muscle strength, and ambulation capacity at discharge.

Figure 4. Photo showing a patient receiving mechanical ventilation via an endotracheal tube while ambulating in the ICU with a physical therapist. Data from [14].

Early mobilization in mechanically ventilated critically ill patients is feasible and safe [27, 33, 34] and can decrease duration of mechanical ventilation and intensive care unit length of stay. It is also associated with a shorter duration of delirium, a better functional outcome at hospital discharge, and lower 1-year mortality [35, 36]. Early mobilization has multiple benefits including improved ventilation, perfusion, muscle strength, and functional capacity. Several surveys reporting mobilization therapy practice are published worldwide, and despite all of the reported benefits, the prevalence of early mobilization in ICU patients is still low [37]. Together with other chest physiotherapy modalities, early mobilization can result in a decreased extubation failure rate and shorter durations of mechanical ventilation and ICU stay [38]. **Figure 4** illustrates a patient receiving mechanical ventilation via an ETT and ambulating with a physical therapist in the ICU.

Late initiation may reduce the efficacy of mobilization as the beneficial effects of physical therapy have been found in studies in which the treatment was started early after ICU admission [39]; however, definition of “earliness” remains undefined.

3.3. Special patient groups (ECMO)

Patients undergoing mechanical ventilation or extracorporeal circulation systems such as extracorporeal membrane oxygenation (ECMO) or hemofiltration need particular attention.

The use of ECMO for patients is increasing globally, including in patients with heart or respiratory failure awaiting organ transplantation, and there is an increasing need to assess the safety, feasibility, and functional prognosis regarding the performance of early mobilization in this patient population [40].

Early ICU physiotherapy while on ECMO for ARDS as a bridge to recovery is feasible; Munshi et al. suggest that there may be an association with improved ICU mortality. However, this needs to be further corroborated in future studies. Future research is needed to identify potential barriers, optimal timing, dosage, and safety profile.

4. Barriers and facilitators to mobilization

Although there is a growing base of evidence showing short-term benefits of early mobilization, such as shorter delirium duration, more ventilator-free days, and decreased ICU and hospital LOS, translation into clinical practice remains difficult for this complex intervention. Dubb et al. provided some common barriers that medical staff may encounter regarding early mobilization as well as possible ways to overcome them.

4.1. Cultural barriers

In many medical centers, present ICU culture is a crucial and potentially modifiable barrier to the initiation of ICU physical therapy and early mobilization. However, inadequate care coordination, timing conflicts with different medical procedures, and priorities for treatment are common barriers [41, 42]. Lastly, probably one of the most important barriers to address in implementation of early mobilization is in relation to the culture of the ICU, reported as an obstruction in almost 60% of studies [43]. This encompasses staff not viewing early mobilization as a priority for the patient and/or inadequate knowledge about the benefits of early mobilization and techniques available to implement it at the appropriate level for the patient.

Overcoming these barriers requires a structured multidisciplinary effort, with clear communication and recognition of the importance of early mobilization and rehabilitation [44].

4.2. Sedation

The widespread use of sedation in the ICU can be a major barrier to mobilizing patients who are critically ill. Implementation of early mobilization into practice was however limited, mostly due to sedation practices and clinician’s safety concerns. Sedation minimization can be combined with early mobility via implementing the ABCDE bundle [45], in which all patients

undergo daily coordinated spontaneous awakening trials, spontaneous breathing trials, sedation and delirium screening, and early mobility and rehabilitation.

4.3. Endotracheal tubes

The presence of an endotracheal tube is another commonly perceived barrier. To assist with overcoming this barrier, Pohlman et al. detailed the steps undertaken to start PT and OT interventions after a median of only 1.5 days of intubation in 49 patients who had undergone endotracheal intubation. There were no catheter-related adverse events [27]. Similar results were obtained in other studies [46, 47].

4.4. Costs

Increased medical staffing needs and costs correlated with early mobilization are commonly recognized barriers. Utilizing a special early mobilization team, Morris et al. did not find an increase in overall costs after accounting for additional costs related to this team, and they also found a significantly reduced risk of readmission or mortality in the year after ICU discharge.

There are multiple facets to ensuring the effective implementation of early mobilization, with interdisciplinary coordination and communication being an integral component (Table 6).

Patient-associated factors

- Unstable physiologic stability (cardiovascular, respiratory, neurologic)
- High sedation levels
- Presence of delirium/agitation
- Pain
- Medical limitations (procedures, orders)

Structural issues

- Low staff numbers
- Inexperienced clinical staff
- Deficiency in staff training
- Lack of defined EM programs

Process factors

- Absence of coordinated review for suitability for EM
- No EM leadership
- Poor communication

Cultural factors

- Nonexistent education regarding risks and benefits of EM
- Lack of prioritizing EM in daily care plans
- Deficient knowledge regarding EM techniques and equipment

Data from [48].

Table 6. Barriers to implementation of early mobilization in intensive care unit barrier.

5. Discussion

This chapter shows that early mobilization in the ICU demonstrated benefits on patient outcomes. The intervention improves ventilation, circulation, perfusion, muscle metabolism, and alertness and decreases rates of venous stasis and deep vein thrombosis.

Based on the findings from these reviews, no study has shown any adverse events associated with early mobilization. When safety consensus guidelines are followed and a team approach is used to ensure safety in clinical practice, early mobilization in the ICU may be an appropriate treatment strategy. It is important to recognize that not all potential safety events carry equal clinical importance (e.g., high blood pressure vs. cardiac arrest).

Patient safety is paramount in any exercise intervention. Patients with high illness severity, coma, and/or delirium are particularly vulnerable, and the utmost care must be undertaken during exercise interventions in these high-risk patients. Although many studies have shown that early mobilization is safe and feasible and one study indicated that healthcare professionals are able to identify the benefits associated with early mobilization, only 21% of physicians and 18% of nurses believe that the potential risks outweigh the benefits of early mobilization.

The optimal timing for initiation of mobilization has yet to be defined. ICU-AW can begin within the first 48 h of ICU admission; early mobilization of ICU patients, defined as mobilization within 72 h of ICU admission; and late mobilization as starting after the first 3 days of ICU admission. Other research suggests that early mobilization is currently defined as occurring within the first 2–5 days of ICU admission. Arriving at a definitive definition of early mobilization is difficult because the condition of a patient in the ICU changes daily. Further research is required to provide a conclusive definition of early mobilization.

6. Conclusion

Early mobilization and physical rehabilitation of critically ill patients appears to be safe, with a low risk of potential safety events, even when implemented as part of routine clinical practice. Early mobilization, defined as mobilization within 72 h of ICU admission, is well tolerated and feasible and should be the standard of care. However, implicating early mobilization is arduous and may need a cultural change in intensive care with an interprofessional approach.

Increases in ICU-associated neuromuscular dysfunction are resulting in both short- and long-term physical functional impairments. Bed rest and restraint play a major role in the development of neuromuscular disorders and functional ability impairments, and early mobilization and physical therapy may be able to mitigate these problems. The complete effects of early mobilization in the ICU need to be evaluated using a standardized execution protocol to determine the optimal timing, exercise dosage, and progression of mobilization, as well as determining criteria for the intensity and duration of physical therapy most likely to optimize patients' physical condition during critical illness [49].

Achieving early mobilization in practice requires a shift in unit culture and dedication from mobility champions following individualized procedures and protocols to ensure its safe application.

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