International Journal of Allied Medical Sciences and Clinical Research (IJAMSCR)
IJAMSCR |Vol.12 | Issue 4| Oct - Dec -2024
www.ijamscr.com
ISSN: 2347-6567
DOI : https://doi.org/10.61096/ijamscr.v12.iss4.2024.610-617

Review

Effects of Extended Bed Rest in ICU Immobilization and Inactivity

Asit Kumar Bhoi[1], Sushree Smitanjali [2], Dwarikanath Rout[3], Priyabrata Dash[4], Nihar Ranjan Mohanty*[5]

[1]Physiotherapist, KIMS School of Physiotherapy, KIMS, KIIT-DU, Bhubaneswar, Odisha
[2]Physiotherapist, KIMS School of Physiotherapy, KIMS, KIIT-DU, Bhubaneswar, Odisha
[3]Assistant Professor, KIMS School of Physiotherapy, KIMS, KIIT-DU, Bhubaneswar, Odisha
[4]Associate Professor cum Principal (I/C), KIMS School of Physiotherapy, KIMS, KIIT-DU, Bhubaneswar, Odisha
[5]Associate Professor, KIMS School of Physiotherapy, KIMS, KIIT-DU, Bhubaneswar, Odisha

*Author for Correspondence: Nihar Ranjan Mohanty
Email: drnrm1989@gmail.com

Published on: 11 Dec 2024

Published by:
DrSriram Publications

Abstract

Extended bed rest, often a necessity for critically ill patients in Intensive Care Units (ICUs), has traditionally been prescribed to prevent complications, conserve metabolic resources, and ensure patient comfort. Historically, bed rest was considered beneficial for various medical conditions, including acute rheumatoid arthritis flares, cavitary tuberculosis, acute myocardial infarction, and acute low back pain. However, modern research through randomized controlled trials has largely debunked these benefits, revealing that prolonged immobilization can lead to significant adverse effects, thereby complicating recovery processes. This study delves into the extensive impact of prolonged bed rest on multiple physiological systems, emphasizing the severe consequences on muscle integrity, cardiovascular and respiratory functions, endocrine balance, and overall rehabilitation outcomes.

Keywords: Bed rest, ICU, muscle atrophy, cardiovascular complications, respiratory function, endocrine imbalance, rehabilitation, early mobilization.

INTRODUCTION

Rationale for bed rest

Bed rest is commonly prescribed for critically ill patients with the belief that it can prevent complications, conserve metabolic resources, and provide comfort. This practice assumes that minimizing physical activity allows the body to focus its energy on healing and recovery. Historically, bed rest has been a standard recommendation for various medical conditions. For example, patients experiencing acute rheumatoid arthritis flares have been advised to rest to reduce inflammation and pain. Similarly, those with cavitary tuberculosis have been prescribed bed rest to minimize the spread of the infection and conserve energy. In cases of acute myocardial infarction, bed rest was believed to reduce cardiac workload and prevent further damage to the heart. Acute low

back pain has also been a common reason for recommending bed rest, with the intention of reducing strain on the spine and allowing inflammation to subside.

However, the validity of bed rest as a therapeutic intervention has been increasingly questioned. Randomized controlled trials (RCTs), which are considered the gold standard in clinical research, have largely demonstrated that bed rest does not provide significant benefits for these conditions. In many cases, prolonged inactivity has been found to be detrimental rather than beneficial.

Adverse effects of bed rest

Despite its intended benefits, prolonged bed rest can lead to a multitude of complications that may hinder or delay recovery from critical illnesses. One of the most significant consequences is muscle atrophy from disuse. When muscles are not regularly engaged, they begin to weaken and shrink, leading to a loss of strength and functionality. This muscle atrophy can be particularly problematic for patients who are already weakened by their illness.

In addition to muscle atrophy, joint contractures can occur. Joint contractures result from the tightening or shortening of muscles and tendons around a joint, limiting its range of motion and potentially causing permanent deformity. This condition can severely impair a patient's ability to regain mobility and independence.

Thromboembolic disease is another serious complication associated with prolonged bed rest. When patients are immobile, blood flow through the veins slows down, increasing the risk of blood clots forming. These clots can travel to the lungs, causing a potentially life-threatening pulmonary embolism, or to other parts of the body, leading to strokes or other serious complications.

Furthermore, bed rest can contribute to the development of insulin resistance. When the body is inactive for extended periods, its ability to process glucose efficiently is impaired. This can lead to elevated blood sugar levels and an increased risk of developing type 2 diabetes, complicating the patient's overall health status and recovery process.

Causes for bed rest

Several medical conditions necessitate bed rest for patients. Multiple traumas, for example, can result in extensive injuries that require immobilization to allow proper healing. Spinal cord injuries often necessitate bed rest to prevent further damage and to manage pain. Stroke patients may require bed rest during the initial recovery phase to stabilize their condition and prevent additional complications.

Myocardial infarction, or heart attack, is another condition that has traditionally warranted bed rest to reduce the strain on the heart and promote recovery. Rheumatoid arthritis, with its painful joint inflammation, often leads to periods of immobility to manage symptoms.

In addition to these conditions, various other medical issues can result in the prescription of bed rest. Some individuals become immobile due to disabilities that limit their ability to move. In other cases, patients may remain bedridden without any underlying organic problem, often due to psychological factors such as severe depression or anxiety, which can lead to a lack of motivation to move.

While bed rest has been a traditional component of managing many medical conditions, modern research has highlighted its potential drawbacks. Muscle atrophy, joint contractures, thromboembolic disease, and insulin resistance are significant complications that can arise from prolonged immobility. Understanding the nuanced impact of bed rest is crucial for healthcare providers in order to balance the need for rest with the importance of maintaining as much activity as possible to support overall health and recovery.

Muscle complications due to immobilization

Strength loss

Immobilization significantly impacts muscle strength, resulting in a daily strength loss ranging from 1.0% to 1.5%. This decline is alarmingly rapid; within just 7 to 9 days of bed rest, an individual can experience a 20% to 30% reduction in muscle strength. Over a period of five weeks, this strength loss can escalate to approximately 50% of the individual's previous muscle capacity. However, this drastic decline eventually stabilizes, leaving the patient with only 25% to 40% of their original muscle strength. A critical factor in mitigating this decline is muscle activation. Research suggests that engaging in a single muscle contraction per day at 50% of maximum strength can prevent this strength deterioration. The muscles most affected by immobilization include the quadriceps and extensor muscles, which are crucial for activities such as standing and walking. The loss of strength in these muscles severely impacts a patient’s mobility and independence.

Impact on muscle fiber composition

The initial phase of immobilization has a profound effect on muscle fiber composition, particularly on type I fibers, also known as slow-twitch fibers. These fibers are essential for endurance activities as they are highly resistant to fatigue. Immobilization leads to a reduction in the diameter of type I fibers. Additionally, there is a decrease in the proportion of type I fibers and oxidative enzymes within the muscle tissue, resulting in diminished

muscular endurance. This decrease means the muscles tire more quickly and are less capable of sustaining prolonged activity. On average, the weekly muscle mass loss during immobilization ranges from 5% to 10%, highlighting the rapid deterioration of muscle integrity.

Muscular atrophy

Muscular atrophy, or the wasting away of muscle tissue, occurs when muscles are not used regularly. During muscle contraction, intramuscular tension increases, which helps pump blood out of the muscle and facilitates venous and lymphatic drainage. When muscles relax after contraction, arterial blood flow improves, delivering essential nutrients to the muscles. This cyclical process of contraction and relaxation is crucial for maintaining muscle health. However, during periods of immobility, the lack of muscle activity disrupts this process, depriving muscles of necessary nutrition. Consequently, muscles undergo dystrophy or atrophy, leading to significant weakness.

Blood flow and muscle function

Blood flow to muscles is closely linked to their contraction state. At about 10% of maximum voluntary contraction, blood flow begins to reduce, and at approximately 70% of maximum voluntary contraction, blood flow ceases entirely. Therefore, regular cycles of muscle contraction and relaxation are essential for ensuring an adequate supply of blood and nutrients to the muscles. In the absence of these cycles, muscles become starved of nutrients, accelerating atrophy and weakness.

Impact on muscle torque

The effect of immobilization on muscle torque has been well-documented. Gogia et al. (1988) conducted a study on the impact of bed rest on extremity muscle torque in healthy men confined to bed for five weeks, with only brief periods of sitting up for bowel movements. The study revealed a 26% reduction in gastrocnemius muscle torque and a 24% reduction in soleus muscle torque. These findings illustrate the profound effect of immobilization on muscle strength, particularly in muscles crucial for lower limb function and stability.

Extended impact on muscle strength and recruitment

Restricted activities not only affect the muscles near the injury site but also influence muscle strength and recruitment patterns in distant muscles. Beckman and Buchanan (1995) and Nicholas et al. (1976) conducted studies showing that hip muscle strength and recruitment patterns were affected for several months following an ankle sprain. This highlights the systemic impact of immobilization, where muscles far from the injury site also experience strength loss and altered functionality.

In summary, immobilization has a severe and rapid impact on muscle strength, composition, and function. Daily strength loss, significant reductions in muscle mass and endurance, and the disruption of normal blood flow and nutrient delivery to muscles all contribute to muscular atrophy and weakness.

The systemic effects of immobilization extend beyond the immediate site of injury, affecting overall muscle recruitment patterns and long-term functional capacity. Early intervention through muscle activation and rehabilitation exercises is crucial to mitigate these detrimental effects and promote recovery.

Forgetfulness of movement patterns

The brain encodes movement patterns rather than individual muscle actions. These patterns are stored in the motor cortex, and stimulation of this area results in voluntary movement. When a person is immobile, whether due to inability, restriction, or lack of movement, the brain gradually forgets these movement patterns.

As the injury, illness, or disorder heals, the person may struggle to perform voluntary movements and will need extensive motor learning or relearning to regain these abilities. Regular, relaxed passive movements help maintain the memory of these movements, facilitating easier rehabilitation and restoration of function. According to Beevor's theory, the brain retains the pattern of movement, not the specific muscle actions.

To regain voluntary movements, he must engage in extended motor learning or relearning training. This process involves repeatedly practicing specific movements to help the brain and muscles relearn how to work together effectively. Regular, relaxed passive movements, where the limbs are moved gently without active muscle engagement, help reinforce these movements in the client's memory. By repeatedly experiencing these passive movements, the client's brain is better able to recall and recognize the patterns of these movements, making it easier to transition to active movement during rehabilitation.

According to Beevor's theory, the brain is more adept at remembering the overall pattern of a movement rather than the individual muscle actions required to perform it. This means that by focusing on the complete movement pattern during training, the brain can more efficiently coordinate the muscles involved.

As a result, rehabilitation efforts that emphasize movement patterns over isolated muscle actions are likely to be more effective in restoring the client's ability to perform voluntary movements and daily activities.

Bone and joints

Bone Health and Calcium Metabolism When bones lack the necessary stress and tension from weight-bearing activities and muscle contractions, it can lead to a condition called osteopenia, which is characterized by reduced bone density. Osteopenia can subsequently cause hypercalcemia, where excess calcium enters the bloodstream.

This surplus calcium is then excreted through urine and feces, with noticeable increases starting around 2 to 3 days after the onset of immobilization. The peak excretion typically occurs between 3 to 7 weeks. Even after normal physical activity resumes, elevated calcium levels in the body persist for about 3 weeks before gradually returning to normal around 5 to 6 weeks.

When comparing the excretion patterns of calcium with nitrogen and protein in the urine, it's evident that calcium normalization takes the longest. Nitrogen loss is about 2 grams per day, commencing around 5 to 6 days post-immobilization and reaching its peak during the second week.

After resuming activity, nitrogen loss continues for another week before stabilizing in the second week. It takes approximately 4 weeks for nitrogen values to fall below normal and about 6 weeks to return to normal levels.

The excretion of calcium, coupled with phosphorus loss, contributes to muscle atrophy and increases the risk of bone fractures due to weakened structural integrity.

Joint Health and Ligament Changes Prolonged inactivity affects joint health significantly. A decrease in the extensibility of periarticular connective tissues around the joints is observed. The articular cartilage, which covers the ends of bones in synovial joints, begins to deteriorate because it lacks adequate nutritional support. Unlike other tissues, hyaline cartilage in synovial joints does not receive nutrients through blood vessels. Instead, it relies on the diffusion of nutrients from the synovial fluid, facilitated by the mechanical loading and unloading of the joint.

Ligaments, which connect bones to each other and provide joint stability, undergo biochemical changes as early as 2 weeks after immobilization. These changes include a decrease in ligament strength, an increase in compliance (stretchiness), and a higher rate of collagen degradation. For surgically repaired ligaments, immobilization can hinder the healing process and overall strength recovery. The longer a ligament remains immobilized, the more significant these detrimental effects become.

In summary, the lack of physical stress and tension on bones and joints due to immobilization or inactivity leads to several adverse effects. Osteopenia and hypercalcemia can develop in bones, while joints suffer from decreased connective tissue flexibility and cartilage health. Ligaments weaken and lose their structural integrity, making them more prone to injuries and compromising overall joint stability.

Cardiac effects of immobilization

Reduction in Blood and Plasma Volumes Prolonged periods of immobilization can lead to a significant reduction in both blood and plasma volumes. This decrease causes a redistribution of body fluids, which contributes to postural hypotension, a condition characterized by a sudden drop in blood pressure when moving from a lying down to a standing position.

Additionally, venous blood tends to pool in the legs due to the lack of muscle contractions that normally aid in pumping blood back to the heart. As a compensatory mechanism, beta-adrenergic sympathetic activity is increased, which is part of the body's fight-or-flight response aimed at maintaining cardiovascular function.

Decreased Cardiovascular Efficiency Cardiovascular efficiency is markedly reduced during periods of immobilization. Patients often develop an increased resting heart rate (HR) and a decreased stroke volume (SV), which is the amount of blood ejected by the heart with each beat. On average, the heart rate increases by approximately 0.5 beats per minute each day of immobilization, eventually leading to tachycardia (abnormally high heart rate) even with minimal or submaximal physical exertion.

The stroke volume can increase by up to 15% within just two weeks of bed rest due to changes in blood volume and venous pooling in the lower extremities. However, this increase is not sufficient to counterbalance the overall reduction in cardiovascular efficiency.

Reduced Oxygen Extraction There is also a decrease in maximal oxygen extraction (VO2 max), which can occur as early as 3 to 5 days after immobilization begins. VO2 max is a measure of the body's ability to take in, transport, and utilize oxygen during exercise, and its reduction signifies a decline in overall cardiovascular and muscular efficiency.

Postural Changes and Orthostatic Intolerance When an immobilized person transitions from lying down to sitting or standing, they may experience symptoms such as dizziness (reeling of the head) or even fainting. This is due to the body's inability to quickly adjust to the change in posture, leading to orthostatic intolerance.

In healthy, mobile individuals, a rapid drop in blood pressure upon standing is immediately detected by baroreceptors located in the aortic arch and carotid sinus. These receptors send signals to the cardiac center in the brain, which responds by increasing sympathetic stimulation of the heart. This increases cardiac output and raises blood pressure. Simultaneously, the vasomotor center increases sympathetic stimulation of the blood vessels in

the lower limbs, causing partial vasoconstriction and reducing the downward movement of blood. These responses help maintain blood pressure and ensure adequate circulation to the brain.

Summary Immobilization has profound effects on the cardiovascular system, including reduced blood and plasma volumes, venous pooling, and decreased cardiovascular efficiency. These changes lead to increased resting heart rate, decreased stroke volume, and reduced oxygen extraction capacity. Upon resuming activity, patients may experience orthostatic intolerance, characterized by dizziness and fainting. In contrast, healthy individuals have rapid compensatory mechanisms to maintain blood pressure and cerebral circulation when changing posture.

Impeded Mechanisms in Bedridden Patients In bedridden patients, the body's normal compensatory mechanisms for maintaining blood pressure are significantly impaired due to reduced blood volume. This reduction can lead to a more pronounced drop in blood pressure when the patient attempts to stand, a condition known as postural hypotension. The effectiveness of the baroreceptor reflex, which normally helps regulate blood pressure, is blunted. This blunting occurs because the reduced blood volume provides less of a stretch stimulus to the stretch receptors in the blood vessels, causing these receptors to become progressively less sensitive over time.

Reduced venous return and stroke volume

In addition to the reduced blood volume, bedridden patients experience decreased venous return and stroke volume. Venous return refers to the amount of blood that returns to the heart from the body, and a reduction in this volume decreases the amount of blood the heart can pump out with each beat (stroke volume). Prolonged immobility leads to cardiac deconditioning, where the heart muscle weakens and the myocardial walls thin, further limiting the heart's pumping efficiency.

Postural hypotension

This combination of factors—reduced blood volume, decreased venous return, and cardiac deconditioning—contributes to the development of postural hypotension. This condition can occur after as little as 20 hours of bed rest. When a bedridden patient attempts to stand, they may experience significant dizziness, light-headedness, or even fainting due to the sudden drop in blood pressure.

Venous Stasis and Deep Vein Thrombosis (DVT) Venous stasis, or the pooling of blood in the veins, is a common issue in bedridden patients and predisposes them to deep vein thrombosis (DVT). In DVT, platelets and other clotting factors fall from the mainstream of blood flow toward the periphery, where they adhere to the intimal layer of the venous wall. This adhesion initiates the formation of an intravenous clot, or thrombosis. Thrombosis typically develops silently, without obvious symptoms, and can obstruct venous return from the area distal to the clot. The most common site for DVT is the saphenous vein in the calf muscles.

Embolism and its consequences

If a thrombus (blood clot) dislodges from its original site and circulates through the bloodstream, it is known as an embolism. An embolism can travel to various parts of the body, causing serious and potentially life-threatening complications. For instance, a pulmonary embolism occurs when a clot lodges in the arteries of the lungs, obstructing blood flow and leading to severe respiratory issues or even death. Similarly, a cerebral embolism occurs when a clot blocks blood flow to the brain, potentially resulting in a cerebrovascular accident (CVA), commonly known as a stroke. This can cause focal neurological deficits, such as hemiplegia, which is paralysis on one side of the body. Bedridden patients are at a high risk of cardiovascular complications due to impaired compensatory mechanisms caused by reduced blood volume, decreased venous return, and cardiac deconditioning. These issues can lead to postural hypotension, deep vein thrombosis, and potentially life-threatening embolisms. Effective management and preventive measures are crucial in minimizing these risks and maintaining cardiovascular health in immobilized patients.

Pulmonary effects of immobilization

Decreased Ventilation In immobilized patients, there is a notable decrease in overall ventilation, characterized by reduced tidal volume (the amount of air moved into or out of the lungs during a normal breath) and minute ventilation volume (the total volume of air entering the lungs per minute). This reduction in airflow results from the decreased activity and strength of the diaphragm and intercostal muscles, which are crucial for effective breathing.

Diminished Diaphragmatic Movement and Chest Expansion The lack of movement and prolonged immobility lead to diminished diaphragmatic movement and reduced chest expansion. The diaphragm, the primary muscle responsible for breathing, and the intercostal muscles, which assist with expanding and contracting the chest cavity, both lose strength over time. This weakness hampers their ability to adequately inflate and deflate the lungs. Consequently, the patient experiences impaired secretion clearance, which is the body's ability to remove mucus and other secretions from the airways. This impaired clearance further reduces effective ventilation.

Reduced Cough and Bronchial Ciliary Activity In immobilized patients, the ability to cough effectively is compromised. Coughing is a vital reflex that helps clear mucus and foreign particles from the respiratory tract. Alongside this, bronchial ciliary activity, which involves tiny hair-like structures that sweep mucus and debris out of the lungs, is also diminished. The combined reduction in these defensive mechanisms can lead to the accumulation of secretions in the lungs, creating an environment conducive to infections. One common and serious infection that can develop in this context is hypostatic pneumonia, which results from the pooling of secretions and reduced lung ventilation.

Decreased Vital Capacity Vital capacity, the maximum amount of air a person can expel from the lungs after a maximum inhalation, decreases significantly in immobilized patients. This decline is indicative of reduced lung function and respiratory efficiency, further complicating the patient's ability to maintain adequate oxygenation and ventilation.

A-V Shunting and Ventilation-Perfusion Mismatch A-V (arteriovenous) shunting and regional changes in ventilation-perfusion ratios are additional pulmonary complications. A-V shunting refers to blood bypassing the normal lung tissue and thus not being properly oxygenated. This can occur due to regions of the lungs being poorly ventilated but still perfused with blood. The mismatch between ventilation (airflow) and perfusion (blood flow) leads to inefficient gas exchange and can contribute to hypoxemia (low blood oxygen levels).

Effects of the Supine Position When a patient remains in the supine position (lying on their back) for extended periods, both forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV1) are reduced. FVC measures the total amount of air exhaled during a forced breath, while FEV1 measures how much air can be forcibly exhaled in one second. These reductions are due to several factors, including airway obstruction potentially caused by mucus pooling, increased resistance in the airways, and a loss of elastic recoil within the lung tissues. Structural changes in the lungs from prolonged immobility, such as the stiffening of lung tissues, contribute to this loss of elastic recoil and further compromise respiratory function.

Immobility has profound negative effects on pulmonary function. Decreased tidal volume and minute ventilation volume, diminished diaphragmatic movement, and reduced chest expansion lead to impaired secretion clearance and ventilation. The reduction in effective cough and bronchial ciliary activity increases the risk of respiratory infections such as hypostatic pneumonia. Decreased vital capacity, A-V shunting, and ventilation-perfusion mismatches further compromise respiratory efficiency.

The supine position exacerbates these issues by reducing FVC and FEV1 due to airway obstruction, increased resistance, and structural changes in the lungs. These factors collectively contribute to significant respiratory complications in immobilized patients, necessitating careful management and preventive strategies to maintain pulmonary health.

Endocrine complications of immobilization

Basal Metabolic Rate (BMR): During prolonged periods of immobilization, the basal metabolic rate (BMR)—the rate at which the body uses energy while at rest to maintain vital functions—decreases significantly. This reduction reflects the body has lowered energy requirements due to decreased physical activity and muscle mass.

Hormonal Changes Immobilized patients often exhibit depressed levels of aldosterone and antidiuretic hormone (ADH). Aldosterone is responsible for regulating sodium and potassium balance, while ADH controls water retention in the kidneys. The reduction in these hormone levels leads to increased diuresis (urine production), resulting in a decrease in both blood volume and plasma volume. This reduction contributes to the overall decline in cardiovascular efficiency and can exacerbate postural hypotension.

Negative Nitrogen Balance Inactivity leads to a negative nitrogen balance in the body, which means that nitrogen excretion exceeds nitrogen intake and synthesis. This imbalance is a result of increased protein catabolism (breakdown) and decreased protein synthesis.

On average, nitrogen loss through urine can reach up to 2 grams per day, peaking during the second week of immobilization. This loss is indicative of muscle wasting and a general decline in bodily protein stores. Furthermore, urinary stasis—a condition where urine remains stagnant in the bladder—can increase the risk of urolithiasis (kidney stones) and urinary tract infections.

Glucose Intolerance Glucose intolerance is a common complication in immobilized patients. When subjected to a glucose tolerance test, these individuals often exhibit hyperglycaemic (elevated blood sugar) and hyperinsulinemic (elevated insulin levels) responses. This impaired glucose metabolism is indicative of reduced insulin sensitivity and an increased risk of developing insulin resistance or type 2 diabetes over time.

Hypercalcemia and Calcium Loss Immobilization also leads to significant calcium loss from the bones, a condition known as hypercalcemia. The maximum loss of calcium typically occurs during the fourth and fifth weeks of immobility. This prolonged loss contributes to osteoporosis, a condition characterized by weakened bones and an increased risk of fractures. The rate of vertebral mineral loss can reach approximately 1% per week. After 12 weeks of bed rest, bone density can be reduced by about 50%, with long bones being more affected than

other bones. This drastic reduction in bone mass underscores the critical need for early intervention and mobility exercises to mitigate these effects.

Gastrointestinal Complications Immobilization has several adverse effects on the gastrointestinal system. Decreased gastrointestinal motility leads to constipation and a loss of appetite, further contributing to malnutrition and overall physical decline. Additionally, immobilized patients are at risk for skin atrophy and the development of pressure ulcers. These ulcers, also known as bedsores, result from prolonged pressure on the skin, particularly over bony areas, and can lead to severe infections if not properly managed.

Immobilization induces numerous endocrine and metabolic complications, including a decreased basal metabolic rate, altered hormone levels leading to reduced blood and plasma volumes, and a negative nitrogen balance due to increased protein catabolism and decreased synthesis. Patients frequently experience glucose intolerance and hypercalcaemic conditions, contributing to significant bone density loss and osteoporosis.

Gastrointestinal issues such as decreased motility, constipation, and pressure ulcers further exacerbate the patient's condition. Comprehensive care strategies focusing on early mobilization, nutritional support, and meticulous skin care are essential to mitigate these adverse effects and promote overall health in immobilized patients.

CONCLUSION

This study concluded with the information about the impact of prolonged bed rest on multiple physiological systems, emphasizing the severe consequences on muscle integrity, cardiovascular and respiratory functions, endocrine balance, and overall rehabilitation outcomes. These information can help other researchers and readers to get ready hand data about bed rest complications.

ACKNOWLEDGEMENTS

All authors extend their gratitude to all the persons helped in this manuscript preparation.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1. Convertino, V. A. Cardiovascular consequences of bed rest: effect on maximal oxygen uptake. Medicine & Science in Sports & Exercise, 1997; 29(2), 191-196.
2. Hoppeler, H., Vogt, M. Muscle tissue adaptations to hypoxia. Journal of Experimental Biology, 2001; 204(18), 3133-3139.
3. LeBlanc, A. D., Schneider, V. S., Evans, H. J., Engelbretson, D. A., & Krebs, J. M. Bone mineral loss and recovery after 17 weeks of bed rest. Journal of Bone and Mineral Research, 1990; 5(8), 843-850.
4. Paddon-Jones, D., Rasmussen, B.B. Dietary protein recommendations and the prevention of sarcopenia. Current Opinion in Clinical Nutrition & Metabolic Care, 2009; 12(1), 86-90.
5. Gogia, P. P., Schneider, V. S., LeBlanc, A. D., Krebs, J. M., Kasson, C., & Pientok, C. Bed rest effect on extremity muscle torque in healthy men. Archives of Physical Medicine and Rehabilitation, 1988; 69(12), 1030-1032.
6. Trudel, G., & Uhthoff, H. K. Contractures secondary to immobility: is the restriction articular or muscular? An experimental longitudinal study in the rat knee. Archives of Physical Medicine and Rehabilitation, 2005; 86(3), 368-373.
7. Beckman, S. M., & Buchanan, T. S. Ankle inversion injury and hypermobility: effect on hip and ankle muscle electromyography onset latency. Archives of Physical Medicine and Rehabilitation, 1995; 76(12), 1138-1143.
8. Nicholas, J. A., Hershman, E. B., & Colosimo, A. J. The role of exercise in the treatment of ankle sprains. The American Journal of Sports Medicine, 1976; 4(6), 308-313.
9. Topp, R., Ditmyer, M., King, K., Doherty, K., Hornyak, J. The effect of bed rest and potential of prehabilitation on patients in the intensive care unit. AACN Advanced Critical Care, 2002; 13(2), 263-276.
10. Hermans, G., & Van den Berghe, G. Clinical review: Intensive care unit acquired weakness. Critical Care, 2015; 19, 274. doi: 10.1186/s13054-015-0993-7.
11. Bailey, P., Thomsen, G. E., Spuhler, V. J., etal., Early activity is feasible and safe in respiratory failure patients. Critical Care Medicine, 2007; 35(1), 139-145. doi: 10.1097/01.CCM.0000251130.69568.87.

12. Schweickert, W. D., Pohlman, M. C., Pohlman, etal., Early physical and occupational therapy in mechanically ventilated, critically ill patients: a randomised controlled trial. The Lancet, 2009; 373(9678), 1874-1882. doi: 10.1016/S0140-6736(09)60658-9.
13. Truong, A. D., Fan, E., Brower, R. G., & Needham, D. M. Bench-to-bedside review: Mobilizing patients in the intensive care unit--from pathophysiology to clinical trials. Critical Care, 2009;13(4), 216. doi: 10.1186/cc7885.
14. Herridge, M. S., Tansey, C. M., Matté, A., Tomlinson, G., Diaz-Granados, N., Cooper, A., & Canadian Critical Care Trials Group. Functional disability 5 years after acute respiratory distress syndrome. New England Journal of Medicine, 2011; 364(14), 1293-1304. doi: 10.1056/NEJMoa1011802.
15. Needham, D. M., Davidson, J., Cohen, H., Hopkins, R. O., Weinert, C., Wunsch, H., Harvey, M. A. Improving long-term outcomes after discharge from intensive care unit: Report from a stakeholders' conference. Critical Care Medicine, 2012; 40(2), 502-509. doi: 10.1097/CCM.0b013e318232da75.
16. Hodgson, C. L., Berney, S., Harrold, M., Saxena, M., Bellomo, R., & Davidson, B. Clinical review: Early patient mobilization in the ICU. Critical Care, 2013; 17, 207. doi: 10.1186/cc11820.
17. Thomas, P., Baldwin, C., Bissett, B., Boden, et al. Physiotherapy management for COVID-19 in the acute hospital setting: clinical practice recommendations. Journal of Physiotherapy, 2020; 66(2), 73-82. doi: 10.1016/j.jphys.2020.03.011.
18. Winkelman, C. Inactivity and inflammation in the critically ill patient. Critical Care Clinics, 2009; 25(3), 439-458. doi: 10.1016/j.ccc.2009.04.001.