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Abstract
Bed rest often is used to treat a wide variety of medical conditions. However, bed rest results in profound deconditioning of the body. Bed rest reduces the hydrostatic pressure gradient within the cardiovascular system, reduces muscle force production, virtually eliminates compression on the bones, and lowers total energy expenditure. This review focuses on the deconditioning that occurs in the cardiovascular, muscular, and skeletal systems following bed rest. Reduction in plasma volume reduces cardiac preload, stroke volume, cardiac output, and ultimately, maximal oxygen consumption. Skeletal muscle volume, muscle cross sectional area, and fiber cross sectional area decrease, which results in diminished muscular strength. These changes are most pronounced in the antigravity muscles. Increased bone resorption leads to a negative calcium balance and eventually decreased bone mass, particularly in the lower limbs. Diminished bone mass coupled with decreased muscular strength increases the risk of bone fractures, even with minor falls. It is important for clinicians to recognize these negative consequences of bed rest, which can be explained independent of disease or disorder. With this in mind, bed rest can be minimized as much as possible and early ambulation and physical activity may be prescribed to limit the deconditioning effects of bed rest.

Keywords
Deconditioning, Inactivity, Disuse, Bedrest

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Review

THE PHYSIOLOGICAL CONSEQUENCES OF BED REST

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ABSTRACT

Stuempfle, KJ, Drury, DG. The Physiological Consequences of Bed Rest. ***JEP****online* 2007;10(3):32-41. Bed rest often is used to treat a wide variety of medical conditions. However, bed rest results in profound deconditioning of the body. Bed rest reduces the hydrostatic pressure gradient within the cardiovascular system, reduces muscle force production, virtually eliminates compression on the bones, and lowers total energy expenditure. This review focuses on the deconditioning that occurs in the cardiovascular, muscular, and skeletal systems following bed rest. Reduction in plasma volume reduces cardiac preload, stroke volume, cardiac output, and ultimately, maximal oxygen consumption. Skeletal muscle volume, muscle cross sectional area, and fiber cross sectional area decrease, which results in diminished muscular strength. These changes are most pronounced in the antigravity muscles. Increased bone resorption leads to a negative calcium balance and eventually decreased bone mass, particularly in the lower limbs. Diminished bone mass coupled with decreased muscular strength increases the risk of bone fractures, even with minor falls. It is important for clinicians to recognize these negative consequences of bed rest, which can be explained independent of disease or disorder. With this in mind, bed rest can be minimized as much as possible and early ambulation and physical activity may be prescribed to limit the deconditioning effects of bed rest.

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TABLE OF CONTENTS

INTRODUCTION

Bed rest is a long standing treatment for managing acute and chronic injury and illness. It may have started with Hippocrates, the father of medicine, who suggested that "In every movement of the body, whenever one begins to endure pain, it will be relieved by rest" (1). Bed rest was emphasized in the 19th century as the primary treatment for many disorders (2). However, in the 20th century, physicians and scientists became increasingly aware of the harmful effects of prolonged bed rest (3). Classic bed rest studies following World War II documented the deconditioning that occurs following bed rest (3). The beginning of manned space flight in the 1960's increased the number of bed rest studies, further revealing the detrimental physiological effects of inactivity (3). Allen et al. conducted an exhaustive search of the medical literature from 1966 to 1998, which provided additional evidence for the harm of bed rest for any medical condition (4). In 15 trials that investigated bed red rest as a primary treatment for a variety of conditions, no outcomes improved significantly and nine worsened significantly (including acute low back pain, labor, proteinuric hypertension during pregnancy, myocardial infarction, and acute infectious hepatitis) (4). In 24 trials that investigated bed rest after a medical procedure, no outcomes improved significantly, and eight worsened significantly (including lumbar puncture, spinal anesthesia, radiculography, and cardiac catheterization) (4).

Confinement to bed causes a reduced hydrostatic pressure gradient within the cardiovascular system, unloading of forces on skeletal muscles and bones, and reduced total energy expenditure. The resultant physiological adaptations negatively affect most organ systems of the body (3). This paper focuses on the effects of bed rest on the cardiovascular, muscular, and skeletal systems, the organ systems that exhibit the most pronounced deconditioning.

EFFECTS OF BED REST ON THE CARDIOVASCULAR SYSTEM

The cardiovascular system functions optimally while counteracting gravity in an upright position (5). A coordinated interaction between the cardiovascular and nervous systems ensures adequate blood perfusion to the brain and other organs. When the body assumes a horizontal position for an extended period of time during bed rest, deconditioning of the cardiovascular system occurs (5).

Maximal oxygen consumption ($\dot{V}O_2max$) commonly is used to assess cardiovascular function in both health and disease. Bed rest decreases $\dot{V}O_2max$, and the extent of the loss depends on the length of the bed rest, with $\dot{V}O_2max$ decreasing approximately 0.9% per day over 30 days of bed rest (6). The decrease in $\dot{V}O_2max$ during bed rest appears to be independent of gender and age (7-9). However, more fit individuals may experience a greater absolute decrease in $\dot{V}O_2max$ compared to less fit individuals (7,8,10,11).

The decrease in $\dot{V}O_2max$ following bed rest can be attributed to both cardiac and peripheral effects, although cardiac effects predominate (Figure 1).

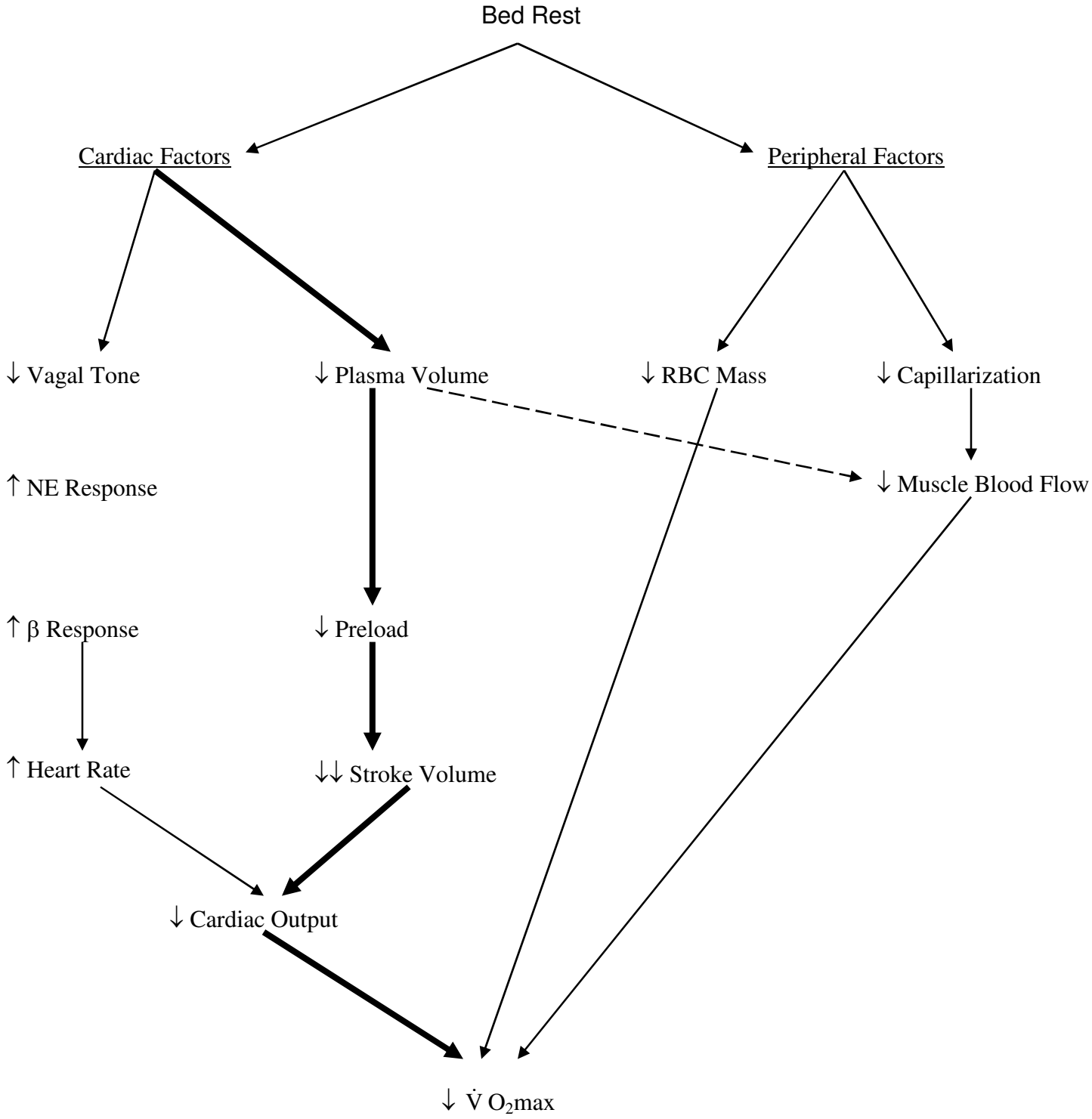

Figure 1. Cardiovascular mechanisms affecting $\dot{V}O_2max$ following bed rest. (Abbreviations: NE, norepinephrine; RBC, red blood cells). Figure modified from Convertino (6).

A 26% decrease in $\dot{V}O_2$max in five men after 20 days of bed rest was accompanied by a similar 26% decline in cardiac output (11). Similarly, a 17% decrease in $\dot{V}O_2$max following 10 days of bed rest in 12 men resulted from a 23% reduction in cardiac output (6). A change in heart rate is not responsible for the decreased cardiac output following bed rest. In fact, both resting and maximum heart rate have been observed to increase following bed rest (6). The increase in resting heart rate may be due to a decrease in vagal tone (12), and the increase in maximum heart rate may be caused by an increased release of norepinephrine and an increased sensitivity of cardiac β adrenergic receptors (6). The primary cause of decreased cardiac output and $\dot{V}O_2$max following bed rest is a reduction in stroke volume (6). The reduction in stroke volume is not caused by a change in contractility. In fact, contractility and ejection fraction appear to increase following bed rest due to increased sensitivity of cardiac β adrenergic receptors (13). Instead, the primary mechanism for the reduction in stroke volume following bed rest is decreased preload due to a reduction in plasma volume (6). Rapid diuresis occurs within the initial 24-48 h of bed rest, resulting in a 10-20% reduction in plasma volume (14). Additionally, venous compliance increases by 20-25% with bed rest, which results in venous pooling in the lower extremities when an upright posture is resumed and a reduction in stroke volume (15,16).

Although decreased stroke volume and cardiac output are the primary causes of the diminished $\dot{V}O_2$max following bed rest, peripheral factors may also contribute (Figure 1). Prolonged bed rest resulted in a 9% decrease in red blood cell mass, compromising the oxygen-carrying capacity of the blood, and perhaps contributing to the reduced $\dot{V}O_2$max (17). Furthermore, decreased capillarization and muscle blood flow following bed rest also may diminish $\dot{V}O_2$max (16).

In addition to reduced $\dot{V}O_2$max, bed rest results in additional complications with the cardiovascular system, including alterations in orthostatic tolerance, and increased frequency of venous thrombi. Orthostatic hypotension occurs following bed rest, and may be caused by a decrease in plasma volume (14,18,19). Venous thromboembolism can be a serious complication of bed rest (20). Patients with venous thrombi have a 50% chance of developing pulmonary emboli (21), and the mortality from untreated pulmonary embolism is 20-35% (22). Bed rest duration is directly related to the frequency of venous thrombosis (20).

EFFECTS OF BED REST ON THE MUSCULAR SYSTEM

Like the cardiovascular system, the muscular system functions optimally when supporting the body in an upright position against gravity. The antigravity muscles of the neck, lower back, abdomen, buttocks, thighs, and calves are especially important to the maintenance of an upright posture (5). Bed rest results in the disuse of these muscles, which leads to deterioration in muscle structure and function (5).

The predominate response of skeletal muscle to decreased use during bed rest is atrophy. Atrophy progressively increases with the duration of bed rest (23), as reported by changes in muscle volume, muscle cross sectional area and fiber cross sectional area during and following bed rest (Figure 2).

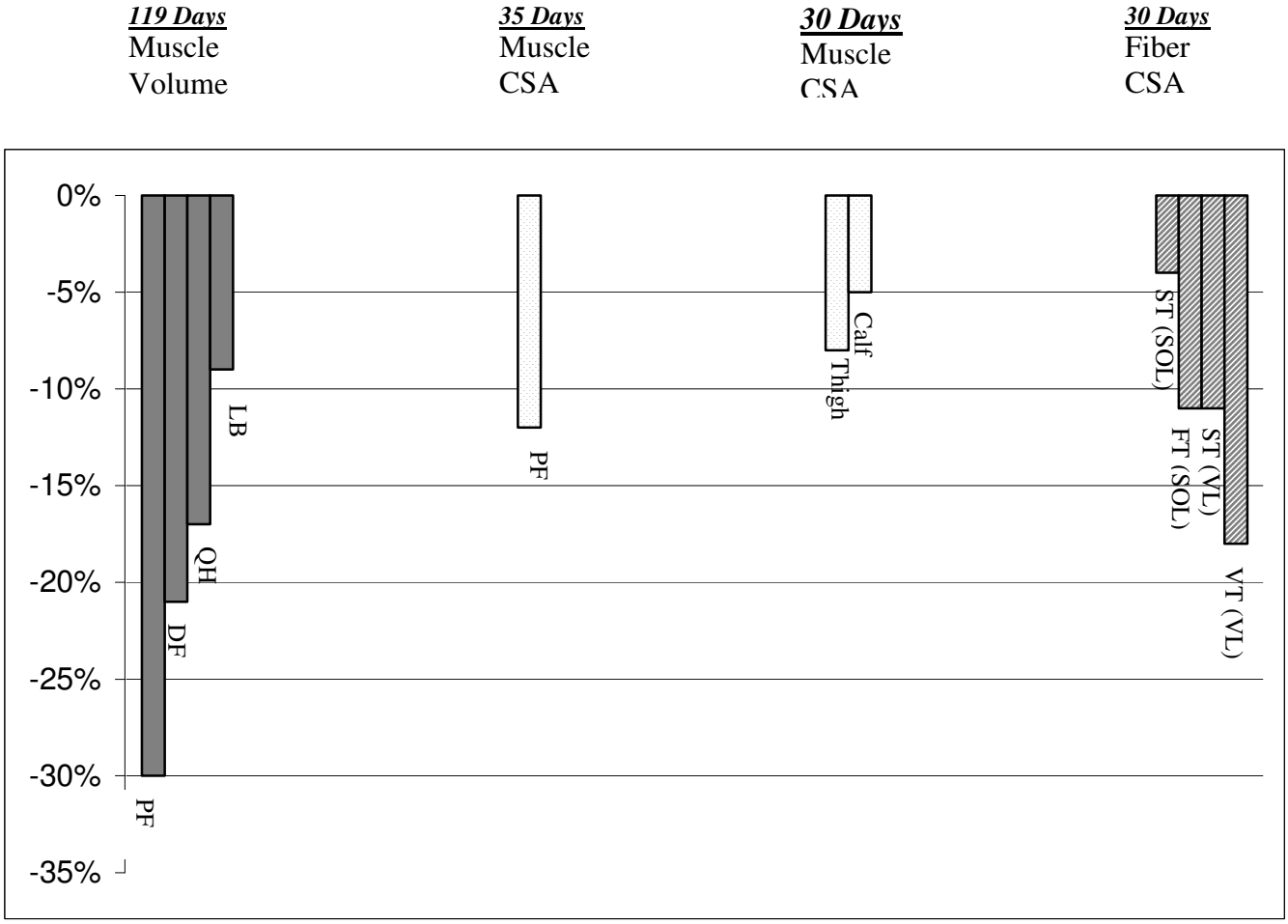

Figure 2. Changes in measures of muscle mass following bed rest. (Abbreviations: CSA, cross-sectional area; PF, plantar flexors; DF, dorsi flexors; QH, quadriceps and hamstrings; LB, lower back; ST, slow twitch; SOL, soleus; FT, fast twitch; VL, vastus lateralis). 119 day data from LeBlanc (24), 35 day data from LeBlanc (25), 30 day muscle cross sectional area data from Convertino (16), and 30 day fiber cross sectional area from Hikida (26).

Following 119 days of bed rest, muscle volume decreased in the ankle plantar flexors (-30%), ankle dorsi flexors (-21%), quadriceps and hamstrings (-16 to –18%), and intrinsic lower back muscles (-9%) (24). Muscle cross sectional area of the ankle plantar flexors decreased by 12% after 35 days of horizontal bed rest (25). Similar atrophy occurred at the thigh (-8%) and calf (-5%) after 30 days of head-down tilt bed rest (16). Muscle biopsy data after 30 days of head-down tilt bed rest revealed an 8% decrease in slow twitch fiber cross sectional area, and a 15% decrease in fast twitch fiber cross sectional area (26). Negative nitrogen balance, indicative of an imbalance between protein synthesis and degradation of skeletal muscle, is an early marker for the muscle atrophy that occurs with bed rest (23). Urinary nitrogen excretion increases significantly by the fifth day of bed rest (23), and peaks during the second week of bed rest at 21-40% above baseline (27).

Besides atrophy, bed rest results in additional changes to skeletal muscle. Muscle biopsies of the vastus lateralis and soleus following 30 days of bed rest revealed a number of ultrastructural changes, including Z-line streaming, myofibril disorganization, cellular edema, and mitochondria in the extracellular space, suggesting damage to the sarcolemma (26). Deficits in oxygen delivery and utilization also occur following bed rest. Thirty days of bed rest resulted in a 38% decrease in

maximum blood flow to the calf (16) and diminished activity of skeletal muscle oxidative enzymes in both the vastus lateralis and soleus (26).

As would be expected, the decrease in muscle mass following bed rest is accompanied by a decrease in muscle strength (Table 1).

Table 1. Change in maximal muscle strength following bed rest.

Number of Days	Muscle Group	% Change	Reference
30	Knee flexors	-6	Dudley (28)
	Knee extensors	-19	
35	Plantar flexors	-26	LeBlanc (25)
35	Plantar flexors	-25	Gogia (29)
	Dorsi flexors	-8	
	Knee flexors	-8	
	Knee extensors	-19	
	Elbow flexors	-7	

The greatest decreases in strength are in the antigravity muscles (28,29). Maximal strength of the knee flexors (-6%) and knee extensors (-19%) decreased following 30 days of bed rest (28). Thirty-five days of bed rest caused decrements in maximal strength of the ankle plantar flexors (-25%), ankle dorsi flexors (-8%), knee flexors (-8%), knee extensors (-19%), and elbow flexors (-7%) (29). Similarly, maximal strength decreases of 26% were observed in the ankle plantar flexors after 35 days of bed rest. (25).

Some of the decrement in muscle strength following bed rest may be the result of a reduction in muscle electrical efficiency (23). After seven days of space flight, there is an increased ratio of EMG activity to unit force production in the ankle extensors, suggesting an increase in the amount of neural activity required to elicit the same muscular force output following disuse. This altered electrical efficiency may be caused by changes in motor unit recruitment (23).

The effects of decreased muscle strength and neuromuscular changes on posture, balance, and gait may be a significant concern. In the first few days after returning from space, astronauts exhibit increased postural sway, gait changes, and impaired kinesthetic sense (30,31). These factors also contribute to an increased risk of falls in the elderly (23).

EFFECTS OF BED REST ON THE SKELETAL SYSTEM

Similar to the cardiovascular and muscular systems, the skeletal system also functions optimally when exposed to gravity. Bone integrity is maintained by the mechanical loads imposed by weight bearing in an upright position and the contraction of skeletal muscle. Maintaining normal bone mass requires a balance between the formation of new bone by osteoblasts and the resorption of old bone by osteoclasts. Normally, the rates of these two events are equal, and bone mass remains constant. However, the removal of normal weight bearing activity during bed rest disrupts this balance, and resorption is favored, resulting initially in an alteration in calcium balance, and later in bone loss (5).

Increased resorption transiently increases serum calcium, resulting in increased urinary excretion of calcium. Hypercalciuria is routinely observed in the first week of bed rest , and urine calcium peaks at 60% above normal values between the fifth and seventh weeks of bed rest (23). Fecal calcium also increases during the first week of bed rest, contributing to the negative calcium balance (23). Fecal

calcium increases due to a reduction in intestinal calcium absorption (23). Calcium absorption decreases from 31% to 24% of dietary intake over 17 weeks of bed rest (32).

Loss of bone calcium during bed rest is the result of increased bone resorption by osteoclasts and not endocrine changes. Parathyroid hormone (PTH) promotes the release of calcium from bone, and stimulates the kidneys to release the active form of Vitamin D, 1,25-dihydroxyvitamin D, which increases intestinal calcium absorption (33). PTH decreases (34-36) or does not change (32) during bed rest, and 1,25-dihydroxyvitamin D decreases (32,34-36).

The negative calcium balance caused by bed rest eventually results in decreased bone mass (Figure 3).

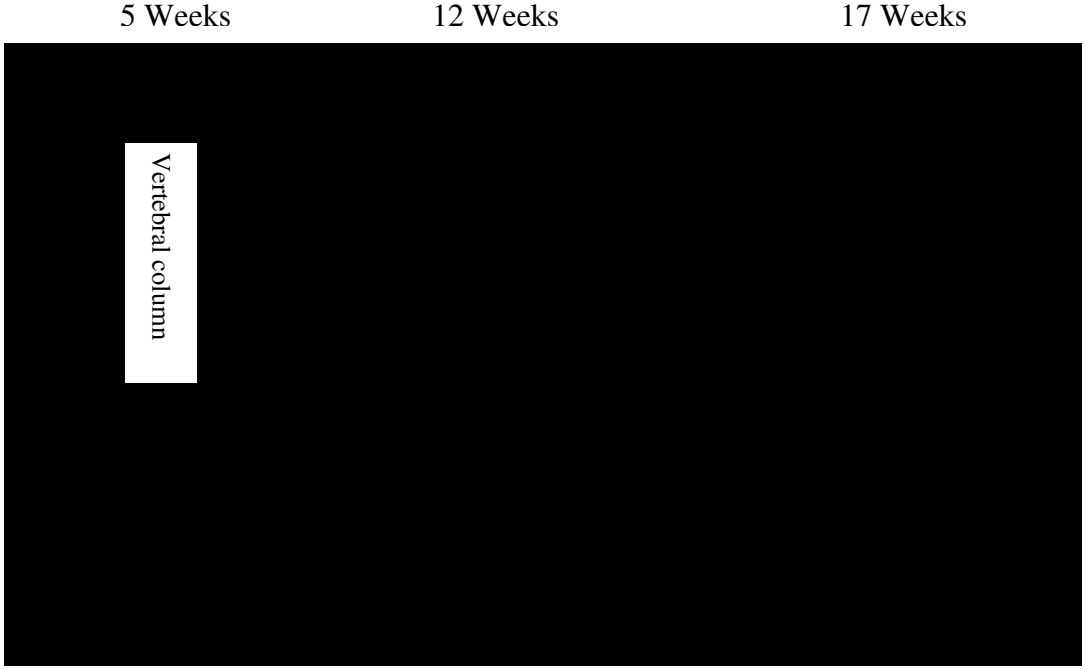

Figure 3. Change in bone density at various sites observed following bed rest. Five week data from LeBlanc (37), 12 week data from Zerwekh (34), and 17 weeks from LeBlanc (38).

The bones of the lower limbs are the most susceptible to decreased bone mass (23). Bone mass in the vertebral column decreased 0.9% following five weeks of bed rest (37). Greater trochanter bone mass decreased 4% and spine bone mass decreased 3% after 12 weeks of bed rest (34). Seventeen weeks of bed rest resulted in significant losses in bone mass of the calcaneus (-10%), greater trochanter (-5%), femoral neck (-4%), lumbar spine (-4%), and tibia (-2%), whereas no significant change occurred in the radius or ulna (38).

Changes in biochemical markers of bone resorption and formation can be used to monitor changes in the skeleton following bed rest. Bone resorption markers such as pyridinoline, deoxypyridinoline, and N-telopeptide increased following bed rest (32,34,39,40). In contrast, no change in bone formation markers (alkaline phosphatase, serum osteocalcin, Type I procollagen propeptide) occurred after bed rest (34,40).

Histomorphometric analysis of bone from biopsy samples is the only direct way to determine whether changes in bone mass following bed rest result from increased resorption of old bone or decreased

formation of new bone. Iliac crest biopsies after 120 days of bed rest revealed a significant increase in bone resorption surface, indicating increased activity of osteoclasts (41). In contrast, no significant change in formation of nonmineralized bone matrix occurred, although subsequent mineralization of the newly formed bone matrix appeared to be impaired (41).

Change in bone mass may not have an immediate impact on an individual's functional capacity following bed rest, as do changes in the cardiovascular and muscular systems. However, the individual does have an increased risk of bone fracture (23). Each bony site appears to have a specific bone mass that constitutes a "fracture threshold". When bone mass falls below this threshold, the bone is very susceptible to fracture, even with minimal trauma (42). Decreases in bone mass following bed rest, coupled with decreases in muscle strength and possible changes in balance and gait (43), significantly increase the risk of bone fractures with even minor falls (23).

SUMMARY

From the available research, it is clear that prolonged bed rest has adverse physiological effects on the cardiovascular, muscular, and skeletal systems. This bed rest deconditioning can be explained independent of disease or disorder. Many of the negative effects begin within days of confinement, but their consequences can last much longer. It is important for clinicians to recognize these deleterious effects, and to limit bed rest as much as possible. Furthermore, it is important to realize that early ambulation and physical activity may help to limit the deconditioning effects of bed rest.

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