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# Bed Rest in Older Adults: "A Catabolic Crisis" for Muscle Mass and Function

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Significant reduction in muscle mass, control and function among elderly population occurs during extended bed rest inactivity periods, as observed by numerous studies. The adult geriatric population is more susceptible to lean muscle mass loss (Sarcopenia) as compared to the young. Many intertwined factors play a role in such muscle degeneration among the elderly. Physical inactivity coupled with meager nutrition as is inevitable in bed rest morbidity cases, heighten and accelerate the progression of sarcopenia. During post middle age, adults on an average lose muscle mass at the rate of 5 percent in each decade of their life span. Lean muscle tissue loss impairs the locomotor skills, contractile power, muscular coordination and efficacy. They eventually lead to worsened quality of life in the form of hampered daily activities, movement hindrance, loss of flexibility and agility and increased proneness to accidents and falls. Eventually in older adults, it leads to increased incidence of readmission and frequent institutionalization. With changed life styles of the 21st century world, preponderance of white collar jobs, decreased outdoor pursuits and a shift in the diet paradigms from traditional to modern; sarcopenia is highly prevalent in developed and developing countries with a male preponderance. This trend is interesting as females would normally be considered ideal candidates for sarcopenia owing to their inherently low myogenetic androgens. This can perhaps be attributed to the inherent homeostatic resilience of the female gender as compared to the males and has been encoded in their blueprint down the years of evolution. Prolonged inactivity as is common in hospitalized patients in chronic morbid disease states, is a very strong and inevitable indicator for subsequent sarcopenia and much greater likelihood of disability. Therefore, commanding patients to uninterrupted bed rest might carry much greater risk than benefit unless there is a solid medical condition indicating absolute inactivity in the form of bed rest. Thus, in the elderly subjects; even a transient mandatory bed rest period must be a very well reflected and deliberated practice by the attending physicians

as it could initiate a fast downhill and often irreversible process in terms of muscular function.

Accelerated muscle loss during bed rest appears to be driven primarily by a reduction in muscle protein synthesis; stemming from augmented muscle protein breakdown among the elderly (while it remains 'unaltered 'among the younger population), "anabolic resistance" or a blunted protein synthetic response to mixed nutrient meals leading to a sluggish amino acid transport and a decrease in post-absorptive muscle protein synthesis coupled with an anabolic resistance to feeding. Increased sympathetic mediated stress response secondary to physical crisis like injury or co-morbidity also augments degeneration of lean muscle tissue by inflammatory mediators.

For the patient, the effects of functional incapacity and performance reduction is more consequential in terms of life quality than the morphologic changes in lean muscle mass parameters. Key measures of functional capacity such as floor transfer time, stair ascent power and standing plantar flexion characteristically decrease over the initial weeks of bed rest. Meager outcomes post 'bed rest' among older adults as compared to their younger counterparts is an inevitable physiological process owing to the 'lesser lean muscle tissue' at their start off point. Also, the compensatory myogenesis among the geriatric population subjected to bed rest is much slower than the young.

Walking, reaching out for daily living practices are essential constituents of individual and personal freedom. Researchers have found a strongly positive correlation between 'bed restduration' and the motor function decline and social activity.

Resistance training and gradual onset weight bearing exercises cannot totally prevent; but definitely diminish muscle loss and hence enhance functional stability. Myogenesis can also be triggered by pharmacologic interventions such as specific protein and amino acid intake. Integrated regimes encompassing 'nutritional balance' with 'standardized exercise routines' under trained physiotherapists yield better results in terms of restoring motor function than either approach when adopted in isolation. Literally stating; patient compliance here is a pivotal variable that might "make or break the muscle" in terms of suggest increase in skeletal muscle cross-sectional area, Type II muscle fiber hypertrophy, increased percentage of Type IIa fiber area and isokinetic knee extensor strength.

Hence, it is the eminent duty of physicians to ensure enhanced cooperation of their patients by clearly defining to them the benefits of combined therapy incorporating increased lean protein in diet coupled with low intensity, short and frequent duration exercise like weight bearing and walking or gradual jogging in bouts and at moderate speed. This approach can be used as an intermittent therapeutic regimen in 'bed rest' conferred patients where the consistency of hospi-inactivity should be interrupted by guarded exercise interventions in order to protect skeletal muscle mass and function during bed rest.

The key tip off point that marks an onset of the downhill process in terms of muscular loss varies from person to person and hence, the intensity threshold of resistance training needed yet remains unclear and cannot be objectively outlined in therapy regimes. High intensity workouts may be medically contraindicated or simply not possible in severely ill patients. The ideal evidence based practice would be to consistently monitor locomotor outcomes and go backwards to modulate the intervention based on outcomes [1-5].

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