Osteoporosis refers to bones that lose minerals more quickly than they are replaced[1]. Over time, the structure of the bone becomes impaired and leads to bone weakening. Therefore, "normal" loading or loading that would have been tolerated prior to this condition, ranging from falls to loaded spinal flexion, may cause adverse events such as fractures.

Osteoporosis can be prevented or slowed through multiple avenues, including exercise. Physical activity and weight-bearing exercises positively improve bone health by creating a mechanical stimulus that causes adaptations at the cellular level that results in changes to the shape, mass, and strength of the bone[2]. However, patients with spinal cord injuries have limited to no ability to activate the muscles below the level of their injury, resulting in significant declines in weight-bearing activity especially in the lower extremities. Therefore, due to the nature of spinal cord injuries, these patients experience "rapid, severe osteoporosis with increased fracture risk" due to the "increased osteocyte expression of sclerostin, suppressed bone formation, and indirect stimulation of bone resorption.[3]"

Ashe et al. describe bone changes beginning within the first few days following injury by noting that patients experience hypercalciuria, which is indicative of excessive bone resorption, at levels 2-4 times that of a patient on prolonged bed rest without spinal cord injury[4]. Bone mass continues to decrease over the first 1-2 years as 50% of the patients living with a complete spinal cord injury experience osteoporosis one-year post-injury and more than 80% at long-term follow-up.[5] These prevalence rates are in stark contrast to that of the general population which only shows 12.6% of adults 50 and older have osteoporosis in their hip, spine, or both.[6] To further exacerbate the negative impact of osteoporosis the average age at injury of patients with spinal cord injuries is only 42 years old[7]. Therefore, this population is experiencing significantly

higher rates of osteoporosis, and resulting fracture, at much younger ages and at much greater rates.

Due to the immediate and stark changes to bone structure, young age at onset, and changes to muscle activation, prevention of bone mineral density loss becomes vital for the health and prognosis of patients with spinal cord injuries. There is evidence to suggest that some pharmacological treatments can be effective in preventing excessive bone loss[4]. The medication regimen in the first year post-injury and after the first year post injury should be different[4]. However, this is limited and variable evidence surrounding the use of activity-based physical therapy, which may include functional electrical stimulation (FES), rowing, resistance training, standing, and walking. Sutor et al. conclude that there is no intervention that uniformly attenuates bone loss or increases bone mineral density[8]. The author indicates that some studies demonstrate that FES modalities may attenuate bone loss in the acute and subacute phases at the distal femur and/or proximal tibia9. In addition, some studies also indicate that weight-bearing without FES shows no change to bone density in the distal femur or proximal tibia, but an increase in bone density in other areas of the lower extremity[8].

The limited agreement surrounding the best approach to treatment makes it difficult to maximize bone mineral density outcomes for this patient population. This difficulty is further exacerbated by the sheer amount of variability in injury type and subsequent muscle function. Therefore, based on current evidence, it appears that interventions that focus on upright weight-bearing positioning and muscle activation to muscles with paralysis or paresis are a good approach to attempt to attenuate bone mineral density loss. Unfortunately, unlike patients with osteoporosis without spinal cord injury, there appears to be a limited number of patients who will experience an increase in bone mineral density. Further research needs to be done to explore

parameters for interventions that maximize effects for bone mineral density. Therefore, the physical therapist must use their clinical judgment and principles of overload to prevent further bone loss. Education about lifestyle choices that increase rates of bone mineral density loss becomes vital as compounding negative factors could be devasting for prognosis. The physical therapist should adequately educate the patient about tobacco cessation, moderate alcohol consumption, and a diet high in vitamins and calcium[9].

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