

The Benefits of Passive Standing

Passive standing with the aid of braces, tilt table or standing frame has traditionally been recommended as part of a comprehensive therapeutic exercise program to maintain the health and physical fitness of spinal cord injured (SCI) individuals (Axelson et: al., 1987; Nyquist, 1962). During acute rehabilitation, tilting in bed or on a tilt table is initiated as soon possible to establish orthostatic tolerance for future wheelchair mobilization and, if indicated, gait training (Lopes & Figoni, 1981). In the V. A. health care system, periodic standing in a standing frame or long leg brace is frequently prescribed later for a variety of potential physiological benefits. Generally, these therapeutic claims include reduction of spasticity (Odeen & Knuttson, 1981), prevention of hypercalciuria, renal calculcsis, osteoporosis, and pathological fractures (Abramson, 1948; Abramson & Delagi, 1961; Comarr, 1955; Hattner & McMillan, 1968; Kaplan et: al., 1981), increased flexibility and prevention of contractures of hip, knee and ankle joints (Abramson & Ebel, 1953) orthostatic and cardiovascular conditioning (Lopes, Figoni & Parkash, 1984; Odeen, 1979), improved urinary drainage and renal function (Gould et: al., 1955), and pressure relieved and prevention of pressure sores on seated weight bearing areas (Abramson & Ebel, 1953). Furthermore, recent proliferation of mobile passive seating aids and stand-up wheelchairs has increased the opportunities for SCI wheelchair users to assume upright postures for more frequent and extended periods of time at work and at home.

Statement of Need:

People confined to wheelchairs as a result of degenerative neurologic conditions or trauma commonly develop spasticity, contractures and disuse osteoporosis in their legs. These morbid conditions can significantly affect the person's level of function and general well being. Spasticity may interfere with passive or active range of motion of the legs making dressing and transfers difficult, if not impossible (1). If severe enough, spasticity may cause limbs to move in a violent fashion injuring them or even causing the person to be thrown from the wheelchair. Contractures of the major joints of the paretic limbs are also commonly encountered and likely result from the fact that the joint are not actively ranged in wheelchair-bound persons (2). Functionally, contractures can interfere with dressing of the legs and feet; transfers, and standing. Lastly, osteoporosis is routinely seen in paretic legs and increases the risk of fracture (3) Minor trauma in this setting can result in fracture of a major bone(s) in osteoportic limbs (4). Treatments routinely employed for these conditions have included drugs and rehabilitative techniques (5). Spasticity has been managed with the use of antispasmodics such as baclofen, diazepam and dantrolene sodium. These drugs are often

associated with significant side effects such as drowsiness and liver toxicity. Furthermore, they are minimally effective in some people (6). Osteoporosis of the neurogenic type has not been shown to respond to bone enhancing drugs to date although calcium compounds are sometimes prescribed as well as vitamin D, particularly in the setting of a new fracture. Standard rehabilitative therapies for spasticity have included range of motion of muscles and joints in an effort to prevent contracture formation. However, these techniques seem to be most effective early in paralysis and become less effective over time as is evident with the development of spasticity, and contractures in the population of neurologically impaired, wheelchair-bound people (2). Most recently, Functional Electrical Stimulation (FES) has been used to drive paretic muscles into active movement; however, data is sparse as to outcome effect on spasticity, contractures, and osteoporosis (5). Also the cost and need for skilled supervision of subjects during FES limits its application. As a consequence, a more traditional but aggressive rehabilitative approach to these problems involves the patient standing supported in a frame which allows body weight to be transmitted through the muscles, joints and bones of the paretic lower extremities (2). It has been reasoned, but not shown experimentally, that this intervention will load the extremities and thus induce conditions that will reduce spasticity, contractures and osteoporosis in a safe, cost-effective manner, that avoids the lack of efficacy or troublesome effects of drugs used for these problems. It is hoped that this study will validate or refute the usefulness of "standing" or weight bearing in the treatment of spasticity, contractures, and neurogenic osteoporosis.

Literature Review and Work Accomplished:

Spasticity in paralyzed people:

Spasticity is an increased excitability of the muscle stretch reflex that leads to exaggerated tendon jerks, clonus and abnormally high resistance to passive movements (10). After transection the spinal cord reorganizes its reflex patterns. There is abundant information available regarding manifestations of this phenomenon (11). Loss of suprasegmental inhibition (12) is considered an important determinant of hyperexcitability of the spinal cord. In incomplete lesions an imbalance of excitatory and inhibitory brain stem influences affect the amount of spasticity exhibited (13,14). The Hoffman reflex (H-reflex) is known to undergo changes in humans with spinal cord injury (8, 12, 15). This reflex can be easily inhibited shortly after spinal injury but once spasticity is clinically apparent, it is less inhibited with vibration (16). The ratio of amplitude of the H-reflex to the Compound Muscle Response (M-wave) increases as Spasticity develops (8). Besides the use of the H-reflex and clinical exam of muscle tone and

deep tendon reflexes, it is now possible to measure torque and EMG in passive and active movements of extremities using isokinetic dynamometry with transducer and goniometer attachments (Kincom technology) (9). This technique has the advantage of precise reproducibility of the test movements and adaptation to sudden spasms and movements to exclude the risk of injury (10). Basic treatment begins with adequate ranging of spastic extremities (17). Therapeutic exercise, biofeedback, topical cold and splinting have been used. Electrical stimulation of peripheral nerves has been applied for the treatment of spasticity (17) and effects are reported to last for hours after cessation of the stimulus. A recent report evaluates the effects of surface stimulation of muscle (18). Baclofen, an analog of gammaaminobutyric acid (GABA), is an effective agent although its effects do not seem related to GABA "a" receptors (19). Diazepam (Valium) is widely used to treat spasticity although its action is not fully understood (20,21). It appears that Diazepam facilitates the post-synaptic effects of GABA. Dantrolene (Dantrium) blocks excitation-contraction coupling in skeletal muscle. Other drugs such as clonidine, glycine, threonin and chlorpromazine (10) have been reported to have efficacy in reducing spasticity but generally are not routinely used in the clinical setting.

Contractures of the legs in paralyzed people.

Contractures are a common problem in paretic legs. Hips are normally contracted in flexion; knees lose extension and ankles are plantar flexed. Prolonged sitting in a wheelchair with hips and knees flexed may contribute significantly to the shortening of the muscle-tendon unit resulting in restriction of joint movement and capsular tightening. Inability to dorsiflex ankles when supine and imbalance of muscle strength in plantar flexors over dorsiflexors in spasticity, contribute to ankle plantar flexion contractures. In normal, standing provides active forces to counteract these adverse effects of bed and wheelchair positioning as well as stretching ankle plantar flexors (2).

Osteoporosis in paralyzed people:

Osteoporosis occurs when bone resorption is more extensive than bone deposition (22). It causes bone mass loss that alters the structure but not the composition of the tissue (22). It is characterized by an increase porosity of cancellous bone and a decrease in cortical thickness in compact bone. Often the disease etiology is unclear; however, many factors are associated with the condition. These factors include hormonal, dietary, exercise, malabsorption of calcium, corticosteroids, hyperparathyroidism, hyperthyroidism and immobilization (23). The association with neurogenic conditions such as spinal cord injury,

hemiplegia and multiple sclerosis has not been as thoroughly studied as other varieties of the condition but is thought in part to be a variant of the immobilization or disuse variety. Within the literature there has also been a debate about whether disuse is primarily responsible for the condition, or if in fact, lack of muscle activity is not equally a factor in its genesis (5,24). Several observers have noted that spasticity in muscles seems to reduce the amount of osteoporosis in spinal cord injured subjects (25,26). Osteoporosis is a diagnostic problem early in its course as routine x-rays will show abnormality only after 30% or more of bone mass is lost (22). Dual Photon Absorptiometry (DPA) is accurate, reproducible and involves only a low dose of radiation (10 mR) in assessing bone density (27). Most of the reported work with DPA has involved the spine and proximal femur. Because DPA measures calcium in osteophytes, aorta nodes or stones, it is not useful for the initial diagnosis of osteoporosis in persons with osteoarthritis, diffuse idiopathic skeletal hyperostosis or facet joint disease. (27). Quantitative computed tomography is another available and accurate techniques for measuring bone density but requires relatively high radiation doses (500-900 mR) (27) and has been applied primarily to the study of the spine (28). Other techniques such as radiographic photodensitometry, quantitative digital radiography, total body calcium by neutron activation analysis and measurement of ultrasound velocity have limiting factors associated with their use such as site-specificity or cost (28).

Preliminary Studies:

We have studied the effect of "standing" on spasticity (Clinical Assessment and H-reflex), osteoporosis (dual photon absorptiometry) and contractures (range of motion) in neurogenic paralysis was studied over time (mean=224 days) in six males (means = 49 years) with paralysis (mean duration = 24 years) confined to wheelchairs (mean duration = 19 years) Subjects stood in standing frames an average of 144 hours over a mean duration of 135 days. Clinical Assessment (CA) utilized a 5 point grading scale: 0=reflex, tone or clonus; 1= barely perceptible reflex and tone, or less than 4 beats of clonus (BOC); 2=decreases reflex and tone, or 4-6 BOC; 3=normal reflex and tone, or 7-9 BOC; 4=increased reflex and tone, OR 10-12 box; 5=marked increase reflex and tone or greater than 12 BOC. At 8 weeks following completion of the study 67% of subjects continued to stand voluntarily. On a scale of 1 (low) to 10 (high), these subjects reported feeling more relaxed (mean=5.5), healthier (mean=6.75), and recommended (mean=8.75) "standing" for others. A significant decrease in bone density was observed in femoral neck, although lumbar spine values were normal.

Literature References:

Young, R. & Shahani, B.T. (1986) in Management of Spinal Cord Injuries (Bloch, R.F. & Basbaum, M., eds.) pp 241-283, Williams & Wilkins, Baltimore.

Kottke, F.J., Pauley, D.L. & Ptak, R.A. (1966) Arch Phys Med Rehab 47, pp 345-352.

Petrofsky, J.S. & Phillips, C.A. (1984) Central Nervous System Trauma 1, pp 57-74.

Comarr, A.E., Hutchinson, R.H. & Bors, E. (1962) American Journal Surg 103, pp 732-739.

Cybulski G.R., Penn R.D. & Jaeger R.L. (1984) Neuro surgery 15, pp 132-146.

Keenan, R.E., Kolb, M.E. & Horne, M.L. (1977) Clin Therapeutics 1, pp 48-55.

Kraij, Bajd, T., Turk, R. & Benko, H. (1986) Paraplegia 24, pp 221-230.

Little, J.W. & Halar, E.M. (1985) Arch Phys Med Rehab 66, pp 19-22.

Knutsson, E. & Martensson, A. (1980) Scand J Rehab Med 12, pp 93-106.

.Knutsson, E. (1983) in Motor Control Mechanisms in Health and Disease (Desmedt, J.E., ed.) pp 1013-1034, Raven Press, New York.

Ashby, P. & McCrea, D.A. (1987) in Handbook of the Spinal Cord (Davidoff, R.A., ed.) pp 119-143, Marcel Dekker, Inc., New York.

Lance, J.W. (1980) in Spasticity: Disordered Motor Control (Feldman, R. G., Young, R.R. & Koella, W.P., eds.) pp 185-203, Symposia Specialists, Chicago.

Dimitrijevic, M.R. (1987) Paraplegia 25, pp 205-208.

Magoun, H.W. & Rhines, R. (1947) Spasticity: The stretch-reflex and extra pyramidal systems, Springfield, IL.

Delwaide, P.J. (1973) in New Developments in Electromyography and Clinical Neurophysiology (Desmedt, J.E., ed.) pp 508-522, Karger, Basel.

Taylor, S., Ashby, P. & Verrier, M. (1984) J Neurol Neurosurg Psychiatry

47, pp 1102-1108.

Katz, R.T. (1988) American Journal Physical Med & Rehab 67, pp 108-116.

Robinson, C.J., Kett, N.A. & Bolam, P.M. (1988) Arch Phys Med Rehab 69, pp 598-604.

Davidoff, R.A. (1985) Ann Neurol 17, pp 107-116.

Cook, J.B. & Nathan, P.W. (1967) J Neurol Science 5, pp 33-37.

Verrier, M., Ashby, P. & MacLeod, S. (1977) Arch Phys Med Rehab 58, pp 148-153.

Berg, E. & Moyle, D. (1988) J Musculoskeletal Med 5, pp 64-81.

Smith, R. (1987) Br Med J 294, pp 329-332.

Gross, M., Roberts, J.G., Foster, J., Shankardass, K. & Webber, C.E. (1987) Arch Phys Med Rehab 68, pp 158-161.

Abramson, A.S. & Delagi, E.F. (1961) Arch Phys Med Rehab 42, pp. 147-151.

Cowell, L.L., Squires, W.G. & Raven, P.B. (1986) Med Sci Sports Exerc 18, pp 501-508.

Griffiths, H. (1987) J Musculoskeletal Med 4, pp 59-71.

Chestnut, O.H. (1988) Triangle 27, pp 37-45.