Bone loss is one of the inevitable complications of spinal cord injury. Since people with paralysis don’t typically put weight or pressure on their bones, they tend to lose bone density and often develop osteoporosis. But while osteoporosis in the SCI population first was studied in relation to calcium metabolism and the hypercalciemia and renal calculi that followed, there appear to be differences between SCI-induced osteoporosis and other causes of bone loss, such as prolonged bedrest and space travel.

Hypercalciuria (calcium dumped from the bones to the urine) is seen by 10 days following the SCI and reaches a peak 1-6 months postinjury. This level of hypercalciuria is 2-4 times that of persons without SCI who undergo prolonged bedrest. This increase is the direct result of a metabolic imbalance between bone formation and bone resorption.
This model at the skeletal level following SCI resembles the high bone turnover rate seen in postmenopausal women.

Bone loss following SCI occurs throughout the skeletal system, with the exception of the skull. The distal femur and proximal tibia are the bones most affected, followed by the pelvis and the arms. The amount of demineralization in the skull, pelvis, and lower limbs is independent of the neurologic level.

In people with injury less than 1 year, reduction in bone mineral densities has been noted in the femur as much as 43 percent, as compared with controls. Over 50 percent of bone content in these regions is demineralized at the 10-year anniversary of the injury. (Bone fractures usually do not occur until bone mass is 30-40% below normal. Data from the Model SCI Systems show that 14 percent of people with SCI get fractures five years after injury. This increases to 28 percent after 10 years and 39 percent after 15 years. The frequency of fractures increases with age and completeness of injury, and is higher in women than men.)

The arms and trunk actually increase bone content after the 4-month point; the net effect is an approximate 10-21 percent loss of bone at the 10-year point. The trunk has a net gain in mineral content by 12 years post-injury.

As far as treatments go, there are drugs available to reduce the risk of broken bones. These drugs can slow or stop bone loss or rebuild bone. With drugs and exercise such as on an FES bicycle, osteoporosis can be lessened and bone density regained.

Web Sites

**Osteoporosis and Spinal Cord Injury**


Medscape Reference: Osteoporosis and Spinal Cord Injury

http://sci.washington.edu/info/forums/reports/osteoporosis.asp

Northwest Regional Spinal Cord Injury System: Osteoporosis in SCI

This page has text and video of an October 9, 2007 presentation by Jelena Svircev, MD, assistant professor in the Department of Rehabilitation Medicine at the University of Washington.

http://www.uab.edu/medicine/sci/daily-living/managing-personal-health/secondary-medical-conditions/osteoporosis

Spinal Cord Injury Information Network: Osteoporosis (Bone Loss)

http://www.uab.edu/medicine/sci/uab-scims-information/secondary-conditions-of-sci-health-education-video-series


The 19-minute Bone Health video discusses aspects of heterotopic ossification (classifications, etiology, diagnosis, prevention and treatment options) and osteoporosis
(initial bone loss after traumatic injury, impact of aging, impact of menopause, prevention and treatment options). The video can be downloaded or streamed online.

**Osteoporosis in General**

Medline Plus: Osteoporosis

National Osteoporosis Foundation (NOF)
251 18th St. S
Suite 630
Arlington, VA 22202
Phone: 703-647-3000, 800-231-4222 (Toll-free)

NIH Osteoporosis and Related Bone Diseases National Resource Center
2 AMS Circle
Bethesda, MD 20892-3676
Phone: 202-223-0344, 800-624-2663 (Toll-free), 202-466-4315 (TTY)
E-mail: NIHBoneInfo@mail.nih.gov

American Academy of Orthopaedic Surgeons: Osteoporosis

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