Biomechanical properties of human tibias in long-term spinal cord injury

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Abstract—Long-term spinal cord injury (SCI) profoundly alters skeletal structure and function. In this study, the biomechanical properties of tibias from persons with SCI and from individuals closely matched in age and size but without SCI were quantified at both the structural and material levels. Nondestructive torsion tests were performed to determine apparent shear moduli for the tibia. The cortical thicknesses and polar moment of inertia were determined numerically. Four-point bending tests were performed to determine flexural modulus of elasticity on cortical bone specimens of the tibia. The apparent shear moduli of the SCI tibias were found to be lower than the non-SCI tibias (p<0.05). The cortical thicknesses of the SCI tibias were significantly thinner than the control tibias (p<0.05), while the polar moment of inertia showed no significant differences between control and SCI tibial cross sections (p>0.5). The flexural modulus of elasticity of the cortical bone specimens were lower in the SCI tibias than the controls (p<0.05). These differences suggest that tibias may undergo micro-structural changes as well as structural adaptation following SCI, which alter their mechanical properties.

Key words: biomechanics, bone, spinal cord injury, tibia.

INTRODUCTION

Permanent damage to the spinal cord is a tragic injury, with devastating manifestations in multiple organ systems, including bone structure and metabolism (1–3). Not only is motor function irreversibly lost, but significant attrition of bone mass occurs below the level of the cord lesion (4–6). It is empirically known that the osteoporosis below the level of the spinal cord injury (SCI) far exceeds the normal attrition of bone mass associated with aging, bed rest, immobilization, or disuse due to disorders other than paralysis (7). In persons with SCI, bone undergoes resorption as evidenced by increased urinary excretion of calcium, magnesium, phosphorus, and hydroxyproline immediately after injury (8–10). Bone mineral and density studies have shown that most bone loss occurs in the lower limbs. Loss of bone mass is most rapid in the first 4 months following complete SCI, and falls to two-thirds of the original bone mass at 16 months (11). Other density studies have shown that bone mineral content in persons with chronic SCI falls to 25 percent less than normal in the proximal femur, and 50 percent less in the proximal tibia (12).

This loss of bone mass results in significant decrease in the structural integrity of the skeleton, and the risk of pathologic fractures is greatly increased. Investigations of bone mineral density using dual photon absorptiometry indicate a much higher incidence of fractures among persons with SCI, with fracture rates reaching 10 times that...
of the general population (13). For the remainder of their lives, these persons are susceptible to bony injury from the trivial forces encountered in daily living. Fractures are a serious additional disability to the persons with chronic SCI, whose remaining abilities are already maximized to retain the highest possible self-sufficiency in daily life. That margin of independence can be overcome when self-care and transfer capabilities are encumbered by limb immobilization or surgical procedures required following a pathological fracture. Such pathologic fractures may also lead to long-term complications such as skin breakdown, malunion, and deformity, and in some cases, infection and gangrene.

Investigations into the severe osteoporosis of persons with SCI have focused on the specific characteristics of the fractures and the persons affected, and on metabolic and bone density alterations found in these persons (10,13–16). To date, the biomechanical changes in the individual bones of persons with SCI have not been described or quantified in the literature. The hypothesis of this study was that the bone in persons with chronic SCI undergoes alterations that severely degrade its biomechanical properties. The tibia provides an excellent specimen for study of pathologic bone in SCI, since the most severe osteopenia is found in the lower limbs of both persons with quadriplegia and paraplegia (11,12). Also, 18 percent (13) to 41 percent (16) of all the fractures in persons with SCI occur in the tibia. The objectives of this study were to quantify the structural, geometric, and material properties of human tibias of persons with chronic SCI and compare them to control specimens closely matched in age and size.

METHODS

Overview

Three types of biomechanical properties were investigated. Structural properties were determined using a nondestructive torsion test. Geometric properties, including cortical thicknesses and polar moment of inertia, were measured at four different levels of tibial cross sections. Material properties of the cortical bone were determined for each quadrant at three different levels of the tibia using a four-point bending test.

Specimens

Four pairs of fresh frozen whole tibias closely matched in size and age were used for this study (Table 1). All tibias were macroscopically intact with no apparent abnormalities. Specimens were also radiographed to screen for previous fractures or destructive lesions. Four of the tibias were amputated from persons with long-term SCI, and four (controls) from persons without SCI. All specimens were male, and all amputations were due to peripheral vascular disease.

Nondestructive Torsion Tests

Nondestructive torsion tests were performed to determine apparent shear moduli for the entire diaphyseal

Table 1.
Specimen information.

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Years</th>
<th>Length</th>
<th>Weight</th>
<th>Weight/Length</th>
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</thead>
<tbody>
<tr>
<td>SCI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>42</td>
<td>39.5</td>
<td>351</td>
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<td></td>
<td>68</td>
<td>48</td>
<td>44.5</td>
<td>536</td>
<td>12.0</td>
</tr>
<tr>
<td></td>
<td>68</td>
<td>48</td>
<td>44.5</td>
<td>535</td>
<td>12.0</td>
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<td></td>
<td>68</td>
<td>36</td>
<td>39.0</td>
<td>425</td>
<td>10.9</td>
</tr>
<tr>
<td>Mean</td>
<td>66.0</td>
<td>43.5</td>
<td>41.9</td>
<td>461.8</td>
<td>11.0</td>
</tr>
<tr>
<td>SEM</td>
<td>2.0</td>
<td>2.9</td>
<td>1.5</td>
<td>45.2</td>
<td>0.7</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>N/A</td>
<td>36.0</td>
<td>318</td>
<td>8.8</td>
</tr>
<tr>
<td></td>
<td>69</td>
<td>N/A</td>
<td>42.0</td>
<td>581</td>
<td>13.8</td>
</tr>
<tr>
<td></td>
<td>62</td>
<td>N/A</td>
<td>41.0</td>
<td>615</td>
<td>15.0</td>
</tr>
<tr>
<td></td>
<td>68</td>
<td>N/A</td>
<td>36.5</td>
<td>392</td>
<td>10.7</td>
</tr>
<tr>
<td>Mean</td>
<td>67.3</td>
<td>N/A</td>
<td>38.9</td>
<td>476.5</td>
<td>12.1</td>
</tr>
<tr>
<td>SEM</td>
<td>1.8</td>
<td>N/A</td>
<td>1.5</td>
<td>72.1</td>
<td>1.4</td>
</tr>
</tbody>
</table>

Years = since SCI; length in cm; weight (wet) in g; weight/length in g/cm; SEM = standard error of the mean.
region of the tibia as well as for its proximal middle and distal regions. Each tibia was thoroughly cleaned of all soft tissue and was kept moist by regular irrigation with 0.9 percent saline. The total length of each tibia was determined by measuring from the intercondylar eminence to the tip of the medial malleolus. Tibias were divided into five regions, each consisting of 20 percent of the total length (Figure 1). Between each region, 0.889 mm threaded Kirschner wires (K-wires) were inserted into the bone to measure the regional angular deformation of the tibia. The tibia was then mounted onto a custom jig specifically designed to hold the specimen so that the axis of rotation coincided with the long axis of the bone (Figure 2). The proximal and distal ends of the tibias were rigidly fixed in the jig using fixation screws and then potted using dental stone. Before each of the torsion tests was performed, a pre-load of 3 N-m was applied in torsion and then cycled 10 times from 0.5° to 2.5° at a frequency of 0.125 Hz for pre-conditioning. The specimen was allowed to recover for 30 min in an unloaded state.

At the end of the recovery period, a pre-torque of 3 N-m was again applied to the tibia and then cycled 10 times from 1.0° to 5.0° at the same frequency of 0.125 Hz. Torsion tests were performed for both internal and external rotation, using the MTS machine (MTS Systems Corporation, Minneapolis, MN) with a 30-min recovery period between each test.

Measurements of torque, rotation, and time were recorded at a frequency of 5 Hz by LabVIEW® (National Instruments, Austin, TX), an analog-to-digital program installed on a Macintosh IIci (Apple Computer, Cupertino, CA). Angular deformation was measured using a video digitizing system that recorded the movement of reflective markers attached to the K-wires inserted into the bone (17). The video digitizing system (VDS) utilizes a high-resolution video camera, a studio-grade video cassette recorder (Panasonic AG-6300), a high-resolution monitor (Panasonic TR-124MA), a microprocessor (Motion Analysis VP-320) and a 486 PC computer to run the data acquisition software. The VDS software calculates the centroid of the reflective markers, and digitizes their displacement within ±5 μm. Previous tests have documented the accuracy of the VDS (17).
Geometric Assessment

Transverse sections of the tibia were cut perpendicular to the long axis of the bone using an ISOMET (Buehler, Lake Bluff, IL) low speed diamond wheel saw in constant 0.9 percent saline lubrication. After sectioning, trabecular bone was removed from the center of the cross section. These bone cross sections were directly scanned using a Hewlett Packard Scan Jet IIC scanner (Hewlett Packard, Palo Alto, CA). The scanned images were then analyzed using a modified SLICER program, developed by Nagurka and Hayes, to determine the cortical thicknesses in the anterior, posterior, medial, and lateral quadrants, and to calculate the polar moment of inertia of the tibial cross sections (18).

Calculation of the Apparent Shear Modulus

The data from the nondestructive torsion test and the geometric cross-section data were then used to calculate the apparent shear moduli for the proximal, middle, and distal regions of the tibia using the following equation:

\[
G = \frac{(T)(L)}{(\phi)(J)}
\]

\(G\) = apparent shear modulus
\(\phi\) = angle of twist measured by the video digitizing system
\(T\) = torque measured by the MTS
\(L\) = length of the region,
\(J\) = polar moment of inertia measured by Slicer program

Four-Point Bending Tests

The flexural modulus of elasticity of the cortical bone specimens were determined by performing four-point bending test to failure (Figure 3). Match-stick type strips were obtained from the 4 quadrants (anterior, posterior, medial, lateral) at each of the 3 mid-diaphyseal regions, yielding a total of 12 match sticks per tibia. The strips were cut from cortical bone using an ISOMET diamond saw, then sanded using an ISOMET grinder/polisher to produce the match-stick-type specimen measuring 3×3×60 mm. Each was subjected to a four-point bending test to failure, using an Instron machine at a loading rate of 50 mm/min in a constant temperature saline bath. The direction of loading was from the endosteal cortex to the periosteal cortex, and all tests were performed at 22 °C.

\[
I = \frac{bh}{12}
\]

\[
E = \frac{Pa}{6I} \left( a + \frac{3L}{2} \right)
\]

\(I\) = moment of inertia
\(b\) = width of the beam
\(h\) = height of the beam
\(E\) = flexural modulus of elasticity
\(P\) = pressure of crosshead

Figure 3.
A schematic drawing showing the origin of the matchstick type specimen representing each quadrant. These specimens were used for four-point bending tests and the configuration of the four-point bending test used for the calculation of flexural modulus of elasticity is also shown.

Statistical Analysis

The data were analyzed using multivariate ANOVA with a significance level of 0.05.

RESULTS

Nondestructive Torsion Test

As indicated in Figure 4, the apparent shear moduli of the SCI tibias were found to be smaller than that of the control tibias (p<0.05). The individual comparison between the SCI tibias and the control tibias for region one (proximal), region two (middle), and region three (distal) of the tibial diaphysis showed a statistically significant decrease in apparent shear moduli of the SCI tibias only for the proximal region during internal rotation (p<0.05). The mean values for the apparent shear moduli of the control tibias exceeded those of SCI tibias for all
Figure 4.
A graph showing the apparent shear moduli for proximal, middle, and distal regions of the tibial diaphysis. The apparent shear moduli measured from both the internal and external torsion are shown. Note that the values for SCI specimens are consistently lower than those of the control specimens.

Geometric Assessment

The anterior, posterior, medial, and lateral cortical thicknesses for both the SCI and control tibias are shown in Table 2. Overall, the cortical thicknesses were significantly thinner for SCI tibias compared to the controls (P<0.05). The cortical thicknesses of the tibial cross sections for both groups decreased significantly from cross section one (proximal) to cross section four (distal) of the tibia (P<0.05). Individual comparison of the cross sections and quadrants between SCI and control tibias only demonstrated statistical significance in the posterior quadrant of cross section one, three (middle/distal), and four, and in the anterior and medial quadrants of cross section four (P<0.05).

In both SCI and control tibias, the polar moment of inertia decreased from the proximal to the distal cross section (P<0.05). However, no significant differences (P>0.5) were found between SCI and control tibias in the polar moment of inertia measurements at each cross sectional level (Figure 5).

Four-Point Bending Test

As shown in Figure 6, the flexural modulus of elasticity of all quadrants was lower for SCI tibias when compared to controls (P<0.05). Region and quadrant comparison between groups only showed a statistical significance between SCI and control tibias in the medial and lateral quadrants of region 2 (P<0.05).

DISCUSSION

Pathophysiology of Spinal Cord Injury Osteoporosis

Current understanding of the pathophysiology of osteoporosis following SCI is based upon considerations of mechanical stress deprivation, metabolic and hormonal changes, and neurological and autonomic alterations. It
Table 2.
Cortical thickness.

<table>
<thead>
<tr>
<th></th>
<th>Anterior</th>
<th>Posterior</th>
<th>Medial</th>
<th>Lateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slice 1</td>
<td>4.6±1.7</td>
<td>2.6±0.2*</td>
<td>2.8±0.8</td>
<td>2.7±0.6</td>
</tr>
<tr>
<td>Slice 2</td>
<td>5.9±1.8</td>
<td>3.6±0.1</td>
<td>3.5±0.2</td>
<td>3.9±0.7</td>
</tr>
<tr>
<td>Slice 3</td>
<td>7.1±2.0</td>
<td>3.7±0.6*</td>
<td>3.4±0.3</td>
<td>3.9±0.7</td>
</tr>
<tr>
<td>Slice 4</td>
<td>3.3±0.7*</td>
<td>2.4±0.4*</td>
<td>2.4±0.2*</td>
<td>3.0±0.6</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slice 1</td>
<td>6.7±1.7</td>
<td>4.3±0.6*</td>
<td>3.3±0.5</td>
<td>2.7±0.1</td>
</tr>
<tr>
<td>Slice 2</td>
<td>9.7±1.1</td>
<td>5.4±0.6</td>
<td>4.1±0.4</td>
<td>3.7±0.2</td>
</tr>
<tr>
<td>Slice 3</td>
<td>9.8±1.4</td>
<td>6.2±0.6*</td>
<td>4.5±0.5</td>
<td>4.6±0.4</td>
</tr>
<tr>
<td>Slice 4</td>
<td>5.3±0.4*</td>
<td>4.7±0.2*</td>
<td>3.5±0.1*</td>
<td>4.3±0.5</td>
</tr>
</tbody>
</table>

Thickness in mm; means ± SEM (standard error of the mean); * = statistically significant difference between groups (p<0.05).

is well known that mechanical forces influence the structure and function of bone. According to Wolff’s law, bone remodels over time in response to mechanical loading, and dependent on its direction, rate, and magnitude (19). Thus, the structural integrity of the skeleton is maintained in response to gravity and mechanical loading, and diminished in response to immobilization or disuse. In persons with paraplegia and quadriplegia, who are unable to bear weight on their lower limbs and who are sitting or recumbent the majority of the time, the limbs are not exposed to the forces that are required to stimulate bone formation. Some investigators have speculated that the absence of muscle tension and weight bearing may not only fail to stimulate any osteoblastic activity, but may also trigger massive osteoclastic resorption (11). Treatments and preventative measures based on these mechanical factors, however, have not been effective in reversing SCI osteoporosis (12).

In addition to mechanical factors, metabolic and hormonal alterations may be responsible for the SCI osteoporosis. There is direct evidence of bone mineral and matrix resorption in urinary excretion studies of calcium, phosphorus, hydroxyproline, and other components (1,3,8,9). Documented alterations in hormones, such as parathyroid hormone, calcitonin, and glucocorticoids, potentially contribute to bone mass attrition, yet it is unclear if the alterations are primary or secondary mechanisms (1,2). Abnormal hydroxylation of proline in bone collagen also has been demonstrated (8). Treatments to modify bone metabolism, such as disphosphonates and calcium supplementation, however, have not significantly altered the bony resorption. Also, changes in the autonomic nervous system are proposed to cause attrition of SCI bone, via changes in vascular tone and flow (1,9). Sympathetic denervation in SCI may cause arteriovenous shunts and a slowdown of intraosseous blood flow, thus increasing bone resorption.

Despite many causative factors, bone loss following SCI is severe and rapid. Bone mineral and density studies have demonstrated that most bone loss is in the lower limbs and occurs rapidly in the first 4 months after complete cord injury, falling to two-thirds of the original bone...
mass at 16 months (11). Other density studies showed that bone mineral content in persons with chronic SCI falls to 25 percent lower than normal in the proximal femur, and 50 percent lower in the proximal tibia (12). This loss of bone mass results in significant weakness in the structural integrity of the skeleton, and the risk of pathologic fractures is greatly increased. The high incidence of fractures in persons with complete SCI is well demonstrated, with their fracture rate being 10 times that of the general population (13).

Pathologic Fractures in Persons with Spinal Cord Injury

Clinical data on fractures in persons with SCI became more evident in the literature in the 1950s and 1960s as survival beyond the acute spinal injury became possible. In 1963, Eichenholtz reported 4 percent of their entire SCI population had long bone fractures (15). Treatments acceptable to “normal” persons resulted in serious complications, such as skin slough, pressure sores, and amputation. Comarr et al. found that over half of all of fractures occurred in the lower limbs (14). Freehafer et al. found an overall incidence of fractures of 3 percent (16,20). McMaster and Stauffer classified subjects into three categories: acute fractures concomitant with the SCI, pathologic fractures in osteoporotic limbs, and the relatively uncommon high energy injuries in osteoporotic extremities (10). Nottage found a majority of SCI fractures in chronically pathologic bone (21).

Ragnarsson et al. noted long bone fractures in 4 percent of their SCI population, most commonly in the supracondylar distal femur (33 percent), femoral shaft (30 percent) and tibial shaft (18 percent). The study also noted a tenfold higher risk of pathologic fractures in complete SCI compared to incomplete injury, and that subjects with paraplegia had an even higher rate than those with quadriplegia (13). Sex, age, time from injury, or spastic versus flaccid paralysis had no bearing on the incidence of fractures. Garland et al. found that nonoperative treatment of acute fractures concomitant to the acute spinal injury resulted in a higher complication rate such as deformities, nonunions, and pressure sores (22).

Results of Biomechanical Investigation

The research in the SCI osteoporosis has been focused on the specific characteristics of the fractures along with persons affected, and on metabolic and bone density alterations found in almost all persons with SCI. To date, the biomechanical changes in the individual bones of persons with SCI have not been well described or quantified. With the affiliation of a large SCI center at the VA Medical Center, Long Beach, this study had the opportunity to examine in detail the biomechanical properties of tibias from persons with SCI. This study presents the structural, geometric, and material properties of these tibias and their comparison with control specimens closely matched in age and size.

The results of this biomechanical study are consistent with the basic science and clinical research into the bone atrophy following SCI. Both the structural and material properties and cortical thicknesses of SCI tibias were significantly inferior to control specimens, and appear to be consistent with the high incidence of loss of bone density and pathologic fractures. The apparent shear moduli showed greater difference in the proximal region between the SCI tibias and the control tibias. Although it cannot be delineated whether this is an adaptation at the structural level or the material level, the adaptive changes due to SCI were greater in the the proximal tibia as compared to the distal tibia. The cortical thicknesses of SCI specimens were significantly thinner than the controls (p<0.05). However, in region-specific analyses, the statistically significant differences were seen in slice four, the most distal cross section for anterior, posterior, and medial quadrants. The remaining differences were seen in the posterior quadrant, of cross-sectional slices one and three. The data show that the posterior quadrant underwent the greatest amount of cortical thinning due to SCI as compared to the other regions. Also, anterior and posterior cortical thickness were no more inferior distally than proximally, again demonstrating no regional differences in level of bone atrophy. The polar moments of inertia were neither inferior nor superior to controls. This indicates that some cortical thinning was compensated for by the increase in the cortical diameters to achieve similar value in polar moment of inertia. The proximal regions of the tibial diaphysis of the SCI specimens were not any less affected than distal regions in material properties as indicated by the flexural modulus of elasticity. If the alterations in blood flow are a causative factor in SCI bone atrophy, one might assume that variations in perfusion across proximal-to-distal segments might cause increased atrophy proximally. No such regional difference in bone strength was apparent in this investigation.

It is hoped that this baseline information on the tibias of persons with SCI will advance the understanding of the mechanisms behind the profound bony changes of SCI. Further basic science and clinical investigation may
bring progress toward the effective prevention and treatment of this difficult complication of a devastating injury.

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REFERENCES


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