Bone mineral content and fatigue failure of lumbar motion segments in simulated flexed lifting: Does specimen age influence the relationship?

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This paper describes a comparison of the fatigue failure responses of older versus younger lumbar spine cadaver motion segment specimens. These specimens were repeatedly subjected to loads simulating those experienced by the spine in lifting a 9 kg load in different torso flexion postures (0, 22.5, and 45 degrees of flexion). An older sample of 36 motion segments (average age 81 years ± 8 SD) is compared with a younger sample of 18 motion segments (average age: 47 years ± 9 SD) with respect to the number of cycles to failure and results of survival analyses evaluating the effects of flexion and bone mineral content with and without the younger data set. Compared to the older sample of spines, the younger sample exhibited many more cycles to fatigue failure in all the torso flexion conditions (10020 versus 8267 average cycles to failure in 0 degrees flexion, 7124 versus 3262 in 22.5 degrees flexion, and 3229 versus 263 cycles to failure in 45 degrees flexion). The increased cycles to failure in young specimens is likely due to the increased bone mineral content (BMC) in younger motion segments (30.7 g ± 11.1 g per motion segment versus 27.8 ± 9.4 g). Cox regression analyses modeling both flexion effects and BMC indicate that betas are similar when just older spines are modeled and when younger specimens are included in the analysis. Betas for survival analysis models with and without younger specimens generally show little change for flexion (2.716 versus 2.756 for 22.5 degrees [0 degrees referent], 3.926 versus 3.380 for 45 degrees [0 degrees referent]). Bone mineral content shows a similar protective effect in both models (Betas: -0.127 versus -0.087). Overall, comparison of the models indicate that risk ratios are similar for 22.5 degrees flexion, slightly increased for 45 degrees flexion and slightly more protective influence predicted for BMC in the combined sample versus the older sample alone.

I. Introduction

A vertebral fracture will result when the strength of a vertebra is exceeded by a transitory load or by cyclic, repetitive loads [Brinckmann et al. 1988, Hansson et al. 1987]. The strength of a vertebra, and therefore its ability to withstand the stresses imposed on it, is primarily determined by the amount of mineralized tissue of which the vertebra is comprised [Bartley et al. 1966, Hansson and Roos 1981, White and Panjabi 1990]. Several studies have found that the relationship between the ultimate compressive strength of a vertebral body and bone mineral content is generally linear [Bartley et al. 1966, Bell et al. 1967, Galante et al. 1970, Hansson and Roos 1981].

There is a decrease in vertebral strength with age, particularly beyond the age of 40 years [Bell et al. 1967]. This decreased strength is associated with a reduction in bone mineral density. Specifically, there [White and Panjabi 1990] appears to be a significant loss of horizontally-oriented trabeculae – particularly in the central portion of the vertebral body [Atkinson 1967]. The disappearance of the horizontal trabeculae deprives the vertical trabeculae of important fortifications, and the load-bearing capability of the center of the vertebral body becomes degraded as a result [Bogduk 1997]. The decreased strength of the cancellous core of the vertebral body as aging takes place is associated with a shift in the load-bearing responsibilities of cancellous and cortical bone. In

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spines less than 40 years of age, the cancellous bone bears approximately 55 percent of the compressive load, while the cortical shell bears 45 percent of the load [Rockoff et al. 1967].

The age-related changes detailed above may also impact the vulnerability of the endplate to failure. Specifically, the weakened trabecular network underlying the endplates permits increased deformation of the cartilaginous endplates, and may increase the propensity to fracture [Bogduk 1997]. The finding of increased fracturing in the endplates and vertical trabeculae with increasing age tends to support this notion [Vernon-Roberts and Pirie, 1977]. The purpose of the present investigation is to compare and contrast the fatigue failure responses of older motion segment specimens to a younger cohort, and to evaluate the influence of bone mineral content on fatigue failure of both groups.

2. Methods

2.1 Cadaver material

Three replications of an incomplete block experimental design were performed using a total of eighteen lumbar spines, each of which was dissected into three motion segments (L1-L2, L3-L4, and L5-S1). The first two reps (12 spines or 36 motion segments) consisted of an older cohort of specimens (averaging 81 years ± 8 SD). The third rep (6 spines or 18 motion segments) was performed using a younger group of specimens (mean age: 47 years ± 9 SD).

2.2 Determination of bone density

The bone density for vertebral bodies L1-L4 of each spine was measured by means of dual x-ray absorptiometry (DXA) using a Lunar DPX machine (Lunar, Madison, WI). Specimens were positioned on a bed of rice (to simulate body tissue) with the anterior aspect of the spine facing up. The bone mineral density (BMD) of the specimen was expressed in grams divided by the projected area of the bone. Bone mineral content (BMC) was derived from the BMD and area measurements. Bone mineral density, projected area of the vertebral bone, and bone mineral content for L1-L2 and L3-L4 segments were provided in standard reports.

2.3 Determination of Simulated Lifting Loads in Torso Flexion

A dynamic EMG-assisted biomechanical model [Granata and Marras, 1995] was used to develop appropriate loads and load rates associated with lifting a 9 kg load starting at three trunk flexion angles: 0 degrees (neutral), 22.5 degrees (partial flexion), and 45 degrees (full flexion). These spinal load estimates were obtained from an existing large database of lifting tasks. Resultant loads were 1300 N, 2400 N, and 3150 N for these torso flexion angles, respectively. Since the spine is composed of viscoelastic tissues, it was considered critical that the rate of loading also be correctly modeled in each posture. Average model estimates of load rate for the three postures were 700 N/s, 2100 N/s, and 4800 N/s, with increasing load rates in more flexed postures. Motion segments were flexed according to endplate angles obtained in a radiographic and MRI studies of subjects flexing in vivo [Chen 2000].

2.4 Testing Procedure

Motion segments were potted in trays containing polymethylmethacrylate (PMMA), the flexion angle of each motion segment being confirmed by endplate measurements obtained via multiple radiographs during the fixation process. This flexion angle was maintained throughout the cyclic loading regimen for a given specimen. All tests were conducted in a humidified environmental chamber at a temperature of 37 deg C. Motion segments were creep loaded for 15 minutes (at 500, 750 or 1050 N depending on torso flexion angle) and then cyclically loaded at 0.33 Hz until failure or the maximum number of cycles (10,020) was completed using a hydraulic materials testing machine (MTS Systems Corporation, Eden Prairie, MN). Failure was defined as a displacement of 10 mm after termination of the period of creep loading. The primary dependent measure was the number of cycles to failure.
2.5 Statistical analyses

Comparisons of the fatigue failure responses of older and younger specimens were achieved via different survival analysis techniques (Kaplan-Meier Product Limit Estimation and Cox Regression Analysis). Kaplan-Meier Product Limit Estimation and mean survival times were obtained separately on the younger specimens (18 motion segments) and older specimens (36 motion segments). Plots of survivorship functions for each group were obtained. Variables included in the Cox regression models included flexion angle, lumbar level, bone mineral content, bone mineral density, and weight of the motion segment. As Cox regression analyses incorporated bone mineral data, L5-S1 segments (from which bone mineral content data was not obtained) were omitted from the analyses. It was not possible to derive Cox models for the smaller sample of younger specimens (insufficient degrees of freedom), thus comparisons were made between Cox regression coefficients of models with and without the younger specimens included.

3. Results

3.1 Mean Survival Times and Censored Observations

Kaplan-Meier Product-Limit analyses demonstrated longer survival times and an increased number of censored observations for the younger cohort of specimens compared to the older group. Figures 1A and 1B illustrate the survivorship functions s(t) at each angle of torso flexion for older and younger spines, respectively. As can be seen from these figures, the increased load and load rates associated with greater torso flexion are associated with decreased survival of specimens in both groups. However, the survivorship functions for younger spines show a dramatically improved survival for the simulated lifting task at all torso flexion levels. Table 1 details the difference between these groups in terms of censored observations (i.e., motion segments surviving the entire 10020 cycles of the test) and in terms of mean survival time. In all cases, survival was significantly enhanced in the younger sample. On the average, younger specimens enjoyed an increase of approximately 3000 cycles to failure compared to older specimens, and an increase of about 35% in the number of censored observations.

3.2 Cox Regression Models

Table 2 provides the results of Cox regression models examining the effects of flexion angle and bone mineral content using just the older cohort of spines, and using the entire sample of spines (old and young). These analyses do not include any L5-S1 segments, as bone mineral scanning software did not have an algorithm for determining the bone mineral content of the sacrum. Younger spines were found to have approximately 3 additional grams of bone mineral per motion segment than older spines (30.7 g ± 11.1 SD versus 27.8 ± 9.4 SD).

As can be seen in the first analysis presented in Table 2, both flexion angle and bone mineral content were significant predictors of the risk of failure for older motion segments. Compared to the referent posture (0 degrees flexion), simulated lifting in 22.5 degrees flexion had a 15 fold increase in risk, while the 45 degree condition saw a 29-fold increase in risk compared to the referent. As can be seen from the negative exponent, increasing BMC exerted a protective influence. According to the model, every gram of BMC resulted in an 8.3% increase in cycles to failure for this older sample.

When the same analysis was performed on all eighteen spines (young and old, 36 motion segments), results were quite similar. The estimates of risk for the 22.5 degree flexion condition were almost identical to the all old sample (15.127 versus 15.741); however, the risk ratio for the 45 degree condition was appreciably higher in the combined sample (50.5 as opposed to 29.4). Bone mineral content again was again found to be protective, but this influence was found to be slightly stronger when the younger motion segments were included in the model (RR: 0.881 versus 0.917 for the old specimens). Thus, each additional gram of bone mineral content resulted in an approximate increase of 12% in cycles to failure for the combined sample.

4. Discussion

Survival analyses of the fatigue failure responses of older versus younger spines to
simulated lifting loads in three torso angles illustrate some significant differences in response as well as some similarities. Undoubtedly, the most notable difference is the large increase in cycles to failure observed at all flexion angles in the young cohort. At each flexion angle, it took approximately 3000 more cycles to achieve failure in younger spines than older specimens. The number of censored observations also increased significantly in each flexion condition for young motion segments. Notably, none of the younger specimens failed at the 0 degree torso flexion simulated lift. Furthermore, while none of the older specimens survived the 45 degree torso flexion simulated lift, one-third of younger specimens were able to last the entire 10020 cycles in this condition. Clearly, young spines have a dramatically increased tolerance to repeated loading compared to older specimens. The increased tolerance exhibited by younger spines is undoubtedly due, at least in part, to the increased bone mineral content found in younger spines. While the absolute difference in BMC is small (less than 3 g per motion segment), the protective influence appears to be quite significant.

While the absolute difference in cycles to failure is significant, Cox models incorporating the flexion effect and the influence of BMC are similar in terms of the estimates of each effect. The one exception is that the risk ratio for the most extreme flexion condition was estimated to be significantly higher in the combined sample. This may reflect the increased disparity between failure experience in the 0 degree condition (where no samples failed) and the 45 degree condition (where most specimens failed) in the young sample. However, other than this exception, the models were quite similar, and it is notable that the standard errors for each risk ratio estimate are reduced in the full sample, indicating increased confidence of each estimate.

Results of this study suggest that exposure to spinal loads experienced in flexed lifting of a given weight are likely to result in much more rapid fatigue failure compared to the lighter loads experienced in upright lifting. These findings provide strong support for minimizing the amount of forward bending performed in lifting tasks.

These results also suggest that older workers may experience fatigue failure much more quickly, primarily due to the reduced bone mineral content in their spines. This suggests that lifting limits may need to consider age-related changes when determining appropriate workloads for the older worker.

Certain limitations should be considered regarding the data presented here. These include the possibility that preexisting medical conditions or loading histories might have differentially affected the specimens. The sample size of the younger spines is also somewhat small, and the average age is still middle-aged.

In conclusion, results of this analysis demonstrate much greater tolerance to cyclic loading for younger specimens. However, if bone mineral content is taken into consideration, Cox regression models regression coefficients for the flexion effect and bone mineral content are quite similar in the combined versus the old sample, with the exception of increased risk in the most extreme flexion condition.

References

Table 1. Comparison of censored observations and mean survival times for old and young spine motion segments using spinal loads predicted when lifting a 9 kg load in three torso flexion postures.

<table>
<thead>
<tr>
<th></th>
<th>0 degrees</th>
<th>22.5 degrees</th>
<th>45 degrees</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% Censored</td>
<td>Mean Survival</td>
<td>% Censored</td>
</tr>
<tr>
<td>Older Specimens (n=36)</td>
<td>67% 8267</td>
<td></td>
<td>25% 3261</td>
</tr>
<tr>
<td>Younger Specimens (n=18)</td>
<td>100% 10020</td>
<td></td>
<td>67% 7124</td>
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</table>

Table 2. Results of Cox regression models incorporating effects of simulated torso lifting loads at different angles of flexion and bone mineral content of the specimens. A model incorporating old spines is compared with a model incorporating both young and old specimens.

<table>
<thead>
<tr>
<th>Sample</th>
<th>df</th>
<th>Beta</th>
<th>SE</th>
<th>P</th>
<th>Risk Ratio</th>
<th>95% CI for RR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Old Spines (24 specimens)</td>
<td>2</td>
<td>0.001</td>
<td></td>
<td>0.001</td>
<td>15.127</td>
<td>15.127-83.487</td>
</tr>
<tr>
<td>Flexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>22.5 degrees</td>
<td>1</td>
<td>2.716</td>
<td>.872</td>
<td>0.002</td>
<td>15.127</td>
<td>2.741-83.487</td>
</tr>
<tr>
<td>45 degrees</td>
<td>1</td>
<td>3.380</td>
<td>.941</td>
<td>0.000</td>
<td>29.371</td>
<td>4.641-185.903</td>
</tr>
<tr>
<td>BMC</td>
<td>1</td>
<td>-0.087</td>
<td>.038</td>
<td>0.022</td>
<td>0.917</td>
<td>0.851-0.988</td>
</tr>
<tr>
<td>Old + Young Spines (36 specimens)</td>
<td>2</td>
<td>0.001</td>
<td></td>
<td>0.001</td>
<td>15.741</td>
<td>15.741-85.309</td>
</tr>
<tr>
<td>Flexion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>22.5 degrees</td>
<td>1</td>
<td>2.756</td>
<td>.862</td>
<td>0.001</td>
<td>15.741</td>
<td>2.905-85.309</td>
</tr>
<tr>
<td>45 degrees</td>
<td>1</td>
<td>3.923</td>
<td>.921</td>
<td>0.000</td>
<td>50.530</td>
<td>8.310-307.245</td>
</tr>
<tr>
<td>BMC</td>
<td>1</td>
<td>-0.127</td>
<td>.036</td>
<td>0.001</td>
<td>0.881</td>
<td>0.820-0.946</td>
</tr>
</tbody>
</table>

Figure 1. a) Survivorship function for old spines by torso flexion angle (n = 36); b) survivorship function for young spines by torso flexion angle (n = 18).