Cortical-Trabecular Load Sharing and High-Risk Tissue Distribution in the Human Proximal Femur

+1Nawathe, S; 1Romens, A L; 2Fields, A J; 2Roberts B; 2Bouxsein M L; 1Keaveny, T M
+University of California, Berkeley, CA, 1Beth Israel Deaconess Medical Center, Boston, MA
Senior author tmk@me.berkeley.edu

INTRODUCTION:

Hip fractures are the most serious type of osteoporotic fracture in terms of mortality, morbidity, and social cost [1]. While the cortical and trabecular bone both contribute to femoral strength, there remains an incomplete understanding of cortical-trabecular load sharing within the femoral neck, how such load-sharing varies along the femoral neck, the distribution of tissue at high risk of initial failure, and the general sensitivity of this overall behavior to variations in the elastic modulus of the cortical and trabecular bone tissue. These fundamental issues are potentially important clinically from both diagnostic and therapeutic perspectives. Both aging and drug treatment can differentially alter cortical and trabecular bone tissue properties [2], and improved knowledge of cortical-trabecular load-sharing within the femoral neck, and insight into failure distribution and the associated micromechanics could eventually lead to improved methods for non-invasive assessment of bone strength in a clinical setting using lower resolution but targeted imaging modalities.

In this context, our goal in this study was to quantify the cortical-trabecular load sharing in the human femoral neck and identify the regions of most highly loaded tissue, i.e. the “high-risk” tissue [3], within the proximal femur. We also sought to determine the sensitivity of these outcomes, and the overall femoral stiffness, to variations in the elastic modulus of the cortical bone tissue. This study is unique since it is the first to quantify such load sharing using micro-CT-based high-resolution finite element analysis.

METHODS:

High-resolution CT scans (isotropic voxel size of 61.5 microns on a side; XreemCT, Scanco Medical AG, Switzerland) of one elderly human cadaveric proximal femur were used to generate a 3D finite element model having over 200 million 8-noded cube-shaped brick elements. In our baseline model, all elements were assigned the same tissue-level isotropic elastic modulus (E = 18.0 GPa) and boundary conditions were applied to simulate a sideways fall onto the greater trochanter [4,5]. Linear elastic finite element analysis was performed using customized parallel code running on a Sun Constellation Cluster supercomputer (Ranger, TACC) having 2176 processors. The main outcomes were: a) load sharing between cortical and trabecular bone along the femoral neck, as characterized by ratio of axial force (along the femoral neck axis) acting on cortical bone to the axial load acting on the entire cross-section; b) the amount and location of high-risk tissue, defined as the 10% of most highly strained tissue within the model [3]; and c) whole-bone stiffness. In a parameter study to assess the sensitivity of variations of the tissue modulus of the cortical bone with respect to the trabecular bone, the elastic modulus of the cortical tissue was varied from 14.4 GPa to 21.6 GPa while the elastic modulus of the trabecular tissue remained unchanged at 18 GPa.

RESULTS:

While the cortical shell supported the majority of the axial load over most of the femoral neck, there was a substantial variation in load sharing along the neck axis (Figure 1). Overall, the shell carried over 90% of the load near the base of the neck but only about 30% in the subcapital region, with an approximately linear variation in between. This load sharing was relatively insensitive to changes in the elastic modulus of the cortical tissue, particularly at the narrowest cross-section where the cortical load sharing was over 80%.

The patterns of high-risk tissue were very different for the cortical vs. trabecular tissue (Figure 2). For the cortical tissue, the high-risk regions occurred in tension primarily along the inferior cortex, and in compression along the superior aspects of the neck and neck-trochanteric junction. By contrast, for the trabecular tissue, most of the high-risk tissue was associated with high levels of compressive strain, in an almost continuous band extending from the loading region in the femoral head through the superior cortex in the neck and on into loading region at the lateral trochanter. Changing the cortical shell elastic modulus from 14.4 GPa to 21.6 GPa did not alter these distributions but increased whole-bone stiffness from 6618 KN/m to 7800 KN/m.

DISCUSSION:

These findings demonstrate the highly complex nature of the mechanical behavior of the proximal femur. Even within the femoral neck there is substantial variation in cortical-trabecular load sharing, and overall the spatial distribution of high-risk tissue is substantially different between the cortical and trabecular tissue and between tension and compression. While our findings cannot be generalized due to our analysis of just a single bone, these findings beg the question as to how the load sharing in the neck and the distributions of high-risk tissue vary with aging and between men and women. The sensitivity of overall femoral stiffness to the elastic modulus of the cortical tissue suggests that treatment-induced effects that target only the cortical bone may have alter overall strength since trends in whole-bone stiffness often track trends in strength, but fully non-linear analysis is required to properly address this issue. In terms of clinical assessment of femoral strength, these results suggest that neck fractures are associated primarily with failure of the cortical bone and that intertrochanteric fractures are associated primarily with trabecular failure — consistent with results from cadaver biomechanical testing studies [6] but requiring confirmation in further studies.

REFERENCES:


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