The spine research group at the Orthopedic Biomechanics Laboratory, located at Research North, 1 Overland Street, Boston, MA, is part of the research activity performed within the Department of Orthopedic Surgery at Beth Israel Deaconess Medical Center. Our research is directed at understanding the effect of age, disease and trauma on the structural properties and mechanical behavior of constituent musculoskeletal tissues underlying the structure and function of the vertebra and the intervertebral disc. These interests extend to the assessment of spinal instrumentation, current and novel treatment modalities and the development of imaged based diagnostic tools. The following sections present past and current work aimed at elucidating the processes underlying the biomechanics of vertebral failure.

1. MECHANICS OF VERTEBRAL FRAGILITY FRACTURES:

Vertebral fractures caused by bone fragility result in a patient population exhibiting prolonged and intractable pain and significant morbidity. Though extensive research highlighted the effect of osteoporosis on bone quality as an important mechanism in the etiology of vertebral fractures, the structural mechanisms underlying the initiation of fractures at specific vertebral levels within the thoracolumbar spine, demonstrating significantly higher propensity to undergo fracture independent of bone density, are not clear. Significantly, little is known about either the effect of various fracture patterns on the kinematic and residual structural stability of these vertebrae once failed, or the effect of the structural damage within the vertebra on the residual load carrying capacity of these vertebrae under functional loads.

Initial research efforts focused on quantifying the effect of severe fracture, defined as 50% reduction in the anterior height of the vertebral body, on the residual load carrying capacity of single osteopenic thoracolumbar vertebrae.[1] In response to un-constrained compression-flexion loading, the vertebrae demonstrated a complex multi-planar failure process independent of initial vertebral geometry or the distribution of density within the vertebrae. Notably, though clearly “mobile” under the applied load, once recovered, osteopenic vertebrae exhibited a spontaneous and significant recovery of their geometry and load carrying capacity often reaching up to 75% of their intact state. With the support of AO, NASS and OREF foundation grants, this work was extended to three-level osteopenic thoracolumbar spinal units. Using a custom six-degree-of-freedom testing device developed in our group, the failed spines were found to exhibit significant changes in kinematic response and a significant reduction in structural competence.[2] Importantly, although radiologically the fractured vertebrae were classified as demonstrating a “stable” fracture, under functional flexion-extension radiographs, the affected spines showed considerable “mobility” and marked changes in the kinematics of the neural canal.

With epidemiological studies suggesting that up to 50% of vertebral fragility fractures are caused by a fall event, we investigated the role of high rate loading conditions simulating a fall event on the mechanics of vertebral fracture.[3] This novel study revealed that, under high loading rate, both the structural response and the mechanism of failure were significantly different from that previously observed under commonly used low rate tests. Moreover, under flexion-extension radiographs, the failed segments could sustain only a small degree of loading prior to exhibiting high deformation/angulation values resulting in severe loss in the dimensional space afforded by the neural canal.

The results of these studies highlight the need for further research into the role of constitutive vertebral structural tissue in affecting the complex-multiplanar process underlying the failure of human vertebrae within the context of the biomechanics of the whole spine. Our current work aims to identify the role of regional differences in vertebral anatomy, kinematical constraints and the loading conditions on the role of each constitutive tissue in the process of vertebral failure. The outcome of such studies will help to better define whether osteopenic fractures can be truly classified as “stable” fractures and may suggest target for improved treatment modalities for the prevention of re-occurring as well as new fractures within the spine.

2. MECHANICS OF PATHOLOGICAL VERTEBRAL FRACTURES:

Annually, more then one million new cases of cancer occur in the US[4] with 7% to 27% of these patients ultimately afflicted with skeletal metastasis.[5] Vertebral bone is the most common site of metastasis in breast cancer and is the leading cause of death among women 40-44 years of age.[6] Pathologic vertebral frac-
tures, occurring in up to 50% of vertebral bone metastases, [7] are often associated with intractable pain, loss of function and other morbidities depending on the age of the patient, skeletal site and coexistent pathology such as osteoporosis.[8] Despite the extensive development of radiotherapy and systemic treatments,[9-12] current clinical guidelines for estimating fracture risk in adults with metastatic bone defects are poorly defined and have low specificity for predicting pathological fracture.[13, 14] Though retrospective clinical studies suggested defect geometry, pain, age, anatomic site, lesion type, and activity levels to be predictors of fracture risk, the effect of bone strength and applied loads in determining the risk for pathologic fracture remains unclear.

Previous work in our laboratory investigated the failure process of single vertebrae with a 40% non-contained defect within the vertebral body under compression-flexion loading.[15] Under increased loading, the vertebrae demonstrated a complex multi-planar failure process with the process initiated by local buckling of the cortex at the site of the defect followed by increasing involvement of the vertebral body as a whole. Notably, in contrast to vertebrae with no lytic defect, little or no restoration of vertebral geometry was observed for vertebrae with metastatic defect.

Using a novel computer controlled, CT compatible, mechanical testing system work has now been extended to investigate the effect of osteolytic defects on the structural response and failure processes of a complete spine with particular emphasis on the role of axial torsion as a determinant factor. Within a 64 array high resolution CT imager, test carried out under compressive loads (0-750N) using L2-L3 spinal unit instrumented with strain gauge rosettes, revealed the inclusion of non-contained defect to result in significant changes in the response and failure processes of a complete spine with paramechanical testing system work has now been extended to the process of single vertebrae with a 40% non-contained defect within the vertebral body under compression-flexion loading.[15]

References
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