Torsional Properties of Distal Femoral Cortical Defects

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abstract

The optimal management of pathologic long bone lesions remains a challenge in orthopedic surgery. The goal of the current study was to investigate the effect of defect depth on the torsional properties of the distal femur. A laterally placed distal metaphyseal cylindrical defect was milled in the cortex of the distal femur in 20 composite models. The proximal extent of the defects was constant. By decreasing the radius of the cylinder that intersected this predefined cord, 4 different radii defining 4 different depths of resection of the distal femur were created for testing: 17%, 33%, 50%, and 67% cortical defects, when normalized to the width of the femur at the level of resection. Each femur was mounted into a hydraulic axial/torsion materials testing machine and each specimen underwent torsional stiffness testing and torsional failure in external rotation. The specimens with less than a 33% cortical loss consistently demonstrated a superiorly oriented spiral fracture pattern, while the specimens with greater than a 50% cortical loss consistently demonstrated an inferiorly oriented transverse fracture pattern. The cortical defects were all statistically ($P<.05$) less stiff in torsion as the defect grew larger. There was a strong linear correlation between the mean torsional stiffness and cortical defect size ($r^2=0.977$). This observation is supported by finite element analysis. The amount of femur remaining is crucial to stability. This biomechanical analysis predicts a critical loss of torsional integrity when a cortical defect approaches 50% of the width of the femur. [Orthopedics. 2014; 37(3):158-162]

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The optimal management of pathologic long bone lesions remains a challenge in orthopedic surgery. Critical size bone defects from benign or malignant tumors occur in both adults and children. These defects cause pathologic fractures and result in a considerable source of pain and disability. According to 2007 data from the Centers for Disease Control and Prevention, the yearly incidence of tumors that typically metastasize to bone (ie, breast, prostate, lung, thyroid, and kidney) is approximately 231/100,000 and the incidence of multiple myeloma is approximately 5/100,000.\(^1\) Seven percent to 27% of the patients with a primary carcinoma will develop a bone defect.\(^2\) Hence, with the US population of 310 million, 65,000 to 210,000 new bone defects are diagnosed yearly in the United States. Accurately diagnosing the defects at high risk for fracture would facilitate timely and appropriate intervention.

Several investigators have described risk factors associated with pathologic fracture, such as patient age, associated pain, the lesion size, anatomic location, geometry, appearance, and metabolic activity.\(^3\)\(^-\)\(^6\) These risk factors are largely formulated from retrospective clinical data, and their clinical application does not correlate with the diagnosis of a high-risk bone defect.\(^7\) Possibly the best system for diagnosing high-risk bone defects in metastatic disease is Mirels’ rating system. Mirels’ rating system evaluates the radiographic amount of cortical erosion as 1 of 4 criteria.\(^6\) This system offers an 81% to 91% sensitivity and 33% to 35% specificity.\(^6\)\(^,\)\(^8\)\(^,\)\(^9\) It is a good screening tool but a poor diagnostic tool. Mirels’ rating system has a high false-negative rate, resulting in the unnecessary stabilization of 2 out of every 3 patients prophylactically stabilized.

Cortical destruction of more than 50% is another commonly referenced cutoff for the prophylactic stabilization of long defects. This recommendation is based on the initial evaluation of only 4 patients in 1970 with presumed impending fractures.\(^10\) This assumption was retrospectively evaluated and only 1 of 44 (2.3%) fractures occurred with less than 50% cortical destruction, while 35 of 44 (80%) fractures occurred with greater than 80% cortical destruction.\(^4\) Biomechanical data support a 12.7% to 62% reduction in torque at failure after 50% cortical loss in the diaphysis of canine femora.\(^11\)\(^,\)\(^12\) Retrospective evaluation of 110 femoral lesions with 14 fractures demonstrated that more than 50% cortical involvement was predictive of fracture, while Mirels’ score and other risk factors were not sufficient.\(^13\) The goal of the current study was to investigate the effect of defect depth, in the setting of cortical loss, on the torsional properties of the lateral distal femur.

**MATERIALS AND METHODS**

Fourth-generation composite femurs (Sawbones; Pacific Research Laboratories, Vashon, Washington) were used to decrease the specimen variability often seen in cadaveric specimens. A power analysis revealed that a sample size of 5 would offer the ability to detect a difference of less than 1 Nm (nanometer) between samples with 95% confidence and a statistical power greater than 80%.\(^11\)\(^,\)\(^14\)\(^,\)\(^15\) A total of 21 fourth-generation composite large left femurs were randomly divided into 5 groups, 1 group with 1 specimen and the other 4 groups with 5 specimens. A laterally placed distal metaphyseal cylindrical defect centered 90 mm proximal to the lateral femoral condyle was precision milled (pro-LIGHT Machining Center, Light Machines Corp, Manchester, New Hampshire). The diameter (ie, proximal-distal extent) of the cylindrical defects was 30 mm and constant. By decreasing the radius of the cylinder that intersected this predefined cord, 5 different radii defining 5 different depths of resection of the distal femur (ie, maximal lateral to medial distance) were selected for testing: 0-mm radius (intact, 0 cm, n=1), 135.5-mm radius (17% cortical defect, 0.70 cm), 78.2-mm radius (33% cortical defect, 1.40 cm), 63.8-mm radius (50% cortical defect, 2.10 cm), and 60.1-mm radius (67% cortical defect, 2.80 cm) (Figure 1). Cortical defect percent was determined by dividing the maximal depth of resection by the width of the femur at that location (Figure 1).
Each femur was mounted into a custom polymethylmethacrylate (PMMA) mold that applied force and torque to the distal femoral condyles distal to the defect. The proximal end of the femur was mounted in another custom PMMA mold. The femurs were mounted into a hydraulic axial/torsion servo hydraulic materials testing machine (Model 809, Axial-torsional Load Transducer, Model 662.20C-04, Multi-purpose Testware in the FlexTest GT controller; MTS Systems Corp, Eden Prairie, Minnesota) (Figure 2). Each specimen underwent axial compression and torsional testing to determine stiffness as well as torsion to failure. Previous studies evaluating the failure of femoral defects have focused on torsion.11,12 The choice of using external rotation was based on several studies that implicated torsion as the most likely failure mechanism of the femur, along with the fact that an external rotation of the tibia is also most likely when falling.16 Force and torque were applied through the anatomic axis of the femur to simplify the interpretation and extrapolation of data with respect to each defect. Torsional stiffness testing was then performed by loading each specimen from 0.5 to 12 Nm for 3 cycles at 1 Nm/s with axial preload held at 200 N. The range of torque applied was determined as 50% of the maximum torque in the linear range via preliminary testing of a 67% cortical defect to failure. Finally, each sample was failed in torsion at a rate of 1 Nm/s with axial preload held at 200 N. Failure was defined as composite bone fracture. Axial and torsional stiffness were calculated from the linear portion of the load-displacement and torque-angle curve, respectively. Data were analyzed via a Student’s t test with a Tukey Kramer adjustment for multiple comparisons. Errors are reported as standard error of the mean for stiffness incorporating all cycles or standard deviation for torque at failure. A P value less than .05 was considered statistically significant.

Finite element models were prepared from cylinders with a diameter, radius, and depth of resection similar to the femoral specimens described above (Figure 1). The diameter of the model was matched to the diameter of the unnotched femur at the site where the notches were placed. The models had a uniform modulus of 10,000 MPa and a Poisson’s ratio of 0.3. The mesh was created using ImageJ (National Institutes of Health, Bethesda, Maryland) at a 90×90×300 cubic resolution at 0.5 mm per element edge.17 The problem was solved using finite element software written in the authors’ laboratory.18,19 The polar moment of inertia was calculated for the smallest and circular cross-sections using the BoneJ plug-in to the program ImageJ.17 Ten degrees of torsion was applied to the top of the cylinders using rigid body displacements. The bottom of the cylinder was fixed in torsion but both the top and bottom surfaces were allowed to warp in the vertical direction.

RESULTS
Representative images of the fractured groups after testing are shown in Figure 3. The femurs with a 17% or 33% defect
consistently demonstrated a superiorly oriented spiral fracture pattern. The femurs with a 50% or 67% defect consistently demonstrated an inferiorly oriented spiral fracture pattern. The intact femur was determined to be too strong for failure with the testing apparatus used; however, the authors were able to obtain torsional stiffness measurements.

There was a strong linear correlation between the mean torsional stiffness and defect size ($r^2=0.977$, Figure 4A). The 17%, 33%, 50%, and 67% defects were all statistically distinct and less stiff in torsion as the defect grew larger ($P<.05$). The intact femur had a stiffness of 13.99 Nm/degree. The 17% defect had a stiffness of 11.58±1.05 Nm/degree (17.12% lower than the intact femur). The 33% defect had a stiffness of 7.17±0.52 Nm/degree (48.7% lower than the intact femur and 38.1% lower than the 17% defect). The 50% defect had a stiffness of 5.00±0.43 Nm/degree (64.2% lower than the intact femur, and 56.8% lower than the 17% defect, and 30.3% lower than the 33% defect). The 67% defect had a stiffness of 3.00±0.26 Nm/degree (78.6% lower than the intact femur, 74.1% lower than the 17% defect, 58.2% lower than the 33% defect, and 40.0% lower than the 50% defect).

There was no statistically significant difference in the torque at failure between the 17%, 33%, and 50% defects ($P>.05$, Figure 4B). However, there was a statistically significant decrease in torque at failure in the 67% defect group ($P<.05$, Figure 4B). There was a 20.9% decrease in the failure torque between the 50% and 67% defect, from 46.8±8.4 Nm to 37.0±1.6 Nm, respectively.

A simplified cylindrical finite element model was created of the intact femur as well as each defect to simulate the force distribution at each defect site during the torsional loading (Figure 5A). There was a progressive concentration of forces at the point of maximal defect. The torsional stiffness of the finite element models and the experimental stiffness values were correlated ($r^2=0.941$, Figure 5B). The normalized torsional stiffness of the experimental samples correlated very tightly with the torsional (ie, polar) moment of inertia relative to the polar moment of the undamaged cross-section of the cylinder ($r^2=0.983$, Figure 5C).

**Discussion**

The biomechanical data support the “50% rule” or “Harrington’s criteria” in cortical destruction. The torsional stiffness, torque at failure, and fracture pattern change significantly once the cortical defect reaches or crosses this threshold. This biomechanical observation supports data regarding the natural history of cortical defects with few pathologic fractures occurring with less than 50% cortical destruction and the majority of pathologic fractures occurring with more than 80% cortical destruction.4

The fracture pattern switches from a spiral pattern to a transverse pattern with 50% cortical loss. There is a qualitative decrease in torsional strength, an increase in angular displacement at failure, and an inability of the bone to resist the applied load. Finite element analysis supports the observation that resistance of torsional

![Figure 4](image1.png)

**Figure 4:** Correlation of defect size with torsional stiffness (error bars reported as standard error of the mean) (A). Torque at failure for each defect (error bars reported as standard deviation) (B). The asterisk indicates a statistically significant difference ($P<.05$).

![Figure 5](image2.png)

**Figure 5:** von Mises shear stress in each finite element model at 10° of rotation. The solution was linear, using 8 node trilinear brick elements. The samples are, from right to left, 67% cortical width defect, 50% cortical width defect, 33% cortical width defect, 17% cortical width defect, and intact (A). Linear correlation of the experimental femur torsional stiffness to the predicted torsional stiffness of the undamaged cross-section of the cylinder (B). Linear correlation of the normalized torsional stiffness of the finite element model (left) and the experimental femurs (right) to the torsional (ie, polar) moment of inertia relative to the polar moment of the undamaged cross-section of the cylinder (C).
load is linearly related to the polar moment of inertia around the remaining bone. This observation is also reproduced in the measurement of ultimate load; once the defect exceeds 50% cortical loss, there is a rapid and dramatic decrease in torque to failure. The torque at failure was well above the amount of peak axial torque (7 Nm) experienced by the femoral shaft during level walking. However, 40 Nm may not be sufficient for more strenuous activity or a fall.

Transcortical defects have been investigated in sheep femora. The reduction in torsional strength was linearly related to cortical defect size, while cortical wall thickness and bone curvature had little to no effect on torsional strength. In addition, circular defects in canine femora resulted in a linear decrease in torque to failure with defect sizes from 20% to 60%. However, if the entire range is considered, these data appear sigmoidal in nature. The current authors observed dramatic losses in torsional stiffness from 33% to 50% cortical loss. A 50% cortical defect had a torsional stiffness of 5.00±0.43 Nm/degree, which was 64.2% lower than the intact femur.

Although this does not mean that 50% cortical loss will predict fracture risk, it implies that the ability of the bone to resist normal torsional loads will remain intact up to 50% cortical loss. While the amount of femoral disruption in a cortical breach appears to be critical for resisting fracture in torsion, it is not the only determinant of prophylactic stabilization. Activity level, pain, lesion size, anatomic location, geometry, and metabolic activity play a critical role as well. Despite the difficulty in defining the extent of a cortical lesion clinically, it is clear that the amount of cortex remaining is critical.

Synthetic bones may not reproduce the failure mechanism of a living or cadaveric femur. Their mechanical properties have previously been demonstrated to be within the range of cadaveric femora under axial loading, bending, and torsional tests. In addition, the inter-femoral variability was 20 to 200 times higher in cadaveric femora compared with synthetic femora, which makes synthetic preferable to cadaveric in biomechanical analyses with a small sample. The use of composite femoral models precludes an analysis of the possible benefits of oral or intravenous bisphosphonate therapy and current multidisciplinary systemic treatments in the setting of metastatic disease. Isolating failure in external rotation represents a sufficiently simple and relevant biomechanical setup to address a clinical scenario. However, the authors acknowledge that this does not and cannot address all modes of clinically relevant failure.

**REFERENCES**