ABSTRACT

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"Stress Taper" Strategy in Plate Fixation Increases Failure Strength in a Cadaveric Femur Model

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Abstract

Purpose:

To potentially limit peri-implant fractures, our institution commonly implements a fracture fixation strategy known as a "stress taper," in which the screw lengths toward the end of a construct are incrementally decreased. The premise is to dissipate the stress as one moves proximally in order to avoid a focal stress-riser when loaded. The aim of this study was to determine if the stress taper strategy increases torsional strength and fails in a simpler fracture pattern than the bicortical locking construct when biomechanically tested in a cadaveric femur model.

Methods:

Including an assessment of the failure mechanism Seven matched pairs of cadaveric femora were randomly assigned to 1 of 2 distal femur fixation groups: plating with stress taper strategy or bicortical fixation. Both strategies used locking plates and the same number of locking screws in the same positions. Specimens were first cyclically loaded in axial compression and torsion. Specimens were then axially rotated to failure under 800 N of compression. Peak torque at failure and degrees of rotation at failure were calculated and compared using paired t tests. Fractures were categorized with the assistance of fluoroscopy according to the OTA 32.

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Biography: Jeremy Raducha's research program is focused on un- derstanding the epigenetic neural gene control mechanisms that govern regulation of higher order brain function via chro- matin packaging control in neurons. Her research group focuses on understanding the role(s) of specific HATs in cognition and neurogenerative disorders such as Alzheimer's disease (AD). Her research group generated a robust Drosophila model sys- tem that enables them to modulate Tip60 HAT levels in neural circuits of choice under AD neurodegenerative conditions, in vivo. Its use led to their exciting discovery that Tip60 is critical for cognitive processes and protects multiple cognitive neu- ral circuits impaired in the brain during early AD progression. Her group is currently deciphering the mechanisms underlying Tip60 HAT action in neuroprotective gene control using fly and mouse AD models and determining how these Tip60 epigeneticprocesses go awry in the brains of human AD patients.