

was the inserts choice ( $\beta = -0.08$  mm/N,  $p = 0.012$ ). In extension, the difference between estimated gaps and measured gaps was  $> 1$  mm for 36% of all assessments and 91% of gaps were underestimated. Only one measure, however, was underestimated by  $> 2$  mm. In flexion, gap estimates were  $> 1$  mm for 35% of all measurements and 59% of all measurements were overestimated. Four measures were overestimated, and one was underestimated by  $> 2$  mm. DISCUSSION/SIGNIFICANCE OF IMPACT: We found that the applied forces varied among surgeons and a negative association between insert thickness and forces in extension valgus exam. We also found that error in gap estimates among surgeons was  $> 1$  mm a third of the time and that underestimation is more common in full extension, which may lead to using smaller inserts that affect knee stability. CONFLICT OF INTEREST DESCRIPTION: The corresponding author has no COI but my coauthors had the following COI:

1. Royalties from a company or supplier: Zimmer; Stryker; Exactech, Inc; Lima; Mathys Ltd.
2. Speakers bureau/paid presentations for a company or supplier: Acelity; Flexion Therapeutics; Smith & Nephew; Exactech, Inc; Mallinckrodt Pharmaceuticals; Stryker.
- 3B. Paid consultant for a company or supplier: Acelity; DePuy Synthes; Exactech, Inc; Flexion Therapeutics; Intellijoint; Smith & Nephew; Zimmer; Stryker
4. Stock or stock options in a company or supplier: Imagen; Insight Medical; Intellijoint; Parvizi Surgical Innovation; OrthAlign; Orthobond.
5. Research support from a company or supplier as a Principal Investigator: Acelity; Exactech, Inc; Intellijoint; Smith & Nephew; Mallinckrodt Pharmaceuticals; Stryker; Lima.
6. Royalties, financial or material support from publishers (The following conflicts were disclosed) Exactech, Inc.
7. Medical/Orthopaedic publications editorial/governing board: Bone and Joint Journal 360; Journal of Orthopaedics and Traumatology; Techniques in Orthopaedics.
8. Board member/committee appointments for a society: Knee Society; Eastern Orthopedic Association.

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### Recovery Time is Exaggerated in Individuals with Degenerative Cervical Myelopathy Following Standing Lateral Waist Pulls

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OBJECTIVES/GOALS: The aim of this study was to quantify balance impairments in stance in individuals with degenerative cervical myelopathy (IwDCM) in response to external perturbations. IwDCM have damage to their spinal cord due to degeneration of the cervical vertebral column, but little is known about balance. METHODS/STUDY POPULATION: Recovery time following a perturbation may be an important measure of balance. Changes in recovery time were measured in 7 IwDCM (2m, 58.59±15.00y) and 6 controls without DCM (2m, 56.91±11.04y) as they stood on an instrumented treadmill and received cued (predictable) and uncued (unpredictable) lateral pulls to the waist at 12% (high) and 6% (low) pull magnitudes. Individuals stood with feet together,

shoulder width, and wide. Recovery time was defined as the time following pull onset when the absolute value of the center of pressure velocity returned to  $< 1x$  baseline standard deviation. Repeated measures ANOVA was performed on recovery time. RESULTS/ANTICIPATED RESULTS: We anticipate that feet together standing, unpredictable, higher magnitude perturbations will be most challenging evidenced by longer recovery times. For waist pull recovery time, there was a trend for a Group x Predictability x Magnitude x Stance Width interaction ( $p = 0.1$ ) which we anticipate being greater with additional participants. There were significant Group x Predictability x Stance Width ( $p = 0.01$ ) and Group x Magnitude x Predictability ( $p = 0.01$ ) interactions. IwDCM had exaggerated recovery times in narrow and wide stances with unpredictable pulls. IwDCM recovered more slowly in response to unpredictable higher magnitude pulls. DISCUSSION/SIGNIFICANCE OF IMPACT: Balance responses in IwDCM are most impaired in narrow stances and when perturbations are unpredictable. Rehabilitation should focus on shortening latency of response timing and increasing power utilization during balance response to promote quicker recovery.

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### Refined structure of human ferroportin using restraints from mass spectrometry

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OBJECTIVES/GOALS: Mammals require iron for hemoglobin, respiration, immunity and as cofactor in enzymes. But free iron is toxic from the production of reactive oxygen species. Ferroportin is the sole exporter of cellular iron and it crucially determines cellular and systemic iron levels. Labile iron must be tightly regulated. This requires structural understanding. METHODS/STUDY POPULATION: We built structure of human ferroportin (FPN1) using the ab initio prediction approaches of Rosetta/Robetta and by comparative modeling with distance restraints in MODELLER. Templates selected were from solute carrier protein families of distantly related orthologs and homologs including a proton coupled peptide transporter (PDB ID: 4IKV) and the bacterial iron transporter in outward-open and inward-open states, (PDB ID: 5AYM, 5AYO). Each model was validated by experimental mass spectrometry data. The energy minimized structural model was inserted into a lipid bilayer, placed in a rectangular simulation box, covered with TIP3P water solvent balanced with counterions and conditioned. Finally, we carried out 350 nanoseconds molecular dynamics simulations. RESULTS/ANTICIPATED RESULTS: Our first model of FPN1 (571aa), using Rosetta/Robetta *ab initio* approach, resembles the structure of the proton-dependent transporter, POT and consists of 12 transmembrane helices. The membrane spanning helices veer away from the orientation in the structure of 4IKV. The alternate model using MODELLER and the method of satisfaction of constraints, returned one template, the structure of *Bdellovibrio bacteriovorus* iron ( $Fe^{2+}$ ) transporter homolog (5AYN, 440aa) with sequence identity of 19%. Aligning FPN1 on the template sequence incorporating structural information revealed better conservation (29%). This model also comprises 12 transmembrane helices in two bundles separated by a large intracellular loop. The iron binding site predicted in both models match