INTRODUCTION:
Proximal junctional kyphosis (PJK) is being reported with increasing frequency following posterior spinal instrumentation. Recent studies have reported the incidence at 26% (7.3 years postoperatively) for adolescent idiopathic scoliosis (Kim et al, Spine 2005) and 26% (5.3 years postoperatively) for adult spine deformity (Glattes et al, Spine 2005). The principle variables that may be influencing this increased incidence appear to be implant construct stiffness, upper instrumentation vertebral level selection, residual alignment, disruption and derervation of the dorsal musculature, and disruption of stabilizing posterior thoracic spine elements. The purpose of this study was to determine the effect of upper-instrumented vertebral anchor site preparation, with resultant posterior structure destabilization, on thoracic spine motion segment stability.

METHODS:
Thoracic spine (T1-10) from 6 fresh cadavers, average age 78 (62-99) years, were separated into 30 motion segments. The connecting ligaments and intervertebral disc of each motion segment were left intact while all extraneous soft tissue and musculature was dissected away. If spanning osteophytes were present around the anterior and/or lateral sides of an intervertebral disc, they were removed with a rongeur to facilitate physiological anterior column motion. Each motion segment was then mechanically mounted onto two aluminum plates (one mounted to the superior vertebral body, and one mounted to the inferior vertebral body of the motion segment) using a series of threaded rods, I-bolts, and nuts. Each motion segment was then mounted into the testing machine. A total of 23 motion segments were tested. Seven motion segments were excluded due to difficulties encountered during dissection and mounting of the specimens.

Flexion testing was performed on each intact motion segment and following each of a series of 6 destabilizing procedures: bilateral suprtransverse process hooks (Supra TP), bilateral supra-lamina hooks (Supra LH), bilateral pedicle screw placement (PSI), screw removal (PSO), supra and interspinous ligament transection (S & 1 spinous), and complete transection of all remaining posterior structures (All Posterior). Order of testing was held constant with variability being restricted by the increasing destructiveness of the procedures.

A pure flexion bending moment was applied to each motion segment using a MiniBionix Testing System augmented with an additional two-axis Spine Simulator (MTS Systems Corporation, Eden Prairie, MN) (See Figs 1 and 2). Each motion segment was flexed to an angle of approximately 3.2 degrees at a cyclic rate of 0.1 Hz while corresponding torques were recorded. Each motion segment was conditioned by flexing the specimen a total of 4 times and data was collected for analysis on the fourth cycle. Flexion angle data used in the analysis was recorded independently using a video extensometer (ME46, Messphysik, Austria). A constant between-test interval of three minutes was maintained. For each specimen, one of the tests following a single procedure was randomly selected to be repeated as a sham to check for repeatability and time dependent effects.

RESULTS:
As percent change of total intact motion segment stiffness (Fig. 3), each of the first 5 destabilizing procedures produced a small but significant reduction in mean motion segment stiffness when compared to zero (p<0.05, T-tests). Together, the first 5 destabilizing procedures resulted in a mean 23% (p<0.0001, T-test) loss of total motion segment stiffness (Fig. 4). Resection of the remaining posterior structures (facet joints and all other posterior soft tissue structures) produced an additional loss of approximately 45% of the intact motion segment stiffness. Interestingly, the anterior column provided only 32% of the total motion segment stiffness.

None of the first 5 destabilizing procedures produced a loss in motion segment stiffness that was significantly more than any other procedure (p<0.05, ANOVA, Tukey HSD test). It was noted, however, that the transection of all remaining posterior structures produced a loss in motion segment stiffness that was significantly more than any of the first 5 destabilizing procedures (p<0.001, ANOVA, Tukey HSD test). Based on the sham tests, no substantial cumulative fatigue effect was observed in the testing.

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DISCUSSION:
Limitations of this study include the cumulative nature of disruptive testing, possible order effect, and the specimen population. Age and condition of spines might not fairly represent a younger adolescent population whose discs would contribute much less to total flexural stiffness.

Overall, the results of this study suggest that the posterior anatomical structures do play a significant role in stabilizing thoracic motion segments during flexion. Although each of the commonly used destabilizing procedures alone did not contribute immensely to the overall loss in motion segment stiffness, it does appear that a combination of these procedures could play a clinically significant role in destabilizing the upper instrumented motion segment following posterior spinal instrumentation. These findings support a practice of preserving posterior junctional structures during posterior instrumentation surgery.