

WHIPLASH CAUSES INCREASED LAXITY OF CERVICAL CAPSULAR LIGAMENT

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INTRODUCTION

Whiplash neck injuries, caused by relative acceleration between the head and thorax during motor vehicle collisions, produce acute and chronic neck pain, headache, dizziness, and parasthesias in the upper extremities. Clinical studies have targeted the cervical facet joints and capsular ligaments (CLs), as sources of chronic pain in whiplash patients (Barnsley et al., 1995; Lord et al., 1996). The purpose of this study was to determine whether whiplash caused increased CL laxity by applying quasi-static loading to whiplash-exposed and control bone-CL-bone preparations.

METHODS

Facet-CL-facet specimens were prepared from 12 osteoligamentous whole cervical spines (6 whiplash-exposed and 6 control) (Tominaga et al., 2006). The whiplash-exposed spines had been previously rear impacted using the incremental trauma protocol at a maximum peak T1 horizontal acceleration of 8 g (Ivancic et al., 2005). All spines had no history of any disease that could have affected the osteoligamentous structures. The spines were divided into two equal groups with each group consisting of three whiplash-exposed and three control spines. Facet-CL-facet specimens were prepared using C2/3, C4/5, and C6/7 spinal levels in the first group and C3/4, C5/6, and C7/T1 spinal levels in the second group. Left and right facet-CL-facet specimens from each spinal level were prepared

separately and then mounted for mechanical testing (**Figure 1**). In total, 66 facet-CL-facet specimens were prepared.

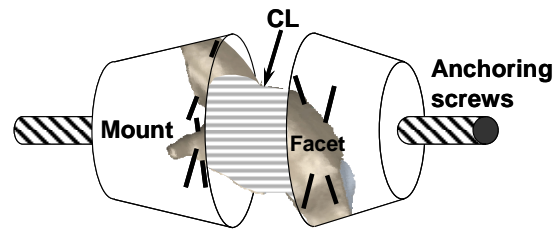


Figure 1. Facet-CL-facet preparation.

Each specimen was mounted in a custom designed, displacement-controlled mechanical testing apparatus (Panjabi et al., 1996). To standardize the neutral CL position, the facets were preloaded to 1 N of compression immediately prior to testing and this was defined as zero CL elongation. The facet-CL-facet specimen was elongated at 1 mm/s in increments of 0.05 mm until a tensile force of 5 N was achieved and subsequently was returned to neutral position. Force and elongation data were recorded at each motion step following a 0.5 second rest period. Four pre-conditioning cycles were performed and data from the load phase of the fifth cycle were used for subsequent analyses.

CL elongation was computed at tensile forces of 0, 0.25, 0.5, 0.75, 1, 2.5, and 5 N. Data from left and right CLs and all spinal levels were combined within each group. Students unpaired t-tests ($P < 0.05$) were performed to determine significant differences in CL elongation between the whiplash-exposed and control groups.

RESULTS

The force-elongation curve is shown for each whiplash-exposed CL (**Figure 2A**), control CL (**Figure 2B**), along with the average curves (**Figure 2C**).

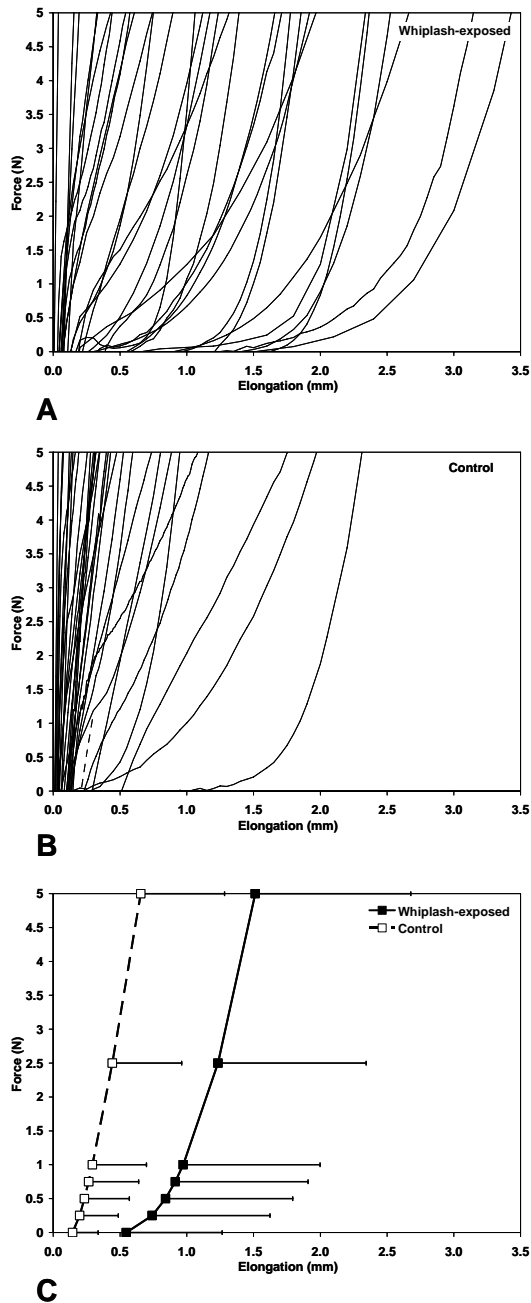


Figure 2. CL force-elongation curves: A) whiplash-exposed, B) control, C) averages.

The average elongation of the whiplash-exposed CLs was significantly greater than that of the control CLs at each tensile force. The difference between the average elongation of the whiplash-exposed and control CLs progressively increased from 0.4 mm ($P=0.021$) at 0 N to 0.9 mm ($P=0.007$) at 5 N.

DISCUSSION AND CONCLUSIONS

Whiplash injuries and the causes of the resulting chronic symptoms are not fully understood. The present study documented statistical increases in CL laxity due to whiplash. The average force-elongation curves were nonlinear, with greater flexibility at low forces and increasing stiffness at higher forces. Greater flexibility was generally observed in the whiplash-exposed CLs, as compared to the control CLs, particularly at low forces. Increased CL laxity due to whiplash may lead to chronic pain and clinical instability.

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