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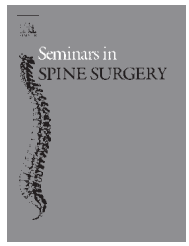

Patient Mechanisms of Injury in Whiplash-Associated Disorders

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ABSTRACT

Whiplash-associated disorders constitute a large proportion of cervical injuries. The posteroanterior acceleration loading from rear-end motor vehicle crashes results in motions of the head–neck complex. This manuscript describes the various mechanisms of injury, including hyperextension kinematics, hydrodynamic forces (pressure gradient in the spinal canal), eccentric contraction (muscle focus), and combined axial and shear loading (local variations in the kinetics of different segments of the cervical spinal column). The anatomic components of the cervical spine that may be involved in functional alterations and that result in acute and chronic symptoms are discussed along with demographic and vehicular factors.

A majority of whiplash injuries occur in low-speed rear-end motor vehicle impacts.[1] During the impact, the seated occupant experiences single-cycle posterior-to-anterior (PA) acceleration loading. The dynamic load is applied to the torso and head–neck complex of the occupant through the seat. The sequence of the external insult is as follows: upper back and shoulders are loaded initially, followed by head contact with the head restraint, and subsequently, the head accelerates forward and rebounds. Although a single mechanism of whiplash injury has not been conclusively accepted, different mechanisms have been proposed to result from each of these events. Vertebral fractures are uncommon because of the lack of the necessary axial force to the osteoligamentous cervical spinal column.[2-4] However, soft-tissue components such as ligaments, facet joints, muscles, and/or disk annulus can sustain subcatastrophic structural damage, often not able to be diagnosed by computed tomography and magnetic resonance imaging, even acutely after the impact event. Long-term consequences of these injuries have significant societal impact in the Western world, with annual estimated costs exceeding billions of dollars in the United States alone.[5,6] Early degeneration and enhanced segmental mobility can lead to surgical options. Based on the loading and kinematics of the head–neck complex, theories have been advanced on the mechanisms of soft-tissue injuries. Mechanisms of whiplash injuries from anatomical and structural perspectives using biomechanical principles and clinical observations are reviewed in this article.

1. Overall Motions of the Head–Neck Complex

The external PA acceleration loading imparts motions to the head–neck complex, which may result in injuries. The overall motions of the belted occupant with respect to the seat and head restraint are shown (Fig. 1). Occupant kinematics can be described in 3 stages. The first stage involves loading of the upper torso and shoulders from the rear through the back of the seat, and during this initial acceleration phase, the head remains stationary. The forward motion of the thorax and shoulders with respect to the stationary head results in head

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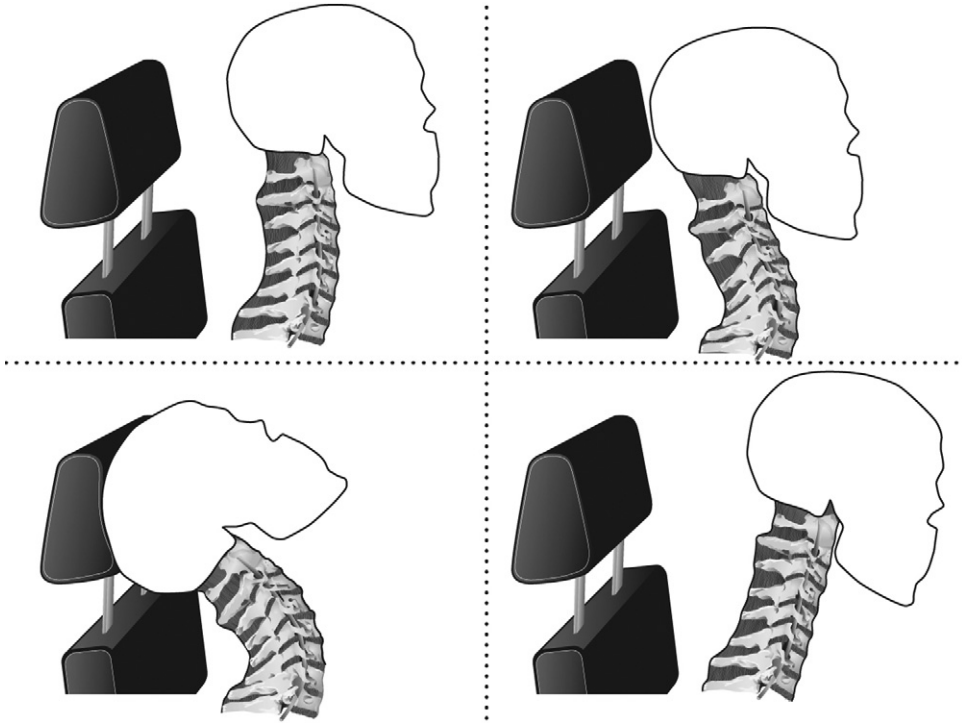

Figure 1 – Phases of head–neck and cervical spine kinematics: initial position (top left), S-curve (top right), extension (bottom left), and rebound (bottom right).

lag, a phenomenon documented in human volunteer and human cadaver biomechanical studies (Fig. 1, top left).[1,5,7-10] During this retraction phase of the PA acceleration loading event, forceful extension of the lower cervical spine occurs, whereas the upper segments sustain flexion, resulting in a transient nonphysiological curvature (ie, S-curve) of the cervical spine (Fig. 1, top right). As the torso continues to displace anteriorly relative to the head, the cervical spine transitions into extension and the head rotates backward to strike the head restraint (Fig. 1, bottom left). Depending on the seat and head restraint design, PA acceleration loading during this intermediate phase results in varied degrees of extension of the lower cervical spine with concomitant extension of the upper segments (ie, C-curve). The third and final stage involves the rebound phase of the head–spine complex motion, wherein the head rebounds from the head restraint and cervical curvature transitions into flexion (Fig. 1, bottom right). The local effects of flexion and extension signatures of the cervical spinal components are often used to delineate mechanisms of injury, as described later in the text.

2. Whiplash Tissue Injury Mechanisms

Hyperextension Mechanism

This mechanism is based on the extension signature of the cervical spine, focusing on the second stage of the PA acceleration loading event. Extension results in stretch of the anterior longitudinal ligament and anterior annulus of the intervertebral disk (Fig. 2). Thus, injuries stemming from this mechanism are primarily confined to, or initiate in, the anterior column of the spine. Instabilities can occur due to increased laxity in the anterior regions of the disk joint(s) after subcatastrophic failure. The degree of local extension during the middle stages of the applied PA acceleration loading influences the degree of instability and chronicity of symptoms. Due to laxity or local tears of the anterior longitudinal ligament, outer layers of the annular fibers may have decreased stiffness at the segmental level under subsequent physiolog-

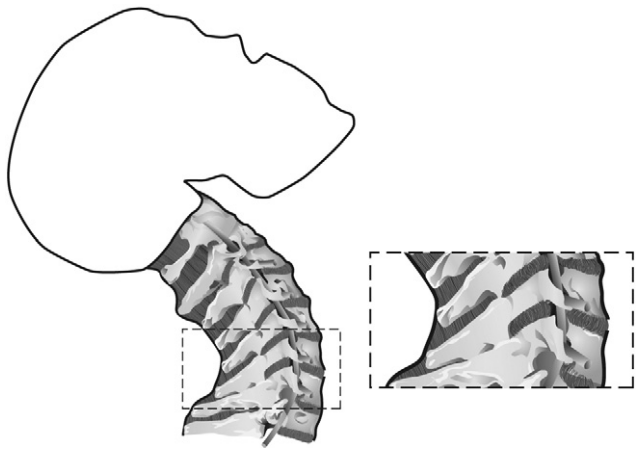

Figure 2 – Hyperextension mechanism of injury. Localized segmental extension before or during head restraint contact can distract anterior column soft tissues beyond their physiological limits, resulting in sub- or catastrophic tissue injury.