The Role of Torsion in Cervical Spine Trauma

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A dynamic servocontrolled torsion machine has been used to characterize cervical injury due to pure rotation of the head. Resultant force moment, torque, and applied rotation have been measured. Torque applied to the base of the skull resulted in injury to the atlantoaxial joint. No evidence of lower cervical injury was observed by computed tomography, magnetic resonance imaging, *in situ* fluoroscopy, or visual inspection. Torque applied directly to the lower cervical spine induced ligamentous injury and unilateral facet dislocation; however, the torque to injure the lower cervical spine was significantly greater than the torque to injure the atlantoaxial joint. It was concluded that pure rotation of the head does not mediate lower cervical ligamentous injury because of the comparative weakness of the atlantoaxial joint. [Key words: torsion, lower cervical spine, kinematics, injury]

TORSION HAS RECEIVED considerable attention as one of the principle mediators of ligamentous injury and dislocation in the lower cervical spine.[15,19] In the clinical setting, loads are typically transferred to the cervical spine as a result of forces applied to the head. However, no experimental study has been performed in which lower cervical injury has been produced as a result of torsional loads applied directly to the head.

To study the torsional responses of the spine, many authors have used static weight-pully systems.[16,30] These systems have successfully described the stiffness and kinematic responses of the spine, but are unsuitable for injury analysis given the importance of viscous effects.[17] Dynamic test systems typically provide the necessary control for repeatable injury production, but they constrain the specimen to motions defined by the hydraulic actuator. The need to reproduce physiologic motions while using such dynamic devices is fundamental to the creation of clinically observed injury models.

This paper describes the use of a dynamic test machine to measure the kinematic parameters associated with torsion, and to determine the role of torsion in cervical spinal injury.

BACKGROUND AND SIGNIFICANCE

Kinetics

The motions of the cervical spine have been studied in various ways,[8,16,22,28] and range of motion has been reported by a number of authors.[8,12,21,28]

The occipitoatlantal joint is irrotational.[24] In contrast, the atlantoaxial joint shows striking mobility, accounting for approximately 50% of normal cervical rotation.[29] Normal rotation occurs about the odontoid process. Atlantoaxial rotation is coupled to both axial displacement and lateral bending.[12–14,25]

The kinematics of the lower cervical spine are more complicated. Coupling of lateral bending and rotation has been observed.[16] This coupling causes the spinous processes to rotate into the convexity of the lateral curve.

An orthogonal radiographic technique has been used to study the lower cervical center of rotation.[16,22,23,27] The radiographic method, developed by Lysell[16] and used by White and Panjabi,[29] identified the center of rotation as the anterior portion of the vertebral body along the midsagittal line. Although accurate, this technique requires a considerable amount of data reduction and is suitable only for static domain testing.

Adams and Hutton[1] used a dynamic method to determine a center of rotation for lumbar segments. They defined the point of minimum stiffness on the midsagittal line as the center of rotation, but they had difficulty identifying a midsagittal axis of twist. Subsequent work by Yang et al[30] reported the existence of two centers of rotation in the lumbar spine away from the midsagittal line, one for each direction of rotation. Adams and Hutton's difficulty identifying an axis of twist appears to have evolved from the direction-specific nature of the lumbar center of rotation.

Injury

The importance of torsion in spinal injury has been described previously.[10,15,19] It is thought to influence both the modality and the ease with which injury occurs.[4,15,19,29] Torsion is also thought to play a fundamental role in the development of lumbar disc degeneration.[6,7]

In the upper cervical spine, rotation of the head causes rotary dislocation of the atlantoaxial joint at low loads.[11] This represents luxation of the lateral mass contralateral to the direction of rotation of the head, with subsequent locking secondary to muscle spasm. The lesion is thought to damage only the capsular ligaments of the dislocated lateral mass; however, odontoid fracture or transverse ligament rupture can occur, and may result in gross neurologic deficit. Rotational injury to the occipitoatlantal joint has not been observed.[23] Goel et al[9] produced capsular ligament and alar ligament damage by applying pure torque to isolated upper cervical spines (O–C2).

In the lower cervical spine, unilateral facet dislocation and posterior ligamentous injury have been attributed to rotation; however, controversy exists. Roaf[19] reported that torsion was required to produce ligamentous injury and dislocation. In his experiments, load was applied manually, directly to the lower cervical spine. Huelke et al[15] noted that rotation produced unilateral dislocation. Rogers[20] and White and Panjabi[28] noted that the unilateral dislocation was the result of an exaggeration of normal coupling of lateral bending and rotation, but did not state the load required to produce the lesion. Braakman and Vinken[4] suggested that unilateral dislocation was the result of combined flexion and rotation. Torg[24] suggested that the lesion resulted from compression. Bauze and Ardran[2] produced dislocation in cadaveric specimens

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by applying combined flexion and compression to the occiput and artificially constraining the lower cervical spine by inserting a peg in the neural canal at the desired level of injury. Their apparatus applied no torque to the specimen, yet lower cervical rotation was observed before unilateral dislocation.

Despite claims that unilateral dislocation is caused by rotational loads, the lesion has not been produced in an experimental study of rotational loads to the head. We therefore chose to investigate this issue, hypothesizing that it is impossible to induce lower cervical ligamentous injury or dislocation in rotation because of the relative weakness of the atlantoaxial joint.

MATERIALS AND METHODS

The test system used has been described in a previous publication, and is briefly summarized below.[18]

Cadaveric Material and Casting Procedure. Six intact, unembalmed cervical spines were obtained from cadaveric specimens aged 68–80 years. Specimens included the base of the skull through T1, with all ligamentous structures intact. Specimens were sealed in plastic bags and stored at −20 C. At the time of testing, the specimens were thawed to room temperature and allowed to equilibrate fully in a 100% humidity environment. Specimens were cast in aluminum cups with reinforced polyester resin. The resting lordosis of the spine was preserved. The centers of the cups were aligned with the center of the neural canal. The distance from the cup center to the dens (d) in the rostral cup and the distance from the cup center to the anterior of the vertebral body (L) in the caudal cup were recorded.

Apparatus. Tests were conducted with an MTS (Materials Testing Systems, Minneapolis, Minnesota) servocontrolled hydraulic torsion testing machine. The base of the skull was rotated using a hydraulic rotational actuator (Figure 1). Rotation of the base of the skull was quantified with a rotational variable differential transformer (RVDT) mounted directly on the rotary actuator. A system to permit load-free changes in axial length of the specimen was implemented with a linear bearing to couple the base of the skull to the rotational actuator. The caudal end of the specimen (T1) was not allowed to rotate. Torque, force, and moment were measured at the caudal end of the specimen by the use of a six-axis array of strain-gauge load cells. A dial gauge was used to align the specimen along the axis of twist and identify the location of the axis of minimum stiffness. *In situ* fluoroscopic images were recorded on videotape. Digital and analog data acquisition systems were used to determine the deformation-time, force-time, and force-deflection histories.

Experimental Methods. Pretest anteroposterior and lateral radiograms were performed before casting to evaluate existing pathology. Each specimen was mounted in the load frame and aligned to place the dens along the axis of twist of the actuator. As an initial estimate, the lower cervical spine was mounted to align the anterior portion of the C7 vertebral body with the axis of twist. A cyclic torsion test was performed with a 1-Hz sine function for 50 cycles to exercise the specimen and place it in a mechanically stabilized (reproducible) state.[17] The angle of twist was estimated to produce 10–20% of the expected injury torque.

A minimum stiffness protocol was performed to identify the center of rotation in the lower cervical spine. The lower cervical spine was mounted such that the axis of twist lay on the midsagittal line anterior to the C7 vertebral body. A ramp-and-hold rotation was applied for 0.5 seconds, and the dynamic torsional stiffness was recorded (stiffness = torque/twist angle [K = T/θ]). This was repeated two times and the results averaged. The axis of twist was moved 0.08 cm (0.2 in.) posteriorly, and the stiffness tests repeated. The procedure was performed at points located from the anterior of the vertebral body through to the center of the neural canal. A third-order polynomial was least-squares fitted to the data to determine the point of minimum stiffness along the midsagittal line. This point was defined as the lower cervical center of rotation, and it was placed along the axis of twist for the remaining tests. The specimen was then injured by applying a ramp-to-failure at approximately 500°/sec. Magnetic resonance images (MRI) and computed tomographic (CT) scans were obtained to identify bony and ligamentous injuries. Torque to injury was also measured.

All injuries produced were confined to the atlantoaxial joint. The joint was dissected and the injuries described. Since no evidence of lower cervical injury was observed, the specimens were recast to isolate the lower cervical spine (C2–T1) and a second failure test performed. Lower cervical torque to injury was measured and compared with torque to injury in the upper cervical spine. CT, MRI, and physical dissection were used to quantify the lower cervical injuries. The strength ratio, R, was defined as the ratio of torque to injury in the lower cervical spine over the torque to injury in the upper cervical spine.

RESULTS

Kinetics

A sample of the torque-angle response used to determine the dynamic stiffness, K, is shown in Figure 2. The center of minimum stiffness in the lower cervical spine was found to lie in the anterior portion of the vertebral body. A sample plot of normalized torsional stiffness (K/Kmax) versus normalized midsagittal position (X/L), used to identify the location of the center of minimum stiffness, is shown in Figure 3. The symbol X represents the distance from the center of the neural canal

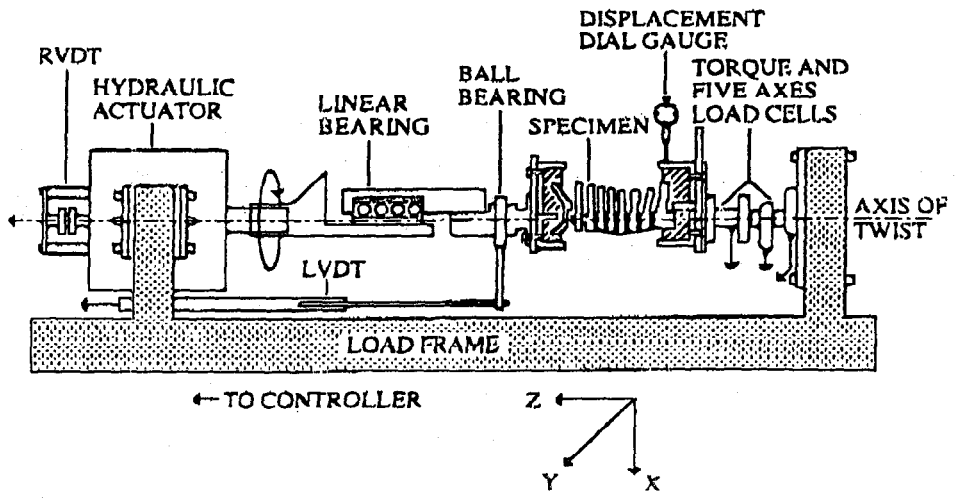

Fig 1. Test apparatus

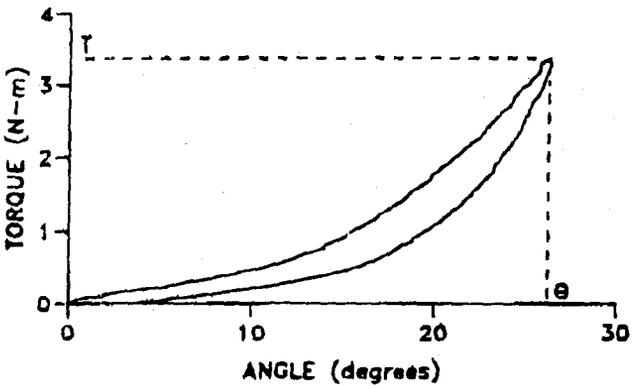

Fig 2. Torque-angle response of the cervical spine, showing the values of T and θ used to determine the torsional stiffness.

(X = 0) to the anterior surface of the vertebral body (X = L), as defined in Figure 4. The location of the center of rotation for each specimen, as determined by the center of minimum stiffness, is listed in Table 1. Mean center of rotation was found to lie at X/L = 0.83 ± 0.16. Referenced against the vertebral body, the mean center of rotation was found to lie about a point one-fifth the anteroposterior diameter of the vertebral body from the anterior of the vertebral body (Figure 4, Table 1, b/B = 0.2). The center of rotation was determined for each specimen within 0.13 cm (0.05 in.). Large flexion–extension moments were observed when rotation occurred at centers other than the center of minimum stiffness. The peak magnitude of the moment increased with increasing distance of the axis of twist from the center of minimum stiffness, and it tended to zero at the center of minimum stiffness.

Initial tests in which the axial length of the specimen was held fixed during rotation resulted in large axial compression forces (Fz = 1200 N) and nonphysiologic injuries. Specifically, ligamentous disruption occurred at the insertion of the specimen in the cup ends. Since the injuries produced were not consistent with clinically observed injuries and the injuries occurred in the specimen ends, the results were discarded and therefore omitted from this article. Subsequent testing, which allowed for changes in the length of the specimen with rotation (Fz < 75 N), produced the clinically observed injuries in the six specimens reported in this article.

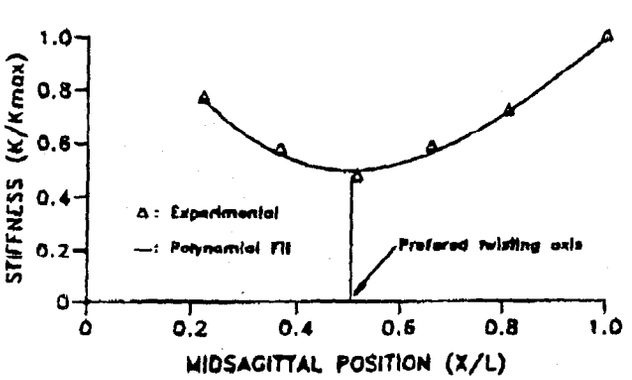

Fig 3. Torsional stiffness versus midsagittal position used to identify the axis of minimum stiffness.

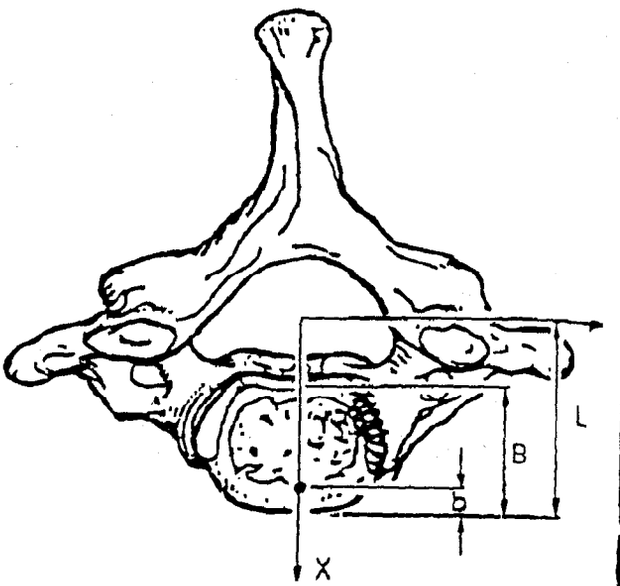

Fig 4. Cervical vertebra with reference axis for determination of the location of the center of rotation.

Injury

Injury was defined as a decrease in torque with increasing angle of twist, or by catastrophic failure of the specimen (Figure 5). Torques to failure are summarized in Table 2. To test the accuracy of these results, the torque transducer was calibrated before and after testing. In addition, previous studies reported mean torsional stiffnesses of 0.25–0.75 Nm/deg.[3,26] Mean torsional stiffness in this study was 0.48 Nm/deg, which agreed favorably with these previously reported values.[18]

In each of the six cervical spines, load applied to the base of the skull resulted in rotary atlantoaxial facet dislocation. Greater than 90° of rotation was observed in the joint, as well as increased compliance in the direction of rotation and a permanent rotation at no load. No evidence of injury was observed in the lower cervical spine by visual inspection, CT, or MRI. Further, fluoroscopy of the specimen during testing indicated only slight twisting of the lower cervical spine at the time of injury. Ligamentous injury consisted of tearing of the capsular ligaments of the anteriorly displaced lateral mass. The alar-transverse ligament complex and the odontoid process were grossly intact. Mean torque to injury was 17.2 ± 5.1 Nm, which was similar to the 13.6 ± 4.5 Nm reported by Goel et al.[9] Because the injury was confined to the atlantoaxis, the joint was dissected, and the injuries described.

Table 1. Location of the Center of Rotation

Specimen No.	Midsagittal Position X/L	b/B
1	0.83	0.23
2	0.85	0.15
3	0.61	0.47
4	0.90	0.01
5	1.00	0.00
6	0.69	0.37
Mean ± SD	0.83 ± 0.16	0.20 ± 0.19

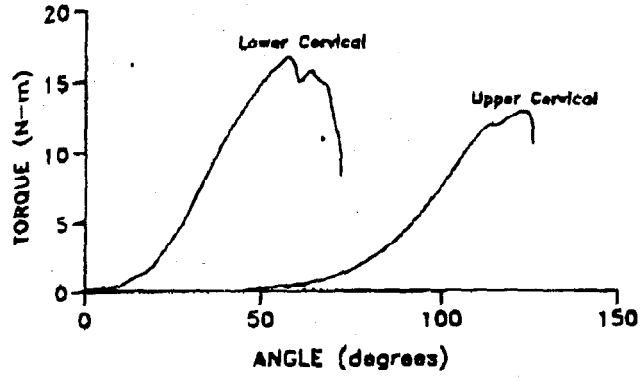

Fig 5. Cervical spine torque to failure, comparing torque to lower cervical injury with torque to atlantoaxial injury.

The lower cervical spines (C2–T1) were recast, mounted in the load frame, and injured. Unilateral facet dislocation with locking was produced in each specimen with significant ligamentous injury (Table 3). All injuries occurred more than one motion segment away from the cup ends, removing the possibility of end effects. Table 2 lists the torques to injury and the strength ratio, R (the ratio of lower cervical to upper cervical torque to injury). Mean torque to lower cervical injury was 21.0 ± 5.4 Nm. Torque to unilateral dislocation was greater in the lower cervical spine than torque to rotary dislocation in the upper cervical spine for each specimen tested (Figure 5, Table 2, $R > 1$). This indicates increased torsional strength in the lower cervical spine ($R_{mean} = 1.24 \pm 0.151$, $P = 0.05$.)

DISCUSSION

Kinetics

The use of a dynamic test system affords many advantages in the study of the spine. It also creates a challenge in that the motions of the hydraulic actuator are not necessarily the *in vivo* motions of the spine. In this experiment, allowing changes in axial length of the specimen was required to produce clinically relevant injuries. It was also necessary to align the spine along the actuator's axis of rotation. By considering the spine as a redundant (structurally indeterminate) structure, we applied Castigliano's theorem[5] to define the best center. That is, motions resulting from an unconstrained structure are such that the energy of the structure is a minimum.

Since energy in an elastic system is $1/2K\theta^2$, the minimum energy for any given angle of twist (θ) occurs at the point of minimum stiffness. We then defined the dynamic stiffness ($K = T_{max}/\theta_{max}$), found the minimum, and hypothesized that this minimum identified the axis about which unconstrained motion would occur *in vivo*. The parabolic shape of the stiffness curves (Figure 3) allowed for easy and accurate identification of the lower cervical center of rotation in each of the six specimens.

The validity of the minimum stiffness method is supported by its agreement with static domain radiographic studies on unconstrained specimens. Specifically, the location of the center of rotation in the anterior of the vertebral body (Table 1, $b/B \approx 0.20$) is similar to the results of White and Panjabi,[29] and explains Lysell's observation[16] of minimal motion of the anterior surface of the vertebral body with axial rotation. The development of large flexion–extension moments at nonminimum stiffness centers of rotation, coupled with large interspecimen variation in center of rotation indicates the need to determine the location of the center of rotation for each specimen tested in a dynamic actuator.

Table 2. Torque to Injury

Specimen No.	Upper Cervical (Nm)	Lower Cervical (Nm)	Strength Ratio (R)
1	11.74	17.10	1.456
2	17.65	20.20	1.144
3	16.05	16.86	1.051
4	12.46	16.87	1.353
5	25.65	29.72	1.160
6	19.66	24.48	1.296
Mean ± SD	17.17 ± 5.13	21.03 ± 5.40	1.24 ± 0.152

Table 3. Description of Lower Cervical Injury

Specimen No.	Injury	Ant Long Ligament	Post Long Ligament	Disk	Interspinous Ligament	Capsular Ligament	Flaval Ligaments	Bony Facets	Degree of Injury
1	C5 UFD	Partial R tear	Intact	R lateral tear	Torn	R & L torn	R partial tear	R sup. avulsion	+
2	C6 UFD	Partial R tear	Intact	Complete rupture	Intact	R & L torn	Intact	Intact	++
3	C6 UFD	Complete rupture	Intact	Complete rupture	Torn	R & L torn	R & L tear	R sup. crush	++++
4	C4 UFD	Partial R tear	Intact	R lateral tear	Torn	R & L torn	R & L torn	R sup. & inf. abrasion	++
5	C6 UFD	Complete tear	Torn	Complete rupture	Torn	R & L torn	Intact	R sup. abrasion	+++++
6	C4 UFD	Partial R tear	Intact	R lateral tear	Torn	R & L torn	R torn	R sup. crush	++

UFD = unilateral facet dislocation.

Injury

Application of torsional load to the head resulted in atlantoaxial rotary dislocation in all of the six specimens tested. The agreement between this experimentally produced injury, Greeley's reported clinical experience, and the experimental results of Goel et al[9] supports the validity of this injury model. Little rotation was imparted to the lower cervical spine, and no evidence of lower cervical injury was observed by *in situ* fluoroscopy, CT, MRI, or visual inspection. Considering that each motion segment of the spine carries the same torsional load, the injury of the atlantoaxial joint without lower cervical injury demonstrates that the atlantoaxis is the weakest joint in torsion in the cervical spine.

This result alone demonstrates that pure rotation of the head does not mediate lower cervical injury, as the atlantoaxial joint is injured at lower torques. This postulate is further supported by the statistically significantly larger torque to injury observed in the lower cervical spine (strength ratio, R = 1.24 ± 0.151), which occurs when torque is applied directly to the lower cervical spine. This ratio is larger despite the fact that the lower cervical spine suffered increased damage associated with a longer test battery and increased handling during preparation.

We conclude from these results that pure rotational loads to the head produce atlantoaxial facet injury. Pure rotation of the head, however, does not mediate ligamentous or bony injury in the lower cervical spine because of the comparative weakness of the atlantoaxial joint.

In contrast, this experiment does not determine the effect of torsion in increasing the ease of injury from other types of loading (compression or flexion). Because the clinical injury environment typically represents a combination of loading conditions that occur simultaneously (ie, combined flexion, compression, and torsion), the role of torsion as a contributing factor in cervical injury should be a topic of further experimental investigations.

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