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Injuries to Cadavers Resulting from Experimental Rear Impact

Low-velocity rear-end collisions frequently produce relatively minor damage to vehicles and perplexing injuries to occupants. These collisions are an ever-increasing hazard, especially at intersections, at interchanges, and to occupants of parked or stalled vehicles. In 1976 20% of all motor vehicle accidents were rear-end collisions involving two vehicles, but these accidents constituted only 4% of all motor vehicle fatalities [*1*]. The neck is the most frequently injured portion of the body, with 10% more cervical injuries reported in females than in males [*2,3*].

To provide additional insight into the injuries and the kinematic response of human subjects under simulated rear impact, six unembalmed cadavers were subjected to a rear impact that simulated a standard-size car at rest being struck from the rear by a car of equal weight with a velocity of 14 m/s (32 mph). The Daisy Decelerator Sled Facility at Holloman Air Force Base, Alamogordo, N. Mex. was used under the direction of the Physical Science Laboratory of the New Mexico State University, Las Cruces, for these tests. This study was performed under the terms of a National Highway Traffic Safety Administration contract.

The results of the simulated rear impact tests with cadaver test subjects are presented and discussed in this report. Anthropomorphic dummies were also tested and the engineering data of those tests were presented elsewhere [*4*].

Methods

Six unembalmed cadavers were donated to The University of New Mexico School of Medicine, Department of Anatomy, for medical research. The terms of the donations met the criteria of the Uniform Anatomical Gift Act. Each cadaver was screened for height, weight, age, and physical condition according to guidelines prior to being accepted as a test subject. Five cadavers were male and subject ages ranged from 52 to 64 years (Table 1). Four of the subjects died of atherosclerotic heart disease and two died of metastatic

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carcinoma. None had been bedfast prior to death nor had a history of cervical spine pathology.

Following external examination and precrash cervical X-rays in Albuquerque, the bodies were transported via a refrigerated vehicle to the test site. Precrash cervical X-rays did not reveal significant degenerative changes in the cervical spines of any of the subjects.

The cadavers were subjected to simulated rear impacts at the Daisy Decelerator Sled Facility, which includes a special pneumatic piston sled accelerator that can produce velocities from 1 to 54 m/s (3 to 120 mph) and a water brake-type decelerator. The sled is mounted on a track that has two tubular rails separated by a distance of 1.5 m (5 ft) and extending for a length of 61 m (200 ft). Sled propulsion is provided by a pneumatic piston that accelerates the entire test assembly over a distance of 13 m (42 ft) to the desired velocity. The test assembly then coasts down the track until a 1.8-m (6-ft) cylindrical piston mounted on the front of the sled enters the water brake cylinder. The most important mechanical component of the device is the water brake, which has two major parts: a stationary cylinder and a piston attached to the sled. The piston enters the cylinder, resulting in transfer of kinetic energy from the moving sled to the water. The braking force can be varied as a function of the sled piston penetration.

The subjects were instrumented in a manner similar to that devised by Ewing and Thomas [5] to study human head and neck response to forward impact. Although some triaxial sensors were used, the analyses were limited to the mid-sagittal plane. The third-axis sensors were used to monitor the severity of the off-plane motion.

The most complicated sensor modules were those used to measure head motion with respect to the first thoracic vertebra (T1) (Figs. 1 and 2). The head module was positioned on the back of the subject's head and consisted of three orthogonally mounted single axial accelerometers mounted with sensitive axes *R-S-T* in and perpendicular to the mid-sagittal plane. Only *R* and *T* accelerometers along with the rate gyro in the mouth module were used for the head center of gravity kinematic analysis. Accelerometer *S* was used only to monitor the degree of off-plane motion.

The mouth module was positioned on a bite plate and consisted of three single axial accelerometers and a rate gyro (Figs. 1 and 2). The sensitive axis of the miniature rate gyro (85 g or 3 oz) was perpendicular to the mid-sagittal plane. Head center of gravity kinematics can be obtained from calculations using the head *R-T* accelerometers and the rate gyro data, the mouth *R-T* accelerometers and the rate gyro data, or the head and the mouth *R-T* accelerometers.

The spine module was similar to the mouth module and was positioned over the posterior spinous process of T1 (Figs. 1 and 2). The module was constructed by making and cutting to fit anatomically a pressure mold of the spinal column of the subject and was secured by a harness that maintained the module in the selected location under considerable pressure. A smooth cover plate was used to prevent the spine module from being stripped off by the headrest while the subject was ramping up the back of the seat. The spine module was the base of a light aluminum fork that served as a photographic target.

Table 1—*Cadaver physical data.*

Cadaver Number[a]	Age, years	Sex	Cause of Death
C1D	64	m	atherosclerotic heart disease
C2D	60	m	metastatic carcinom . of the colon
C3R	52	f	metastatic carcinoma of the cervix
C4R	64	m	acute myocardial infarct
C5D	57	m	atherosclerotic heart disease
C6R	61	m	atherosclerotic heart disease

[a] D = deflecting back of the seat and R = rigid back of the seat.

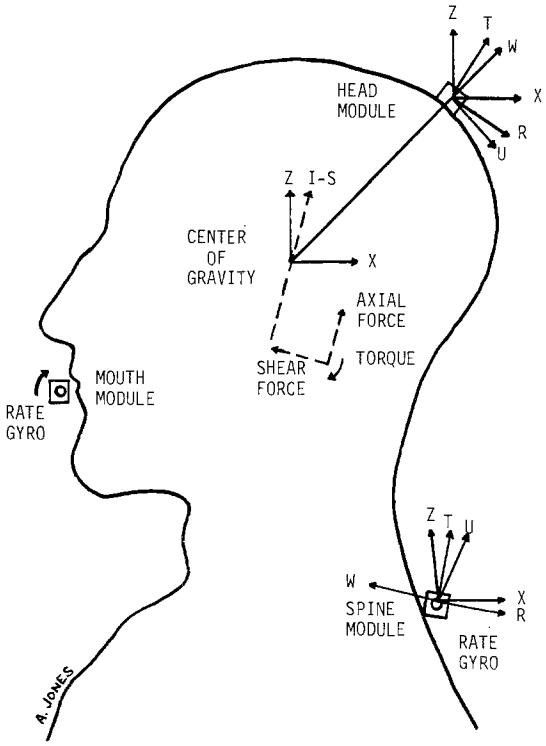

FIG. 1—*Schematic drawing of human head demonstrating module placement and test axes.*

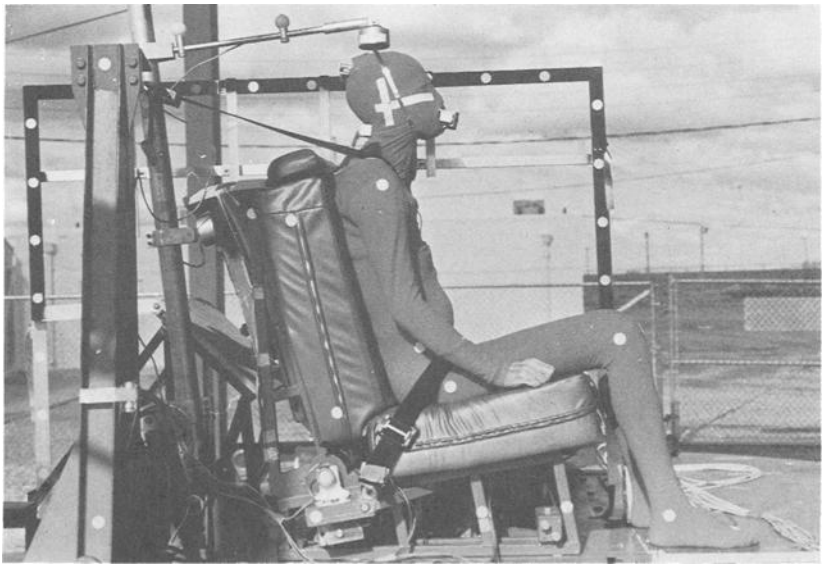

FIG. 2—*Test subject clad and positioned on a standard seat. White disks are camera targets.*

Two single axis accelerometers were used to measure the chest and back accelerations. The first sensor was attached to the sternum and the second sensor was attached posteriorly to the spine opposite to the first sensor. The former measured the chest acceleration and the difference between the former and the latter measured the chest acceleration with respect to the spine.

Three accelerometers mounted on the sled provided three-dimensional sled acceleration measurement. The X sensor measured the horizontal forward acceleration (during power stroke phase), deceleration (during coasting phase), and the impact deceleration. The Y and Z sensors measured the ride smoothness and impact side components.

The head instrumentation attached to the mouth bite plate and to the head plate were held in position by a truss-type strap system. This strapping passed inferior to the jaw and around the superior skull at the mid-parietal region. An added strap passed from the strap under the chin along the anterior sagittal line and joined the upper strap at the parietals. This strap arrangement clenched the mandible tight against the bite plate so that the mouth was clamped shut during the backward stroke. In these tests, the anterior tissues of the neck provided part of the resistance to the posterior motion of the head. No attempt was made to assess the alteration of injury potential as a result of holding the mouth closed.

The deceleration at the head center of gravity was computed from the values of acceleration measured by the accelerometers at the head triaxial mount and from the angular rate measured at the rate gyro on the mouth module (Fig. 1). This method of evaluating head forces and their relation to the T1 vertebra was developed by Ewing and Thomas [5]. Figures 3 through 10 show test measurements from a typical subject, C1D (Cadaver 1 tested in a deflecting back of the seat).

Rigor mortis was no longer grossly present in the subjects by the time of testing and the joints were manually flexed before testing to ensure complete lack of rigor.

Two layers of close-fitting clothing, plus a face mask, were placed on the subject to avoid skin abrasion effects and more closely simulate an actual occupant's clothed condition. The leads from the electronic sensors were routed beneath the layers of clothing. Data collected from the 38 sensors with a pulse code modulation data system were fed into a digital computer for automatic data processing.

Camera targets were located at each anatomical instrumentation mount and were visible to three high-speed motion picture cameras used during the impact sequence. Additional camera targets were affixed to the outer layer of the clothing. Targets and cameras were optimally located to minimize target shadowing.

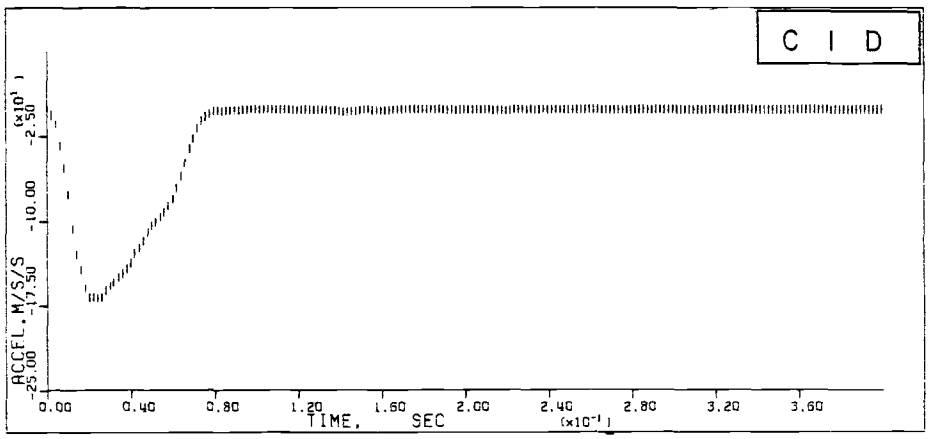

FIG. 3—*Sled deceleration for C1D.*

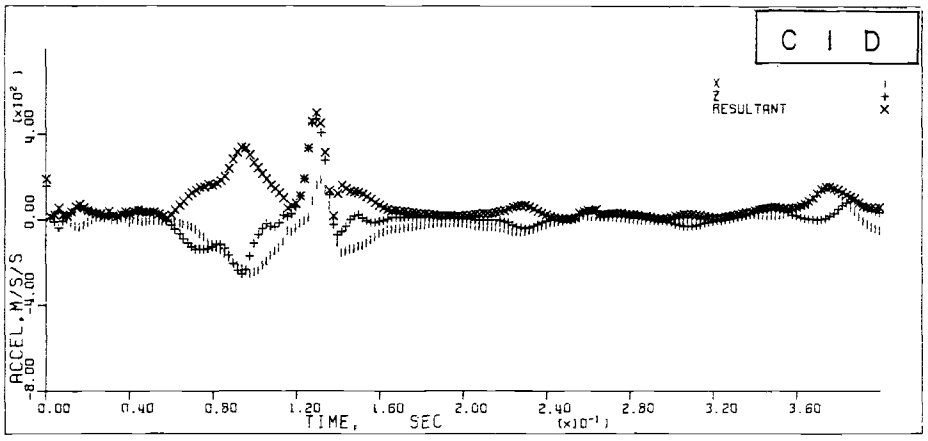

FIG. 4—*Head center of gravity linear acceleration measured in the* X *and* Z *axes and demonstrating resultant linear acceleration for C1D.*

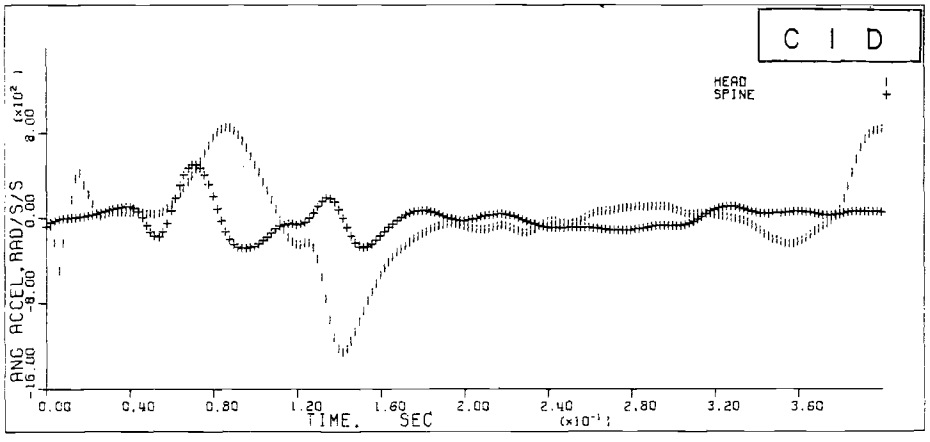

FIG. 5—*Head center of gravity and T1 angular acceleration for C1D.*

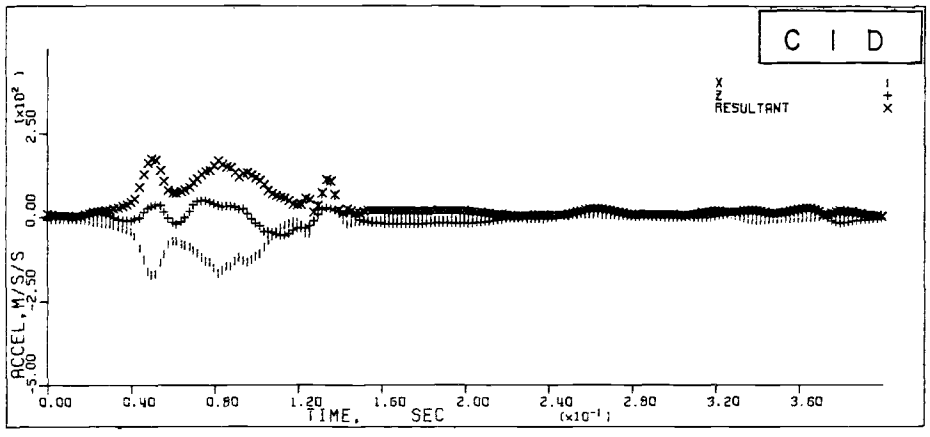

FIG. 6—*Linear accelerations in the* X *and* Z *axes with resultant accelerations measured at T1 vertebra for C1D.*

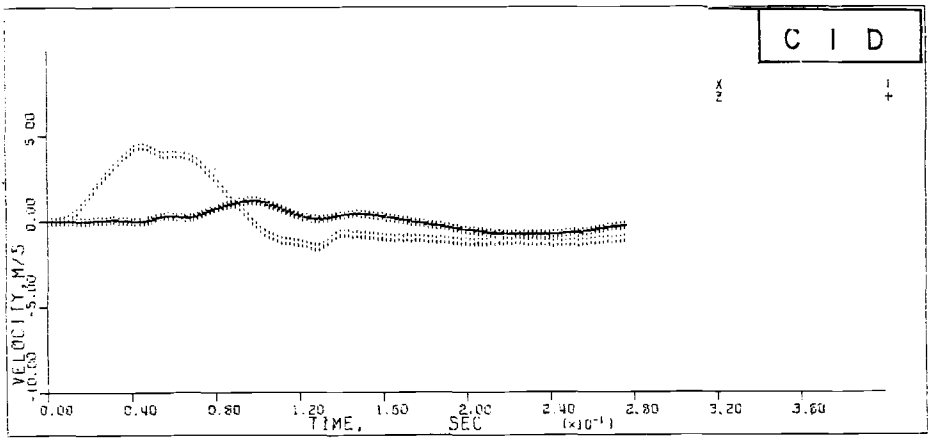

FIG. 7—*T1 vertebral velocity measured for C1D.*

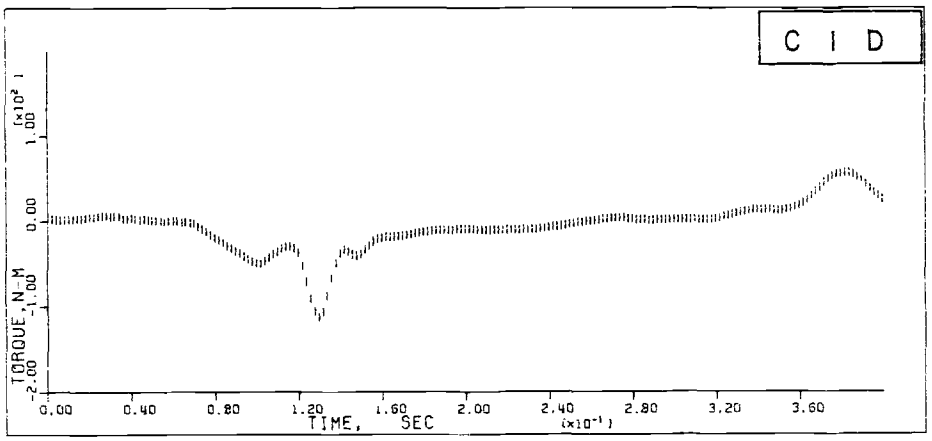

FIG. 8—*C1D head torque measured at occipital condyles.*

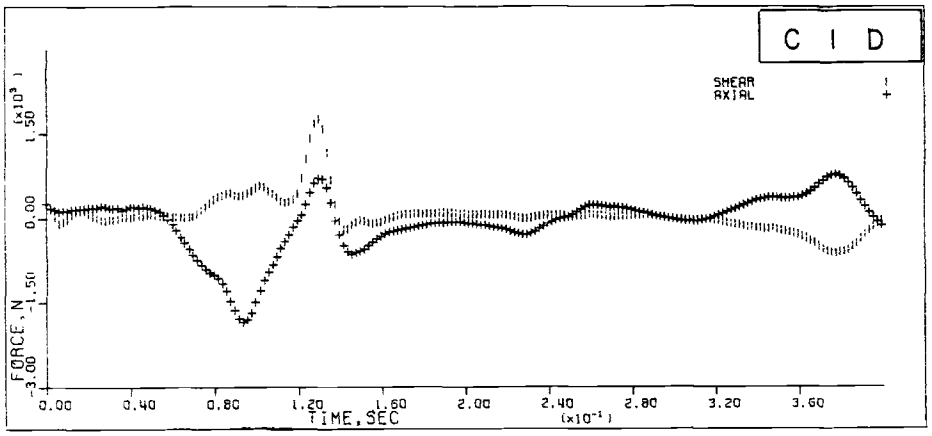

FIG. 9—*C1D shear and axial forces occurring at the occipital condyles.*

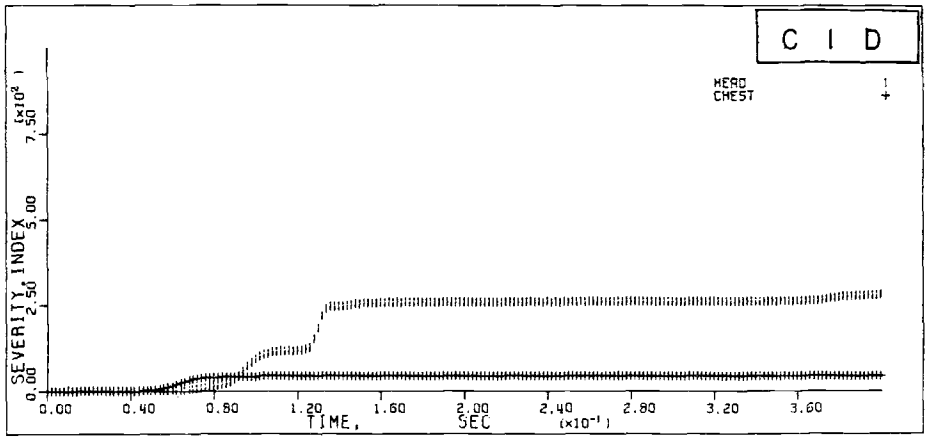

FIG. 10—*Head and chest severity indexes of C1D.*

Each cadaver was placed on a standard seat [*6*] with the headrest at its lowest position with fastened lap and shoulder seat belts (Fig. 2). Three tests had the rotation of the framework of the back of the seat rigidly restrained, while three had the framework designed to rotate backward during impact. Test velocities ranged from 6.8 to 7.8 m/s (15.3 to 17.5 mph) (Table 2).

Following testing, cadavers were refrigerated and transported to the Office of the Medical Investigator in Albuquerque where complete autopsies were performed, with careful dissection of the anterior and posterior neck and cervical spine prior to the examination of the spinal cord and removal of the cervical spine from the head and chest. Prior to autopsy, post-test cervical X-rays were taken (Fig. 11). Tests were usually performed within seven days after the date of death and autopsy was performed the day after the test date. All cadavers demonstrated only slight decomposition at autopsy.

Results

The average sled deceleration was 17.8 *g*, with the individual values measuring 17.0, 18.6, 18.7, 19.0, 19.1, and 14.5 *g*. The deceleration curve for test C1D is demonstrated in Fig. 3 and reveals the representative pulse shape and its respective peak value of 17.0 *g*. The onset rate was 1163 *g*/s in this test and measured approximately 1000 *g*/s in all tests.

The average sled deceleration pulse was 174.7 $m/s \cdot s^{-1}$ and the average sled velocity at impact was 7.46 m/s (Table 3, Fig. 3).

Head acceleration ranged from 458.1 $m/s \cdot s^{-1}$ for test C6R to 602.9 $m/s \cdot s^{-1}$ for C4R with an average value of 485.75 $m/s \cdot s^{-1}$. Head velocity averaged 7.49 m/s and the angular head velocity averaged 31.85 rad/s (Table 3, Figs. 4 and 5).

Resultant acceleration at T1 ranged from 173.3 $m/s \cdot s^{-1}$ to 314.7 $m/s \cdot s^{-1}$, and the average value was 234.83 $m/s \cdot s^{-1}$. Resultant T1 velocity averaged 4.16 m/s (Table 3, Figs. 6 and 7).

Head torque at the occipital condyles averaged 115.7 N·m. The shear on the occipital condyles averaged 1792.0 N and the axial force was 2326.8 N (Table 3, Figs. 8 and 9). The tolerance levels for neck extension for a 50th percentile adult male proposed by Mertz and Patrick [*7*] indicated that ligamentous damage would be expected at 61.4 N·m (42 lbf·ft) of torque at the occipital condyles.

By using a rigid body assumption, a computation was made of the torque occurring at the sixth cervical vertebra (C6) of each of the cadaver subjects. The parameters used in the

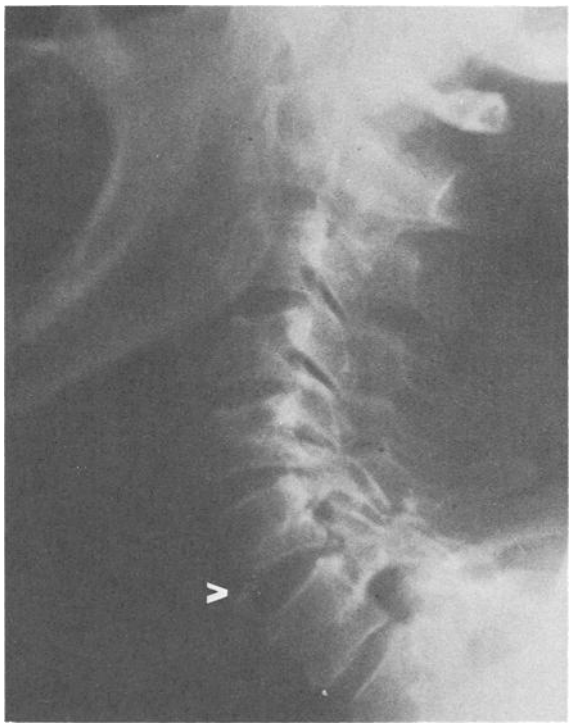

FIG. 11—*Posttest cervical X-ray of C2D, demonstrating an anterior inferior avulsion fracture at C6* (arrow).

computation were the torque found at the occipital condyles and the distance, scaled from X-rays, between the condyles and the middle of the C6 vertebral body. The posterior stroke of the head during impact caused a torque computed to vary between 34.5 and 92.7 N·m (25.3 to 67.9 lbf·ft) on the C6 vertebra. The values agree reasonably well with the mathematical model of Prasad et al [*8*], which demonstrated a torque of 45 N·m at the C7 vertebra during a 10-deg deflection of the back of the seat.

The head and chest severity indexes for C1D were low, with a head index of 281.3 and a chest index of 46.0 (Fig. 10). The average head severity index for all tests was 388.4 and the average head chest index was 78.3 [*9*]. The head injury criterion (HIC) for subject C1D was 160.5 and the average HIC for all tests was 302.7 (Fig. 10, Table 3). If these indexes are greater than 1000, then the acceleration pulse is considered to be life-threatening, but if less than 1000 the acceleration pulse is considered not to be dangerous to life [*9,10*].

A review of the high-speed motion pictures indicated that the subjects initially experienced rearward motion into the seat cushion and against the headrest during the simulated rear impact tests. The cadavers were clad in two layers of close-fitting clothing that had little frictional resistance with the material on the back of the seat and allowed the subjects to freely ramp up the back of the seat until the lap belt restrained them. The subjects eventually rebounded forward. The lower cervical spine and upper thorax contacted the headrest during impact, but the heads of the subjects did not strike any part of the headrest or seat during the impact sequence. Hyperextension of the neck occurred first, followed by hyperflexion on rebound.

Five of six cadavers had cervical injuries without identifiable spinal cord damage (Table 2). One subject had a "bull neck" and sustained no identifiable injuries on X-ray or autopsy

TABLE 2—*Summary of cadaver test conditions and results.*[a]

Cadaver Number[b]	Height, in.	Sitting Height, in.	Weight, lb.	Test Velocity, mph	Sled Deceleration, *g*	AIS Code	Post-Crash Injuries
C1D	70	35.0	126	16.7	17.0	3	ruptured anterior longitudinal ligament at C6-C7; subluxation between C5-C6 and C6-C7; retroperitoneal hemorrhage, minimal
C2D	69	31.3	134	16.6	18.6	3	ruptured anterior longitudinal ligament at C6; anterior inferior avulsion fracture of C6; ruptured thoracic esophagus
C3R	64	32.5	132	17.0	18.7	3	ruptured anterior longitudinal ligament at C6; compression fracture of C6; fractures of C5 and C6 left vertebral arches
C4R	69	34.7	159	17.1	19.0	3	ruptured anterior longitudinal ligament at C6-C7; ruptured C6-C7 intervertebral disk
C5D	72	35.5	195	17.5	19.1	3	ruptured anterior longitudinal ligament at C6-C7; ruptured C6-C7 intervertebral disk
C6R	71	34.5	165	15.3	14.5	0	none

[a] 1 in. = 25 mm; 1 lb = 0.45 kg; and 1 mph = 0.45 m/s.
[b] D = deflecting back of the seat and R = rigid back of the seat.

TABLE 3—*Tabulation of cadaver test parameters.*[a]

Parameters (1)	Notes	Measured Values C1D	C2D	C3R	C4R	C5D	C6R	Mean	Standard Deviation
Sled deceleration pulse, $m/s \cdot s^{-1}$	...	166.8	182.3	183.9	186.2	187.0	142.0	174.7	17.65
Sled velocity at impact, m/s	...	7.46	7.44	7.60	7.62	7.80	6.82	7.46	0.38
Sled stopping distance, m	...	0.263	0.246	0.268	0.268	0.260	0.282	0.265	0.012
Head acceleration, $m/s \cdot s^{-1}$	2 3 5	500.3	491.1	481.3	602.9	380.8	458.1	485.75	71.8
Head velocity, m/s	2 3 5	7.000	8.900	6.992	7.655	7.557	6.877	7.49	0.76
Head displacement, m	2 3 5	0.308	0.316	0.192	0.214	0.283	0.243	0.260	0.05
Head angular acceleration, $rad/s \cdot s^{-1}$	4 5	1333	1423	1657	1648	976	1436	1412.2	250.1
Head angular velocity, rad/s	4 5	28.99	33.08	30.26	33.65	31.88	33.22	31.85	1.86
Head angular displacement, rad	4 5	1.57	1.57	1.34	1.28	1.19	1.50	1.41	0.16
T1 acceleration, $m/s \cdot s^{-1}$	3	174.2	173.3	314.7	262.5	184.5	299.8	234.83	65.4
T1 velocity, m/s	3	4.400	5.146	2.943	3.909	5.762	2.823	4.16	1.17
T1 displacement, m	3	0.241	0.261	0.090	0.137	0.376	0.102	0.201	0.11
Head torque, N·m	6	113.2	148.9	115.7	139.8	71.2	105.3	115.7	27.5
Head force—shear, N	6	1771	1982	1966	2195	1133	1705	1792.0	366.36
Head force—axial, N	6	1845	2261	2496	3312	1503	2544	2326.8	626.34
Torque at C6, N·m	7	71.9	34.5	92.7	82.1	66.0	60.8	68.0	20.0
Head severity index	...	281.3	422.0	365.5	591.1	269.7	400.8	388.4	117.0
Head injury criterion	...	160.5	338.6	288.5	496.8	189.3	342.2	302.7	121.5
Chest severity index	...	46.0	39.4	166.1	126.9	24.1	67.1	78.3	56.01

[a]**Key:**

1. Maximum values (parameters do not occur simultaneously during an impact).
2. Value at center of gravity of head.
3. Resultant.
4. Mid-sagittal plane.
5. Head center of gravity relative to T1 (first thoracic vertebra).
6. At occipital condyles.
7. Computed from measurements scaled from lateral X-ray (accuracy ±25%).

examination. The five subjects with injuries had ruptured anterior longitudinal ligaments at the level of underlying cervical spine injury. An anterior inferior avulsion fracture of C6 was present in one subject, and a uniform compression fracture of the C6 vertebral body was observed in another. Two subjects had ruptured C6–C7 intervertebral disks. Anterior subluxations at C5–C6 and C6–C7 intervertebral joints were present in one test subject. Two esophageal perforations were found in one subject. Left posterior vertebral arch fractures of C5 and C6 were sustained by one cadaver. No soft tissue hemorrhages, muscle lacerations, or spinal cord damage was present. Only one subject demonstrated retroperitoneal hemorrhage, possibly resulting from lap belt rebound pressure. Rupture of the posterior longitudinal ligament was not produced in any of the subjects.

All posttest injuries determined at autopsy were rated according to 1974 American Medical Association–Society of Automotive Engineers–American Association for Automotive Medicine Revision of the Abbreviated Injury Scale (AIS) [*11*] and appropriate numbers were assigned to each injury. The cervical injuries were all AIS Code 3 (severe, not life-threatening) since no underlying spinal cord damage was identified. The esophageal perforations were assigned AIS Code 4 (severe, life-threatening, survival probable) and the retroperitoneal hemorrhage was assigned AIS Code 3. Subject C6R was assigned AIS Code 0 since no injuries were visible on X-ray or autopsy examinations.

No major differences in injury patterns were noted in the subjects with deflecting versus rigid seats.

Discussion

The cervical spine is extremely mobile, allowing flexion, extension, lateral bending, and rotation, but it does not possess great structural stability [*12*]. Bailey [*12*] states that the most important anatomical structures providing stability to the cervical spine are the cervical musculature, ligamentum nuchae, and the intervertebral disks. The apex of the lordotic curve is at C3 through C6 vertebrae and these vertebrae receive the maximum stress in cervical trauma [*13*]. Maximum cervical mobility is between C5–C6 and C6–C7; it is therefore apparent that C6 and its superior and inferior intervertebral disks would be susceptible to injury from rear impact as simulated in this study.

Hyperextension in living humans can produce rupture of the anterior longitudinal ligament, rupture and avulsion of the intervertebral disks, joint subluxation, and rupture and avulsion of the posterior longitudinal ligament [*12–15*]. These injuries occur more commonly than compression fractures of the posterior articular processes. Although damage can occur at any level [*16*], the most frequent level of involvement is at the C5–C7 vertebral segment, which is the area of greatest mobility [*17*]. De Palma and Subin [*18*] state that the cervical disk spaces most frequently involved were C5–C6 and C4–C5, while C6–C7 was the least involved in 310 patients with cervical injury and that the older the patient, the lower the level of involvement. The C1 (atlas) and C2 (axis) vertebrae may also be injured by hyperextension, but damage at this level is uncommon [*19*]. Axial compression of the skull and cervical spine with hyperextension can shear the posterior arch of the atlas [*20*]. Fractures of the neural arches of the axis are more common than isolated fractures of the axis resulting from hyperextension [*20*]. The odontoid process of the axis may be fractured as the anterior arch of the atlas is forced against it, and this fracture may be produced by hyperextension or hyperflexion [*16*]. Sprain or laceration of the transverse atlas ligament resulting in anterior atlas displacement may occur following hyperextension [*19*]. Fracture of the C2 body is uncommon, but an avulsion fracture or teardrop fracture of the lower C2 vertebra is not uncommon following hyperextension [*19*].

Clemens and Burow [*21*] subjected 13 incomplete human torsos to hyperextension forces without headrests at a test velocity of 7.0 m/s (15.6 mph or 25 km/h). The torsos had the head and skin removed and were mounted on rigid plates to avoid uncontrolled move-

ments of the lumbar spine and pelvis. Intervertebral disk damage was the most common injury, followed by ruptures of the anterior longitudinal ligament and vertebral fractures. A few torsos experienced ruptures of the ligamenta flava and posterior longitudinal ligaments. All injuries were concentrated at the C5–C6 and C6–C7 levels, while the upper cervical spine was hardly affected. Average test deceleration was 15.0 *g*. The results, test velocity, and deceleration of these experiments are similar to those reported herein with the exception of the differences in subject preparation.

Hyperextension produces traction on the anterior longitudinal ligament and the ligament may be stretched, ruptured, or avulsed and a portion of vertebral bone may be avulsed from the vertebral bodies [*16*]. An anterior inferior avulsion fracture of C6 was present in test subject C2D (Table 2). In a study of the mechanics of spinal injuries, Roaf [*22*] subjected portions of healthy cervical and thoracic vertebrae removed soon after death to forces applied with a standard Denniston testing machine and found that the anterior longitudinal ligament could not be ruptured with pure extension alone but required rota tion as well to produce a rupture of the ligament. The forces applied in his study, however, were applied slowly for ease of analysis.

Soft tissue injury resulting from hyperextension in the living include muscle lacerations and hemorrhages with or without damage to large neurovascular structures [*16,23*]. Prevertebral hematomas may displace the esophagus and trachea forward, producing dysphagia [*23*]. The vertebral arteries may be damaged or temporarily occluded by an unstable atlanto-occipital joint, causing basilar artery insufficiency [*24,25*]. Evidence of soft tissue injuries of the cervical muscles and neurovascular structures was not present in any of our subjects, but this is not to imply that such injuries would not have been sustained in living subjects. Subject C2D experienced two esophageal perforations (Table 2). Hyperextension forces can stretch and tear the muscularis of the esophagus, resulting in perforation [*26*]. The trachea, cervical musculature, and sympathetic chains may also be damaged, depending on the severity of the hyperextension [*14*]. Minimal retroperitoneal hemorrhage was present in subject C1D, possibly because of lap belt rebound pressure.

Radiological evidence of cervical injury may be lacking or demonstrate straightening or reversal of the cervical lordotic curve, thinning of one or more vertebral disks, fractures of vertebrae, displacement of the esophagus and trachea, or some combination of these injuries [*18*]. In a series of 309 patients with acute flexion-extension cervical injury, the usual radiographic appearances were normal except for cervical curvature straightening [*27*].

A discrepancy exists between the degree of vertebral displacement and severity of spinal cord damage [*15*]. Paralysis may not be present in a case of complete dislocation, whereas tetraplegia may accompany cervical trauma without radiologic evidence of bony or joint disturbance [*11,15,28,29*]. Barnes [*28*] reported 21 cases of paraplegia resulting from cervical spine injuries. Only one third of his cases were a result of hyperextension injuries and two thirds were due to flexion injuries. He observed that the spinal cord damage in the hyperextension cases was not as severe as in those patients injured by flexion forces. In a series of 77 patients with cervical fractures or dislocations or both, Rogers [*30*] observed that 27 patients initially demonstrated no symptoms of spinal cord or nerve root function; however, 10% of the patients later developed symptoms of cord compression. Most cervical fractures occur at C4 through C7 and serious spinal cord damage is more often associated with hyperflexion and flexion-rotation than hyperextension [*31*]. Durbin [*32*] found that the most common site of fracture-dislocations was at C4–C6 and paraplegia was present with damage at the C3–C6 level. No correlation exists between the degree of cervical fracture dislocation and degree (or absence) of neurological damage [*33*]. With hyperextension the ligamentum flavum can bulge into the spinal canal, resulting in narrowing of the canal by 30% [*34,35*]. The bulging ligamentum flavum can press on the posterior columns of the spinal cord, resulting in neurological symptoms [*23*]. In animals, cord trauma produces hemorrhagic lesions and edema in the central gray matter and anterior horns

within 30 min; these damaged areas become confluent in 4 h, followed by cord necrosis in 24 h [*31*]. Localized nervous tissue disruption and hemorrhage under the site of vertebral fractures were produced in monkeys subjected to hyperextension [*36*].

Morphological evidence of spinal cord damage was not observed in any of the subjects in this study. The lack of damage is in agreement with the findings of Marar [*37*], in which seven cadavers were subjected to manually produced hyperextension forces. All seven cadavers had fractured vertebral bodies, three had fractures of C4, one had a fracture of C5, and three had fractures at C6. The anterior longitudinal ligaments in all subjects were ruptured at the level of fracture. One subject had an incomplete rupture of the C6-C7 disk. The posterior longitudinal ligaments and interspinous ligaments were intact in all seven cadavers. Spinal cord damage was not observed in those four cadavers subjected to only hyperextension forces. Marar [*37*] found that a backward displacement force at the fracture site was necessary to produce posterior subluxation and spinal cord constriction. The lack of spinal cord damage in our cadaver studies should not be construed to indicate that spinal cord damage would not have occurred in the living subject under similar conditions.

Concussion, subdural hemorrhage, dizziness, loss of balance, and other central nervous system related injuries have been described after hyperextension, and these injuries may be the result of pressure gradient effects, shearing forces, and mass movements of the cranial contents [*3,16*]. Brain damage was not present in any of the six subjects in this study.

Hyperflexion forces may produce rupture and avulsion of the ligamentum nuchae, interspinous ligaments, ligamentum flavum, and the posterior longitudinal ligaments [*12*]. Compression fractures of the vertebral bodies, disk rupture and avulsion, and spinal cord damage may also occur, depending on the severity of the injury. Vertebral arches and facets may also fracture because of shear forces [*16*]. Subject C3R sustained a compression fracture of C6 and fractures of left C5 and C6 vertebral arches as well as rupture of the anterior longitudinal ligament at C6. The compression and vertebral arch fractures can be accounted for on the basis of hyperflexion forces occurring on rebound.

Subject C6R experienced no cervical injuries that could be determined by radiological or autopsy examination. In comparison to the other five subjects, Cadaver 6 had generalized increased fatty deposits and the neck was large, being the same circumference from the chin to shoulders, with little or no chin prominence. The thyroid was enlarged and weighed 85 g and revealed nodular hyperplasia. The sled test velocity was 6.8 m/s (15.3 mph) and the deceleration was 14.5 *g*, which was below the crash severity of the other five tests.

Conclusions

The findings indicate that the forces ocurring in a rear impact such as simulated by these tests tend to cause injury to the anterior portions of the cervical spine rather than to the posterior portions. Compression fractures of the anterior portions of the vertebral bodies were produced in preference to tension injury of the posterior intervertebral ligamentous tissue during rebound.

Summary

Six unembalmed cadavers donated for medical research were subjected to low-velocity simulated automotive rear impact with the subjects restrained with lap and shoulder belts and with seat headrests at the lowest position. Five subjects had experimental injury at the C6 level without spinal cord damage. One cadaver had no experimental injuries. The results of the simulated rear impacts were discussed.

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