Annu. Rev. Biomed. Eng. 2000. 02:55–81
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FUNDAMENTALS OF IMPACT BIOMECHANICS: Part I—Biomechanics of the Head, Neck, and Thorax

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Key Words injury mechanisms, mechanical response, human tolerance

■ **Abstract** This is the first of two chapters dealing with some 60 years of accumulated knowledge in the field of impact biomechanics. The regions covered in this first chapter are the head, neck, and thorax. The next chapter will discuss the abdomen, pelvis, and the lower extremities. Although the principal thrust of the research has been toward the mitigation of injuries sustained by automotive crash victims, the results of this research have applications in aircraft safety, contact sports, and protection of military personnel and civilians from intentional injury, such as in the use of nonlethal weapons. The reader should be keenly aware of the wide variation in human response and tolerance data in the cited results. This is due primarily to the large biological variation among humans and to the effects of aging. Average values are useful in design but cannot be applied to individuals.

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1523-9829/00/0825-0055$14.00

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INTRODUCTION

The aim of this chapter is to provide the reader with the fundamentals of the biomechanics of impact injury and an up-to-date literature source that can be used for a more detailed study of the subject. The entire body region will be covered under three of the four main branches of impact biomechanics, which are injury mechanisms, mechanical response, tolerance, and the biofidelity of surrogates.

Injury Mechanisms

In this branch of impact biomechanics, tests and analyses are conducted to determine the mechanical parameters involved in causing a certain injury. Generally, several reasonable hypotheses are proposed and tested, and the one that most consistently produces the same injury emerges as the mechanism of injury. Failure of a long bone occurs as the result of a tensile load being applied to it. In most cases, the tension would come from bending of the bone, and thus the mechanism of bony fracture of a long bone is frequently due to the application of a bending moment. In some cases, two different loads or motions can cause the same injury. For example, in the brain, diffuse axonal injury (DAI) can result from angular as well as linear acceleration of the head. In that sense, the injury mechanism for DAI is not unique. However, both of these motions can cause shear stresses to develop in the brain, and thus the injury mechanism can very well be shear stress or strain. It is not clear at this time if any injury has a unique mechanism. The mechanisms described in this chapter can be found in the literature or are based on recent research results. In the latter case, there is a cautionary note that no archival literature supporting the mechanism is available.

Mechanical Response

Mechanical response data obtained from impacts to animals and human cadavers are needed to design more human-like dummies. Response data can also be compared with their human equivalent to determine if that particular region of the animal is a good human surrogate. As in the study of injury mechanisms, response data are collected from individual body regions such as the head, neck, chest, abdomen, and the lower extremities. Such data are just becoming available for the upper extremities, and some side-impact data are available for the shoulder. In general, response data take the form of a load-deformation curve where the load can be either a force or a moment and the corresponding deformation is either a

linear or angular displacement. Early data obtained by biomechanical researchers, such as Yamada (1), provided static response. However, it was determined that the response of many body tissues is dependent on the rate of loading, and dynamic tests at various rates of loading were carried out.

Other forms of response data include force histories, displacement histories of an impact or force, and acceleration as a function of a relevant independent variable, such as drop height. If two sets of histories are available, a cross-plot of the data would yield a force-deflection curve. Thus, histories are rudimentary response curves that are used in the absence of load-deformation curves. In orthopedic biomechanics, the response of individual tissues, such as compact bone, cartilage, and ligaments, are obtained in the form of force-deflection curves and/or stress-strain curves. These data are usually more basic than what are needed in automotive impact biomechanics.

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Human Tolerance or Injury Risk to Impact

This is the third area of study in impact biomechanics and is closely tied to rule-making as well as to the design of instrumentation used in the anthropomorphic test dummies (ATD) to ensure that the parameters measured are in the injury range. It is also the most difficult area of study because of the large variation in mechanical properties of human tissue due to age, gender, weight, and geometry. All of these factors are in addition to the normal biological variation in tissue strength and the level of injury we are willing to accept. For example, frontal crashes do not cause serious neck problems unless the deceleration is very high. However, minor rear-end collisions can result in long-term neck pain. There are several levels of tolerance. They range from the "Ouch" level for volunteer subjects to the LD (Lethal Dose) 50 level at which half of the subjects would suffer a fatality. To define a reasonably safe level for the average car occupant without having to make the car unaffordably expensive, a moderate to severe level of injury is chosen as the tolerance level. That is, the injuries sustained by the average occupant should not be life-threatening. There is an Abbreviated Injury Scale (AIS), developed by emergency room physicians and physicians in other medical specialties to quantify the severity of an injury to each body area. Severity is defined as threat to life and is not based on disability or impairment. On the AIS scale, any injury greater than AIS 4 is life-threatening. The severity levels of AIS are shown in Table 1.

Although an injury can be quantified by the use of the AIS, it is still not possible to provide a quantitative description of the agent that caused the injury. Thus, injury criteria are needed by biomechanical engineers to describe the relationship between one or more physical parameters and the injury they caused. These criteria usually take the form of a simple mathematical expression or a force or acceleration level. Generally, the physical parameter should be consistent with the mechanism of injury that most likely caused the injury.

It is perhaps necessary to bring out the difference between an injury criterion and a safety standard, particularly standards set by national or state governments

TABLE 1 The Abbreviated Injury Scale (AIS)

AIS	Severity level
0	No injury
1	Minor
2	Moderate
3	Serious
4	Severe
5	Life threatening
6	Maximum
9	Unknown

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which control the sale of cars. In theory, safety standards should be based on scientifically established injury criteria. However, many standards are no longer scientifically sound because much more knowledge on injury mechanisms and injury criteria are now available and standards, once implemented, are hard to change. Examples of this inconsistency are highlighted in this chapter.

The specific body regions covered in this chapter are the head, neck, and thorax. The abdomen, pelvis, and the lower extremities will be discussed in the next volume of the *Annual Review of Biomedical Engineering*. Data on upper extremities are just becoming available, and very little is known regarding the response and tolerance of the shoulder.

BIOMECHANICS OF THE HEAD

Head Injury Mechanisms

The major mechanisms of brain injury are discussed in a review article by King et al (2). They are positive pressure, negative pressure, and shear due to pressure gradients or relative motion of the brain with respect to the skull. At the site of impact or the coup site, a positive pressure or compressive stress is developed and a negative pressure occurs at the contrecoup site. These pressures are accentuated by the deformation of the skull: in-bending at the coup site and out-bending at the contrecoup site. Positive pressure can contuse the brain, whereas the mechanism of injury for negative pressure can be due to either tensile loading or cavitation, which is compressive loading due to the collapse of vapor bubbles formed as a result of negative pressure. Injuries due to relative motion of the brain inside the skull are based on contusions seen on the surface of the brain and on a diffuse form of brain injury, DAI. This term was first coined by Strich (3), who observed retraction balls forming along injured axons that make up the bulk of the white

matter in the brain. She attributed this injury to shear, which can be caused by pressure gradients as well as by motion of the brain inside the skull. This motion is more pronounced when there is a large angular acceleration component of head motion. However, quantitative data of relative brain motion are just becoming available through the work of Hardy et al (4), who used a bi-axial high-speed X-ray unit to track radio-opaque targets in an intact and freshly dead cadaveric brain. There is an indication that although relative motion occurs even at mild levels of impact, the extent of the motion is less than that observed in physical models of the brain as well as in previous animal or cadaveric models, such as the 'lexan calvarium' described by Ommaya et al (5), the sagittal hemisection of Gurdjian & Lissner (6), and the rotational models of Thibault et al (7). Because of the complex structure of the brain and a possible difference in material properties between the gray and white matter, the development of shear strains or stresses in the brain is not easily visualized. However, axons are apparently stretched in the process, and the injury process leads to the enlargement of the axons at locations where there is damage to the microtubules within the axons. Microscopically, this is seen as either retraction balls or enlargements of sections of the axon with the use of appropriate biochemical stains. It has been shown by Gennarelli et al (8) that although DAI takes a few hours to develop in an injured brain, it is the result of the impact and is not a secondary injury due to other causes, such as ischemia or increased intracranial pressure. Research needs to continue to confirm the injury mechanisms attributed to relative brain motion, as described above. Another severe form of brain injury due to relative brain motion is the rupture of bridging veins that drain blood from the brain into the dural sinus. This short section of vein is stretched when the head experiences a large angular acceleration and the extent of the stretch is particularly large for veins that are directed more anteriorly than medially. This statement is based on a computer model by Zhou et al (9) simulating brain impact. The model predicted that a sudden angular deceleration of a head rotating rearward would tend to cause a bridging vein rupture, such as when a quarterback is sacked and lands on his back, sustaining an occipital impact with a large angular deceleration component.

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Mechanical Response of the Head

Mechanical Response of the Skull Early response data of head impact consisted of the response of the entire head as a function of drop height or impact speed. Hodgson & Thomas (10) provided such response data for embalmed cadaveric heads impacting a variety of rigid and padded surfaces in terms of peak force and peak acceleration as a function of free-fall drop height. McElhaney et al (11) impacted unembalmed heads with a pendulum and obtained response data as a function of the impact speed of the pendulum. The two sets of data are not directly comparable, indicating the wide variation in response in biological specimens. Data obtained from impacts against rigid surfaces are particularly useful for the design of dummy heads, which are usually metal headforms covered by a soft

vinyl cover. The stiffness of the cover can be tuned to mimic head impact against a rigid surface to produce a dummy head with a human-like response. The Hybrid III dummy head was developed based on these data (12).

Impact Response of the Brain Although many attempts have been made to determine the relative of motion of the brain inside the skull as a result of a blunt impact, reliable quantitative data were made available by Hardy et al (4) only very recently. Previous studies using the lexan calvarium, sagittal hemi-sections, and physical models lacked the assurance that the data would be the same if an intact brain was used. Hardy et al (4) obtained quantitative data by the use of a high-speed bi-axial X-ray machine that produced X-ray pictures of an instrumented cadaveric brain at 500 frames/second (fps). Neutral-density accelerometers (NDA) and low-density radio-opaque targets were inserted into an intact unembalmed cadaveric brain that was exposed to room temperature for a total of less than 24 hours after death. Preliminary data are encouraging. Figure 1 shows a single frame of the X-ray image in which the two NDAs (*small squares*), the two pressure transducers (*ovals*), and low-density targets (*small dots*) can be clearly seen. Because bi-axial images were obtained, the precise three-dimensional coordinates of each object could be obtained from each frame. The absolute displacement of the brain with

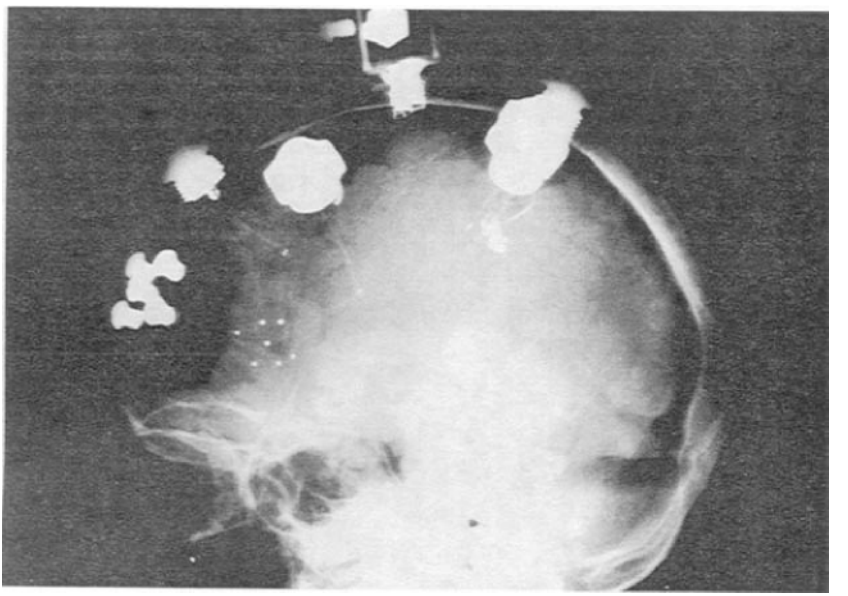

Figure 1 One frame of a high-speed X-ray video of a cadaveric brain, instrumented with neutral density accelerometers (squares), pressure transducers (ovals) and reduced density targets (small dots). The larger metallic pieces are skull-mounted hardware to seal the holes made during insertion (4).

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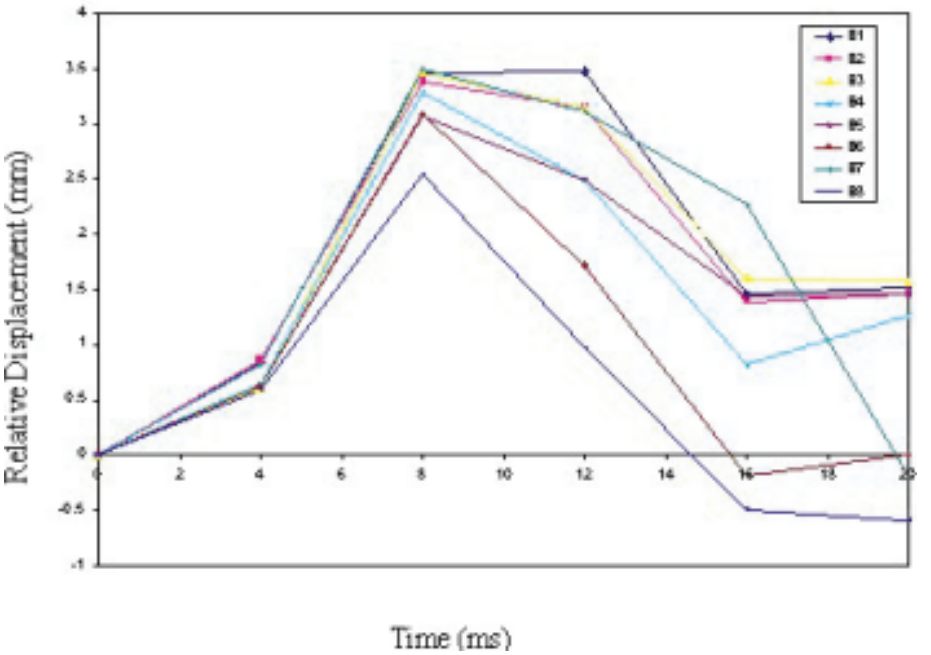

Figure 2 Relative motion of the brain with respect to the skull, measured from X-ray images (A1-Bsharat et al, 13).

respect to the skull measured from the X-ray images matched that computed from acceleration data from one of the neutral-density tri-axial accelerometers. For a low-level occipital impact by a pendulum at 2.7 m/s, the displacement of the brain relative to the skull is shown in Figure 2 (see color insert) (13).

Head Injury Tolerance

The principal concern in head injury is brain injury. One of the first attempts to define tolerance of the brain to linear acceleration was the Wayne State Tolerance Curve (WSTC), proposed by Lissner et al (14). A more comprehensive version of this curve, taken from McElhaney et al (15), is shown in Figure 3, which shows that the brain can tolerate higher accelerations if the duration of the pulse is shorter. It was based on cadaveric skull fracture data and concussive data from animals as well as on long-duration human sled experiments. Very few data points were used to plot this curve, and the instrumentation used was questionable. However, Ono et al (16) confirmed the validity of the curve through a series of experiments on subhuman primates. The WSTC was converted to a severity index by Gadd (17, 18) that was based on the WSTC. The equation for the Gadd Severity Index (GSI) is

$$\mathrm{GSI} = \int_0^T a(t)^{2.5}\, dt \leq 1000 \qquad (1)$$

where a(t) is the acceleration of the head center of mass, t is the time, and T is the duration of the impact pulse.

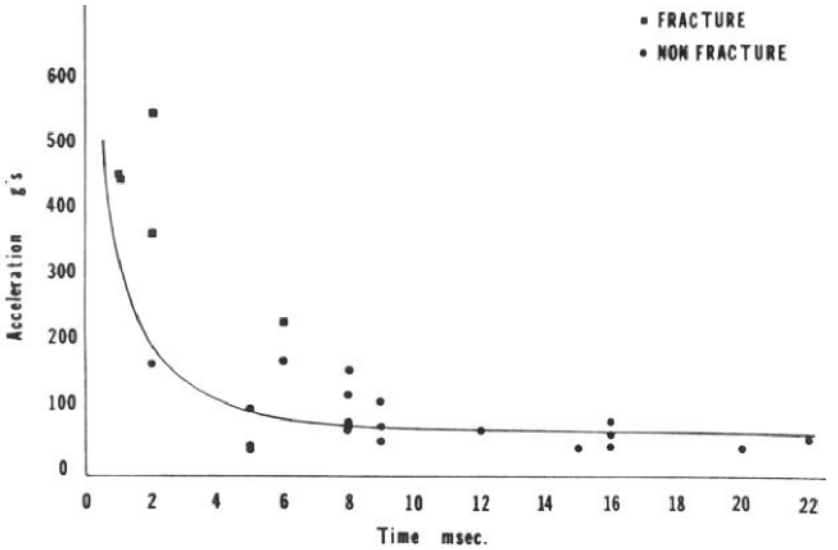

Figure 3 The Wayne State Tolerance Curve (15).

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The exponent 2.5 was taken from the WSTC, which could be approximated by a straight line with a negative slope of 2.5 if it was plotted on a log-log scale. Gadd picked this value of 2.5 as a weighting factor for his integral relationship and determined that a limit of 1000 matched some of the data generated at Wayne State. The GSI was subsequently modified to the Head Injury Criterion (HIC) by Versace (19), but the limit remained at 1000. The final form of HIC adopted by the US federal government as the head injury standard for Federal Motor Vehicle Standard 208 is

$$\mathrm{HIC} = \left[\int_{T_1}^{T_2} a(t)\,dt\right]^{2.5} \Big/ (T_2 - T_1) \leq 1000 \qquad (2)$$

This criterion is valid for linear acceleration impacts, but in most head impacts both linear and angular acceleration are present, and no validated injury criterion for angular acceleration is currently available. Despite its deficiencies, no substitute criterion has proven to be acceptable to the government or the biomechanics community. More animal research is needed to find a more effective criterion, but such research is limited by ethical and other issues.

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BIOMECHANICS OF THE NECK

Neck Injury Mechanisms

Neck injuries can range from mild to catastrophic. Generally, the injuries involving the spinal cord at the higher cervical levels are life threatening whereas those at the lower levels can result in paralysis. To injure the cord, it is necessary to disrupt the alignment or integrity of the cervical column. Burst fractures of cervical vertebral bodies can propel fragments into the cord and cause permanent cord damage. Subluxation of one vertebra over another decreases the size of the spinal canal, again causing cord damage. It is not necessary to sever the cord to produce quadriplegia. If the cord is impacted or crushed temporarily, sufficient damage can be done to paralyze the extremities. In the upper cervical area, separation of the atlas from the occiput is generally a fatal injury. Other life-threatening injuries to the upper column are multiple fractures of the arches of C1 and fractures through the pars interarticularis of C2 (hangman's fracture). Milder forms of cervical injury include the so-called whiplash syndrome caused by a rear-end collision. Although clinical literature frequently describes it as a real injury, the picture is confused by a multitude of claims of an injury for which the etiology is unknown.

Because the neck is a slender column that can be subjected to a variety of bending loads in association with an axial load, the injury modes can be classified as compression, tension-extension, tension-flexion, compression-extension, compression-flexion, and lateral bending.

Compression (and Bending) Injuries These injuries result from crown impacts to the head which produce a high compressive load on the neck accompanied

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by bending loads that can depend on the initial orientation of the head, initial configuration of the neck, and surface friction. They are not common automotive injuries but can occur in ejections and rollovers. The compression comes from the mass of the body following the head, which is stopped by a resisting surface. It was shown by Pintar et al (20, 21) that for burst fractures to occur, it was necessary to have the neck in the flexed position initially. That is, a high compressive load can be transmitted from the head to the neck only if the cervical spine was straight. Such burst fractures cause fragments of the vertebral body to move out radially in all directions. Those that move posteriorly toward the spinal cord are likely to cause permanent cord injury. Chang et al (22) used a hydraulic sensor to demonstrate that the extent of travel of the fragments exceeds that seen on X-rays and scans following the injury. Nightingale et al (23) showed that fracture dislocation of the cervical spine occurs within the first 10 to 20 ms of the impact and that the subsequent motion of the head depends on its initial orientation as well as on the manner in which the neck buckles and the magnitude of friction between the head and the surface it impacts (24).

Tension-Extension Injuries The whiplash syndrome is frequently associated with this type of injury. It is generally assumed that the injury is result of the hyperextension of the neck, which is also apparently in tension as the neck is stretched out over the headrest or seatback. However, the cervical spine is initially placed in compression by the seatback pushing on the kyphotic thoracic spine. Because the thoracic spine tends to straighten out, it pushes up on the cervical spine and down on the lumbar spine, applying a compressive load on both spines in the process. As a result, the ligaments and tendons holding the cervical spine together are loosened while the vertebrae are required to transmit a large shear force from the torso to the head. Yang & Begeman (25) postulated that this is the mechanism causing soft tissue injuries of the cervical spine. That is, the injury occurs well before the head and neck go into hyperextension. Unpublished data from cadaveric tests at Wayne State University show that there is stretch of the facet capsules within 40 ms after the onset of the impact. Moreover, Bogduk & Marsland (26), Aprill & Bogduk (27), and Lord et al (28) have clinical evidence that the reported neck pain is coming from the facet joint capsules of the neck. Recent volunteer tests by Matsushita et al (29) also show initial compression of the cervical spine, and there is anatomical evidence that the facet capsule contains nerve endings that can sense pain (30). If the compression/shear hypothesis is shown to be valid, the most effective manner of preventing whiplash injuries is to redesign the headrest so that it is in close proximity with the head so that it can push the head forward at the same time as the seatback is pushing the torso to minimize the shear force in the neck.

In more severe impacts, hyperextension injuries do occur. They include tear-drop fractures of the anterior-superior aspect of the vertebral body and separation of the disc from the anterior vertebral endplate. At times, a diagnosis of a herniated or bulging cervical disc is made after prolonged complaints of neck pain. In the

opinion of this author, such findings are coincidental and are the result of a pre-existing disc condition that may have been asymptomatic prior to the crash or the pain was not constant or severe enough to warrant a visit to the doctor. The basis for this opinion is that intervertebral discs cannot be ruptured as the result of a single loading event in the absence of massive bony fractures of the adjacent vertebrae (31).

With the advent of the airbag, severe life-threatening injuries to the cervical spine can occur to out-of-position front seat occupants or drivers of short stature. The tension-extension injury is caused by the deploying airbag, a part of which gets under the chin and separates the head from the neck at the atlanto-occipital junction or the C1/C2 junction. A preliminary indication of this hazard was reported by Cheng et al (32), who produced this injury in cadavers using a pre-deployed airbag that was placed on a vertical steering wheel.

Tension-Flexion Injuries These are relatively uncommon because complaints of chronic or persistent neck pain by belted occupants involved in frontal crashes are rare. In very severe frontal crashes, atlanto-occipital and C2/C2 separation can occur. Thomas & Jessop (33) produced these injuries in subhuman primates that were fully restrained and were subjected to a frontal deceleration of 120 g.

Compression-Extension Injuries These injuries can occur to unrestrained front seat occupants involved in a frontal crash. When the head impacts the windshield, the neck is placed into extension and compression simultaneously. Such occupants are likely to sustain fracture of one or more spinous processes as well as symmetrical lesions of the pedicles, facets, and laminae. If there is a fracture-dislocation, the inferior facet is displaced posteriorly and upward and appears to be more horizontal than normal on X-ray (34).

Lateral Bending Injuries Lateral bending occurs when there is a side or oblique impact. This is usually accompanied by shear and axial loading. Injuries characteristic of this type of loading are lateral wedge fractures of the vertebral body and fractures to the posterior elements on one side of the vertebral column. Avulsion of the brachial plexus can also occur. When the neck is subjected to twisting, unilateral facet dislocations or unilateral locked facets are seen (35). However, pure torsional loads on the neck are rarely encountered in automotive crashes.

Mechanical Response of the Neck

The response of the cervical spine to loads from various directions has been studied by Mertz & Patrick (36, 37), Patrick & Chou (38), Schneider et al (39), Ewing et al (40), and more recently by Nightingale et al (41). Mertz et al (42) elected to quantify response in terms of rotation of the head relative to the torso as a function of bending moment at the occipital condyles and obtained loading corridors for flexion and extension. These data form the basis for the design of the Hybrid III

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dummy neck. Volunteer data on neck bending were obtained by Patrick & Chou (38). Neck response curves for sagittal flexion and extension, from four test subjects, were found to fall within the loading and unloading corridors established by Mertz & Patrick (36, 37), upon which the loading corridors of Mertz et al (42) were based. Schneider et al (39) have studied the lateral flexion response properties of the human neck. They tested 96 male and female volunteers, ranging in age from 18 to 74 years. They also documented their head and neck anthropometric information. The three-dimensional range of motion of the head relative to the torso was measured, and the response of the head and neck to low-level acceleration was studied.

The voluminous data acquired at the Naval Biodynamics Laboratory in New Orleans constitute a valuable source of neck response data for volunteers who were tested to relatively high acceleration levels. Some of data have been analyzed by Wismans & Spenny (43, 44), but the bulk of the data have not. Frontal response data were analyzed and compared with cadaveric responses by Wismans et al (45). Head trajectories were found to be comparable, but the cadaveric head rotation was larger than that of the volunteer. A new MADYMO neck model simulating volunteer head-neck response was recently reported by van der Horst et al (46). Curved lines were used to simulate the lines of action of muscles, and a delay time for muscle activation was incorporated into the model. However, this delay was not based on muscle stretch or stretch rate. The model was validated against available lateral and frontal impact data. It was concluded that the delay in muscle activation was an important element in producing realistic results and that active muscle behavior was essential for an accurate description of human-neck response.

There have also been several recent studies involving neck extension in simulated rear-end collisions. Matsushita et al (29) and Ono et al (47) obtained X-ray movies of volunteer neck motion at 90 frames per second. It was found that the neck was in compression due to the upward ramping of the torso on an inclined seatback and that there was more relative rotation in the lower cervical vertebrae (C5-6), leading to the hypothesis that the injury could be in the facet capsules in low-speed rear-end collisions. More recent results obtained by Deng (48) from cadavers and through the use of a bi-axial high-speed X-ray camera device at a framing rate of 250 per second demonstrated that compression began early in the impact, even if the seatback was vertical, and that there were both relative translation and rotation between adjacent cervical vertebrae.

Camacho et al (24) studied the response of the cervical spine due to a crown impact to the head. High-speed video pictures were obtained to show the buckling mode of the cervical spine. Figure 4 (see color insert) shows the time course of the buckling for a crown impact against a rigid surface.

Neck Injury Tolerance

There is no widely accepted tolerance for the various loading modes on the neck. The reasons for this inability to set tolerance levels are many. Some of the more

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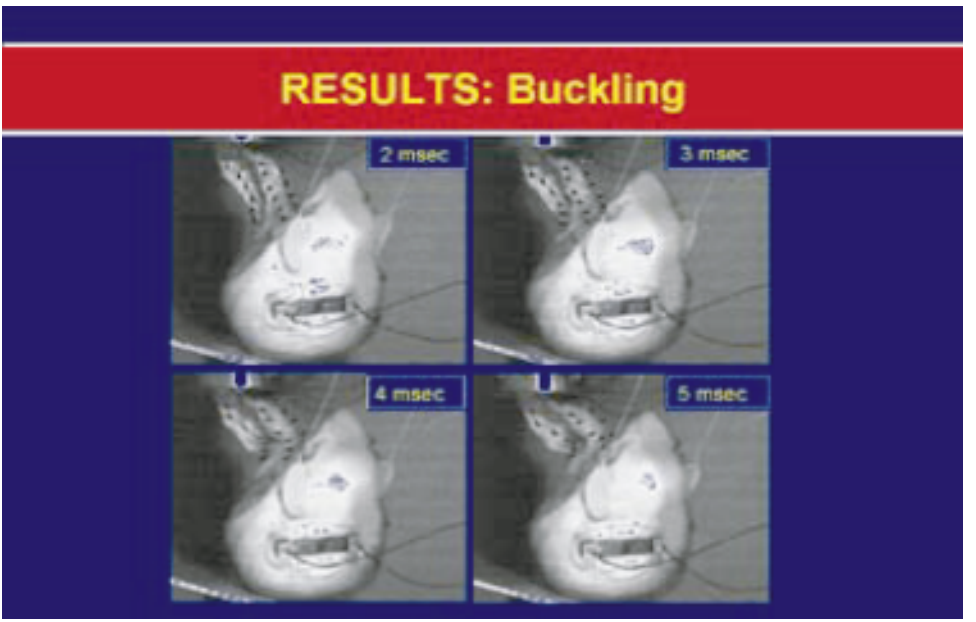

Figure 4 Time course of neck buckling during a crown impact (Nightingale et al, 41).

significant ones were provided by McElhaney & Myers (49). The spine is a multi-segmented column with nonlinear structural properties. Its geometry is complex, it produces large strains at physiologic loading, and its constituent elements have nonlinear material properties. Cervical injury mechanisms have been shown to be sensitive to the initial position of the neck, the direction of loading, the degree of constraint imposed by the contact surface, and possibly the rate of loading. These factors are in addition to the normal biological variation in the strength of human tissue and the level of injury we are willing to tolerate. For example, serious neck problems are not encountered in frontal crashes unless the g-level of impact is very high. However, in rear-end collisions, long-term neck pain can result, even though the impact is of low level. Tolerance of the cervical spine is next discussed for different loading modes, based largely on an excellent review by Myers & Winkelstein (50).

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Tolerance of the Neck in Flexion-Extension There is ample evidence that the neck can take a fairly high frontal deceleration without injury. Ryan (51), using himself as a test subject, withstood a 23-g impact without injury. He was wearing a single belt with tighteners. Ewing et al (52) conducted volunteer tests at 10 g and reported only belt contusions from the military harness used. Head accelerations at the mouth mount reached 38.6 ± 6.8 g. Similar tests with male volunteers performed by Cheng et al (53) reached a maximum sled deceleration of 10 g with no reported injuries. However, in the same series, only one of three female volunteers was willing to reach the 10-g level. The main reason given for discontinuing the tests was the intolerable whipping of the head due to weakness in the neck. Volunteer test results reported by Mertz & Patrick (36, 37) are the most frequently cited and widely used. The only volunteer was Prof. Patrick himself. He withstood a flexion moment of 59.4 N.m (Newton-meters) (43.8 ft-lb) with neck pain. This was defined as the pain threshold. At 87.8 N.m (64.8 ft-lb), he had an immediate onset of pain and prolonged soreness. This was defined as a flexion injury threshold.

Many cadaveric studies on neck flexion have been reported. Lange (54) produced a variety of neck injuries at high levels of sled deceleration in both frontal and rear-end impacts. The injuries were above human tolerance, and the only reason for citing this paper is to emphasize the fact that the observed "disc ruptures" were, in fact, disc separations from the endplates, accompanied by rupture of the longitudinal ligaments.

Tolerance of the Neck in Extension A large number of studies has been conducted to study the problem of whiplash-associated disorders (WAD). These were mostly at low impact levels aimed at understanding the causes of neck pain resulting from minor rear-end crashes. On the other hand, there were the studies by Clemens & Burow (55), who created disc injuries that were frequently associated with anterior longitudinal ligamentous rupture. There were also some joint capsular tears and bony fractures. Because of the overly severe input used (approximate sled acceleration of 25 g), it was not possible to establish a threshold for any of the injuries that were documented. The work of Lange (54) is also at the severe

level (19–29 g sled acceleration), and the injuries found were consistent with those reported by Clemens & Burow (55).

Returning to the work of Mertz & Patrick (36, 37), we find that the static limit for Prof. Patrick was 23.7 N.m (17.5 ft-lb) and the average static limit from 10 volunteers was 21.2 N.m (15.6 ft-lb). Their dynamic results show that the moment tolerated at the base of the skull was 16.7 N.m (12.3 ft-lb) for Prof. Patrick. The proposed noninjurious limit is 47.4 N.m (35 ft-lb) or twice the static limit of Prof. Patrick, and the proposed ligamentous injury limit is 57 N.m (42 ft-lb). This limit is based on ligamentous damage to a small cadaver at 33.4 N.m (24.6 ft-lb), which when scaled to the size of Prof. Patrick was 57 N.m. The scaling method used was proposed by Mertz & Patrick (36). These limits may be too high for neck pain associated with whiplash. Pain can occur without any visible damage to the soft tissue. Microscopic examination of the tissue may be necessary to establish a basis for whiplash-induced pain.

Tolerance of the Neck in Lateral Bending There do not appear to be much tolerance data of the neck in lateral bending. Analysis of volunteer data obtained by Ewing et al (56) by Wismans & Spenny (43) show that there were no obvious injuries from runs made at 5 to 10 g. These tests resulted in a lateral bending moment of 20 to 60 N.m and lateral rotations of 52 deg. Cadaveric studies have been conducted by Kallieris et al (57) simulating three-point belted near-side occupants. The 58 cadavers tested ranged in age from 19 to 65 years and the impact speeds were between 40 and 60 km/h (25 to 38 mph). A variety of injuries, ranging from AIS 1 soft tissue damage to AIS 3 or higher bony fractures were found, frequently at the C6 level. The maximum head resultant acceleration for these tests was 163 g. Far-side lateral impacts were studied by Horsch et al (58) and by Kallieris & Schmidt (59). When an in-board shoulder belt was used, some AIS 1 cervical injuries were found in both studies. However, in the older cadavers used by Horsch et al (58), they found transverse clefts of cervical discs as described above. It is not clear whether the tolerance values from this study are valid or not. The delta V for the Horsch experiments was between 33 and 37 km/h (21 to 23 mph). In the second study by Kallieris & Schmidt (59), the cadavers used were younger and only AIS 1 injuries were found for a delta V of 50 km/h (31 mph).

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BIOMECHANICS OF THE THORAX

Thoracic Injury Mechanisms

The thorax houses organs essential to life. It is protected to a certain extent by the rib cage and the thoracic spine, but the forces encountered in a severe automotive crash are frequently large enough to fracture the ribs and sternum as well as tear the main arteries within the thorax or injure the walls of the heart. At times, high-speed blunt impacts can cause the heart to go into ventricular fibrillation. The lungs can

be contused by impact with the chest wall or by the passage of compression waves through the alveolar tissue. They can also be lacerated by the ends of fractured ribs. The various injury types appear to be dependent on the rate of loading because of the viscoelastic nature of the tissues involved. At low rates of loading, the injury is due to deformation of the rib cage or crushing. At very high rates of loading, such as a pressure wave from an explosion or impact to the chest by a high-speed blunt object (baseball or nonlethal munitions), the injury is a function of the speed of the wave or of the missile. For automotive crashes, both deformation and rate of loading play a role in injury causation.

Low-Speed Crush Injuries For impact speeds below 3 m/s (about 5 mph), the contents of the thorax are injured by the crushing of the rib cage. As is shown below, the amount of crush necessary to induce injury is around 35% of the depth or width of the thorax (34). The injury mechanism is compression of the organs.

Automotive-Related Chest Injuries In the range of 5 to 30 m/s, the cardiovascular injury mechanism is compression accompanied by shear and tensile loading. Aortic ruptures can occur in both frontal and lateral impacts to the chest, although in the cadaver, such ruptures have not been reproduced in simulated frontal impacts. That is, the blood vessels are apparently more vulnerable in a side impact. Since the rupture tends to occur at attachment points of inter-arterial ligaments, it is postulated that these points constitute areas of stress concentration. As for the lung, the extent of damage depends not solely on the degree of compression of the chest but also on the speed of impact. This conclusion is based on studies by Viano & Lau (60), who chose the liver as a target organ and performed impacts with different combinations of speed and compression to establish the dual dependency in this speed range. Animal models were used for this study.

Injury Mechanisms of the Thoraco-Lumbar Spine The frequency of injury to the thoraco-lumbar spine is very low (<1%), based on relatively old data reported by King (61). However, if the injury involves the spinal cord, paraplegia can result. To understand the mechanisms of injury to the thoraco-lumbar spine, it is necessary to review briefly the contributions made by many investigators in the area of spinal injuries resulting from pilot ejection in which the spine is subjected to a caudocephalad (tail-to-head) acceleration. Anterior wedge fractures of the lower thoracic and upper lumbar are seen in pilots who eject. Vulcan et al (62) determined that the mechanism of injury for these wedge fractures is a combined compressive and bending load. The bending is due to the fact that the center of mass of the torso is anterior to the spine. Prasad & King (63) demonstrated that there are two load paths down the spine to transmit vertical (axial) compression generated by inertial loading, depending on the orientation of the lumbar spine. The two load paths are the discs and the articular facets that transmit compressive load by bottoming the tips of the inferior facets onto the laminar of vertebra below. Belted occupants involved in severe frontal crashes can also sustain anterior wedge fractures of the

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thoraco-lumbar spine due to the same mechanism but from a different loading source. In this case, there is no vertical inertial loading but the compressive load is generated by the kyphotic thoracic spine, which tends to straighten out against the lap belt during the crash. This mechanism is the same as the one that imposes a compressive load on the cervical spine during a rear-end collision. However, it was first confirmed experimentally by Begeman et al (64) after it was predicted by a two-dimensional spinal model developed by Prasad & King (63).

In terms of other forms of soft tissue damage, there is a frequent complaint of low back pain following an automotive collision. The impact severity can range from a minor fender bender to a very high speed crash. In some cases, a herniated disc is diagnosed and the treating physician will almost invariably attribute the rupture to the crash. However, predominant findings in the literature indicate that disc rupture is a slow degenerative process and, thus, an extremely violent single loading event is needed to cause the nucleus pulposus to extrude from the side of the disc. That is, a single load cannot rupture a disc unless there are concomitant fractures of the adjacent vertebrae. Intervertebral discs do not herniate like a balloon and back pain is not solely due to the disc. There are many sources of back pain, including the facet capsules, and a causal relationship between an impact and a ruptured disc does not exist. A more detailed discussion is provided by King (31).

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Mechanical Response of the Thorax

Frontal Thoracic Response *Response to Flat Impactors.* In the automotive environment, the thorax can be loaded frontally by a rigid or deformable surface, such as an instrument panel, steering wheel, or shoulder belt. Frontal response was first studied by Patrick et al (65), who tested several embalmed cadavers statically and dynamically. Antero-posterior static stiffness was found to vary from 32.4 to 70.0 kN/m (185 to 400 lb/in) when the chest was loaded by a 102-mm (4-in)-wide bar. Dynamic stiffness prior to rib fracture, based on impacts with a 152-mm (6-in) diameter padded pendulum, was approximately 175.1 kN/m (1000 lb/in) for loads up to 4 kN (900 lb). The stiffness dropped markedly after the rib fracture, but it recovered to about half the pre-fracture stiffness when the internal organs were compressed. The stiffness increased with loading rate. The static stiffness of an unembalmed thorax was found to vary from 6.3 to 10.9 kN/m (36 to 62 lb/in). Dynamic data were obtained by Nahum et al (66) and Kroell et al (67) using a 152-mm (6-in) impactor, weighing 227 N (51 lb) or 191 N (43 lb).

Response to Belt Loading. The diagonal shoulder belt elicits a response from the thorax quite different than a 152-mm (6-in)-diameter impactor. Schmidt et al (68) and Patrick & Levine (69) have reported asymmetric rib fracture patterns in which all of the fractures were in the sternum or the lower in-board rib cage. This difference can be exemplified by static volunteer data acquired by Fayon et al (70). Belt loading stiffness at the sternum ranged from 17.5 to 26.3 kN/m (100 to 150 lb/in), whereas that for disc loading ranged from 8.8 to 17.5 kN/m (50 to

100 lb/in). The belt loading stiffness of the second rib was from 17.5 to 26.3 kN/m, whereas that of the ninth rib was from 8.8 to 17.5 kN/m (50 to 100 lb/in). L'Abbe et al (71) provided additional volunteer belt-loading stiffness data. For loads of up to 650 N (150 lb) and deflections of 10 mm (0.4 in), the static stiffness under the belt was 67.6 kN/m (386 lb/in) at the mid-sternum, 40.0 kN/m (228 lb/in) at the seventh rib, and 94.8 kN/m (541 lb/in) at the clavicle. Their dynamic loads peaked at 3.6 kN (810 lb), and their dynamic deflections were twice the static values. The dynamic belt-loading stiffness averaged 137.5 kN/m (785 lb/in) at the mid-sternum, 123.2 kN/m (703 lb/in) at the seventh rib, and 200 kN/m (1142 lb/in) at the clavicle.

Walfisch et al (72) reported a dynamic belt-loading stiffness of about 70.0 to 161.1 kN/m (400 to 920 lb/in). The data are approximate because the deformation was given as a percentage of chest deflection and a 9-in chest depth was assumed. The mean stiffness was 119.4 kN/m (682 lb/in).

It is not clear why the static stiffness values provided by Fayon et al (70) were much lower than corresponding data by L'Abbe et al (71). One possible explanation is that the supine posture of the subjects used by Fayon et al (70) caused the spine to assume a curved shape, which could have lowered the apparent stiffness of the rib cage.

Side Impact Thoracic Response Stalnaker et al (73) conducted the first side impact tests on the left side of six unembalmed cadavers. A 152-mm (6-in) diameter flat impactor with a mass of 10 kg (22 lb) was used at two impact speeds—6.1 and 8.8 m/s (20 and 29 ft/s). The initial dynamic stiffness ranged from 273.6 to 437.8 kN/m (1563 to 2500 lb/in) at 6.1 m/s (20 ft/s). At the higher velocity, the values were 437.8 and 790.0 kN/m (2500 to 4500 lb/in).

Drop tests were then conducted by Stalnaker et al (74) in which the thorax, abdomen, and pelvis of unembalmed cadavers were dropped on their sides against rigid or padded surfaces instrumented with load cells. The drop heights were 1.0 m (3.3 ft) for rigid impacts and 2.0 m (6.6 ft) for padded impacts. The dynamic stiffness of the thorax was found to range from 96.8 to 255 kN/m (553 to 1457 lb/in).

Pendulum impacts conducted by Viano et al (75) were directed at a 60-deg angle from the antero-posterior axis to ensure that the photographically measured deflection did not involve any whole-body rotation of the rib cage. The flat impactor used had a rigid surface. It was 152 mm (6 in) in diameter and had a mass of 23.4 kg (51 lb). The three impact speeds used were 4.3, 6.7, and 9.5 m/s (10, 15, and 20 mph). Stiffness values were not computed, but the suggested corridors had an initial stiffness of 100 to 140 kN/m.

Thoracic Injury Tolerance

The study of human tolerance of the chest to blunt impact was initiated to protect the unbelted driver involved in frontal crashes. The information was needed to design energy-absorbing steering wheel columns and deformable steering wheels.

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Later studies involved the interaction of the lapbelt with the rib cage, and more recent work concentrated on thoracic tolerance to side impact. Because there are abdominal organs within the rib cage, it is often difficult to separate the discussion of injuries to the thoracic viscera from those of the abdomen. In this chapter, tolerance of the upper abdominal organs such as the liver and spleen are discussed.

Frontal Thoracic Tolerance Stapp (76, 77) demonstrated that the chest can sustain decelerations of up to 45 g when fully restrained with a double shoulder harness. The computed pressure under the harness was 252 kPa (36.5 psi). Peak accelerations of 30 g at a rate of 1000 g/s were not tolerated. Mertz & Kroell (78) reviewed other early frontal tolerance data. Patrick et al (65, 79) obtained force tolerance values based on tests on unrestrained but embalmed cadavers. At a peak dynamic load of 5.96 kN (1340 lb) one cadaver sustained 4 rib fractures, whereas at 8.23 kN (1850 lb), extensive fractures were observed. These data were supplemented later by Kroell et al (67, 80), who performed a large number of frontal chest impacts, using a 152-mm (6-in)-diameter rigid pendulum, as described above. They found that chest compression correlated well with AIS ($r = 0.730$) whereas the maximum plateau force did not ($r = 0.524$).

The current FMVSS 208 on chest compression is based on a recommendation by Neathery (81), who analyzed the Kroell data and found that a compression of 76 mm (3 in) would result in an AIS of 3. The compression values in terms of percent of chest depth and compression for a 50th percentile male and the corresponding AIS values are shown in Table 2. An AIS of 4 was assigned to those cases in which the number of rib fractures was sufficient to cause a flail chest, in the opinion of the investigators. Viano et al (82) recommended that the chest compression limit should be 32% because at 40%, there was severe injury to the thoracic viscera and that at 32%, there would not be adequate rib stability to protect the internal organs.

Viano & Lau (60) defined an alternate injury criterion for the chest. It is called the Viscous Criterion (V^*C) and is the instantaneous product of chest wall velocity (V) and chest compression (C) expressed in percent of chest depth. This criterion was developed based on studies carried out by Lau & Viano (83), who impacted rabbit livers at speeds of 5 to 20 m/s (16.4 to 65.6 ft/s) at a C_{max} of 16%. Liver injury

TABLE 2 Chest Compression Injury Criteria

Chest compression (%)	50th percentile chest compression (mm)	Abbreviated Injury Scale (AIS)
30	69	2
33	76	3[a]
40	92	4

[a]Estimated by Neathery et al (81).

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was found to increase with increasing impact velocity. In a second study involving frontal impacts to the chest of 123 anesthetized rabbits, Lau & Viano (84) observed that lung injury increased with C_{max} at velocity levels of 5, 10, and 18 m/s (16.4, 32.8, and 59.1 ft/s). The alveolar region of the lung was more sensitive to velocity than regions of vascular junctions.

Lateral Thoracic Tolerance *Velocity- and Compression-Based Injury Criteria.* The cadaveric experiments reported by Viano et al (75) produced reasonable correlation between injury and VC_{max} as well as peak compression, C_{max}. For a 25% probability of injury, VC = 1.5 m/s (4.9 ft/s) and C = 38%. These data were confirmed by tests reported by Cavanaugh et al (85), who found that VC and C were more predictive of injury than force- or acceleration-based criteria. A total of 17 tests were eventually conducted in this series, and it was found that a VC_{max} in excess of 1 m/s (3.3 ft/s) would result in thoracic injuries of AIS 4 to 5 (86).

Acceleration-Based Injury Tolerance. The most well-known acceleration-based injury criterion is the Thoracic Trauma Index (TTI) proposed by Eppinger et al (87). The formula to compute TTI for a cadaveric test is given below:

$$\mathrm{TTI} = 1.4 \times \mathrm{Age} + 0.5 \times (\mathrm{Rib_y} + \mathrm{T12_y}) \times \mathrm{Mass/Mass_{50}} \qquad (3)$$

where Rib_y is the struck side rib 4 peak acceleration, $T12_y$ is the 12th thoracic vertebral peak acceleration, Mass is the mass of test subject, and $Mass_{50}$ is the mass of a 50th percentile male subject.

This criterion was based on an earlier analysis of data from 30 cadaveric tests reported by Eppinger et al (88). In 27 of these tests, the cadaveric subjects were instrumented with the 12-accelerometer array on the chest and spine, as specified by NHTSA for all tests done under US Department of Transportation contracts (Eppinger et al, 89). The analysis concentrated on the peak accelerations of the struck 4th rib and that of the T12 lateral acceleration. Marcus et al (90) normalized the AIS observed in these tests to a 45-year-old subject, using the following relationship:

$$\mathrm{Normalized\ AIS} = \mathrm{AIS} - 0.025 \times (\mathrm{Age} - 45)$$

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DISCUSSION

Biomechanics of the Brain

Several head injury mechanisms have been proposed, including pressure, shear, and relative motion. There is evidence that these mechanisms do indeed cause concussion, DAI, surface contusions, as well as bridging vein ruptures. However, it is still not clear how an axon is injured by shear. It appears to be too flexible

to be sheared, except at the neuron/axon level, but it can be injured by the tensile component of a shear tensor, or it can be stretched by a tensile force. This exact mechanism has not been fully explored. Injury mechanisms at the molecular level are still not fully understood. It is known that a traumatic event in the form of mechanical, thermal, or electrical energy opens up calcium channels in the membrane, allowing calcium ions to enter the cell. At the end of the traumatic event, the excess calcium ions cause an enzyme to be formed, killing the cell. It is not clear why the calcium pump could not keep up with the influx of calcium as a result of the trauma or how the formation of the enzyme can be stopped.

Although quantitative high-speed X-ray data of cadaveric brain response to impact are becoming available, the response of the entire brain to impact will take a long time to map. One possible way to speed up the process is to combine the experiment with a validated computer model that can predict the response of every part of the brain and simulate a large variety of impact situations that are difficult to accomplish experimentally.

Historically, brain injury tolerance was divided into tolerance to linear and angular acceleration, even though the head is usually subjected to both types of acceleration in any given impact. The Head Injury Criterion or HIC was developed based on the linear acceleration of the skull, impacting a rigid surface. However, since the standard was adopted about 25 years ago, the automobile industry has used HIC with a certain degree of success even though most impacts involve an angular acceleration component. At present, a reliable angular acceleration tolerance limit has not been established. This value may exceed 16,000 rad/s^2. Future standards may require the use of a computer model to compute the distribution of strain in the brain, and the tolerance limit will be a pre-set strain limit that should not be exceeded.

Biomechanics of the Neck

Neck injury mechanisms of a rear-end impact are still being debated, particularly for low-speed crashes that produce no objective signs of injury. One such mechanism was proposed in this paper, but there are many proposed theories. Research is in progress to establish the precise mechanism, and effective prevention of whiplash injury can only come about when that mechanism is understood.

The response of the neck also requires extensive study, even though there is a substantial body of knowledge on cervical spine response. Much data need to be collected to describe its response fully. The use of a validated model of the cervical spine can extend the study more effectively and in less time. The use of a bi-axial X-ray machine to determine relative vertebral motion will likely provide data that can be the basis for a full understanding of the whiplash phenomenon.

Neck tolerance is defined in terms of the various modes of loading. In the absence of additional data, the data generated by Mertz & Patrick (36, 37) remain

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as the most reliable guide for neck flexion and extension. Reliable dynamic lateral flexion tolerance data do not appear to be available.

Biomechanics of the Thorax

One of the most difficult injuries to reproduce in the cadaver is aortic rupture due to a frontal chest impact. Several unsuccessful attempts were made at Wayne State University, using cadavers with a pressurized arterial system. It has been hypothesized by Viano (91) that aortic tears occurred as a result of the development of high pressures within the artery which, when combined with a large stretch, will cause the artery to fail. Another hypothesis is that the tears occur because of stresses at the junction of ligaments that hold the arterial system together. Such injuries occurred in cadavers that were subjected to a side impact. The ligamentum arteriosum, connecting the aorta with the pulmonary artery, was responsible for this injury, as described by Cavanaugh et al (85). As to why this injury could not be reproduced in frontal impact is still a mystery. Another unknown injury mechanism is the reason for the heart to go into unrecoverable fibrillation after an impact to the chest by a projectile, such as a baseball. Protective gear can be developed only after the injury mechanism is fully understood.

The mechanical response of the shoulder has not been fully characterized. In side impact, its motion has been described by Irwin et al (92). More data are needed to determine the stretch of the ligaments and motion of the bones that make up the glenoid joint.

Frontal thoracic tolerance can be expressed in terms of acceleration, V*C, or displacement. The current standard using the 60-g, 3-ms clip and the 3-in deflection appears to work well, but the V*C limit of 1.0, as proposed by Viano and Lau (60), is statistically a better predictor of thoracic injury. Recently, Bir & Viano (93) have verified that V*C is a good predictor of thoracic injury for impacts in the velocity range of 9.7 to 14.5 m/s. This conclusion was based on 41 swine impact tests performed by Kroell et al (94). For side impact, the competing injury criteria are TTI, V*C, and compression, each of which is based on a different test dummy. The problem lies more with the selection of the dummy than with the injury criterion. It is up to the harmonizers of international standards to settle on a single standard using a single dummy.

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CONCLUSIONS

The body regions covered in this chapter are the most crucial in terms of survival. Needless to say, prevention of injury to the head (brain) is of paramount importance in terms of mental function and coordination of motion. With the introduction of airbags in automobiles and new regulations regarding head protection for other areas of the vehicle, the probability of head injury has been reduced considerably. However, about a quarter of all automobile-related fatalities are associated with

pedestrians, motorcyclists, and pedal cyclists. Prevention of head injury for these groups as well as for people involved in falls and in contact sports remains a substantial challenge to safety professionals.

The prevention of neck injuries is also a difficult task. The fact that the neck can be injured in so many ways and over a large range of input conditions renders the task both daunting and challenging. Catastrophic injury risks can be reduced by the use of restraints in automobiles in the event of a rollover, but in contact sports and diving into shallow pools there is still a need to exercise behavioral control because currently available methods of environmental control are not effective. For whiplash-associated disorders, the mechanism of injury needs to be established before effective treatment and prevention modalities can be introduced.

As for the thorax, the prevention of aortic rupture in severe crashes should be a top priority item in research. A fuller understanding of the mechanism of ventricular fibrillation due to a direct impact to the chest is also needed for the prevention of this devastating injury.

The use of computer models to simulate impact to these body regions is already quite advanced. They can certainly help in the testing of new methods of injury prevention. For example, there is no biomechanical basis for making the liner of a helmet of equal thickness all the way around the head. Perhaps a good brain injury computer model can be used to design a helmet for optimal 360-degree protection. Finite-element models of the neck are particularly useful in the prediction of high stresses in the cervical spine in a large variety of combined loading conditions that would be difficult to determine experimentally. Similarly, the modeling of the heart and great vessels can perhaps extend our understanding of the nature of catastrophic injury to these areas.

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Annual Review of Biomedical Engineering
Volume 2, 2000

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