

INJURY CRITERIA AND HUMAN TOLERANCE FOR THE NECK

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INTRODUCTION

It is curious that only rarely does a standard reference work contain an anatomical definition of the neck. It may help for us to accept the definition of Walker et al. [1]. This uses two anatomical planes, one of which separates the head from the neck and the second of which separates the neck from the torso. No neck vertebrae are included in the head specimen, and no thoracic vertebrae are included in the neck specimen. It is believed that this represents the closest anatomical definition of neck in the literature.

In contrast, Clauser et al. [2] chose to include almost two cervical vertebrae in the head specimen and a neck specimen was apparently not divided from the torso.

Examination of Walker's data shows that, excluding cadavers one, five, and twenty, the mean head-neck weight of the seventeen remaining was 6,075 gm. The mean headweight was 4,463 gm and by difference, the mean neck weight was 1,609 gm or about 3.55 lbs.

This seemingly insignificant portion of the total body weight is important far beyond the ratio of weights, however, since with the exception of the cranial nerves, the entire input to the brain and output from the brain must traverse this organ. Due to its somewhat paradoxical requirements for mobility on the one hand and protection for the neural pathways on the other, a complex structure is found. Several major arteries and veins pass through the neck exterior to the vertebral column which protects the neural pathways (or spinal cord). The autonomic nervous pathways also lie exterior to the vertebral column, as do the portions of the organs of respiration and digestion which pass through the neck.

The internal protection of the neck consists of seven cervical vertebrae with ligament attachments, while external protection consists of skin and underlying muscle.

These anatomical and anthropometric factors are important in considering the tolerance limits of the human neck to impact acceleration of various types.

CRASH CONDITIONS AND NECK INJURY

It is convenient to consider crash injuries to the neck as being of two types: (1) those due to energy transmitted directly to the neck and (2) those due to energy transmitted indirectly to the neck from either the torso or the head.

Direct Energy Transmission

Direct impact injuries to the neck occur from the usual or expected sources, such as impact with objects in the seat's adjacent environment, and from impact with some unusual ones. The usual objects are aircraft seat tray-tables, other portions of adjacent seats, headrests, broken canopies or passenger windows, weapons systems components or subsystems, communication systems, and the instrument panel as well as automobile dashboards and steering wheels.

Prevention of these injuries is rather complex in concept, involving determination of human head-neck dynamic response with a variety of restraint systems and, using those data, design of a minimum clearance envelope around the eye reference point of sufficient size that direct impact with the neck cannot occur. Obviously a variety of human anthropometric sizes must be tested to obtain such data, and each must be tested with the different restraint systems unique to or used in each vehicle. The task becomes enormous if human subjects are to be used in each test, and the hazard to the subjects becomes more severe the closer one approaches the force envelope of normal use for this emergency system.

The unusual causes of direct impact are those caused by the helmet, helmet components, or restraint harness. These are potentially lethal and must be prevented if possible. The impactors of the neck are the trailing edge of the helmet chinstrap, the edge of the helmet shell itself, and the edge of the restraint harness.

If the chinstrap is improperly designed or worn, the strap will come diagonally forward from its attachment points on the shell and its posterior edge will rest upon the hyoid bone. During a crash, the helmet shell, if permitted by retention system design, may rotate forward, causing a direct impact of the chinstrap on the hyoid bone. Fracture of the hyoid is invariably fatal unless emergency surgery is performed within four minutes. It is unfortunately true that the hyoid bone is usually not carefully examined at autopsy. The number of deaths in accidents from this cause is therefore unknown.

Experiments performed by Melvin and Synder et al. [3] at the Highway Safety Research Institute of the University of Michigan on fresh cadaveric material show: "The loads to cause fracture in both the thyroid cartilage and the cricoid cartilage were found to be higher under dynamic loading than under static loading. For the thyroid cartilage, the mean dynamic fracture load was 40.6 pounds and for the cricoid, 55.5 pounds." Under static compressive loading, mean load to fracture all the larynges was 110 lbs.

The helmet shell itself can rotate backward as a result of direct impact to the shell, and has been shown to cause posterior compression fractures of the cervical vertebrae with cord transection at the middle portion of the cervical cord. This can also occur during high-speed ejection due to wind blast. It may also rotate laterally due to direct impact to the shell and may cause transverse process fractures in the cervical vertebrae. The only prevention for this type of injury is development of a test method for evaluating helmet retention systems, and this hinges upon development of a validated human head-neck dummy which will respond as will a human head and neck to multivectorial impact acceleration.

The edge of the shoulder harness may impact the carotid sinus and in some individuals may cause syncope (or fainting). This otherwise innocuous period of unconsciousness represents a hazard when only two minutes are available to escape from a helicopter or other vehicle before the fire or from an immersed vehicle which is sinking. Direct impact of the restraint harness upon the neck has also been shown by Snyder et al. [4] to cause atlanto-occipital separation and spinal cord transection at 30 -Gy measured on the vehicle.

Once direct impact occurs, prediction of injury becomes extremely difficult. Prevention or attenuation of direct impact is the only known means of injury control, and there are very few data as to tolerance limits of the neck.

Indirect Energy Transmission

When energy is transmitted from the torso through the neck to the head, causing dynamic response of the head and neck, injuries may occur. Similarly, energy transmitted from the head through the neck to the torso may cause neck injuries. Furthermore, imperfect restraint systems designed to protect the neck from these events may actually cause neck injury.

It is indirect energy transmission through the neck which truly tests the structural characteristics of the neck and therefore permits the development of tolerance limits. These are imperfectly defined at present but are under intensive study.

One important factor necessary to the development of tolerance limits is measurement of input forces to the neck and determination of the resultant injuries. Generally speaking, these data are almost unavailable for living humans. Some studies have been performed on cadavers [5]. Animals have been more extensively studied. An excellent bibliography containing 2,356 references is available [6]. Some human volunteer data with man-mounted instrumentation at the neck have been published but tolerance limits were not approached [7,8,9,10].

One might classify the results of indirect transmission in terms of bony injuries, such as fractures and dislocations; neuropathology; and soft tissue injuries including the so-called whiplash syndrome. Vector direction and peak acceleration of the impact, relative to torso and/or to head, are probably the most important

predictors of injuries. Duration at peak acceleration is also important. A most important determinant of cervical injury is the type and degree of head-neck restraint.

If one may generalize concerning these determinants of tolerance limits of the neck to impact acceleration, it is that each affects the head-neck dynamic response, which in turn causes or intensifies the injury.

One of the goals of protective devices, therefore, is to prevent or attenuate the dynamic response. Dynamic overshoot during dynamic response may be severe. To give an example: during Stapp's [11] pioneering exposure at 38.1 $-G_x$ measured on the sled on Run 135, reversible injury occurred "with production of a pronounced vasomotor reaction," apparent injuries to the retinas of both eyes and other symptoms almost all of which were referable to the head and neck. This exposure has been widely used as a tolerance limit, and merits a more extensive examination.

In the joint Army-Navy-Wayne State University studies [8] on instrumented volunteers, peak resultant head acceleration on subject 013 was determined for increments of sled acceleration during $-G_x$ acceleration. Table 1 gives the results.

TABLE 1

<u>Sled Acceleration (G)</u>	<u>Peak Resultant Mouth Acceleration (G)</u>
3.3	6.1
5.8	19.2
10.1	47.8

It would appear therefore that Stapp's 38 $-G_x$ run might have caused him to suffer a peak resultant head acceleration (measured at the mouth) of as much as 200 G, which may represent the linear acceleration tolerance limit for the unrestrained head and neck with heavy torso and pelvic restraint.

Sonntag et al. [12] made experimental $-G_x$ exposures of chimpanzees with good head-neck restraint which prevented dynamic response of head, neck, and torso relative to each other from occurring. Two animals were tested at sled accelerations of about 150 G. Neither animal was killed by these high accelerations although minor injuries did occur.

Angular acceleration has been implicated as a cause of injury to the central nervous system by Ommaya et al. [13,14]. His experiments on uninstrumented monkeys with head unrestrained, when scaled to man according to Holbourn's formulas, led him to predict that an angular velocity of 30 rad/sec and/or angular acceleration of 1800 rad/sec² would cause concussion in man. This prediction was later increased to 50 rad/sec and 1800 rad/sec² respectively.

Ewing and Thomas et al. [8] have shown, however, that angular velocity directly measured on instrumented living human heads of 32 rad/sec had no subjective or objective effect on their volunteers in $-G_x$ acceleration exposures with unrestrained head and neck, but with restrained torso and pelvis. Previously unpublished data [15] from recent carefully instrumented human volunteer exposures have shown no adverse effects at 38 rad/sec head angular velocity, nor with head angular acceleration of 2,675 rad/sec². Perhaps the reason for the discrepancy may lie in the lack of direct measurement by instrumentation on the primates.

A recent exposure at 160 $-G_x$ sled acceleration of a 20-lb rhesus monkey with heavy torso and pelvic restraint but unrestrained head and neck resulted in fatal injuries, including transection of the medulla oblongata and the vertebral arteries and complete disarticulation of the atlanto-occipital junction [15]. Surprisingly there were no bony fractures, the skin was not disrupted, and the carotid arteries were uninjured. Peak resultant linear acceleration measured directly on the instrumented head exceeded 220 G. Due to a mechanical failure, the true peak could not be determined. This experiment will be replicated soon. Angular acceleration and velocity were not measured.

Other investigators have found almost identical injuries in other primates at comparable or lower sled acceleration exposures [16,17,18,19]. Clarke et al. [16] found several baboon cervical cord transections with avulsion of the atlanto-occipital articulation at as low as 109.9 $-G_x$ sled acceleration with rate of onset of acceleration of 4,227 G/sec. They also found avulsion of the odontoid process at 92.86 $-G_x$ sled acceleration with rate of onset of sled acceleration of 3,439 G/sec, resulting in fatal cervical spinal cord and brainstem trauma without transection.

Lombard et al. [17] makes the observation that $-G_x$ translational acceleration produces translational shear loading which may result in atlanto-occipital shearing with cord transection in guinea pigs.

Snyder et al. [18] reported an atlanto-occipital separation and transection of the spinal cord at 30 $-G_y$ sled acceleration in a baboon restrained by lap belt only.

Snyder et al. [19] further expanded on these experiments in a later paper in which three instances of cervical fractures with complete atlanto-occipital separation occurred in 14 baboons restrained with lap belt only and subjected to 30 $-G_y$ sled acceleration at rates of onset of acceleration from 2,600 to 3,000 G/sec.

Sonntag et al. [12] reported a case of atlanto-occipital avulsion in an anesthetized baboon restrained by lap belt only in an exposure to 40 $-G_x$ sled acceleration.

In a more fundamental study, Fielding and Cochran [20] used fresh cadaveric material in which the second cervical vertebra (C2) and all structures superior to it were removed from the cadaver to study the forces required to cause failure of the atlanto-occipital junction. C2 was then fixed to a rigid block and an instrumented sling was used to apply loading to C1 in a forward direction approximating the $-x$ vector. Force applied to the sling pulled C1 and the occiput anteriorly in a slightly flexed position while C2 remained stationary.

In these experiments, "failure of the transverse ligament" (the primary stabilizing component of the atlanto-occipital structure) "was sudden as the body of the ligament ruptured with a loud 'crack' in the majority of specimens." "The force required to rupture the transverse ligaments had a mean of 84 Kp for all twenty specimens, with a mean of 111 Kp for rapid loading and 72 Kp for slow loading. In no instance did the odontoid fracture prior to ligament failure." "The force required to fracture the odontoid alone was determined to be 70-180 Kp but never was significantly less than the force that had been required to cause complete failure of all the ligaments in the same specimen" [20].

Following rupture of the transverse ligament, force required to produce 12-mm anterior displacement of C1 (sufficient to cause major spinal cord injury) was found to be a mean of 72 Kp with fast-loading mean 85 Kp and slow loading mean 66 Kp.

These measurements were also made by Spence et al. [21] in a somewhat different way, and good comparability was found. It would appear therefore that these data represent true end points which can be used for tolerance limits for the atlanto-occipital junction.

In a different vector direction, Critz et al. [22] showed that exposure of living human volunteers to 14.5 -G_z sled acceleration in a supine subject resulted in 28.6 G peak mouth acceleration with peak displacement of the head relative to shoulder of 1.01 in. This was not accompanied by incapacitating symptoms or injuries.

Stapp [23] reported that an anesthetized chimpanzee similarly situated and restrained in the supine position was subjected to 122.7 -G_z which resulted in a peak acceleration measured on the sternum of 285.5 G with fatal results in part due to right carotid artery linear lacerations. Displacement of the head relative to shoulder straps is not mentioned.

Both Lombard et al. [17] and Stapp and Taylor [24] agree that head-neck motion relative to each other and to the torso are the limiting factors on human tolerance to impact acceleration. Therefore, if they are correct, tolerance limits of the human neck will be dependent upon the vector direction and the degree of head-neck-torso restraint as well as force inputs.

Present Status of Research and Development in Tolerance Limits of the Human Neck

Several excellent general and specific compilations of known tolerance limits are available [25,26,27]. Most data available, however, are related only to sled or vehicular input and tolerance limits are related to specific restraint systems. While most useful in the interim, final tolerance limits must be related to directly measured input forces or accelerations.

The use of vehicular acceleration as a determinant of tolerance limits would appear to have only limited application. True input to the neck for a given vehicular acceleration is quite dependent upon coupling of the man to the vehicle; to the dynamic response permitted

by various restraint systems; to human dynamic response itself; and to vector directions of vehicle acceleration input. Perfect coupling of human to vehicle is possible only when a rigid body, such as the head or portions of the limbs, can be restrained to a rigid seat or restraint system, such as that used by Sonntag, as designed by Muzzy [9].

As stated earlier, it would appear necessary therefore to measure input to the neck directly, which obviates all these problems. Such direct measures of input represent the net result of the interaction of the vehicle input acceleration, the man-vehicle coupling, and the man's dynamic response remote from the neck. Such directly measured input to the neck can then be related to neurophysiological and neuropathological effects which are the output and which in turn determine the tolerance limits.

Such directly measured input-output data cannot be used directly to relate vehicular acceleration to output effects or tolerance limits, since coupling between man and vehicle remains a variable. Another variable is the difference in coupling between vehicle and man for identical restraint systems caused by physical structure of the living man. For example, a large man with a thick fat pad in the buttocks will have a different coupling than a man with a thin fat pad in the buttocks if a belt systems is used, even with identical preloading of the restraint system on each and in the presence of a rigid seat which is used experimentally but rarely used for vehicular design. The same applies to other bony projections used as restraint system load points, such as the clavicles and the pelvic bones. The forcedeflection curves for such tissues overlying the bony skeletal restraint load points remains to be determined for the human race or a representative sample thereof, to reduce this source of error in determining the coupling of man to vehicle.

The use of input-output data concerning human neck tolerance limits, then, is for construction of a mathematical analog of man's response to peak impact acceleration from all vector directions, rates of onset (up to some reasonable point), and duration at peak acceleration, covering a reasonable sample of the anthropomorphic sizes of the human race, along with definition of those portions of the dynamic response envelope which cause detrimental, injurious, and/or lethal physiological effects.

Such a model must be analytical rather than theoretical, must permit one segment of the man's skeleton to be driven with a particular acceleration envelope, and must allow valid prediction of the human response at a distant site, such as the upper or lower end of the neck along with prediction as to the injury potential of that response.

Availability of such a validated analog would also result (as a spinoff) in development of performance criteria for a three-dimensional human dummy which could be used in actual crash testing.

The utility of the validated mathematical and dummy analogs would lie in using a mathematical model of a vehicular response to impact acceleration through all impact vector directions and

magnitudes, with different rates of onset and duration at peak of impact acceleration to drive the mathematical model of human response, using coupling of man to vehicle as a variable. Such use would permit selection of one or two of the most promising coupling or decoupling means cheaply and rapidly in advance of actual expensive hardware design and construction, with actual expensive crash testing used to select the most effective means for achieving the purpose.

Such approaches are not new. Vehicular, restraint system, and human response models have been developed by several organizations and individuals too numerous to be reported here. The innovation is the availability of directly measured validated input data to the neck.

The Navy laboratory at New Orleans has developed and validated the anatomical and transducer mounts to perform this task. A large-scale determined effort using instrumented volunteers is under way at this time, with completion scheduled in the next five years.

The Navy approach is to use instrumented subjects to measure living human head-neck response independent of restraint in response to increments of peak acceleration, rate of onset of acceleration, duration at peak, for numerous different vector directions and subject sizes up to the limit of voluntary human tolerance and then to model the living human response envelope.

Human analogs (subhuman primates) are then identically instrumented and subjected to the same profiles as the man, and the primate response is then modeled.

A comparison model can then be prepared showing the correspondence between man and primate, again up to the limit of voluntary human tolerance.

The primates are then subjected to increasing increments of the noted variables beyond the limit of voluntary human tolerance. Man's response envelope extending from the noninjury levels up to and including injury levels can then be extrapolated using the correspondence model between man and primate, developed as noted above and the analytical model of human head-neck response prepared and validated.

Such a model can also be used with a three-dimensional, digitized vehicular cockpit or workspace in which the mathematical model of man is restrained by a validated mathematical model of a restraint system. In this case, the seat impact forces, magnitudes, and vector directions can be obtained by exercising a mathematical model of the vehicle at different impact velocities, attitudes, and vector directions, and the resulting probable direct impact to the neck throughout an entire envelope of crash conditions can be determined, and personnel hazard identified. Required modifications both to vehicular cockpit or workspace clearance envelope can be determined, along with necessary modifications to restraint systems design to prevent impact within a given clearance envelope.

SUMMARY

Measured input-output data for the neck are required to determine absolute tolerance limits to indirect impact acceleration. Considerable data are available as to vehicular output in crash situations. However, very little directly measured data concerning input to the man's neck are available. Determination of absolute tolerance limits is necessary but probably will not be completed for several years. Tolerance limits of the human neck to direct impact are unavailable.

NOTE

Opinions or conclusions contained in this report are those of the author and do not necessarily reflect the views or the endorsement of the Navy Department.

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