

The Biomechanics of "Whiplash" in Low Velocity Collisions

Richard P.Howard, Richard M. Harding and Scott W. Krenric

Biodynamic Research Corporation

Copyright © 1999 Society of Automotive Engineers, Inc.

ABSTRACT

Recently, it has been demonstrated experimentally that the so-called "whiplash" response during low velocity rear-end collisions may produce a spontaneously resolving strain injury to muscles of the neck, but that injury to other spinal elements is biomechanically improbable. This paper reviews the results of these studies as a means of addressing the longstanding controversy which surrounds "whiplash" and the claims that the "whiplash" response produces more extensive injuries. It is concluded that there are no objective, experimentally-based scientific data to support the concept that the low-velocity "whiplash" response is capable of producing any injuries beyond those to the cervical musculature.

INTRODUCTION

The purpose of this paper is to explain the importance of the findings reported and conclusions reached by current research into the biomechanics and etiology of the so-called "whiplash" disorders associated with low velocity rear-end automobile collisions. The paper describes the extent of the problem before moving to a brief reprise of the relevant anatomy and physiology, presents a consideration of the vehicle dynamics and occupant kinematics associated with low velocity rear-end collisions and of the somewhat conflicting results of research in these fields, and concludes with a discussion of injury causation mechanisms and the part played in this area by clinicians.

The commonly used term describing a rapid extension-flexion motion of the neck is "whiplash". As a matter of convenience, the term will be used in this paper, although in low velocity vehicular collisions the term "whiplash" is misleading in that it implies a mechanical response of the neck which has not been validated experimentally.

Low velocity automobile collisions in some societies result in frequent whiplash injury claims. In Japan, approximately fifty percent of car-to-car collisions result in neck injuries, with the incidence being greatest at low impact speeds [1]. In Canada, the reported annual incidence of compensated whiplash cases varies from 70 per 100,000 inhabitants in provinces with no-fault insurance systems to 700 per 100,000 inhabitants in prov-

inces subject to a civil tort system [2]. In the United States, the incidence of whiplash claims exceeds 10,000 per year [3], while in Lithuania, where these injuries are not compensated by insurance, the diagnosis of whiplash is virtually unknown [4].

"WHIPLASH" -- ANATOMY AND PHYSIOLOGY

The whiplash response following a collision to the rear of a vehicle was initially described as a rapid rearward bending of the neck which occurred as a result of forward acceleration of the torso by the vehicle seat back [5]. The unsupported head was assumed to lag behind the torso in its forward motion, causing the neck to hyperextend. A forward elastic cervical response was then thought to occur, causing the head to move rapidly forward, producing marked flexion of the neck. Various authors have attributed both hyperextension and hyperflexion excursions to the whiplash response, thus taking cervical structures beyond both physiologic and anatomic limits and thereby causing injury. The range of these movements within the whiplash response was postulated, consequently, to produce tensile injuries to muscular and ligamentous tissues and compressive injuries to the cervical zygapophyseal joints. Injuries to the intervertebral disc capsules as a consequence of the whiplash effect have also been claimed [6]. A related, but less frequently cited, injury-producing flexion-extension response has also been postulated to occur in low velocity frontal collisions [7]. In this case, the proposed mechanism of injury is an initial hyperflexion followed by a rebound hyperextension of the neck.

The relevant anatomy of the neck in the context of its response to mechanical stresses has previously been described by Mertz and Patrick [8], among others. In brief, the bony anatomy consists of seven cervical vertebrae. The first vertebra, the atlas, provides direct support to the skull and articulates with the occipital condyles to provide the principal sagittal rotation of the head. The second cervical vertebra, the axis, articulates with the atlas around a pivot-like process, the odontoid, to provide the principal horizontal rotation of the head. The remaining cervical vertebrae are similar to each other in shape and function, and they provide limited rotational freedom. Each vertebra articulates with adjacent vertebrae

through synovial joints held in place by strong fibrous ligaments. Fibrocartilaginous intervertebral discs lie between the end-plates of the vertebral bodies. Longitudinal ligaments adhere to the anterior and posterior surfaces of the vertebral bodies, are continuous along the length of the spine, and their fibers interlace with capsular fibers of the intervertebral discs.

Movement of the head and neck is accomplished by muscle pairs attached to the skull, the vertebral processes, and the rib cage and clavicles. The mass of the posterior vertebral muscles is much greater than that of the pre-vertebral pairs. Moreover, the centroid of the posterior muscle attachments is further displaced from the occipital condyles than that of the pre-vertebral muscles. These anatomic arrangements permit a much greater resistance to forward flexion than to extension of the neck. The sternocleidomastoid muscles can function both as extensors and flexors of the head upon the neck because of their attachments to the mastoid processes. With the head positioned forward, or anteriorly, the vector of the forces generated by the sternocleidomastoids passes superiorly to the occipital condyles to produce a flexing force. When the head is positioned rearward, or posteriorly, the vector passes inferiorly to the condyles to produce an extension force. The cervical musculature acts to limit extension, flexion, and rotation excursions of the neck, thereby protecting the joint structures of the upper vertebral column.

The range of motion of the cervical spine varies from person to person, and females generally have a greater degree of flexibility than males. Total extension-flexion excursion for young subjects (15 to 24 years of age) averages 139 degrees, while for older subjects (55 to 64 years) the average diminishes to 116 degrees [9]. The extension excursion is usually slightly greater than that in flexion.

LOW VELOCITY REAR-END COLLISIONS -- THE IMPORTANCE OF CHANGE IN VELOCITY

Vehicle structural dynamics in low velocity rear-end collisions have been altered over the years by such developments as refined seat design, with the incorporation, in the 1950's, of adjustable head restraints (which upon their introduction were usually inappropriately adjusted to the seated height of the occupant and frequently continue to be so today) and, in the last decade, by the increasing use of integrated seat back/head restraint designs. Bumper design has also evolved from the original spring steel structures to devices made of force managing materials and incorporating energy dissipating mechanisms.

Despite these design improvements, low speed rear-end collisions (defined here as an impact-related change in velocity [ΔV ; ΔV] of $12 \text{ km}\cdot\text{h}^{-1}$ or less) continue to result in claims of whiplash injury, with complaints which may include headache, neck pain, upper extremity radicular symptoms, and temporomandibular joint problems.

These (and indeed any other injuries) can only be produced if the forces acting during the event are sufficient in magnitude, direction, and duration to produce stresses which exceed the tolerance levels of the tissues which are affected. The use of the impact-related ΔV provides a quantitative approach to injury causation potential since the ΔV occurs over a characteristically short period of time and can be associated with an acceleration profile, the forces resulting from which produce the potentially injurious tissue stresses.

Typically, the vehicles involved in such a collision may sustain little or no visible damage, and their occupants exhibit no objective signs of injury. Often, the impact-related ΔV of the struck vehicle cannot be precisely determined, since at these low speeds there is no convenient method by which lack of structural damage can be related to a specific ΔV . Herein lies the dilemma, and the need for scientific study to elucidate the manner in which vehicles and their occupants behave during such events.

VEHICLE DYNAMICS AND OCCUPANT KINEMATICS RESEARCH

Severy et al. [10] were the first to define the biomechanical effects of high speed rear-end collisions, using 1940's vintage motor vehicles and anthropometric test dummies (ATDs). Of particular relevance to this discussion, however, were the several low speed validation tests carried out by these authors using live human volunteer subjects. These tests provided the earliest truly scientific description of whiplash responses to low speed rear-end collisions. Although a wealth of live human test subject acceleration and tolerance data has been obtained by the armed forces since that time, no definitive further testing to measure head/spine accelerations in the low velocity vehicle impact environment was conducted in the United States until 1991, when a series of vehicle-to-vehicle collisions was undertaken by McConnell et al. [11] using instrumented human volunteer test subjects. A similar approach was taken in Canada by West et al. [12], and other groups have subsequently conducted tests and published results which have broadened our understanding of cervical dynamics and the likelihood of injury during such low velocity events [13] [14].

Between the initial research of Severy et al. and the new test research of the 1990's, a multitude of articles has been published in the scientific literature. Much of this interim research related to the dynamics of high velocity rear-end collisions and utilized surrogates for human occupants, including ATDs, cadavers, and anesthetized animal subjects. Data from this research and from the work of Severy et al. formed the experimental basis for mathematical models which predicted dynamic responses and injury likelihood of surrogates in collision environments. Such models were used in part to explain whiplash injury mechanisms, but they were not based on objective biomechanical force measurements [15] [16]. Thus, in retrospect, neither these various forms of surrogate testing nor the proposed mathematical models

based thereon satisfactorily described the responses of live human subjects, particularly in low velocity collisions [17]. Nevertheless, many of these articles have found their way into the medical literature, where associations have been drawn between low energy collisions and a subsequent history of subjective symptoms including chronic neck pain, headache, and cognitive impairment.

The plethora of whiplash-oriented literature produced over the past several decades should therefore be read with caution. Indeed, in 1995, the Quebec Task Force on Whiplash-Associated Disorders described that body of literature as "seriously deficient" [2] (p. 41S). This assessment was made after a review of over 10,000 biomechanical, clinical, and other related publications, of which only 346 survived the scientific scrutiny of the Task Force. With specific regard to the description of occupant kinematics during low velocity rear-end collisions, the Task Force recommended the 1993 study by McConnell et al. [11], involving volunteer occupants exposed to test collisions after giving informed consent to participate.

Research efforts in at least four countries have now demonstrated the kinematic response of human volunteer subjects during low velocity rear-end collisions. These studies have been variously conducted in actual vehicles, test sleds, and amusement park bumper cars. While the aim of these studies has been to define human responses, all have additionally reported on any symptoms experienced by the test subjects participating in this work. Nielsen et al. [18] presented a compilation of symptoms as documented in both published and unpublished research involving a total of 364 test runs. Most subjects reported none. In those who did experience symptoms, mild neck discomfort was the most common complaint, while the longest symptom duration was 7 days. Since the compilation by Nielsen et al., additional investigations have been published. Currently, at least 200 men and women ranging in age from 17 to 63 years have participated in some twenty reported studies involving forty-five vehicle types and well over 500 test runs with velocity change exposures up to $16.6 \text{ km}\cdot\text{h}^{-1}$. With one exception, the duration of reported neck symptoms has been 7 days or less. The single exception was a 47 year-old male who experienced 10 weeks of limitation of neck motion following exposure to a velocity change of $11.4 \text{ km}\cdot\text{h}^{-1}$ [19].

THE LIKELIHOOD OF WHIPLASH INJURY AND ITS MECHANISMS

Interaction with the vehicle interior and with the restraint system generates local forces on the occupant which are best measured in the test environment by measuring accelerations. Acceleration of a vehicle occupant imposes specific force patterns on neck structures which may lead to extension injuries. Cervical extension occurs in a rear-end collision as the torso is moved forward by the accelerating seat back. If the seat back incorporates a structurally competent head restraint which is in contact with the occupant's head at the time of impact, little or no

extension of the neck will occur; indeed, rearward facing subjects in sled tests, with the posterior surfaces of their torso and head fully supported, have undergone accelerations of 40 G and above with no head or neck injuries [20]. If the head is unsupported, however, the degree of neck extension is determined by the differential acceleration of the head with respect to the torso, by the resistive action of the muscles and ligaments of the cervical column, and by the structural strength and elasticity of the seat back. In low velocity collisions, the nature of the articulation of the cervical spine to the head, the action of toned cervical musculature, and the subsequent reflex responses of neck flexion muscle groups all combine to limit extension of the neck and to maintain cervical excursion well within the normal physiologic range of motion for extension. Thus far, this limitation of motion within physiologic limits has been demonstrated in human volunteer tests with ΔV 's up to $10.9 \text{ km}\cdot\text{h}^{-1}$ [21] [22]. It is reasonable to conclude that cervical joint structures (including the vertebral end-plates, intervertebral discs, facet joint articulating surfaces, and ligaments) will not undergo unusual bending stress provided that all cervical column structures remain within their physiologic extension-flexion envelope. The imposed forces which bend the cervical column posteriorly are borne initially and principally by the normal tone of the cervical muscles used to keep the head erect, and subsequently by reflex muscular augmentation preventing extension beyond physiologic limits. These muscular responses, the reflex initiation of which is believed to result from a central response mechanism and to be further recruited by muscle stretch, protect the cervical vertebral column from hyperextension, but in doing so the muscles may themselves undergo strain injury [22]. Results from recent studies by several investigators indicate that mild cervical muscle strain occurs at a ΔV threshold of approximately $8 \text{ km}\cdot\text{h}^{-1}$ [11] [12] [14]. This injury threshold is also the case when the head is positioned forward of any head restraint such that some extension occurs before support of the head by the head restraint is gained. Muscle strain injury under these conditions appears to be produced by forced stretching of cervical muscles under tone, and by rate-related motion rather than by the extent of excursion. Rapid increases in muscle tension imposed dynamically may exceed the tensile tolerance limits of individual muscle fibers without stretching the muscle as a whole beyond its normal relaxed or resting length. The muscles which would be expected to sustain a strain-type injury in this scenario would be those which resist extension; that is, the sternocleidomastoid and strap muscles. At ΔV 's in the 10 to $11 \text{ km}\cdot\text{h}^{-1}$ range, soreness of these muscles has been reported [21].

The frequently-cited symptom of posterior cervical pain remains without a clearly understood basis. It has been suggested that forceful compression of the superior facets upon the inferior facets of the zygapophyseal joints during extension may injure their articulating surfaces and cause these posterior symptoms [23]. Compression of the interspinous ligaments and muscles between the

spinous processes, with a possible shearing component, may also be a pain source [21]; however, this mechanism seems unlikely in the absence of hyperextension. Neither of these possible mechanisms of injury has been supported by conclusive radiological or histological evidence.

It seems reasonable that structures sustaining injury at low levels of applied force would undergo progressively greater injury when subjected to greater forces. It is therefore helpful to compare neck extension injury patterns which occur at higher acceleration levels to controlled experimental low velocity events which produce posterior neck pain. Animal tests in which anesthetized primates were subjected to extremely high +Gx ("eye-balls in") accelerations of 70 G to 150 G did not, however, result in injury patterns which suggest progression from a lesser +Gx acceleration condition where zygapophyseal joint compression injuries or shearing injuries caudal to the C2 level may have occurred [24].

In accidents involving humans, neck extension injuries rarely occur at the +Gx acceleration levels experienced by the animal subjects, since only a very rigid seat back structure is able to support the load required to produce accelerations of this magnitude. Instead, modern seat backs deform elastically at low ΔV 's, while at higher ΔV 's they undergo plastic structural yielding when a rearward force of 2,600 to 3,600 Newtons is imposed at the mid-level of the seat back. This observation implies that the inertial load of a subject of average mass would cause a seat back to yield in the 6 to 10 +Gx acceleration range. Thus, the mechanism of cervical extension injury in high force human accidents is not comparable with experimentally-produced low force extension injuries. Typically, as ΔV increases catastrophic extension injuries are caused by secondary collisions of the occupant with structures within the vehicle, rather than by the seat back-torso interaction which creates the classic whiplash mechanism.

It has also been suggested that the principal whiplash injury in low velocity events occurs as a consequence of neck flexion during the phase of rebound from the seat back [6]. Structurally, this is plausible since neck flexion produces tensile stress in the posterior cervical muscles and ligaments. The likelihood that this is an injury mechanism in the low velocity collision environment is, however, remote. Seat backs typically exhibit coefficients of restitution below 0.3; thus, for a ΔV of 12 km·h⁻¹, the relative velocity at which an occupant would tend to rebound toward the front of the vehicle would be no more than 4 km·h⁻¹. The rearward acceleration necessary to halt the body's forward rebound travel is provided by friction, by reflexive bracing both within the torso and through the extremities, and by the lap and shoulder restraint, if worn. However, the contribution of the restraint system to the limitation of the forward displacement of the occupant may be relatively minor, as forward motion of the occupant with respect to the vehicle is not a significant component of the kinematic response to rear-end collisions.

The peak value of this rearward acceleration has been measured at the occupant's head to be 1.0 to 2.5 -Gx for a ΔV of 10.9 km·h⁻¹. Measurements derived from photographic film records of these events revealed the extent of neck flexion to be slightly forward of the neutral position (that is, the position at which the axis of the thoracic spine is normal to the Frankfort plane), with muscle-controlled extension of the upper portion of the cervical spine coexisting with flexion of the lower cervical spine [21]. Neck flexion during the rebound phase is therefore easily resisted, without injury to posterior cervical muscle groups, during the kinematic response to low velocity rear-end impacts. Moreover, human subject testing has demonstrated tolerance to repeated rearward accelerations of 20 G without injury, a finding which demonstrates the resistive competence of the relatively massive posterior cervical muscles to flexion [25].

Croft has suggested that lateral rotation of the head decreases the extension range of the cervical spine by fifty percent and thereby increases the likelihood of extension injury [26]. While such lateral rotation does diminish extension excursion, there is no objective evidence to suggest that an injury pattern of tensile trauma to the vertebral column on the lateral (leading) convexity, or of compressive trauma on the contralateral (following) side, results from such an orientation of the head with respect to the neck in low velocity rear-end collisions. Moreover, tests performed with the subject's head turned laterally to the left at 30 and 45 degrees produced only transient strain soreness of the right sternocleidomastoid muscle at ΔV 's of 8 km·h⁻¹ [21]. Substantially higher "Gy (side-to-side) accelerations similarly failed to produce these injuries in military test subjects [27]. Thus, this postulated mechanism of enhanced injury potential appears to be unsubstantiated for low velocity whiplash injuries other than possibly to the cervical musculature on the leading side of the lateral rotation.

As mentioned above, conclusions drawn from various mathematical models published in the 1980's and early 1990's influenced opinion with regard to whiplash dynamics. While this approach may have represented an improvement over the manner in which the issue had been previously addressed, through totally unsubstantiated opinions and assertions such as those of Gay and Abbott [28], the misconceptions which arose from the more recent models were significant. The models produced grossly inaccurate results for several reasons. For example, based on the quite limited data of Severy et al. [10], the models incorporated an assumed amplification ratio of head acceleration to vehicle acceleration of 2.5:1.0. Unfortunately, the method of measurement of head acceleration utilized by Severy et al. was not described fully in their publication; however, it is clear from their data that head accelerations were not resolved to the center of mass of the head. Consequently, the linear acceleration data from the two live human test subjects in their study contained an unknown tangential component resulting from head rotation. Additionally, joint rotation factors and damping parameters used in the

models were assumed and were not validated by human subject testing. Because of the uncertainties and/or inaccuracies in these inputs, results from the mathematical models bear little resemblance to live human responses measured in later experimental research. As an illustration, the two-dimensional mathematical model of the head, neck, and torso by McKenzie and Williams [16] responded to an input horizontal acceleration of 5 G at the pelvis by computing a head acceleration of 20 G to 28 G, depending upon the seat back stiffness selected. With a 12 G horizontal input at the pelvis, the resultant horizontal head acceleration was 60 G, and the angular acceleration was $4,500 \text{ rad}\cdot\text{sec}^{-2}$. These values are grossly out of agreement with expectations based upon experimental data. While these authors recognized that their model did not account for neuromuscular response and consequently required improvement [29], others have recently accepted these spurious results and so have perpetuated the misconceptions [26].

The extant description of the human kinematic response to a low velocity rear-end collision remains that provided by McConnell et al. in 1993, as refined by the same group in 1995 and summarized here [21]. The authors divided the occupant response into five phases, with time zero signifying the instant of vehicle contact and thereafter followed by an initial response phase (0 - 100 msec); a principal forward acceleration phase (100 - 200 msec); a head overspeed/torso recovery phase (200 - 300 msec); a head deceleration/torso rest phase (300 - 400 msec); and a restitution phase (400 - 600 msec). In these tests, the ΔV 's of the struck vehicles ranged from 5.8 to 10.9 $\text{km}\cdot\text{h}^{-1}$, with the majority between 8 and 10 $\text{km}\cdot\text{h}^{-1}$. Peak accelerations of the head center of mass (CM) ranged from 4 G to 6 G, while local accelerations of points in the sagittal plane of the head were higher and varied from 5 G to 15 G as a consequence of a tangential contribution from the angular acceleration of the head. Based on these results, the ratio of head acceleration to vehicle (pelvis) acceleration of 2.5:1.0 as reported by Severy et al. would appear to have been a consequence of their experimental subjects' having worn an accelerometer in a location which corresponded to a point which experienced a combined local acceleration (linear plus tangential angular components) of 15 G. As noted above, the head acceleration data in the study of Severy et al. were not resolved to the head CM. Recent studies indicate, however, that the actual ratio between head CM acceleration and vehicle (pelvis) acceleration may more nearly approach 1.0:1.0 for low velocity impacts (vehicle acceleration was 4 G to 6 G for ΔV 's of 8 to 10 $\text{km}\cdot\text{h}^{-1}$) [21] [22]. This ratio reflects the significant damping effect of head-stabilizing muscles, an effect which offsets the magnification of the ratio of head acceleration to vehicle acceleration produced by coupling slack between the occupant and the vehicle in the low velocity environment. Although a very high local acceleration to the head may produce brain injury, 15 G is far below any accepted brain injury threshold [25]. Furthermore, if 15 G were indeed the measured acceleration of the head CM, then some of the

dire predictions of earlier mathematical models might be more nearly correct. The error resulting from the use of localized accelerations of points on the head to calculate neck loads is thus seen to be significant. Similarly, a general calculation of peak moment and shear forces in the entire vertebral column from these data is not meaningful. Thus, in addition to measured accelerations of the head and torso/pelvis/vehicle, other variables such as seat height and stiffness, occupant seated height and posture, head restraint position, and neuromuscular response must be taken into account. Clearly, if peak head acceleration occurs during head impact with the head restraint, this value cannot be used in the calculation of cervical moment forces. As another complication, the head does not act with respect to the torso as a solid mass connected by a mass-less segmented two-dimensional column with seven joints of known resistance. The CM appropriate to the calculation of neck forces is found from the sum of the combined masses of the head, neck, and mandible. The position of this effective CM changes as the geometric relationships between these structures change with respect to time throughout the extension-flexion response, and its location is further influenced by variable muscle input over time.

Ono et al. [30] described a possible mechanism of injury to the zygapophyseal joints of the lower cervical spine. They hypothesized that full rotation of the lower vertebral segments produced by the combined effects of compression and bending preceded similar rotation of more superior segments. They deemed the resultant motion narrowing the spaces between opposing facet articulating surfaces to be non-physiologic and hence capable, at some undetermined vehicle velocity change greater than those which they observed experimentally, of producing injury to these structures, or to the anterior longitudinal ligament. The phenomenon which Ono et al. describe was not demonstrated in their volunteer test subjects at velocity changes of 8 $\text{km}\cdot\text{h}^{-1}$, nor was its basis clearly established. Moreover, one of the seats used in their tests was rigid, thus resulting in greater cervical compressive forces and head rotation than would be expected with the use of a standard automobile seat. The relationship of this described joint motion to injury causation therefore remains hypothetical.

Until the variables which contribute to neck dynamics can be accounted for more precisely, it is appropriate to rely upon data obtained from human subject testing to predict occupant responses to low velocity collisions and, by extrapolation, to predict human responses in the low/medium velocity range. Further research, which increases the aggregate numbers of well-characterized experimental exposures involving human subjects to levels of statistical significance, and which broadens the diversity of the experimentally studied population with respect to anthropometry, age, and gender, will further enhance the predictive value of human subject data. Such research will also provide additional means for assessing and improving the biofidelity of surrogates employed in testing at levels of velocity changes equal to

and exceeding those to which human volunteers can be ethically subjected.

One of the principal reasons for the differences between occupant responses to low velocity rear-end collisions as predicted by ATD and cadaveric data and descriptions of responses based on live human testing is the influence of dynamic neuromuscular mechanisms. In the study by McConnell et al. [21], high speed film records revealed that there was no discernable torso motion until at least 50 to 60 msec after impact, no significant movement of the head with respect to T1 until 80 msec after impact, and bulging of the sternocleidomastoid muscles at 110 to 120 msec after impact. Any sternocleidomastoid muscle stretch response must therefore have occurred within approximately 30 msec of the initiation of the relative motion between the torso and the head. Experiments conducted by Szabo and Welcher recorded impact-related accelerations at the head and lumbar spine of test subjects in rear-end collisions producing ΔV 's of 7.5 to 10.0 km·h⁻¹ [22]. Surface electromyographic recordings were taken simultaneously at selected cervical and lumbar muscle group sites, and measures were taken (and compliance confirmed by electromyographic activity levels) to ensure that the test subjects were not braced at the moment of impact. Measurable lumbar spine accelerations began approximately 20 msec after vehicle impact. A centrally-generated reflex-like muscle response of both the cervical and lumbar regions occurred at 90 to 120 msec after apparent triggering of a mechanism which may be related to the lumbar acceleration. It was felt unlikely that the initial recorded cervical muscle response was directly related to the early phase of head motion since head acceleration began only 20 to 30 msec prior to the cervical muscle response. It was concluded that the initial cervical muscle response, affecting spinal joint rotation, occurred independently of anticipation of the impact, and probably occurred as a consequence of a centrally-generated mechanism triggered by the initial lumbar spine acceleration. While it is likely that such a centrally-generated response mechanism exists, the specific initiating event for this response is not yet known. It may be any of several stimuli including the suggested lumbar spine acceleration, or it may involve more generalized proprioceptive or even auditory stimuli. Topics worthy of continuing investigation are the characterization and differentiation of the contributions of both reflexive and voluntary muscular activity to the occupant kinematic response of the alert human subject. Electromyographic evaluations obtained during human volunteer experiments represent a valuable investigative tool in this regard.

Such recent experiments indicate clearly that significant stabilization of the head occurs very early in the occupant response to these events, and that muscle stabilization continues to occur throughout the extension-flexion cycle. McConnell et al. [11] [21] used mid-1980's vintage vehicles with adjustable head restraints which, for the medium and tall subjects, did not prevent neck extension. The posterior occipital portion of the head rose over the

head restraint and pushed it into the fully "down" position. Despite this fact, all test subjects experienced neck extension well within their measured voluntary range of motion, and none came within 10 degrees of this limit. These results indicate that neck extension was controlled and limited by neuromuscular activity, and by head and neck anatomical geometry. Other articulating structures behaved passively since bones, ligaments, and intervertebral discs are unable to generate an active resisting response to motion until the joint approaches its voluntary motion limit. According to current biomechanical thinking, the neck may be regarded as an articulating, axial weight-bearing column, surrounded by a muscular trunk which, in all but extreme conditions, protects the vertebral column against both anticipated and unanticipated forces; it is therefore not surprising that the muscular support is occasionally strained.

In the study by McConnell et al. [21], all seven test subjects experienced varying degrees of transient cervical muscle soreness, and the two subjects who underwent a ΔV of 10.9 km·h⁻¹ both experienced transient pre-vertebral soreness. But all symptoms resolved within 3 days, including those described by subjects exposed to multiple impacts. The tests by West et al. [12] and by Szabo et al. [14] produced less prominent but similar muscle strain injury symptoms in subjects exposed to single impacts. It has been concluded from these experimental studies that some occupants in vehicles involved in rear-end collisions may reach a cervical muscle strain injury threshold at a ΔV of about 8 km·h⁻¹ without head support, while occupants undergoing ΔV 's in the 10 to 11 km·h⁻¹ range may reasonably be expected to experience mild strain of the pre-vertebral muscles with greater frequency. Further, the less distensible structural elements of the vertebral column (that is, the bony vertebrae, the intervertebral discs, and the ligaments) will not ordinarily undergo significant stress during the extension-flexion response to a low velocity rear-end impact. Injury to these structures would therefore not be expected unless these tissues are compromised by pre-existing conditions such as a healing fracture or recent severe ligamentous disruption. Comparison of similar acceleration loadings in other scenarios involving activities of daily living, which are routinely withstood without injury, support these general conclusions [31], although caution must be exercised when using this approach since the direction, duration, and magnitude of any applied force must be directly comparable. Such a comparison, between rear-end automobile collisions and amusement park bumper car rides, was made by Siegmund and Williamson [13].

SOME CLINICAL THOUGHTS

Misconceptions by many medical providers with regard to the injury causation potential imposed by low velocity rear-end collisions often place the patient in a symptomatically driven clinical treatment course dictated by subjective complaints rather than by objective findings and derived diagnoses. The resulting financial burden on the

health care system is ultimately seen by the public in higher insurance premiums and lost work time. But even objectivity can be distorted. For example, decreased lordosis is frequently reported as a finding on cervical spine imaging and is interpreted to indicate the presence of cervical muscle spasm. It has been shown, however, that straightening of the spine is such a sufficiently common variant of normal that the conclusion that it is due to muscle spasm cannot be supported [32]. Since the whiplash mechanism was first described, diagnostic procedures have of course improved greatly, and radiographic techniques are now capable of diagnostic precision inconceivable several decades ago.

Although this paper is not intended to address the diagnosis, prognosis, or treatment of whiplash in detail, a brief summary of the relevant clinical conclusions of the Quebec Task Force on Whiplash-Associated Disorders [2] is considered an essential component of this discussion. The whiplash-associated disorders addressed by the Task Force were classified into five clinical grades, designated 0 (no complaints and no physical signs) to IV (neck complaint and fracture or dislocation). Of these categories, only Grade I (neck complaint but no physical signs) and Grade II (neck complaint and musculoskeletal signs) presentations are of direct interest here, since the vast majority of patients involved in low velocity rear-end collisions will fall into these grades. With regard to treatment of a Grade I condition, the Task Force recommended an immediate return to normal activities following evaluation. Non-steroidal anti-inflammatory and non-narcotic analgesic medications may be offered to those in Grade II, with a return to normal activities within one week if possible. For those in Grade III (neck complaint and neurological signs), non-steroidal anti-inflammatory and non-narcotic analgesic medications are again recommended, with re-assessment in three weeks if return to normal activities has not occurred. Prescribed rest is seldom indicated (and then only for a period of short duration), and the use of a soft cervical collar is not recommended and is uniformly discouraged. And if the patient has not returned to usual activities six weeks (for Grade I) to twelve weeks (for Grades II and III) after the injury, a multi-disciplinary diagnostic and therapeutic approach is recommended.

CONCLUSION

Independent investigators have, on numerous occasions in the past few years, demonstrated experimentally that, at low velocities, the so-called whiplash response produces, at most, a muscle strain injury, and does not cause injury to the other elements of the vertebral column. But Bogduk has stated, "It is unbelievable that were there not a common organic syndrome, there exists among patients a deliberate, international, translanguagel conspiracy that enables them all to consistently report the same symptoms." [23] (p. 99). Compelling as this argument may seem, similar statements have held sway with regard to other medical conditions only to be modi-

fied or proven false later by objective scientific analysis. As a common unifying phenomenon, verified by sound and modern scientific evidence, the extension-flexion response associated with low velocity rear-end collisions can result in transient, self-limited strain and discomfort of the upper paravertebral musculature. There are, however, no scientific data which support the existence of further, i.e. non-muscular, injury under experimental conditions producing similar cervical forces to those seen in low velocity vehicle collisions. Moreover, no other injury-causation link has been established which has withstood scientific scrutiny. As mentioned previously, future efforts to expand the database of human subject observations and to refine the understanding of the contributions of muscular activity to occupant kinematic responses are still needed. Additional goals of research in these areas should also include the acquisition of data to improve the biofidelity of surrogates and the validity of mathematical models. Yet another objective for ongoing human volunteer experimentation should be to determine the extent to which the recently-proposed neck injury criterion (NIC) advanced by Boström et al. [33] can be applied to human subjects. If such future endeavors can establish a link between the human occupant kinematic response to low velocity rear-end collisions and other injury mechanisms, modification of these conclusions will be made accordingly.

REFERENCE

1. Ono, K. and Kanno, M. (1993). Influences of the physical parameters on the risk to neck injuries in low impact speed rear-end collisions. In: Proceedings, 1993 International IRCOBI Conference On the Biomechanics of Impacts, September 8-9-10, 1993, Eindhoven (The Netherlands). Bron (France): International Research Council On Biokinetics of Impacts (IRCOBI) Secretariat.
2. Spitzer, W.O., Skovron, M.L., Salmi, L.R., Cassidy, J.D., Duranceau, J., Suissa, S., et al. (1995). Scientific monograph of the Quebec Task Force on whiplash-associated disorders: redefining "whiplash" and its management. *Spine*, 20:1S-73S
3. Evans, R.W. (1992). Some observations on whiplash injuries. *Neurol. Clin.*, 10:975-997.
4. Shrader, H., Obelieniene, D., Bovim, G., Surkiene, D., Mickeviciene, D., Miseviciene, I., et al. (1996). Natural evolution of late whiplash syndrome outside the medicolegal context. *Lancet*, 347:1207-1211.
5. Crowe, H. (1964). A new diagnostic sign in neck injuries. *Calif. Med.* 100:12-13.
6. Clemens, H.J. and Burow, K. (1972). Experimental investigation on injury mechanisms of cervical spine at frontal and rear-front vehicle impacts. In: Proceedings of Sixteenth Stapp Car Crash Conference. Sixteenth Stapp Car Crash Conference, November 8-10, 1972, Detroit, MI. New York: Society of Automotive Engineers, Inc.
7. Davis, A.G. (1945). Injuries of the cervical spine. *J.A.M.A.*, 127:149-156.
8. Mertz, H.J. and Patrick, L.M. (1971). Strength and response of the human neck. In: Proceedings of Fifteenth Stapp Car Crash Conference. Fifteenth Stapp Car Crash Conference, November 17-19, 1971, Coronado, CA. New York: Society of Automotive Engineers, Inc.
9. Ferlic, D. (1962). The range of motion of the "normal" cervical spine. *Bull. of Johns Hopkins Hosp.*, 110:59-65.

10. Severy, D.M., Mathewson, J.H., and Bechtol, C.O. (1955). Controlled automobile rear-end collisions, an investigation of related engineering and medical phenomena. *Can. Serv. Med. J.*, 11:727-759.
11. McConnell, W.E., Howard, R.P., Guzman, H.M., Bomar, J.B., Raddin, J.H., Benedict, J.V., et al. (1993). Analysis of human test subject kinematic responses to low velocity rear end impacts. In: *Vehicle and Occupant Kinematics: Simulation and Modeling*. International Congress and Exposition, March 1-5, 1993, Detroit, MI. Warrendale, PA: Society of Automotive Engineers, Inc.
12. West, D.H., Gough, J.P., and Harper, G.T.K. (1993). Low speed rear-end collision testing using human subjects. *Accid. Reconstr. J*, 5 (3):22-26.
13. Siegmund, G.P. and Williamson, P.B. (1993). Speed change (ΔV) of amusement park bumper cars. In: *Proceedings of the Canadian Multidisciplinary Road Safety Conference VIII*. Canadian Multidisciplinary Road Safety Conference VIII, June 14-16, 1993, Saskatoon, Saskatchewan.
14. Szabo, T.J., Welcher, J.B., Anderson, R.D., Rice, M.M., Ward, J.A., Paulo, L.R., et al. (1994). Human occupant kinematic response to low speed rear-end impacts. In: *Occupant Containment and Methods of Assessing Occupant Protection in the Crash Environment*. International Congress and Exposition, February 28-March 3, 1994, Detroit, MI. Warrendale, PA: Society of Automotive Engineers, Inc.
15. Martinez, J.L. and Garcia, D.J. (1968) A model for whiplash. *J. Biomechanics*, 1:23-32.
16. McKenzie, J.A. and Williams, J.F. (1971). The dynamic behaviour of the head and cervical spine during 'whiplash.' *J. Biomechanics*, 4:477-490.
17. Scott, M.W., McConnell, W.E., Guzman, H.M., Howard, R.P., Bomar, J.B., Smith, H.L., et al. (1993). Comparison of human and ATD head kinematics during low-speed rear-end impacts. In: *Human Surrogates: Design, Development, and Side Impact Protection*. International Congress and Exposition, March 1-5, 1993, Detroit, MI. Warrendale, PA: Society of Automotive Engineers, Inc.
18. Nielsen, G.P., Gough, J.P., Little, D.M., West, D.H., and Baker, V.T. (1996). Low speed rear impact test summary - human test subjects. *Accid. Reconstr. J*, 8 (5): 40-43.
19. Castro, W.H.M., Schilgen, M., Meyer, S., Weber, M., Peucker, C., and Wörtler, K. (1997). Do "whiplash injuries" occur in low-speed rear impacts? *Eur. Spine J.*, 6:366-375.
20. Snyder, R.G. (1972). Chapter 6. Impact. In: *Bioastronautics Data Book*. Edited by J. F. Parker, Jr., and V. R. West. Falls Church, VA: BioTechnology, Inc.
21. McConnell, W.E., Howard, R.P., Van Poppel, J., Krause, R., Guzman, H.M., Bomar, J.B., et al. (1995). Human head and neck kinematics after low velocity rear-end impacts - understanding "whiplash." In: *39th Stapp Car Crash Conference Proceedings*. 39th Stapp Car Crash Conference, November 8-10, 1995, San Diego, CA. Warrendale, PA: Society of Automotive Engineers, Inc.
22. Szabo, T.J. and Welcher, J.B. (1996). Human subject kinematics and electromyographic activity during low speed rear impacts. In: *Proceedings of the 40th Stapp Car Crash Conference*. 40th Stapp Car Crash Conference, November 4-6, 1996, Albuquerque, NM. Warrendale, PA: Society of Automotive Engineers, Inc.
23. Bogduk, N. (1986). The anatomy and pathophysiology of whiplash. *Clin. Biomechanics*, 1:92-101.
24. Freeman, L.W. and Wright, T.W. (1953). Experimental observations of concussion and contusion of the spinal cord. *Ann. Surg.*, 137:433-443.
25. Stapp, J.P. (1970). Chapter XV. Voluntary human tolerance levels. In: *Impact Injury and Crash Protection*. Edited by E. S. Gurdjian, W. A. Lange, L. M. Patrick, and L. M. Thomas. Springfield, IL: Charles C. Thomas.
26. Croft, A.C. (1988). Chapter 1. Biomechanics. In: *Whiplash Injuries*. Edited by J. W. Pine, Jr. Baltimore: Williams & Wilkins.
27. Weis, E.B., Clarke, N.P., and Brinkley, J.W. (1963). Human response to several impact acceleration orientations and patterns. *Aerospace Medicine*, 34:1122-1129.
28. Gay, J.R. and Abbott, K.H. (1953). Common whiplash injuries of the neck. *J.A.M.A.*, 152:1698-1704.
29. Williams, J.F. and McKenzie, J.A. (1975). The effect of collision severity on the motion of the head and neck during 'whiplash.' *J. Biomechanics*, 8:257-259.
30. Ono, K., Kaneoka, K., Wittek, A., and Kajzer, J. (1997). Cervical injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low speed rear impacts. In: *Proceedings of the 41st Stapp Car Crash Conference*. 41st Stapp Car Crash Conference, November 13-14, 1997, Lake Buena Vista, FL. Warrendale, PA: Society of Automotive Engineers, Inc.
31. Allen, M.E., Weir-Jones, I., Motiuk, D.R., Flewin, K.R., Goring, R.D., Kobetitch, R., et al. (1994). Acceleration perturbations of daily living. A comparison to 'whiplash.' *Spine*, 19:1285-1290.
32. Helliwell, P.S., Evans, P.F., and Wright, V. (1994). The straight cervical spine: does it indicate muscle spasm? *J. Bone and Joint Surg.*, 76-B:103-106.
33. Boström, O., Svensson, M.Y., Aldman, B., Hansson, H.A., Håland, Y., Lövsund, P., et al. (1996). A new neck injury criterion candidate - based on injury findings in the cervical spinal ganglia after experimental neck extension trauma. In: *Proceedings, 1996 International IRCOBI Conference On the Biomechanics of Impact*, September 11-13, 1996, Dublin (Ireland). Paris (France): International Research Council On the Biomechanics of Impact (IRCOBI) Secretariat.