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Clinical Biomechanics 16 (2001) 267–275

CLINICAL BIOMECHANICS

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Review paper

Biomechanics of the cervical spine Part 3: minor injuries

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Received 15 November 2000; accepted 16 January 2001

Abstract

Minor injuries of the cervical spine are essentially defined as injuries that do not involve a fracture. Archetypical of minor cervical injury is the whiplash injury. Among other reasons, neck pain after whiplash has been controversial because critics do not credit that an injury to the neck can occur in a whiplash accident. In pursuit of the injury mechanism, bioengineers have used mathematical modelling, cadaver studies, and human volunteers to study the kinematics of the neck under the conditions of whiplash. Particularly illuminating have been cinephotographic and cineradiographic studies of cadavers and of normal volunteers. They demonstrate that externally, the head and neck do not exceed normal physiological limits. However, the cervical spine undergoes a sigmoid deformation very early after impact. During this deformation, lower cervical segments undergo posterior rotation around an abnormally high axis of rotation, resulting in abnormal separation of the anterior elements of the cervical spine, and impaction of the zygapophysial joints. The demonstration of a mechanism for injury of the zygapophysial joints complements postmortem studies that reveal lesions in these joints, and clinical studies that have demonstrated that zygapophysial joint pain is the single most common basis for chronic neck pain after injury.

1. Introduction

There is no universally accepted definition of what distinguishes major from minor injuries to the cervical spine. Extreme examples can establish the limits of a spectrum, such that a fracture-dislocation with spinal cord injury is obviously a major injury, whereas a sprained muscle is a minor injury. The difficulty that arises lies in establishing the boundary between major and minor injuries.

Effectively, and in clinical practice, the distinction lies in whether or not there has been a fracture or a dislocation. Fractures that constitute major injuries are fractures of the vertebral body, the pedicles, the odontoid process, and the ring of the atlas. Retrospectively, such fractures imply that a major external force was applied to the spine, as do dislocations. Prospectively, fractures and dislocations threaten the stability of the cervical spine and the integrity of its neural contents. These features render the injury serious in both a biomechanical and a clinical sense. Some fractures, however, can be minor, such as those in an articular process or across the anterior edge of a vertebral body. Because they do not threaten the stability of the cervical spine they constitute the "grey zone" between major and minor injuries.

Against this background, minor injuries of the cervical spine are essentially those in which fractures do not occur, or in which fractures are not readily apparent. By default, these injuries are usually classified as "soft-tissue injuries", a term that implies that there has been no injury of bone and that if anything has been injured it must be one or more of the muscles or ligaments of the neck. The definition is no more specific than this because it is based essentially on the plain radiographic appearance of the neck. Since X-rays cannot demonstrate soft tissues, the definition is one of exclusion, i.e., if there is an injury but X-rays exclude a fracture or dislocation, the injury must be in the (invisible) soft tissue injuries of the neck.

Computerised tomography (CT) and magnetic resonance imaging (MRI) do not help in this regard. In the first instance, they are not routinely used to screen for fractures; plain radiographs remain the cardinal tool for that purpose. CT may be used to better define fractures already evident on plain films, or to search for occult fractures, but it does not resolve soft-tissue injuries. MRI has the capacity to resolve certain soft-tissues, but

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0268-0033/01/$ - see front matter
PII: S0268-0033(01)00003-1

no correlations have been established between neck pain and any feature evident on MRI.

Several factors render soft-tissue injuries of the neck controversial. In the first instance, their very invisibility (under X-rays) is grounds for suspicion, e.g., if the injury cannot be demonstrated, perhaps there is no injury. Secondly, on the basis of extrapolation from soft-tissue injuries in the limbs, it is believed that soft-tissue injuries should heal rapidly, in a matter of days or weeks. Therefore, persistence of neck pain is incongruous with an archetypical soft-tissue injury and, in some circles, this is used as evidence that there was no injury and that the symptoms are manufactured or imagined. Compounding these clinical considerations is the influence of litigation. Soft-tissue injuries are often, if not most commonly, associated with insurance claims. The prospect of compensation and monetary gain confounds the clinical considerations. It seems to increase the likelihood that symptoms are manufactured or artificially prolonged in order to achieve gain.

Perhaps the best-known and most studied example of soft-tissue injury to the neck is whiplash. If nothing else it serves as the archetype for this type of injury. Fractures are typically not evident, yet the patient complains of symptoms, and a proportion of patients develop chronic symptoms that last well beyond the expected period in which soft-tissue injuries should have healed. Moreover, these injuries are subject to litigation. Consequently, whiplash injuries inherit all the suspicions that render soft-tissue injuries controversial. However, the more that whiplash has been studied, the more has scientific inquiry dispelled incorrect notions that caused this controversy.

Invisibility is not evidence of absence. Rather, it is an indication that an inappropriate tool has been used to look for the injury. The literature is replete with studies that have shown small injuries to intervertebral discs, zygapophysial joints, and uncovertebral clefts, both in collagenous tissues and in cartilage and bones, that are plainly invisible on plain radiographs [1–5]. Radiography simply lacks the sensitivity to detect these injuries, and therefore, cannot be used to exclude or refute them. Normal radiographs do not mean that there has been no injury.

Extrapolation from the limbs about the nature of soft-tissue injuries and their period of healing is both inappropriate and false. One can expect a sprained muscle to heal and become asymptomatic within a few days. Experimental studies in animals indicate that lesions at the myotendinous junction repair within one week, and develop nearly normal tension by seven days after injury [6]. Recovery from contraction-induced injuries of muscles is usually complete within 30 days [7]. But the behaviour of muscle injuries is not a model for the behaviour of ligaments, capsules, joints, and intervertebral discs. Most sprained ankles recover within two weeks [8,9], but some do not, and many patients are left with chronic symptoms [10] (even though compensation is not involved). Football injuries to knees, involving the menisci, do not all resolve; some can cause chronic pain and disability until surgery is undertaken to rectify the lesion. Small articular fractures, such as a Bennett's fracture of the thumb, can cause chronic disability. Intervertebral discs, like the menisci of the knee, are unlikely to heal spontaneously after injury, probably because of their relatively meagre blood supply. In essence, clinical experience abounds with examples of soft-tissue injuries to the limbs that do not summarily heal. Correctly used, therefore, extrapolation would predict that at least some injuries of the cervical spine would not heal. Whereas muscle sprains should resolve rapidly, some injuries to joints and discs may remain sources of chronic pain.

It is commonly believed that patients with neck pain exaggerate or perpetuate their symptoms for the purpose of financial gain [11–14], but formal studies and reviews refute this conjecture [15–20]. Although, in some instances, fraud does occur because of the monetary compensation that is available, the majority of patients behave as if they do have an injury, not as if they are malingering. Their symptoms persist despite and regardless of settlement of compensation claims [17–19].

At the root of all this controversy is the nature of the injury and how it might arise. Some physicians have difficulty crediting that a seemingly minor incident can inflict an injury that is not detectable radiologically, and which produces lasting and disabling symptoms. Although how and why symptoms persist is another matter, biomechanics research over the last 40 years has at least provided insights into how and where the injuries occur.

2. Modes of investigation

Investigators have used a variety of means in an effort to determine the biomechanics of injuries that might occur in whiplash. They have used mathematical models, finite-element models, physical models, animal models, and experiments using human cadavers.

Mathematical modelling involves programming into a computer, facsimiles of the cervical vertebra and equations that represent the forces exerted on these vertebrae by their ligaments, discs, and muscles. The objectives of mathematical modelling are to depict faithfully the normal kinematics of the cervical spine and to predict and display its behaviour when subjected to abnormal loads, thereby obviating the need for experiments on animal models, cadavers, or human volunteers. Critical to the utility of mathematical models, however, is their biofidelity, i.e., how accurately they depict normal anatomy and physiology. Substantial

progress has been made in the developments of mathematical models of the cervical spine [21–25]. With each generation more anatomical details have been incorporated and with greater accuracy. However, while adequately reproducing the gross kinematics of the head and neck, no model has, as yet, achieved satisfactory reproduction of the segmental movements of the neck under normal or pathological conditions. Until models are perfected and validated they are not substitutes for more traditional forms of experimental studies.

Finite-element modelling is mathematical method to reconstruct the bones, joints, ligaments, discs, and muscles of the cervical spine in terms of one-, two-, and three-dimensional geometric units (elements) like building blocks [26–29]. A ligament might be represented by a line or a rectangle. A vertebra may be represented as a collection of triangular or rectangular prisms. On a computer, each element is programmed to behave in accordance with the biological properties (stiffness, strength) of the tissue that it represents. The model can then be used to depict and study the internal stresses sustained and the response of the tissues and structures when they are subjected to external loads. Modern computer programs can generate dramatic and visually appealing demonstrations on how the spine behaves under load. However, as with mathematical modelling the utility of finite-element models depend on their biofidelity. To date, attractive models have been developed that depict the behaviour of vertebrae under quasi-static loads [29], but the modelling of kinematics is still imperfect [24,26,27] and awaits better representation of the cervical musculature [27].

Physical models involve constructing a facsimile of the human neck, typically in the form of manikins. Such manikins as have been used in the past may have been adequate for studying head injuries, but for the cervical spine they lack biofidelity. Aluminium cylinders connected by rubber discs are poor representations of the cervical vertebrae and their joints, ligaments, and muscles, and do not faithfully represent their behaviours.

Animal models have the advantage of being biologically accurate; but scaling factors limit their applicability. Differences in size and anatomy cannot be adjusted for in any simple mathematical way. Nevertheless, animal experiments do serve, and have served, a useful purpose in demonstrating how necks behave when subjected to impacts, and the nature of injuries that can result. They serve to indicate what can be expected if human material is used.

Human cadaver studies address the problems of biofidelity. The size and structure of the components of the neck are human, and the masses are correct. The limitations that apply concern mainly the state of the muscles in cadavers. Cadaveric muscles impose an artificial stiffness to the neck, for they are not relaxed. In addition, their inability to contract eliminates a possibly protective effect. However, cadavers with muscles removed provide an experimental model that provides valuable, prima facie, evidence of how the cervical spine behaves.

Perhaps the most daring method of studying whiplash is the use of human volunteers. In this method, there are no problems with biofidelity. Only two limitations apply. One is apprehension, in that it can be argued that volunteers expecting an insult might brace themselves to a greater extent than victims of natural motor vehicle collisions. The other is ethics. Human volunteers can be subjected to only minimal impacts, for fear of seriously injuring them. Both these limitations, however, bias experiments towards conservative findings. A braced neck subjected to low impact is unlikely to be injured to the same extent as a relaxed neck. Movements occurring in response to low speed impacts must occur with greater force under high-speed impacts. What occurs in human volunteer studies, therefore, is the very least that might occur in natural injuries.

3. Historical perspective

A seminal study of whiplash mechanics was published by Severy et al. in 1955 [30]. They used human volunteers in two rear-impact tests at 13 and 15 kph, respectively. Accelerometers applied to the heads of the volunteers in the target vehicles recorded peak accelerations of 5 and 3 g, respectively. Most critically, these experiments demonstrated the phasing of acceleration of different components of the target vehicle and the volunteer (Fig. 1). Peak acceleration of the vehicle preceded that of the volunteer's torso, which preceded that of the head. As the torso accelerated the head remained relatively stationary, and its acceleration peaked some 250 ms after impact. This established that whiplash involved

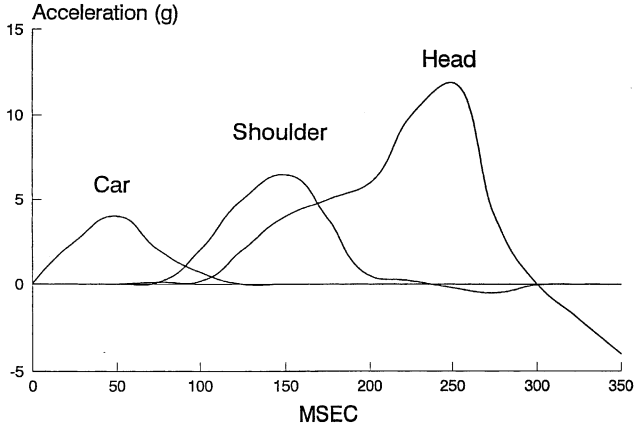

Fig. 1. The magnitude and timing of acceleration of the target vehicle and the shoulders and head of the subject following a rear-end impact. Based on Severy et al. [30]. Positive accelerations are in the forward direction.

inertial loading of the neck, as the torso moved under an initially stationary head.

In subsequent years, whiplash research concentrated on animal experiments. These did not address the biomechanics of injury, but focussed on the nature of lesions that could be produced [31–38]. Their results foreshadowed what would subsequently be found in experiments using human cadavers.

Between 1967 and 1971, Mertz and Patrick [39,40] published the results of experiments on human cadavers and on one human volunteer, exposed to impacts of 16 and 37 kph, with the head either supported or unsupported. They calculated the maximum neck torque, shear forces, and axial compression forces under these conditions, and found all to be below levels that might cause radiographically evident injuries of the cervical spine.

In 1972, Clemens and Burow [41] subjected 21 unembalmed human cadavers to rear-end impacts of the order of 25 kph, with and without head rests. Subsequent dissection revealed injuries to intervertebral discs in 90% of the group without head rests, tears of the anterior longitudinal ligament in 80%, tears of the zygapophysial joint capsules in 40%, and fractures of the posterior vertebral body or a spinous process in 30%. No injuries were found in the group protected by head rests.

During the following 20 years, various investigators continued to quantify the gross kinematics of whiplash movements [42–44] and the effects of head rests and seatbelts [45,46]. Others studied lateral flexion movements [47]. These studies, however, did not substantially alter the prevailing precepts established by earlier studies.

4. Modern studies

Modern studies of the biomechanics of whiplash have used high-speed photography and high-speed cineradiography to determine the kinematics of the cervical spine as a whole and of individual segments, both in cadavers and in human volunteers. The results have been illuminating.

Using high-speed photography, Geigl et al. [48] studied six cadavers and 25 human volunteers undergoing rear-end impacts between 6 and 15 kph. They recorded the pattern of motion of the head and neck, and the timing and magnitude of peak excursions of the head. In a later publication [49] the same laboratory reported observations of 22 volunteers undergoing impacts of 8.5 kph. Similar studies were conducted by Ono et al. [50] on three volunteers at 2, 3, and 4 kph.

Also using photography, McConnell et al. [51] studied four human volunteers each undergoing seven rear-end impacts between 3 and 9 kph, and a further seven volunteers undergoing impacts at 5–11 kph [52]. They provided a detailed description of the movements of the torso, neck, and head throughout the entire whiplash movement.

Yoganandan et al. [53–56], using an intact, cadaveric head–neck preparation including muscles and ligaments, determined overall (head to T1), segmental (head–C2, C2–C5 and C5–T1), and local component (zygapophysial joint) motions to explain the plausible mechanisms of headache and neck pain, the two most common complaints in whiplash. A pinching mechanism in the zygapophysial joint was attributed to neck pain and a posterior stretch in upper head–neck complex in the early stages of whiplash acceleration pulse was attributed to headaches. Grauer et al. [57] and Panjabi et al. [58] developed an artificial head-purely ligamentous cervical spine cadaver model to determine patterns and ranges of segmental motion, using high-speed photography.

Using high-speed cineradiography, Matsushita et al. [59] studied 19 volunteers undergoing rear-end impacts between 2 and 5 kph. Their study provided the first observations of intersegmental motion of the neck, and paved the way for subsequent studies by Kaneoka et al. [60,61] using the same experimental design but providing more detailed analysis of intersegmental motion.

Szabo et al. [62] subjected five human volunteers to 8 kph collisions with the view to determining a threshold for injury. West et al. [63] similarly subjected five human volunteers up to 12 impacts between 4 and 13 kph.

Szabo and Welcher [64] and Pope et al. [65,66] explored the activation of muscles during whiplash. Their results bear on the possible protective role of muscles during whiplash.

These various studies show remarkable consistency and convergence. There is good agreement between cadaver studies and human volunteer studies with respect to overall kinematics of the head and neck. Posterior rotation of the head commences between 60 and 100 ms after impact, and has a peak magnitude of 45° between 100 and 130 ms [49,50,53,54]. In particular, the head does not rotate beyond its physiological limit [51,53,59]. The duration of positive acceleration of the head is between 100 and 110 ms and reaches 13 g [49,54].

There is also good agreement between photographic studies of cadavers and human volunteers and cineradiographic studies of human volunteers with respect to segmental and intersegmental motion of the cervical spine [51–61]. These techniques provide a detailed picture of what happens to the neck and body during whiplash (Figs. 2 and 3).

Between 0 and 50 ms after impact there is no response by the body. At 60 ms the hips and low back are thrust upwards and forwards (Fig. 2). By 100 ms the upper trunk moves upwards and forwards. The upward movement of the trunk compresses the cervical

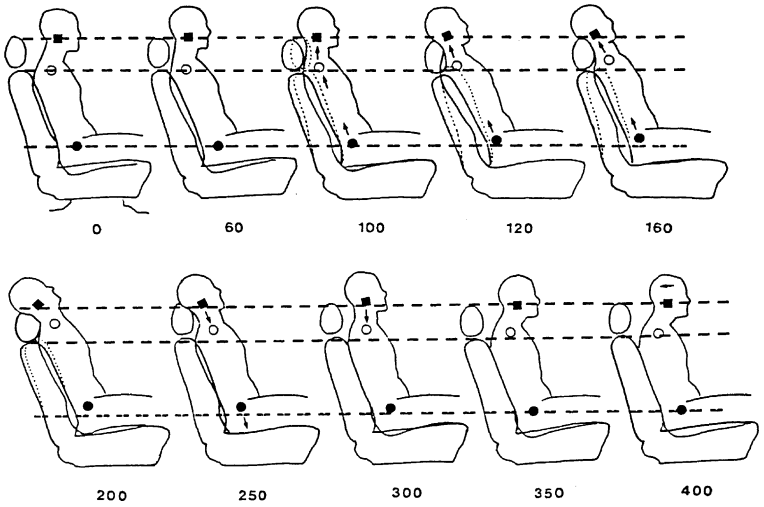

Fig. 2. Movements of the trunk and head after a rear-end impact. Based on McConnell et al. [51]. The symbols represent reference points for the head (square), upper trunk (open circle), and lower trunk (filled circle). The numbers indicate time elapsed, in milliseconds, after impact. By 100 ms the trunk rises and compresses the neck from below. By 120 ms the head rotates backwards. By 200 ms elevation is complete and head rotation maximal. After 250 ms the head and trunk descend. By 300 ms the head rotates forwards, and reaches a maximum excursion by 400 ms, after which, restitution occurs.

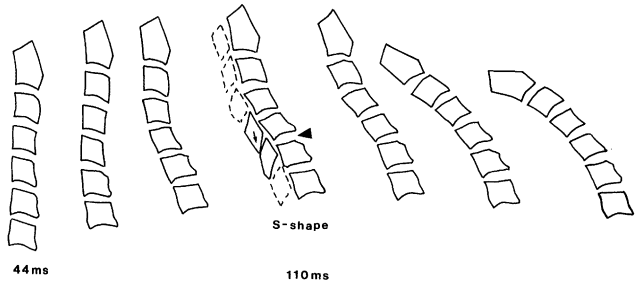

Fig. 3. Tracings of serial radiographs of an individual undergoing a rear-end impact at 4 kph, with no head rest. Based on Kaneoka et al. [60,61]. At 44 ms after impact the cervical spine straightens and is thrust upwards by the rising trunk. By 110 ms it undergoes buckling into an S-shaped configuration, in which the lower cervical segments are extended and the upper segments are flexed. At this time the anterior elements of the lower cervical vertebrae are separated (arrow head), while the articular processes are impacted (arrow). Subsequently, the upper cervical segments extend, and the cervical spine assumes a C-shape.

spine from below, and the forward movement displaces the neck and trunk forwards of the line of gravity of the head. As a result, by 120 ms the centre of gravity of head starts to drop and causes the head to rotate backwards. At about this time, the seat back collapses backwards under the mass of the trunk. By 160 ms the torso pulls the base of the neck forwards, and tension through the cervical spine draws the head forwards. The upward excursion of the torso peaks at 200 ms with an amplitude of 9 cm, and the head reaches its peak posterior rotation of 45°. By 250 ms the trunk, neck, and head are descending, and the descent is complete by 300 ms. By 400 ms the head achieves its maximum forward excursion and begins to rebound. Between 400 and 600 ms restitution of position occurs. At higher impact speeds, the same order of events transpires but the magnitude of the kinematics is greater [51]. The upward acceleration of the neck measures between 1.0 and 1.5 g [51].

Between 50 and 75 ms the cervical spine undergoes a sigmoid deformation as it is compressed by the rising trunk, such that the lower cervical segments undergo extension while upper segments flex [53–57,60,51]. By 120 ms the spine extends into a C-shape (Fig. 2). Although the cervical spine as a whole does not exceed physiological ranges of movement at any stage during its excursion [51,52,57,59], lower cervical segments consistently exceed physiological limits of posterior rotation [58]. Moreover, this rotation occurs around an abnormal axis of rotation [60,61]. The axis is located within the moving vertebra, considerably rostral to its normal location (Fig. 3). This abnormally high location of the axis indicates that the vertebra undergoes no translation; its movement is purely posterior rotation, and arises because the force producing the movement is an upward thrust. No shear force is exerted on the vertebra at this point in time.

As the vertebra extends about this abnormally located axis, its anterior elements rotate upwards and its posterior elements rotate downwards. The upward rotation causes the anterior ends of adjacent vertebral bodies to separate in an abnormal fashion (Fig. 2). Meanwhile, instead of the articular processes of the zygapophysial joints gliding across one another, the inferior articular processes of the moving vertebra chisel into the superior articular processes of its supporting vertebra (Fig. 3). This posterior compression within the zygapophysial joints occurs at about 100 ms after im-

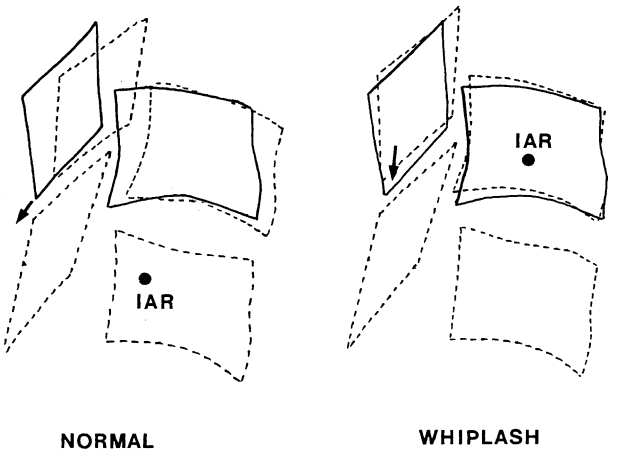

Fig. 4. Tracings of the kinematics of a C5-6 segment based on Kaneoka et al. [60]. During normal movements the instantaneous axis of rotation (IAR) lies below the disc of the segment. As C5 rotates backwards about this axis, its inferior articular facet glides backwards across the superior articular facet of C6 (arrow). During whiplash movement, the IAR is displaced upwards into the moving vertebral body. As C5 rotates backwards about this axis, the tip of its inferior articular process chisels into the surface of the superior articular facet of C6 (arrow).

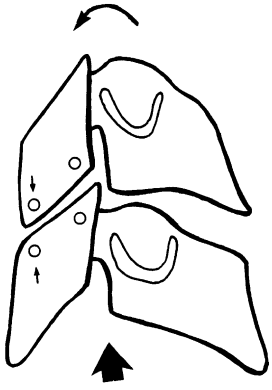

Fig. 5. Movements of the C5 articular processes in a cadaver during rear-end impact in a right lateral view. Based on Yoganandan and Pintar [56]. The circles represent targets implanted into the articular processes. After impact, the C5 vertebra rotates backwards, and the posterior ends of the articular processes are forced together (arrows).

pact. It is evident in volunteers [60,61] and has been measured in detail in cadavers [53–56] (Fig. 4 and Fig. 5).

Muscles are recruited relatively late during the whiplash event. They start to be recruited by 100–125 ms, but may take a further 60 ms to develop tension [58,64]. In the unbraced individual the delay may be 200 ms [67] or even 250 ms [65,66]. Because of their passive stiffness, and to a greater extent if they are activated, muscles might limit the angular excursions of the head, that occur late in the whiplash event. However, by the time muscles are activated, compression of the cervical spine, and the abnormal intersegmental movements that seem critical to injury, have already occurred.

5. Discussion

The critical revision brought about by modern research into whiplash is that it is not a cantilever movement that is injurious; i.e., it is not an extension–flexion movement of the head, as was commonly believed previously. Rather, within less than 150 ms after impact, the cervical spine is compressed. During this period the cervical spine buckles; upper cervical segments are flexed while lower segments extend around abnormally located axes of rotation. It is during this period of compression that the cervical spine is vulnerable to injury.

The cause of the compression is the upward thrust of the trunk. This has been attributed to the seatback extending the thoracic kyphosis, for compression occurs only when the subject sits in a stooped posture [59]. It does not occur if the subject is declined with the head applied to a head-rest. Under those conditions, the torso and the head collapse backwards together as the seatback collapse, and no upward thrust is exerted on the neck [59].

In this regard, the results of whiplash research are concordant with the findings of studies of catastrophic injuries to the neck, e.g., football injuries, diving injuries, and axial head impact. Whereas flexion or extension moments may disrupt joints and cause dislocations, axial compression accounts for the major fractures of the atlas or lower vertebral bodies that occur in such injuries [68]. Moreover, axial compression occurs rapidly after impact, and before bending of the cervical spine occurs, if at all.

Notwithstanding these new insights into whiplash, biomechanics research does not prove, or even suggest, that all victims of whiplash incur injuries. Indeed, the epidemiological literature indicates that only a minority of victims sustain a substantive injury. The vast majority recover with no lasting ill-effects; some 60% recover within six months, and 80% within 16 months [69]. Whether or not a victim sustains an injury is a function of multiple factors: the magnitude of the impact, their posture at the time, their anatomy, and the material strength of the components of their cervical spine.

In this regard, human volunteer tests are designed not to produce injury. In order not to seriously injure the volunteers, impact speeds are kept below a tolerable maximum. Under those conditions, experimental studies reveal qualitatively what occurs in the neck, but they understate the magnitude of the displacements and forces that conceivably might occur in impacts of much greater speed.

Some indication of the threshold for injury is available from the literature. Geigl et al. [48] subjected their 25 human volunteers to impacts of 6–12 kph, and reported that none sustained injuries. However, the same laboratory later reported that one volunteer who when subjected to a 10.5 kph impact developed symptoms for

two weeks, for which reason they reduced their test impact to 8.5 kph [49]. Of the five volunteers subjected to 8 kph impacts by Szabo et al. [62], four experienced headache that resolved immediately, and one reported minor, transient neck stiffness. In contrast, five of the 19 volunteers subjected to impacts of 2–5 kph in the study of Matsushita et al. [59] reported mild discomfort lasting 2–4 days. In the study of West et al. [63] the volunteers were subjected to up to 12 impacts at 4–13 kph, and reported minor neck pain lasting up to 2 days. McConnell's volunteers [51] reported discomfort lasting for 5 h in two cases but three days in another. Although these data indicate that impacts of less than 10 kph are essentially safe, they also suggest that the safe limit is not much above this speed. The incidence of transient symptoms after impacts of 10 kph or less implies that more serious symptoms would be incurred at speeds greater than this.

Most intriguing is the convergence between biomechanics research, postmortem studies, and clinical research. Current biomechanics data indicate that lower cervical zygapophysial joints are impacted during the whiplash movement. This has been demonstrated both in volunteers [60,61] and in cadavers [53–56]. The pattern of movement suggests that the sort of lesions that could occur might range from contusions of intra-articular meniscoids and intra-articular haemorrhage to impaction fractures of the articular cartilage or fractures of the articular processes. These are the very lesions identified in postmortem studies of victims of motor vehicle accidents [1,3]. Meanwhile, clinical studies have established that zygapophysial joints are the single most common source of chronic neck pain after whiplash [70,71]. They account for at least 50% of cases, and perhaps 80% of drivers involved in high-speed impacts [72].

For other cases, the biomechanics data suggest that disruption of an intervertebral disc may be the lesion. As vertebral bodies separate anteriorly, the anulus fibrosus may be torn or the disc may be avulsed from the vertebral body. Such lesions have been produced in experimental studies on cadavers [41] and identified at postmortem [1–3]; but no means have been developed, to date, whereby such lesions can be detected clinically. Rim lesions of the disc have been reported in one study using MRI [73], but subsequent studies have failed to corroborate this finding [74–79]. Disc lesions, therefore, remain a conjecture waiting for a valid diagnostic test before their prevalence can be ascertained.

In essence, most victims of whiplash probably escape substantial injury but for those who develop chronic pain, biomechanics research has revealed a plausible mechanism of injury. As the cervical spine is compressed, the lower cervical vertebrae undergo rotation about an abnormally located axis. Their anterior elements separate while their zygapophysial joints impact. At low impact speeds these movements induce no permanent injury, but it is conceivable that at higher impact speeds, intervertebral disc may be torn or avulsed, and zygapophysial joints may be bruised or fractured. In the context of zygapophysial joint pain, these predictions have been vindicated by clinical research; in the context of discogenic pain they await further study. Nevertheless, for the 10% or 20% of victims who develop chronic symptoms, the available data indicate that their condition is not imaginary or fictitious. The biomechanics data, the postmortem data, and the clinical data agree that injuries can, and do, occur.

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