

INJURY BIOMECHANICS RESEARCH: AN ESSENTIAL ELEMENT IN THE PREVENTION OF TRAUMA

DAVID C. VIANO*¶, ALBERT I. KING†, JOHN W. MELVIN* and KATHLEEN WEBER§

*Biomedical Science Department, General Motors Research Laboratories, Warren, MI 48090; †Wayne State University Bioengineering Center, Detroit, MI 48202; §Section of Pediatric Surgery, University of Michigan Medical School, Ann Arbor, MI 48109, U.S.A.

Abstract—The central aspects of injury biomechanics research are defined and research approaches described. These aspects include the identification and definition of impact injury mechanisms, the quantification of biomechanical response to impact, the determination of impact tolerance levels, and the development and use of injury assessment devices and techniques for evaluating injury prevention systems. The current status of knowledge and technology is then reviewed for the head, cervical spine, thorax, abdomen, and lower extremity. Important gaps are identified, and research priorities emphasizing functional impairment are proposed.

INTRODUCTION

Impact injury to the human body is caused by deformation of biological tissues beyond their recoverable limit, resulting in damage to anatomical structures or alteration in normal function. The structural injuries, such as bone fractures, are likely to heal, but normal motor or sensory function may never completely return if central nervous system tissues are damaged. Even though humans have been known to survive remarkable impact situations, such as falls from extreme heights or severe motor vehicle crashes, mechanical impact is one of the leading causes of injury, fatality, and disability in the United States (American Trauma Society, 1982; Baker *et al.*, 1984; Committee on Trauma Research, 1985; Committee on Trauma & Committee on Shock, 1966; Hartunian *et al.*, 1981; Luchter, 1986; National Safety Council, 1983; Office of the Assistant Secy. for Health & Surgeon General, 1979; Trunkey, 1983; Viano, 1988a). Since accidental injury primarily affects the younger portion of our population (Baker *et al.*, 1984; Perloff *et al.*, 1984), there is an urgency to allocate national resources toward research that will bring us closer to an understanding of the process and effects of injury and thus closer to its prevention (Baker *et al.*, 1984; Morse, 1973; SAE, 1982; Stone, 1977; Toole and Toole, 1984; Trunkey, 1983; U.S. DOT, 1983; Waller, 1984).

Injury biomechanics research uses the principles of mechanics to investigate and explain the physical and physiological responses to impact that result in injury. Both penetrating and non-penetrating injuries may be addressed, but the latter are more complex, less well

understood, and offer greater opportunities for mitigation through technological advances. Trauma research involves a variety of disciplines, including engineering, physiology, medicine, biology and anatomy. For this reason, there is no unified research approach nor single area of education, training, or experience that will prepare scientists for such work, and studies are often conducted by teams of engineering, medical, and other personnel, whose combined expertise is important to the success of the research.

The risk of injury is related to the energy delivered to the body by the impacting object as well as to the object's shape. Penetrating injuries are generated by high-speed projectiles, such as bullets, or by sharp objects moving at relatively lower speeds, such as knives and daggers (Rosenberg *et al.*, 1984). In this type of injury, impact energy is concentrated in a small area of the body, there is little opportunity to dissipate this energy, and the mechanisms of structural damage are fairly obvious (Adams, 1982; Di Maio, 1981). Non-penetrating injuries occur, however, when the body is struck by or strikes a blunt object, such as a vehicle instrument panel, at a moderate velocity, but the force of the impact is distributed over a broad area (Council on Scientific Affairs, 1983; Smith and Coleman, 1984). In this case, impact energy can be absorbed by padding or other crushable materials, which allows the impacting and impacted surfaces to deform and thereby extends the duration of the impact and reduces its severity (Begeman and King, 1975; Blizard and Howitt, 1969; DeHaven, 1969; Kroell *et al.*, 1973; Patrick *et al.*, 1965; Reiser and Chabal, 1969; SAE, 1980; Stapp, 1970).

Because of the inertial resistance of body tissues and the elastic and viscous compliance of body structures, force is developed on the human body during impact. This force generates both deformation and acceleration of the body, and both can cause injury (Viano, 1988a; Viano and Lau, 1988). Compression of the chest can fracture ribs and rupture organs, while head acceleration can result in injury to the brain as its

Received in final form 29 November 1988.

¶The first two authors were members of the National Academy of Sciences' Committee on Federal Trauma Research. This paper amplifies and expands on the core information contained in Impact Biomechanics, Chapter 4 in *Injury in America: A Continuing Health Problem*, National Research Council, National Academy Press, 1985.

¶To whom correspondence should be addressed.

motion lags that of the skull and its soft tissue deforms beyond its recoverable limit. The human body's viscoelastic tissues also protect vital organs from impact injury by absorbing some of the impact energy. As long as the energy delivered to the tissue is below its tolerance limit, recovery from impact deformation occurs.

The broad goal of injury biomechanics research is to understand the injury process and to develop ways to reduce or eliminate the structural and functional damage that can occur in an impact environment. To achieve this goal, researchers must identify and define the mechanisms of impact injury, quantify the responses of body tissues and systems to a range of impact conditions, determine the level of response at which the tissues or systems will fail to recover, develop protective materials and structures that reduce the level of impact energy and force delivered to the body, and develop test devices and computer models that respond to impact in a humanlike manner, so that protective systems can be accurately evaluated. The following sections address the general scope of each of these research aspects and describe the current state of knowledge and technology related to several critical body regions.

INJURY MECHANISMS

A mechanism of injury is a description of the mechanical and physiological changes that result in anatomical and functional damage. Knowledge and understanding of injury mechanisms is fundamental to the science of injury biomechanics, because it provides the basis for determining appropriate measures of response and tolerance to impact in the various parts

of the body. Deformation of tissues beyond their recoverable limit is the general injury mechanism associated with blunt impact (Frankel and Nordin, 1980; Ghista, 1982; Reul *et al.*, 1978; Nahum and Melvin, 1985). This mechanism is measured in terms of strain, defined as the change in a dimension, such as the circumference of an artery, divided by the original dimension. The primary types of strain that damage tissue are tensile strain and shear strain (Fig. 1), which produce lacerations, fractures, ruptures and avulsions (Fung *et al.*, 1972; Mohan and Melvin, 1982, 1983; Viano, 1986). A third type is compressive strain, which produces crushing injuries.

If an artery is stretched beyond its tensile strength, the tissue will break. Organs and vessels can be stretched in different ways, which result in different types of injuries. For example, the motion of the heart during chest compression stretches the aorta along its axis at its points of attachment. This elongation generally leads to a transverse laceration when the strain limit of the tissue is exceeded (Besson and Saegesser, 1983; Mohan and Melvin, 1982). An increase in vascular pressure, on the other hand, dilates the vessel and produces tensile strain in both the axial and transverse directions. If pressures rise beyond the vessel's limit, the tissue will burst outward. Impact along the axis of a femur causes an increase in its curvature, with tensile strain on the anterior surface, and compressive strain on the posterior. Midshaft fracture of the femur occurs when its tensile strain limit is exceeded (Cheng *et al.*, 1984; Evans, 1981; Viano, 1977, 1986; Viano and Stalnaker, 1980). This injury mechanism is also common to the ribs, where compression of the chest causes tensile strain on the outer surface of the ribs. Thus injury to two very

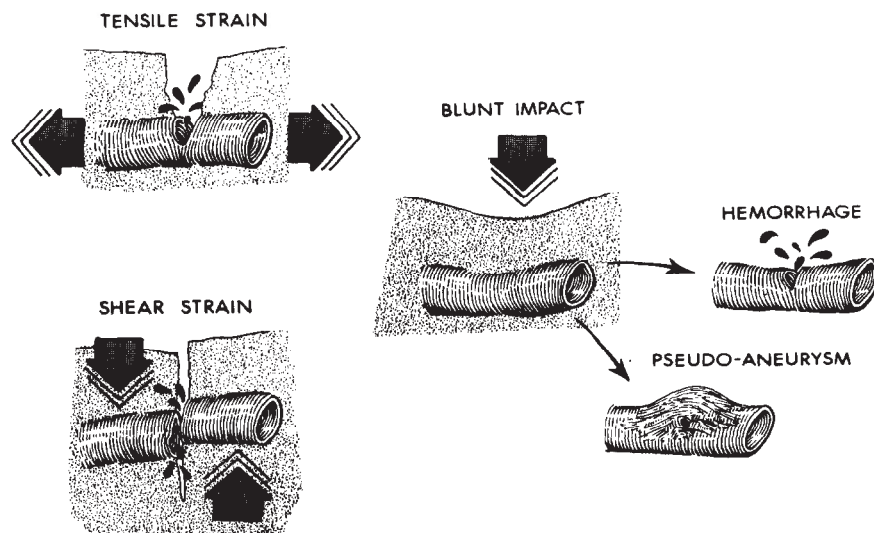


Fig. 1. Stretch of a vessel can tear the tissue (a partial tear shown) with a loss or containment of blood. Opposing forces across the vessel can cause a shear injury (a complete tear shown) again with or without loss of blood. Vessels beneath the skin can be torn by stretch or shear without laceration of the skin. This can cause a bruise or contusion.

different types of tissue, vascular and bony, can be explained in terms of tensile strain and associated failure.

Shear strain occurs when opposing forces act across a tissue, moving it in opposite directions. When the resistive limit is reached, the tissue will separate. During head impact, for example, the differential movement of the brain with respect to the accelerated skull may cause shear strains and eventual failure of the interfacial structures between brain and skull (Gurdjian, 1975; Courville, 1950; Ommaya *et al.*, 1971). Differential motion of lobes of the liver can also shear the attachments at the hilar region and lacerate the major connecting vessels when the strain exceeds the limit of recovery (Lau and Viano, 1981a, b).

The strain mechanism not only explains laceration injuries but is also a principal factor in contusion injury (Fig. 1). In this case, the surface of the tissue is not damaged, but deformation of the body may stretch and shear internal vessels and increase intraluminal pressures, causing internal tissue damage with hemorrhage or blood retained by a pseudo-aneurysm or partial tear of the vessel.

The rate of loading and thus the strain rate is also an important factor in the production of injury (Lau and Viano, 1981a, b; Viano and Lau, 1983; 1988). Because biological tissues are viscoelastic, their response and tolerance to impact is rate sensitive. If, for example, a fluid-filled organ is compressed slowly, much of the applied energy can be absorbed through deformation without tissue damage. When loaded rapidly, however, the organ cannot deform fast enough and rupture may occur before significant change in shape. Compact bone also exhibits rate sensitivity during impact. A femur loaded axially can withstand higher loads as impact velocity increases, but the actual strain (bending) at failure decreases (Cheng *et al.*, 1984; Viano, 1980; Viano and Stalnaker, 1980).

Research addressing injury mechanisms requires a combination of accident investigations, clinical case studies, and laboratory experiments with human cadavers and anesthetized animals (Table 1). Because of extensive clinical and experimental work, mechanisms

for many significant injuries have been adequately described. The sequence of events following blunt thoracic impact that lead to laceration and rupture of the heart and great vessels has been discussed extensively in the literature (Besson and Saegesser, 1983; Liedtke and Demuth, 1973; Symbas, 1976; Viano, 1983), and fracture of the femur and dislocation of the knee joint are generally well understood (Noyes and Grood, 1976; Viano, 1980, Viano *et al.*, 1978). Less is known about injury mechanisms for other organs, bones, and joints, but, more importantly, the mechanisms of functional damage to central nervous tissue have not been well established. Although isolated tissue preparations have provided some information on the changes that occur, many of the mechanisms responsible for these changes are unknown or speculative at best. Research needs to be directed at the biomechanics of impact to central nervous system tissue as the first step toward mitigating its disabling consequences (Journal of American Insurance, 1983; Kraus *et al.*, 1984; NINCDS, 1980).

BIOMECHANICAL RESPONSE

Once a mechanism is described or hypothesized, the next step is to quantify the biomechanical response during impact deformations. Response is a time varying change in shape of the body, an organ, or tissue caused by mechanical loads (Kroell *et al.*, 1973; Stapp, 1970; Nahum and Melvin, 1985). Measurements characterize the elastic and viscous resistance of biological tissue to deformation and the inertial resistance of the body to motion. The quantified responses are used to analyze the injury process, so that response thresholds related to injury can be established, and to develop mechanical or mathematical models that behave like humans under impact conditions.

Ideally, human response measures would be obtained from living human subjects under various impact conditions. For obvious reasons, this is not possible. People involved in accidental impact are not instrumented with electronic measuring devices, and experimental impact of human volunteers can only be

Table 1. Applicability of research tools used in impact biomechanics

Tool/procedure	Injury mechanisms	Impact response	Impact tolerance	Assessment technologies
Crash investigation/reconstruction	***	*	**	**
Clinical study	***	*	*	*
Volunteer test	*	***	*	**
Animal experiment	****	***	****	**
Human cadaver	****	****	****	**
Dummy test	*	**	**	****
Mathematical model	*	**	***	***

- * Limited applicability
- ** Appropriate/useful.
- *** Important/valuable.
- **** Vital/necessary.

done below the pain threshold. There are, in fact, rigid rules and guidelines limiting the acceleration levels that can be used with volunteers. Only a few 'man-rated' impact facilities exist in the United States (Begeman *et al.*, 1980; Ewing *et al.*, 1978; Von Gierke and Brinkley, 1975), most of these being military facilities. As a result, human volunteer data tend to reflect the response of strong young males.

Although measures of response to noninjurious impact can be obtained from volunteer experiments, the primary data on impact response at injury levels must, of necessity, be obtained using human surrogates. These are human cadavers and anesthetized animals, each of which has its limitations for impact biomechanics research. The cadaver is a suitable research model to simulate gross geometric and material properties of the human or to study the mechanical response of a body segment, such as chest deformation or head trajectory. Such surrogates, however, tend to be of an advanced age, have no active muscular response that may influence their realism at lower levels of acceleration, and cannot be used to assess functional changes due to injury. The only way to obtain information on the physiological responses triggered by injury is to subject live, anesthetized animals to experimental impact, and even their physiological responses are influenced somewhat by the anesthetic. Although biological data obtainable only from animal experiments are critical to the study of brain and spinal cord injuries, arrhythmia, and shock, the use of animals for such research is coming under increasing criticism by activist groups who would like to prohibit it altogether.

IMPACT TOLERANCE

At some measurable level of deformation magnitude and rate, the tissue will not be able to recover, and damage or injury will occur. This level of response is the injury threshold and indicates the tolerance of the body, organ, or tissue to impact. Although any injury is of concern, it is common in impact biomechanics to try to identify response thresholds beyond which there is unacceptable damage to tissues or structures, such as gross anatomical lesions or injuries that result in a permanent alteration of normal function. The definition of unacceptable injury is not fixed but is a function of a variety of parameters, including the body region, the type of tissue, and, for experimental purposes, the type of test subject used. Even with the use of surrogates, our current state of knowledge concerning human impact tolerance is very incomplete, and experimental impact data are practically nonexistent for adult females and for children.

Determination of human tolerance levels is complicated by many factors related to the impact configuration, the test environment, and the test subject that influence the outcome of the impact event. These

factors include the velocity, mass, direction and surface area of the impactor and the onset rate and duration of the applied force. In addition, the body orientation, the design and tightness of any restraint system used, and the characteristics of the seating system affect the results. Biological factors, such as age, sex, body size, and physical and possibly mental condition, also influence human injury tolerance as well as the ability to survive particular injuries. Diseases such as hypertension and arteriosclerosis tend to increase the severity of cardiovascular trauma, and osteoporosis reduces the impact resistance of bone. The latter disease is more likely to affect women postmenopause, and is a significant factor in vertebral, rib, and femoral fracture.

Chronic alcohol use interferes with normal body repair processes and is also a significant factor in accident causation. Alcohol in tissues appears to predispose an accident victim to more severe and extensive injury than the non-drinker in a similar impact environment (Anderson and Viano, 1986; Waller *et al.*, 1986). Physiological experiments have demonstrated a higher mortality to blunt thoracic impact with, than without, administration of alcohol, as alcohol enhances the electro-mechanical decoupling of the cardiac excitation-contraction process (Desiderio, 1987) and increases the risk of permanent impairment resulting from spinal cord injury (Anderson, 1986; Flamm *et al.*, 1977). The high incidence of alcohol in the blood of seriously and fatally injured road accident victims (38 and 46%, respectively), indicates that this drug may be one of the more significant factors influencing injury severity.

Along with population variability, individual variability should be considered when trying to determine human tolerance levels. In addition, experimental impacts with a given set of parameters may yield tolerance information for that configuration, but the result may not be applicable to a different set of parameters. Any statement of human tolerance to impact must be accompanied by the specific conditions involved, since there are a number of tolerances depending upon what is being loaded and how the loading occurs, and even a well-controlled impact will generate different results for different subjects. In the end, it may only be possible to identify a distribution of tolerances for a given population and exposure.

Although the most reliable tolerance information comes from experimental impacts, accidental falls can provide some insight into human tolerances to a variety of impact conditions (Foust *et al.*, 1977; Snyder, 1963) particularly at extreme levels beyond which human volunteers are not subjected. Other sources of this type of information are clinical studies of impact trauma and reconstructions of automotive or aircraft crashes (SAE, 1985a). The data obtained, however, are of limited scientific value, because no impact or response measurements are made during the event. Thus, although the injuries may be known, the impact conditions, the biomechanical response,

and the tolerance levels can only be estimated after the fact.

INJURY ASSESSMENT TECHNOLOGY

Blunt impact and acceleration injury can be significantly reduced in the automotive environment through the use of crushable vehicle structures, which absorb impact energy, and restraint systems, which allow the occupant to decelerate more slowly with the crushing vehicle. Other occupant protection technology has been applied to produce a friendly interior, which reduces the likelihood that impact with interior surfaces will be injurious (Viano, 1988a). These protective systems could not be designed, developed, refined, and installed in automobiles, however, without some means of first testing their effectiveness in the laboratory. Although cadavers have been and are still used occasionally for this purpose, they are not easily available nor do they provide repeatable information. A better solution is an anthropomorphic dummy that responds to impact in a humanlike manner, as dictated by average response data generated in laboratory experiments with human surrogates.

For practical reasons, dummies are not actually 'injured', but rather levels of biomechanical response are established that are judged likely to result in injury if the dummy were a human. These response thresholds are called injury criteria, and different criteria are used for different regions on the body (SAE, 1980). Using such a test device along with appropriate criteria in test configurations that represent a range of impact exposures, the risk of injury and disability to the human can be assessed, improvements in protective systems can be made, and the overall risk of injury reduced.

Current anthropomorphic dummies (Foster *et al.*, 1977; Mertz, 1985) are providing more extensive acceleration, deformation, and kinematic data, which in most cases can be correlated somewhat with human impact response. However, their ability to predict anatomical or structural injuries is marginally reliable. More important, dummies are not capable of evaluating functional changes that result in severe cognitive dysfunction, quadriplegia, or fatal ventricular fibrillation.

Another test tool used to assess injury risk is computer simulation of an occupant in a crash situation (King and Chou, 1976; SAE, 1985b). This tool can be used to study the effects of a wide range of design changes and improvements without the time and cost of several laboratory experiments. Although there have been recent advances in the simulation of real-world crashes, mathematical models cannot incorporate all the complexities of the human system.

Anthropomorphic dummies and mathematical models are used extensively as predictive tools, particularly of body kinematics and acceleration during impact. Simulations, however, are only as accurate as

the biomechanical information used in their formulation, and they are probably lacking in accuracy when interactive forces are developed during impact. For most body regions, the lack of basic research data on biomechanical response has retarded the development of more sophisticated models or dummies. Thus, the current tools and techniques for assessing injury are only a first step in the development of a truly adequate evaluation procedure for protective systems. Further advances are dependent on a clear understanding of, and detailed data on, trauma mechanisms, biomechanical responses and tolerance levels of the human. In the following sections, the current state of knowledge regarding these factors is summarized for critical body regions.

HEAD INJURY

A variety of mechanisms have been postulated for mechanical damage to the brain in closed head injury, but all are related to acceleration of the head. The most severe accelerations are usually the result of direct impact to the skull or face. High accelerations can also be generated during abrupt changes in motion without head contact, such as might occur to a restrained occupant in a severe frontal crash, but such acceleration has not been found to produce brain injury without direct impact. The actual mechanisms of injury (Courville, 1950; DOT, 1981; Gennarelli and Thibault, 1982; Gennarelli *et al.*, 1982; Gurdjian, 1975; Gurdjian and Gurdjian, 1975, 1976; Ommaya, 1985; Ommaya *et al.*, 1971; Viano, 1985) may include the following: (1) direct brain contusion from skull deformation at the point of contact; (2) brain contusion from movements of the brain against rough and irregular interior skull surfaces or from the side opposite the impact; (3) brain and spinal cord deformation in response to pressure gradients and motions relative to the skull, resulting in stress in the tissues; and (4) subdural hematoma from movement of the brain relative to its dural envelope, resulting in tears of connecting blood vessels.

Because the brain has its own inertia and is loosely coupled to the skull, its motion will lag that of the skull when the head is suddenly stopped (Fig. 2). The resulting differential displacement causes interfacial shear between the brain and skull, stretches the vessels that tether the brain, and strains the brain tissue as it contacts bony protrusions and membranes. When the skull is impacted directly, the brain also deforms and pressures increase at the site of local skull deformation. These deformations and pressure increases, if of sufficient magnitude, can result in brain laceration or contusion, evidenced by hemorrhage, or diffuse axonal injury, which causes functional changes. Clearly, neural injury is a major source of injury disability (Gennarelli, 1982; Langfitt *et al.*, 1982; Rimel *et al.*, 1981, 1982) and much more research is needed on its mechanism and tolerance to impact.

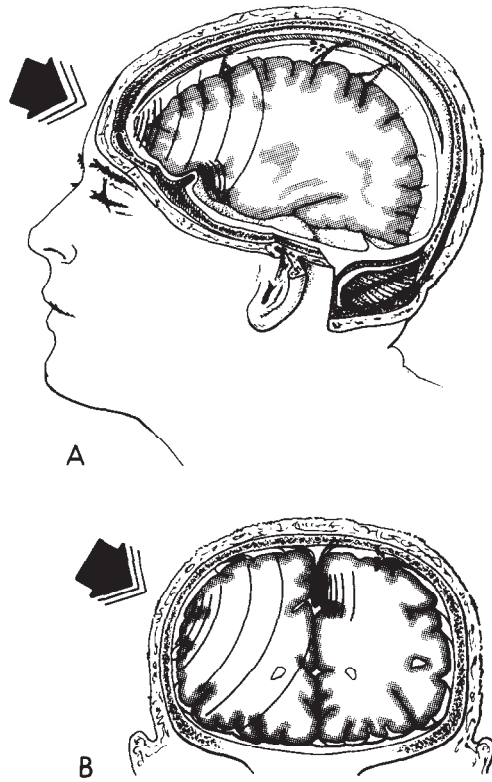


Fig. 2. Blunt impact of the moving head decelerates the skull. Injury of the brain can be caused as its motion lags that of the skull and strain develops in brain tissue and blood vessels.

Although different impacts to the head can produce a variety of complex three-dimensional motions with a wide range of deformations throughout the brain tissue, vascular brain injuries primarily occur on the inferior surfaces of the frontal and temporal lobes, where ridgy protuberances are located (Gurdjian, 1975; Gurdjian and Gurdjian, 1975, 1976). Such injuries occur whether the impact is to the front or the back of the head. At low levels of dynamic loading, there may be only temporary disruption of brain function, or concussion, but the mechanisms described above are still thought to be the source of this dysfunction.

The threshold values for the mechanical impact parameters associated with a particular type of brain injury are not well defined. These parameters include translational and rotational accelerations, rotational velocities and contact forces, and procedures are now available to assess the complete dynamics of the head in test dummies subjected to impact (Viano *et al.*, 1986). In the development of head injury criteria (Hess *et al.*, 1980), emphasis has been placed on the effects of translational acceleration, and tolerance levels have been based on experimental impacts with head contact. The Wayne State Tolerance Curve (Lissner *et al.*, 1960) and the later Japan Head Tolerance Curve (Ono *et al.*, 1980) are based on empirical data and describe tolerable levels of head translational

acceleration as functions of head impact duration. Although the curves are in close agreement for the short-duration, direct-contact impact injuries studied, their application for non-contact, or indirect impact, is open to question.

The Head Injury Criterion (Versace, 1971) is an extension of the Gadd Severity Index (Gadd, 1966) and is based on the general relationship between tolerable acceleration level and the associated duration, as described by the Wayne State Tolerance Curve, but allows the analysis of complex acceleration-time waveforms. The HIC is the most commonly used method of evaluating head impact data at this time. Recently, statistical analysis of direct head impact cadaver test data has been used to define the relationship between HIC values and the probability of sustaining a particular level of injury, thus providing a continuous ability to interpret HIC values (Prasad and Mertz, 1985). A HIC level of 1000 was found to produce an expected 16% incidence of life-threatening brain injury to the adult population.

Injury criteria based on head angular acceleration and angular velocity have been proposed for long-duration, indirect impact situations (Ommaya, 1985), but they lack the extensive evaluation and review that has been given the HIC for short-duration (less than 15 ms) head impacts. Mathematical models of the head hold promise for evolving into injury predictive models, given proper development and evaluation. Simple models, such as the Mean Strain Criterion (Stalnaker and McElhaney, 1970), which are based on translational acceleration, have the potential for describing the dependence of the injury response on impact waveform and direction of impact. The application of the MSC and other brain response models to dummy head accelerations which may be helpful in interpreting injury risks (Viano, 1988b) remains to be developed. More sophisticated finite element models of the brain and skull (see review by Khalil and Viano, 1982) have been developed, but their complexities and the lack of experimental verification of brain injury mechanisms have hampered their development into injury predictive models.

The testing technology available to simulate the response of the head to impact is rather crude (Hess *et al.*, 1980; Hubbard and McLeod, 1974). The data presently available for defining this response are limited to rigid impacts and are predominantly based on embalmed cadaver tests. The response measures are either head acceleration or contact force, and rigid surfaces have been used because response measures may not be repeatable if the impacted surface is allowed to deform. Thus, test dummy heads have been designed to respond in a humanlike manner only to direct forehead impact with rigid surfaces (Foster *et al.*, 1977). Even then, the heads do not contain simulated brains, primarily because there is little data on brain impact response on which to base a simulation. Current head impact data are adequate to define general response to rigid impacts to the front and side

of the head over a range of impact velocities from 1 to 8 m s^{-1} for peak contact force, but only 1 to 5 m s^{-1} for acceleration response. There is, therefore, a need for more studies using unembalmed cadavers at higher impact speeds and using current acceleration measurement techniques. A repeatable and reproducible method for conducting padded head impacts is also needed to define head response to non-rigid impacts.

Another major limitation of current mechanical simulations of head impact is the lack of humanlike facial structures. Skull response is simulated by a metal headform covered by a rubber-like scalp, and the dummy's face is merely the front part of this 'skull'. The fracture and collapse of human facial bones during distributed loading, however, significantly reduce the peak forces and resulting head accelerations, compared with those produced by similar impacts to the skull. Because current dummies have unrealistically rigid faces, indicators of brain impact tolerance that are based on skull acceleration cannot be used to evaluate brain injury when the impact is to the face.

The response of facial structures to impact has been studied to a limited extent, but data on fracture tolerance of facial bones to direct loading is available for individual bones as well as for the face as a whole, (e.g. Hodgson, 1967; Schneider and Nahum, 1972; Tarriere *et al.*, 1981). Soft tissue injuries are also of concern relative to the face, as important nerves can be severed and disfigurement and paralysis result from facial laceration. The failure characteristics of soft facial tissues have been studied in connection with the development of high-penetration-resistant windshields (Patrick and Daniel, 1964), and rating systems for the assessment of laceration severity have been developed (Pickard *et al.*, 1973). There is a need, however, to study the mechanisms of facial laceration from blunt as well as penetrating impact.

CERVICAL SPINE INJURY

Impact to the head may cause the neck to flex (bend forward), extend (bend rearward), or bend laterally, depending on the impact direction (Fig. 3). In addition, tension or compression of the neck may occur along with flexion, extension, or lateral bending, depending on whether the head/neck system is inertially or directly loaded during impact. Each of these loading combinations can result in different injury mechanisms of the cervical spine, including compressive and shear strain at the vertebral bodies that may result in wedge fractures of the bodies and, with increased load, fracture-dislocations of the facets (Holdsworth, 1963, 1970; Kazarian, 1981; Portnoy *et al.*, 1971; Schneider, 1974).

Although this description of the injury mechanisms leading to cervical spine fracture-dislocation is a reasonable hypothesis, it has not been verified experimentally, nor is there any direct correlation between fracture-dislocation and spinal cord damage. Another reasonable hypothesis, however, is that, if the relative displacement between adjacent facets is large, an unstable fracture occurs. Bone and other tissue can then intrude into the spinal canal, causing injury to the spinal cord. Unless the cord is transected, damage is less likely to be lacerative than it is to be contusive to the central grey matter and to cause a transient disruption of axonal function. The hemorrhagic damage expands radially with time and can ultimately permanently disrupt the axonal tracts and alter motor and sensory function. Thus, impact involving head motion initiates a complicated sequence of events that can ultimately result in damage to the vertebral column and intrusion into the spinal cord.

The static and dynamic response of the head/neck system to indirect inertial loading at low crash severities has been studied extensively in volunteers and, to

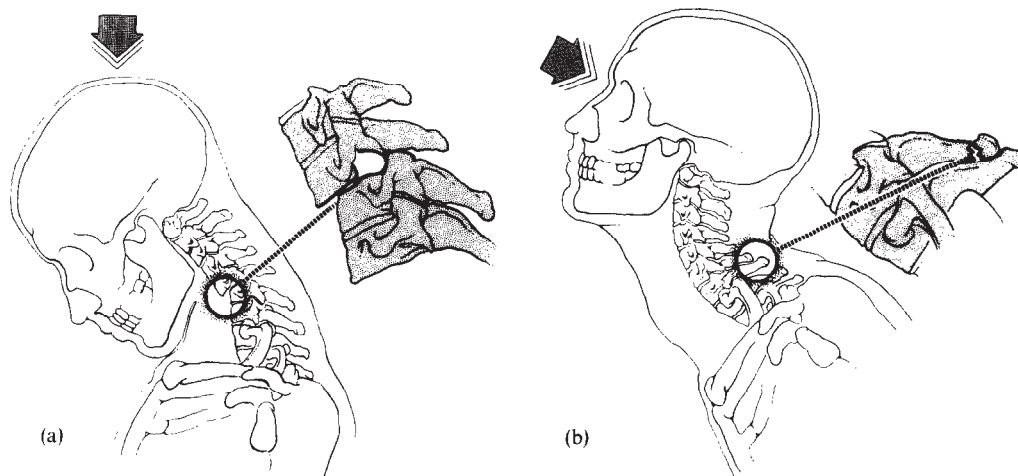


Fig. 3. Downward impact on the head can flex (a) or extend (b) the neck with fracture-dislocation of the vertebrae and possible damage of the spinal cord.

a lesser extent, in cadavers (Collins, 1983; Ewing *et al.*, 1978; Mertz and Patrick, 1967; Patrick and Chou, 1976; Schneider *et al.*, 1975). These studies have included frontal, lateral, and oblique impacts, and a limited data base exists for rear impacts. Specification for suitable neck linkage systems, ranges of motion, and joint resistance characteristics are thus available for dummy design (Mertz *et al.*, 1978, 1973) and mathematical modeling (Bowman *et al.*, 1984; Wismans and Spenny, 1983, 1984).

The status of knowledge on the tolerance of the neck to loading is limited. Of necessity, all volunteer data are below the injury threshold. Additionally, injury mechanisms can be quite different from those mechanisms controlling response. Most injury threshold data are either based on cadaver tests or on reconstructions of accidents with instrumented dummies. As such, the threshold values are subject to the limitations associated with the surrogate used to obtain the data. These data sources have been used to develop tolerance limits for neck bending moments in midsagittal flexion and extension, axial compressive and tensile neck forces, and neck shear forces (Mertz, 1984; Mertz and Patrick, 1971; Mertz *et al.*, 1978; Yamada, 1970). The tolerance of the neck to various combinations of forces and moments, however, has not been established.

The current technology to assess neck injury is to install a multi-axis force and moment transducer at the base of the dummy head. The reaction loads measured are then used individually as indicators of injury severity. The validity of such measures, however, depends on the realism of the head/neck/torso-kinematics. Early dummy necks were merely a column of rubber, but human necks consist of a series of joints. Jointed necks have now been developed to achieve humanlike range-of-motion characteristics (Foster *et al.*, 1977), but, unlike humans, current dummies still have rigid thoracic spines. This lack of torso flexibility affects the response of the neck to impact situations. Without adequate simulations of body (spinal) motion, measurements from the most sophisticated instrumentation may yield inadequate results.

THORACIC AND ABDOMINAL INJURY

Impact to the sternum compresses the rib cage and accelerates the thorax in the direction of the force (Committee on Trauma Research, 1985; Viano, 1983) (Fig. 4). With sufficient compression, ribs can fracture from tensile strain on their outer surface. As the sternum is pushed inward, the heart is compressed and is displaced from its normal position in the thoracic cavity. This motion of the heart can stretch the aorta at the isthmus from its posterior points of tethering. Axial stretch in the aorta causes tensile strain that may result in transverse rupture (Besson and Saegesser, 1983; Viano, 1983). In this case, injury to the aorta is the indirect result of a nonpenetrating thoracic impact, which generates a complex series of relative

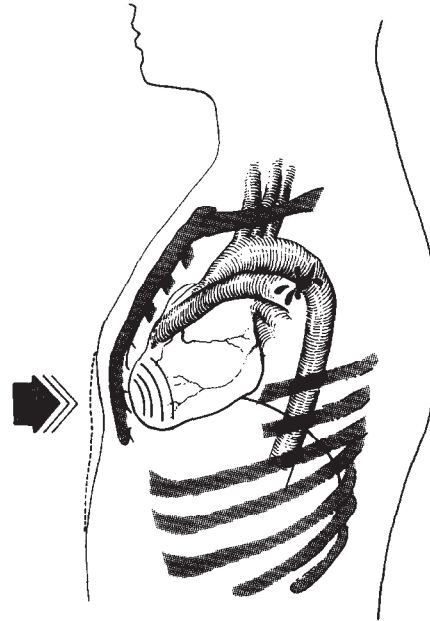


Fig. 4. Chest impact compresses the rib cage, displaces internal organs and may lacerate major vessels by crushing or stretching mechanisms. However, high-velocity impact of the chest can cause major internal organ injury with minimal compression by the viscous injury mechanism.

displacements of body tissues and structures that ultimately cause the non-recoverable axial strain. Compression of the thorax also increases the pressure in the lungs (Lau and Viano, 1981), chambers of the heart (Stein *et al.*, 1982) and aorta (Zehnder, 1960), which incrementally increases the strain in the tissues and adds to injury vulnerability. Although this mechanism of aortic rupture has been well described in clinical literature, there has been no experimental verification of this hypothesis or a tolerable level of compression proposed at this time.

Abdominal contents are even more vulnerable to injury than those in the thorax, because there is little bony structure below the rib cage to provide protection in frontal and lateral impacts. Blunt impact to the upper abdomen can compress and injure solid organs, such as the liver and kidneys, before significant whole-body motion or acceleration occurs (Horsch *et al.*, 1985; Lau and Viano, 1986; Lau *et al.*, 1987; Melvin *et al.*, 1973) (Fig. 5). In the case of the liver, compression increases intrahepatic pressure and generates tensile or shear strains. If the tissue is sufficiently deformed, laceration of the major hepatic vessels occurs, resulting in extensive hemoperitoneum. Abdominal deformation also causes the lobes of the liver to move relative to each other, stretching and shearing the vascular attachments at the hilar region (Lau and Viano, 1981b, c).

Although the most critical upper and lower torso injuries are to the internal organs, most experimental studies using cadavers have assessed thoracic injury in

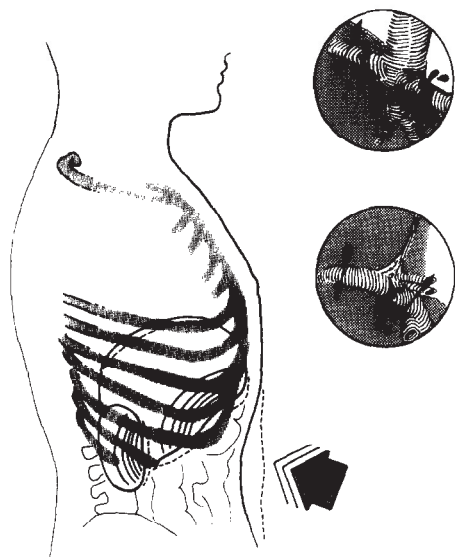


Fig. 5. High-speed impact of the abdomen can injure hepatic tissue without significant abdominal compression or whole-body motion. The liver and spleen are also vulnerable to crushing deformation, and relative motion of the liver and its attachments can rupture vessels.

terms of skeletal damage. Tolerance criteria based on impact force or chest compression, in addition to the traditional acceleration criterion, have thus been suggested. For lower speed impacts, these criteria may be useful. For higher velocity impacts, however, in which certain internal organ injuries have been found to occur prior to significant chest compression (Lau and Viano, 1986; Viano and Lau, 1983, 1985, 1988), a further factor of impact velocity must be considered. The viscous nature of biological tissue and thus the rate-sensitivity of its response to impact loading has been described above, but the concept is particularly important in the assessment of thoracic and abdominal injury. A Viscous Tolerance Criterion (Viano and Lau, 1985, 1988; Lau and Viano, 1986), which takes both velocity and compression into account, provides an injury criterion that is directly related to the biomechanical factors causing injury and indicates that the body's tolerance to compression decreases as the speed of deformation increases.

Tolerance levels for the thorax have been established for each of the types of injury criteria indicated (Hess *et al.*, 1982; Lau and Viano, 1986, Mertz and Gadd, 1971; Neathery *et al.*, 1975; Patrick *et al.*, 1967; Viano, 1978; Viano and Lau, 1985, 1988). The abdomen has received less attention, but tolerance limits based on compression have been suggested (Lau *et al.*, 1987; Lau and Viano, 1981b, c; Melvin *et al.*, 1973). Dummy technology, however, lags somewhat behind the analytical capability. Rigid spines, steel ribs that do not break, a lack of any internal organ simulation, and a foam abdomen with no or minimal instrumentation make reliable injury assessment difficult. The human thorax and abdomen are complex three-

dimensional structures that require complex simulations to realistically respond to impact in all directions. Similarly, instrumentation must be able to make global response measurements to ensure an adequate capability to assess injury potential in impacts from different directions, with different shaped impactors, and at different loading rates.

LOWER EXTREMITY INJURIES

Impact to the knee along the axis of the femur compresses the patella against the soft tissues of the patellofemoral joint, loading and deforming the articular cartilage (Fig. 6). Increased force in the femoral condyles accelerates and deforms the structure. Deformation of the femur is increased by the inertial resistance of the mass of the lower torso, which resists displacement. Because of the curved shape of the femur, deformation increases this curvature through tensile strain on the anterior surface. If the tensile strain exceeds the limit of compact bone, a fracture can begin and rapidly propagate through the structure. Bending is thus the primary injury mechanism for the femur.

The frontal impact response of the upper leg during seated knee impacts has been studied extensively (Evans, 1981; Horsch and Patrick, 1976; Melvin and Stalnaker, 1976; Nahum and Melvin, 1985; Viano and Stalnaker, 1980). This research includes information on the acceleration-time histories, force-time histories, impedance and effective mass. Other studies have defined the geometry of engagement of the knee into crushable padding (Hering and Patrick, 1977; SAE, 1986), and load-deflection data are also available for subluxation of the tibia with respect to the knee joint (Viano *et al.*, 1978).

For injury assessment purposes, femoral response and tolerance limits are generally defined in terms of the force sustained by the femur when it is impacted axially at the knee. This is an indirect response measure since the failure mechanism is due to bending of the bone from knee impact. Measurement of knee impact load, however, can be made with much less effort than that of femoral bending. More research is needed before long-bone tolerance to fracture can be based on parameters directly responsible for its failure.

The current generation of test dummies are instrumented with a femoral load cell that measures the axial force in the dummy upper leg (Foster *et al.*, 1977). More sophisticated load cells are used in dummy femurs (Cheng *et al.*, 1984) for research purposes but are not generally used in the laboratory evaluation of occupant protection technology.

GAPS IN CURRENT BIOMECHANICAL KNOWLEDGE

The status of current knowledge about injury mechanisms, the understanding and quantification of biomechanical response and tolerance to impact, and the

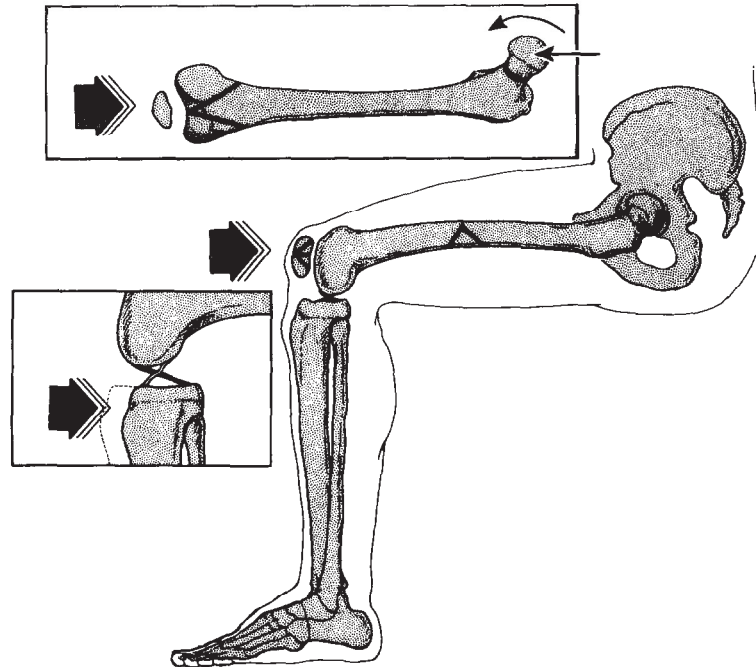


Fig. 6. Knee impact bends the femur with possible midshaft fracture due to tensile strain exceeding tolerance at the midsection. Rigid or sharp contacts may fracture the patella and supracondylar fractures can occur by shearing due to loading through the patella. Load transfer to the pelvis can shear the femoral neck or dislocate the hip, and load transfer to the tibia may injure the posterior cruciate ligament.

availability of meaningful injury evaluation criteria and technology are summarized in Table 2. Although the head and spine house the organs that control life itself, and there is a reasonable understanding of the processes that generate structural injury, very little is known about the mechanisms of functional damage to the brain and spinal cord. Likewise, the mechanical parameters associated with anatomical alterations of thoracic organs are relatively clear, but the disruption of cardiac function after thoracic impact, without apparent structural damage, is only now being determined (King and Viano, 1986; Lau 1985a, b). In order to be able to protect the body from functional impairments, there must be a better understanding of the electro-mechanical changes associated with impact and/or tissue damage.

Research to improve our understanding of, and ability to quantify, biomechanical response to impact, as well as to establish impact tolerance thresholds, has always been handicapped by the obvious necessity of using human surrogates in impact experiments, except at very low severity levels. Human cadavers have provided a reasonable experimental model for skeletal response, especially for the skull, rib cage, and femur, and cadaver research continues to emphasize the response characteristics of the thorax and lower extremities. These studies are supplemented by the use of anesthetized animals for thoracic and abdominal impact experiments, the results of which can be scaled to human dimensions. There is an urgent need, however, to quantify head/neck impact response, because of the

high incidence of brain and spinal injury in motor vehicle crashes and the serious and usually irreversible consequences of brain and spinal cord damage.

In order to develop meaningful measures of the risk of damage to central nervous tissue, a physiological model must be used in experimental studies. The use of animals in biomedical research is encountering increasing opposition from special interest groups, some of whom have resorted to violent means to stop animal research. Guidelines for the ethical treatment of laboratory animals have been drawn up by the National Institutes of Health (Dept of Health Education & Welfare, 1978) and other Federal agencies to assure the highest standards of animal anesthesia, care, and use. The philosophical arguments regarding animal rights and the sanctity of any life form have not been settled, however. A committee of the National Academy of Sciences has concluded that the human cadaver is an essential surrogate for impact biomechanics research, but its use is limited to structural response studies. Animals are thus essential surrogates for the study of the physiological response to impact and injury, so that functional impairment can be understood, treated, and possibly prevented.

Test dummies are the primary tool for predicting injury, yet only a few measures of potential injury are assessed with current techniques during impact tests. The most common ones are the acceleration response of the head and chest and the forces on the femur. Although the measurement of femoral forces is well accepted and used, the mechanisms of femoral injury

Table 2. Status of knowledge in impact biomechanics

Body region	Injury mechanisms	Impact response	Impact tolerance	Assessment technologies
Head				
Skull	****	***	***	***
Face	***	**	**	**
Brain				
Structure	**	*	*	*
Function	**	*	*	*
Spine				
Vertebrae	**	**	*	**
Spinal cord				
Structure	**	*	*	*
Function	**	*	*	*
Thorax				
Rib cage	****	****	****	***
Heart, great vessels				
Structure	***	***	**	**
Function	***	**	**	**
Lungs	***	**	*	*
Abdomen				
Solid organs				
Hollow organs	**	**	*	*
Extremities				
Femur				
Femur	****	****	****	***
Other long bones				
Other long bones	***	**	**	**
Joints				
Joints	***	**	**	**
Muscle				
Muscle	**	*	*	*
Sensory organs				
Skin				
Skin	***	***	**	***
Other				
Other	***	**	**	*

* Unknown/unavailable.

** Hypothetical/inadequate.

*** Somewhat understood and verified/useful.

**** Well known/adequate.

is not axial compression, but rather bending. The head also has a well publicized criterion for evaluation of injury risk (HIC), but the criterion is based on limited experimental verification and has not been correlated with the risk of brain damage. Some experimental data are available for the evaluation of neck injury, but they do not assess functional changes associated with risk of paralysis. Clearly, the two most significant body regions, the head and neck, are not adequately evaluated using current testing technology, yet these are the regions most frequently and severely injured in motor vehicle crashes.

CONCLUSION

As a science, injury biomechanics research is in its infancy. There are large gaps in experimental data, and reliable test techniques have not been developed for important areas of interest. There are also few trained scientists and engineers with background and experience in the mechanics and physiology of trauma, and few universities have developed faculty and curricula to produce new researchers in the field. The recent

establishment of five 'Centers of Excellence' by the U.S. Center for Disease Control is an encouraging step, but it remains to be seen how research priorities will be set, whether the parent organization will be able to maintain an even distribution of engineers, physiologists, and physicians in both its intra- and extramural programs, and how it will achieve meaningful cooperation and exchange of information with existing Federal agencies. It is hoped that the research at these centers and at other universities funded by the program can be undertaken on a long-term basis and directed at an objective understanding of injury mechanisms and biomechanical response, as opposed to short-term research done in support of Federal regulation, which has occurred in the past.

Partially because of a lack of sufficient information on which to base the design of a humanlike dummy, the tools currently in use to assess injury risk are inadequate to indicate the wide diversity of potential anatomical and functional damage. In certain areas, however, there are better data on human impact response than are reflected by current dummies (Melvin *et al.*, 1985; Melvin and Weber, 1985). Some

significant improvements in kinematics and response could therefore be made fairly soon if dummy technology were brought up to date. On the longer term, however, efforts to improve injury assessment capabilities should be consistent with a national research priority to address the most critical injuries, which are functionally disabling injuries to the brain and spinal cord, and to develop ways of preventing, as well as reversing, these injuries.

REFERENCES

- Adams, D. B. (1982) Wound ballistics: a review. *Milit. Med.* **147**, 831–834.
- American Trauma Society (1982) *The Need for a National Trauma Institute*. The American Trauma Society.
- Anderson, T. E. (1985) Spinal cord contusion injury: experimental dissociation of hemorrhagic necrosis and subacute loss of axonal conduction. *J. Neurosurg.* **62**, 115–119.
- Anderson, T. E. and Viano, D. C. (1986) Effect of acute alcohol intoxication on injury tolerance and outcomes. *Proceedings of the 10th International Conference on Alcohol, Drugs and Traffic Safety*. Elsevier, Amsterdam.
- Anderson, T. E. (1986) Effects of acute alcohol intoxication on spinal cord vascular injury. *Cent. Nerv. Syst. Trauma* **3**, 183–192.
- Baker, S. P., O'Neill, B. and Karpf, R. S. (1984) *The Injury Fact Book*. Lexington Books, DC Heath and Company, MA.
- Begeman, P. C. and King, A. I. (1975) The effect of variable load energy absorbance on the biodynamic response of cadavers. 19th Stapp Car Crash Conference, SAE Technical Paper 751168, P-62, pp. 787–806.
- Begeman, P., Levine, R., King, A. and Viano, D. (1980) Biodynamic response of the musculoskeletal system to impact acceleration. SAE Transactions, Vol. 89, 24th Stapp Car Crash Conference, SAE Technical Paper 801312, pp. 477–510, October.
- Besson, A. and Saegesser, F. (1983) *Color Atlas of Chest Trauma and Associated Injuries*, Vol. 1 and 2. Medical Ergonomics Books, Oradell, NJ.
- Blizard, J. R. and Howitt, J. S. (1969) Development of a safer nonlacerating automobile windshield. SAE Technical Paper 690484, May.
- Bowman, B. M., Schneider, L. W., Lustick, L. S., Anderson, W. R. and Thomas, D. J. (1984) Simulation analysis of head and neck dynamic response. 28th Stapp Car Crash Conference, SAE Technical Paper 841668, P-84, pp. 173–194, November.
- Cheng, R., Yang, K.-H., Levine, R. S. and King, A. I. (1984) Dynamic impact loading of the femur under passive restrained condition. 28th Stapp Car Crash Conference, SAE Technical Paper 841661, P-84, pp. 101–118, November.
- Collins, W. F. (1983) A review and update of experiment and clinical studies of spinal cord injury. *Paraplegia* **21**, 204–219.
- Committee on Trauma Research, Commission on Life Sciences, National Research Council and the Institute of Medicine (1985) *Injury in America—A Continuing Public Health Problem*. National Academy Press, Washington, DC, May.
- Committee on Trauma and Committee on Shock, Division of Medical Services, National Academy of Sciences, National Research Council (1966) *Accidental Death and Disability: The Neglected Disease of Modern Society*. National Academy of Science, National Research Council, Washington, DC.
- Council on Scientific Affairs (1983) Automobile-related injuries—Council on Scientific Affairs Report. *JAMA* **249**, 3216–3222.
- Courville, C. B. (1950) The mechanisms of coup–contrecoup injuries of the brain. *Bull. Los Angeles Neurol. Soc.* **15**, 72–86.
- DeHaven, H. (1969) Beginnings of crash injury research. 13th Stapp Car Crash Conference, pp. 422–428, Society for Automotive Engineers, December.
- Department of Health, Education, and Welfare (1978) *Guide for the Care and Use of Laboratory Animals*. Prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council. U.S. Department of Health, Education, and Welfare DHEW Publication No. (NIH) 78–23.
- Department of Transportation, National Highway Traffic Safety Administration (1981) *Head and Neck Injury Criteria—A Consensus Workshop*. DOT-HS 806 434, Washington, DC, March 26–27.
- Desiderio, M. A. (1987) Effects of acute, oral ethanol on cardiovascular performance before and after experimental blunt cardiac trauma. *J. Trauma* **27**, 267–277.
- DiMaio, V. J. (1981) Penetration and perforation of skin by bullets and missiles. *Am. J. Forensic Med. Pathol.* **2**, 107–110.
- Epstein, H. (1973) Traumatic dislocations of the hip. *Clin. Orthop.* **92**, 116–141.
- Evans, F. G. (1981) *A Review of Some Pioneering American Research in Biomechanics*. 1981 Advances in Bioengineering—Winter Annual Meeting of the American Society of Mechanical Engineers, Washington, DC, November.
- Ewing, C. L., Thomax, D. J., Lustick, L. and Muzzy, W. (1978) Effect of initial position on the human head and neck response to +Y impact acceleration. 22nd Stapp Car Crash Conference, SAE Technical Paper 780888, P-77, pp. 101–138, October.
- Flamm, E. S., Demopoulos, H. B., Seligman, M. L., Tomasula, J. J., Decrescito, V. and Ransohoff, J. (1977) Ethanol potentiation of central nervous system trauma. *J. Neurosurg.* **46**, 328–335.
- Foster, J. K., Kortge, J. O. and Wolanin, M. J. (1977) Hybrid III—A biomechanically-based crash test dummy. 21st Stapp Car Crash Conference. SAE Technical Paper 770938, P-73, pp. 973–1014, October.
- Foust, D. R., Bowman, B. M. and Snyder, R. G. (1977) Study of human impact tolerance using investigations and simulations of free-falls. 21st Stapp Car Crash Conference, SAE Technical Paper 770915, P-73, pp. 3–59.
- Frankel, V. H. and Nordin, M. (1980) *Basic Biomechanics of the Skeletal System*. Lea & Febiger, Philadelphia, PA.
- Fung, Y. C. (1981) *Biomechanics—Mechanical Properties of Living Tissues*. Springer, Berlin.
- Fung, Y. C., Perrone, N. and Anliker, M. (Eds) (1972) *Biomechanics—Its Foundations and Objectives*. Prentice-Hall, Englewood Cliffs, NJ.
- Gadd, C. W. (1966) Use of a weighted impulse criterion for estimating injury hazard. 10th Stapp Car Crash Conference, SAE Technical Paper 660793, pp. 164–174, November.
- Gadd, C. W., Nahum, A., Schneider, D. and Madeira, R. (1970) Tolerance and properties of superficial soft tissues *in situ*. 14th Stapp Car Crash Conference, SAE Technical Paper 700910, pp. 356–368.
- Gennarelli, T. (1982) Influence of the type of intracranial lesion on outcome from severe head trauma. *J. Neurosurg.* **56**, 26–32.
- Gennarelli, T. A. and Thibault, L. E. (1982) Biomechanics of acute subdural hematoma. *J. Trauma* **22**, 680–686.
- Gennarelli, T. A., Thibault, L. E., Adams, J. H. and Graham, D. I. (1982) Diffuse axonal injury and traumatic coma in the primate. *Ann. Neurol.* **12**, 564–574.
- Ghista, G. N. (1982) *Human Body Dynamics: Impact, Occupational, and Athletic Aspects*. Clarendon Press, Oxford.

- Gurdjian, E. S. (1975) *Impact Head Injury*. Charles C. Thomas, Springfield, IL.
- Gurdjian, E. S. and Gurdjian, Edwin S. (1975) Re-evaluation of the biomechanics of blunt impact injury of the head. *Surg. Gynecol. Obstet.* **140**, 845–850.
- Gurdjian, E. S. and Gurdjian, Edwin, S. (1976) Cerebral contusions: reevaluations of the mechanism of their development. *J. Trauma*, **16**, 35–51.
- Hartunjan, N. S., Smart, C. N. and Thompson, M. S. (1981) *The Incidence and Economic Costs of Major Health Impairments*. An Insurance Institute for Highway Safety Book, Lexington Books, D.C. Heath & Co. Lexington, MA.
- Hering, W. E. and Patrick, L. M. (1977) Response comparison of the human cadaver knee and a Part 572 dummy knee to impacts by crushable materials. 21st Stapp Car Crash Conference, SAE Technical Paper 770939, P-73, pp. 1015–1053.
- Hess, R. L., Weber, K. and Melvin, J. W. (1980) Review of literature and regulation relating to head impact tolerance and injury criteria. University of Michigan Highway Safety Research Institute, UM-HSRI-80-52-1, July.
- Hess, R., Weber, K. and Melvin, J. W. (1982) Review of research on thoracic impact tolerance and injury criteria related to occupant protection. SAE Technical Paper 820480, Warrendale, PA.
- Hodgson, V. R. (1967) Tolerance of the facial bones to impact. *Am. J. Anat.* **120**, 113–122.
- Holdsworth, F. W. (1963) Fractures, dislocations, and fracture-dislocations of the spine. *J. Bone Jt Surg.* **45B**, 6–20.
- Holdsworth, F. W. (1970) Fractures, dislocations, and fracture-dislocations of the spine. *J. Bone Jt Surg.* **52A**, 1534–1551.
- Horsch, J. D. and Patrick, L. M. (1976) Cadaver and dummy knee impact response. SAE Technical Paper 760799, Warrendale, PA.
- Horsch, J. D., Lau, I. V., Viano, D. C. and Andrzejak, D. V. (1985) Mechanism of abdominal injury by steering wheel loading. 29th Stapp Car Crash Conference, SAE Technical Paper 851724, P-167, pp. 69–78.
- Hubbard, R. P. and McLeod, D. G. (1974) Definition and development of a crash dummy head. 18th Stapp Car Crash Conference, SAE Technical Paper 741193, pp. 599–628.
- Journal of American Insurance (1983) Staggering costs of serious auto crashes continues. *J. Am. Ins.* **59**, 10–13.
- Kazarian, L. (1981) Injuries of the human spinal column: biomechanics and injury classification. *Exer. Sport Sci. Rev.* **9**, 297–352.
- Khalil, T. B. and Viano, D. C. (1982) Critical issues in finite element modeling of head impact. 26th Stapp Car Crash Conference, SAE Technical Paper 821150, P-113, pp. 87–102.
- King, A. I. (1984) The spine: its anatomy, kinematics, injury mechanisms and tolerance to impact. *The Biomechanics of Impact Trauma* (Edited by Chapon, A. and Aldman, B.), pp. 191–226. Elsevier, Amsterdam.
- King, A. I. and Chou, C. C. (1976) Mathematical modelling, simulation and experimental testing of biomechanical system crash response. *J. Biomechanics* **9**, 301–317.
- King, A. I. and Viano, D. C. (1986) Baseball related chest impact. U.S. Consumer Product Safety Commission, Contract CPSC-P-84-1170, Washington, DC.
- Kraus, J. F., Black, M. A. and Hessol, N. (1984) The incidence of acute brain injury and serious impairment in a defined population. *Am. J. Epidemiol.* **119**, 186–201.
- Kroell, C. K., Gadd, C. W. and Schneider, D. C. (1973) Biomechanics in crash injury research. Nineteenth International ISA Aerospace Instrumentation Symposium, Las Vegas, NV, May.
- Langfitt, T. N., Gennarelli, T. A. and Obrist, W. D. (1982) Prospects for the future in diagnosis and management of head injury. *Clin. Neurosurg.* **29**, 353–376.
- Lau, I. V. (1985a) Characterization of impact-induced tachyarrhythmias utilizing propranolol, quinidine and pacing. *J. Electrocardiology* **18**, 151–156.
- Lau, I. V. (1985b) Traumatic heart block and tachyarrhythmias induced by right ventricular impact. *J. Electrocardiology* **18**, 141–150.
- Lau, V. K. and Viano, D. C. (1981a) Influence of impact velocity and chest compression on experimental pulmonary injury severity in rabbits. *J. Trauma* **21**, 1022–1028.
- Lau, V. K. and Viano, D. C. (1981b) An experimental study on hepatic injury from belt-restraint loading. *Avia. Space Environ. Med.* **52**, 611–617.
- Lau, V. K. and Viano, D. C. (1981c) Influence of impact velocity on the severity of nonpenetrating hepatic injury. *J. Trauma* **21**, 115–123.
- Lau, I. V. and Viano, D. C. (1986) The viscous criterion—bases and applications of an injury severity index for soft tissues. 30th Stapp Car Crash Conference, SAE Technical Paper 861882, P-189, pp. 123–142.
- Lau, I. V., Horsch, J. D., Andrzejak, D. V. and Viano, D. C. (1987) Biomechanics of liver injury by steering wheel loading. *J. Trauma* **27**, 225–235.
- Liedtke, A. and Demuth, W. (1973) Nonpenetrating cardiac injuries: a collective review. *Am. Heart J.* **86**, 687–697.
- Lissner, H. R., Lebow, M. and Evans, F. G. (1960) Experimental studies on the relation between acceleration and intracranial pressure changes in man. *Surg. Gynecol. Obstet.* **111**, 329–338.
- Luchter, S. (1986) Traffic related disabilities and impairments and their economic consequences. *Crash Injury Impairment and Disability: Long Term Effects*. SAE Technical Paper 860505, SP-661, pp. 93–114.
- Maltha, J. and Stalnaker, R. L. (1981) Development of a dummy abdomen capable of injury detection in side impacts. 25th Stapp Car Crash Conference, SAE Technical Paper 811019, P-97, pp. 651–684.
- Melvin, J. W. and Stalnaker, R. L. (1976) Tolerance and response of the knee-femur-pelvis complex to axial impact. Report No. UM-HSRI-76-33. The University of Michigan, Highway Safety Research Institute, Ann Arbor, Michigan, 1976.
- Melvin, J. W. and Weber, K. (Eds) (1985) *Review of Biomechanical Impact Response and Injury in the Automotive Environment*. National Highway Traffic Safety Administration, Department of Transportation, Washington, DC 20590.
- Melvin, J. W., Stalnaker, R. L., Roberts, V. L. and Trollope, M. L. (1973) Impact injury mechanism in abdominal organs. 17th Stapp Car Crash Conference, SAE Technical Paper 730968, pp. 115–126.
- Melvin, J. W., King, I. A. and Alem, N. M. (1985) AATD system technical characteristics, design concepts, and trauma assessment criteria. National Highway Traffic Safety Administration, Department of Transportation, Washington, DC 20590. Report No. DOT HS 807 224.
- Mertz, H. J. (1984) Injury assessment values used to evaluate Hybrid III response measurements. General Motors Corporation NHTSA Docket Submission VSG 2284 Part III. Attachment I, Enclosure 2.
- Mertz, H. J. (1985) Anthropomorphic models. *The Biomechanics of Trauma* (Edited by Nahum, A. M. and Melvin, J. W.), pp. 31–61. Appleton-Century Crofts, Norwalk, CT.
- Mertz, H. J. and Gadd, C. W. (1971) Thoracic tolerance to whole-body deceleration. 15th Stapp Car Crash Conference, SAE Technical Paper 710852, pp. 135–157.
- Mertz, H. J., and Patrick, L. M. (1967) Investigation of the kinematics and kinetics of whiplash. 11th Stapp Car Crash Conference, SAE Technical Paper 670919, pp. 267–317.
- Mertz, H. J. and Patrick, L. M. (1971) Strength and response of the human neck. 15th Stapp Car Crash Conference, SAE Technical Paper 710855, pp. 207–255, November.
- Mertz, H. J., Hodgson, V. R., Thomas, L. M. and Nyquist, G. W. (1978) An assessment of compressive neck loads under

- injury-producing conditions. *Phys. Sports Med.* 6, 95–106.
- Mertz, H. J., Neathery, R. F. and Culver, C. C. (1973) Performance requirements and characteristics of mechanical necks. *Human Impact Response: Measurement and Simulation* (Edited by King, W. F. and Mertz, H. J.), pp. 263–288. Plenum Press, NY.
- Mohan, D. and Melvin, J. W. (1982) Failure properties of passive human aortic tissue. I—Uniaxial tension tests. *J. Biomechanics* 15, 887–902.
- Mohan, D. and Melvin, J. W. (1983) Failure properties of passive human aortic tissue. II-Biaxial tension tests. *J. Biomechanics* 16, 31–44.
- Morse, T. S. (1973) The American Trauma Society—lessons learned from cancer and coronaries. *Surgery* 73, 806–809.
- Nahum, A. M. and Melvin, J. W. (Eds) (1985) *The Biomechanics of Trauma*. Appleton-Century-Crofts, Norwalk, CT.
- National Institute of Neurological and Communicative Disorders and Stroke (1980) *The National Head and Spinal Cord Injury Survey*. National Institute of Neurological and Communicative Disorders and Stroke, *J. Neurosurg.* (Suppl.).
- National Safety Council (1983) *Accident Facts*. National Safety Council: Safety in the Transitional, pp. 8–9.
- Neathery, R. F. (1974) An analysis of chest impact response data and scaled performance recommendations. 18th Stapp Car Crash Conference, SAE Technical Paper 741188, pp. 451–466.
- Neathery, R. F., Kroell, C. K. and Mertz, H. J. (1975) Prediction of thoracic injury from dummy responses. 19th Stapp Car Crash Conference, SAE Technical Paper 751151, P-62, pp. 295–316.
- Noyes, R. and Grood, E. S. (1976) The strength of the anterior cruciate ligament in humans and rhesus monkeys. *J. Bone Jt Surg.* 58A, 1074–1082.
- Office of the Assistant Secretary for Health and Surgeon General (1979) *Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention*. Government Printing Office, DHEW Publication No. (PHS) 79-55071.
- Ommaya, A. K. (1985) Biomechanics of head injuries: experimental aspects. *The Biomechanics of Trauma* (Edited by Nahum, A. M. and Melvin, J. W.), Chapter 13, pp. 245–271. Appleton-Century-Crofts, East Norwalk, CT.
- Ommaya, A., Grubb, R. L. and Naumann, R. A. (1971) Coup and contrecoup injury: observations on the mechanics of visible brain injuries in the rhesus monkey. *J. Neurosurg.* 35, 503–516.
- Ono, K., Kikuchi, A. and Nakamura, M. (1980) Human head tolerance to sagittal impact reliable estimation deduced from experimental head injury using subhuman primates and human cadaver skulls. 24th Stapp Car Crash Conference, SAE Technical Paper 801303, P-88, pp. 101–160.
- Patrick, L., Kroell, C. K. and Mertz, H. J. (1965) Forces on the human body in simulated crashes. 9th Stapp Car Crash Conference, SAE Technical Paper, pp. 237–260.
- Patrick, L. M. and Chou, C. (1976) Response of the human neck in flexion extension, and lateral flexion. Vehicle Research Institute Report No. VRI-7-3. SAE Technical Paper.
- Patrick, L. M. and Daniel, R. P. (1964) Comparison of standard and experimental windshields. 8th Stapp Car Crash Conference, SAE Technical Paper, pp. 147–166.
- Patrick, L. M., Mertz, H. J. and Kroell, C. K. (1967) Cadaver knee, chest, and head impact loads. 11th Stapp Car Crash Conference, SAE Technical Paper 670913, pp. 168–182.
- Perloff, J. D., LeBailey, S. A. and Kletke, P. R. (1984) Premature death in the United States: years of life lost and health priorities. *J. Public Health Policy* 167, 183.
- Pickard, J., Brereton, P. A. and Hewson, A. (1973) An objective method of assessing laceration damage to simulated facial tissue. 17th Conference of the American Association of Automotive Medicine, pp. 148–165.
- Portnoy, H. D., McElhaney, J. H., Melvin, J. W. and Croissant, P. D. (1971) Mechanism of cervical spine injury in auto accidents. 15th Conference of the American Association for Automotive Medicine, pp. 58–83. Colorado Springs, CO.
- Prasad, P. R. and Mertz, H. (1985) The position of the United States delegation to the ISO working group 6 on the use of HIC in the automotive environment. SAE Technical Paper 851246.
- Reiser, R. G. and Chabal, J. (1969) Laboratory studies on laminated safety glass and installations on performance. 13th Stapp Car Crash Conference, SAE Technical Paper 690799, pp. 76–116.
- Reul, H., Ghista, D. N. and Rau, G. T. (Eds) (1978) *Perspectives in Biomechanics* Vol. 1 and 2. Harwood Academic Publishers.
- Rimel, R., Giordani, B., Barth, J. and Jane, J. A. (1981) Disability caused by minor head injury. *Neurosurgery* 9, 221–228.
- Rimel, R., Giordani, B., Barth, J. *et al.* (1982) Moderate head injury: completing the clinical spectrum of brain trauma. *Neurosurgery* 1, 344–351.
- Rosenberg, M. L., Gelle, R. J., Holinger, P. C., Zahn, M. A., Conn, J. M., Fajman, N. N. and Karison, T. A. (1984) Violence—homicide, assault, suicide. Position paper for the Carter Center of Emory University Health Policy Consultation, Atlanta, GA.
- Schneider, D. C. and Nahum, A. M. (1972) Impact studies of facial bones and skull. 16th Stapp Car Crash Conference, SAE-Technical Paper 720965, pp. 186–203.
- Schneider, L. W., Foust, D. R. and Bowman, B. (1975) Biomechanical properties of the human neck in lateral flexion. 19th Stapp Car Crash Conference, SAE Technical Paper 751156, P-62, pp. 455–586.
- Schneider, R. C. (1974) Cervical spine and spinal cord injuries. *Mich. Med.* 63, 773–786.
- Smith, G. S. and Coleman, P. (1984) Unintentional injuries: intervention strategies and their potential for reducing human losses. Position paper for the Carter Center of Emory University Health Policy Consultation, Atlanta, GA.
- Snyder, R. G. (1963) Human tolerance to extreme impacts in free fall. *Aerospace Med.* 34, 695–709.
- Society of Automotive Engineers (1980) *Human Tolerance to Impact Conditions as Related to Motor Vehicle Design*. SAE Handbook Supplement HSJ885.
- Society of Automotive Engineers (1982) *Crash Protection*. SAE Special Publication SP-513.
- Society of Automotive Engineers (1985a) *Field Accidents: Data Collection, Analysis, Methodologies, and Crash Injury Reconstructions*. SAE Publication P 159.
- Society of Automotive Engineers (1985b) *Mathematical Simulation of Occupant and Vehicle Kinematics*. SAE Special Publication P-146.
- Society of Automotive Engineers (1986) *Biomechanics and Medical Aspects of Lower Limb Injuries* (Edited by Viano, D. and Levine, R.) SAE Publication P-186.
- Stalnaker, R. L. and McElhaney, J. H. (1970) Head injury tolerance for linear impacts by mechanical impedance methods. ASME 70-WA/BHF-4. American Society of Mechanical Engineers.
- Stapp, J. P. (1970) Voluntary human tolerance levels. *Impact Injury and Crash Protection*. Charles C. Thomas, Springfield, IL.
- Stein, P. D., Sabbah, H., Viano, D. and Vostal, J. (1982) Response of the heart to nonpenetrating cardiac trauma. *J. Trauma* 22, 364–373.
- Stone, W. S. (1977) Trauma: a continuing U.S. health problem. *J. Trauma* 17, 89–92.
- Symbas, P. (1976) Fundamentals of clinical cardiology—cardiac trauma. *Am. Heart J.* 92, 387–396.
- Tarriere, C., Leung, Y. C., Fayon, A., Got, C., Patel, A. and Banzet, P. (1981) Field facial injuries and study of their

- simulation with dummy. 25th Stapp Car Crash Conference, SAE Technical Paper 811013, P-97, pp. 435-468.
- Toole, J. F. and Toole, W. W. (1984) Federal funding for research in stroke and trauma—a clinical investigator's viewpoint. *Stroke* **15**, 168-171.
- Trunkey, D. D. (1983) Trauma. *Sci. Am.* **249**, 28-35.
- United States Department of Transportation (1983) The economic cost to society of motor vehicle accidents. United States Department of Transportation, National Highway Traffic Safety Administration, DOT-HS-806-342.
- Versace, J. (1971) A review of the severity index. 15th Stapp Car Crash Conference, SAE Technical Paper 710881, pp. 771-796.
- Viano, D. C. (1977) Considerations for a femur injury criterion. 21st Stapp Car Crash Conference, SAE Technical Paper 770925, pp. 443-474.
- Viano, D. C. (1978) Thoracic injury potential. *Proceedings of the 3rd International Meeting on the Simulation and Reconstruction of Impacts in Collisions*, IRCOBI, pp. 142-156. INRETS, Lyon, France.
- Viano, D. C. (1980) Femoral impact response and fracture. *Proceedings of the 1980 International Conference on the Biokinetics of Impact*, IRCOBI, pp. 261-272, Birmingham, England.
- Viano, D. C. (1983) Biomechanics of non-penetrating aortic trauma: a review. 27th Stapp Car Crash Conference, SAE Technical Paper 831608, P-134, pp. 109-114.
- Viano, D. C. (1985) Perspectives on head injury research. 1985 *International Research Conference on the Biokinetics of Injury* IRCOBI Secretariat, Lyon, France.
- Viano, D. C. (1986) Biomechanics of bone and tissue: a review of material properties and failure characteristics. SAE Technical paper 861923, SAE Special Publication P-186, *Biomechanics and Medical Aspects of Lower Limb Injuries*, pp. 33-64.
- Viano, D. C. (1988a) Cause and control of automotive trauma. *Bull. N.Y. Acad. Med.* (2nd Ser.) **64**, 376-421.
- Viano, D. C. (1988b) Biomechanics of head injury: toward a theory linking head dynamics motion, brain tissue deformation and neural trauma. 32nd Stapp Car Crash Conference, SAE Technical Paper 881708, pp. 1-20.
- Viano, D. C. and Artinian, C. G. (1983) Myocardial conducting system dysfunctions from thoracic impact. *J. Trauma* **18**, 452-459.
- Viano, D. C. and Lau, I. V. (1985) Thoracic impact: a viscous tolerance criterion. *Proceedings of the 10th Experimental Safety Vehicle Conference*. Oxford, England.
- Viano, D. C. and Lau, I. V. (1988) A viscous tolerance criterion for soft tissue injury assessment. *J. Biomechanics* **21**, 387-399.
- Viano, D. C. and Lau, V. K. (1983) Role of impact velocity and chest compression in thoracic injury. *Avia. Space Environ. Med.* **54**, 16-21.
- Viano, D. C. and Stalnaker, R. L. (1980) Mechanisms of femoral fracture. *J. Biomechanics* **13**, 701-716.
- Viano, D. C., Culver, C. C., Haut, R. C., Melvin, J. W., Bender, M., Culver, R. H. and Levine, R. S. (1978) Bolster impacts of the knee and tibia of human cadavers and anthropomorphic dummy. 22nd Stapp Car Crash Conference, SAE Technical Paper 780896, pp. 401-428.
- Viano, D. C., Melvin, J. W., McCleary, J. D., Madeira, R. G., Shee, T. R. and Horsch, J. D. (1986) Measurement of head dynamics and facial contact forces in the Hybrid III dummy. 30th Stapp Car Crash Conference, SAE Technical Paper 861891, P-189, pp. 269-290.
- Von Gierke, H. E. and Brinkley, J. W. (1975) Impact accelerations. *Foundations of Space Biology and Medicine. Ecological and Physiological Bases of Space Biology and Medicine* (Edited by Calvin, M. and Gzenko, O. G.) Vol. II. Book 1, pp. 214-246, Washington, DC, National Aeronautics and Space Administration.
- Waller, J. (1984) *Injury Control: A Guide to Health and safety Professionals to the Causes and Prevention of Trauma*. Lexington Books, Lexington, MA.
- Waller, P. F., Stewart, R., Hansen, A. R., Stutts, J. C., Poplin, C. L. and Rudgman, E. A. (1986) The potentiating effects of alcohol on driver injury. *JAMA* **256**, 1461-1466.
- Wismans, J. and Spenny, C. H. (1983) Performance requirements for mechanical necks in lateral flexion. 27th Stapp Car Crash Conference, SAE Technical Paper 831613, P-134, pp. 137-148.
- Wismans, J. and Spenny, D. H. (1984) Head-neck response in frontal flexion. 28th Stapp Car Crash Conference, SAE Technical Paper 841666, P-152, pp. 161-171.
- Yamada, H. (1970) *Strength of Biological Materials* (Edited by Evans, F. G.), pp. 75-80. Williams & Wilkins, Baltimore, MD.
- Zehnder, M. A. (1960) Accident mechanism and accident mechanics of the aortic rupture in the closed thorax trauma. *Thoraxchirurgie und Vasculare Chirurgie* **8**, 47-65.