Narayan Yoganandan
Alan M. Nahum
John W. Melvin
Editors

Accidental Injury

Biomechanics and Prevention

Third Edition

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on behalf of Narayan Yoganandan
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Editors
Narayan Yoganandan, PhD, FAAAM, FAIMBE, FASME, FSAE
Chief Editor
Professor of Neurosurgery and Chair, Biomedical Engineering
Professor of Orthopaedic Surgery
Department of Neurosurgery
Medical College of Wisconsin and Zablocki Veterans Affairs Medical Center
Milwaukee, WI, USA

Adjunct Professor
Department of Biomedical Engineering
Marquette University
Milwaukee, WI, USA

John W. Melvin, PhD (Deceased)
President, Tandelta Inc.
Ann Arbor, Michigan, USA

Alan M. Nahum, MD, FACS
Professor Emeritus of Surgery
School of Medicine
University of California at San Diego
San Diego, CA, USA

The Medical College of Wisconsin Inc on behalf of Narayan Yoganandan
Milwaukee
Wisconsin, USA

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To John W. Melvin (1938–2014)

Foreword

This new edition of *Accidental Injury* is a reference and a milestone on the quest for preventing and/or reducing accidental human injuries and deaths associated with vehicular accidents. In many cases this information can be applied to non-vehicular accidental injuries. The mixture of contributors and subject matter is a testament to the comprehensive and multidisciplinary investigation necessary to fully understand and activate preventive measures.

Injury biomechanics has a long and colorful history with pioneers such as H. Yamada who performed laboratory impact experiments with post-mortem human anatomical specimens and Col. John Stapp who designed and performed acceleration experiments on himself.

Dr. William Haddon, the first head of the National Highway Traffic Safety Administration, enumerated the principles of public health policy which were essential to his mission. This included the need to prevent injury even if the accident could not be prevented. This has fostered the development of injury biomechanics. This area of investigation has required the compilation of precise statistical data on accidental injuries and deaths and the mechanisms and relevant circumstances surrounding the injuries. With this information in hand researchers have studied human tolerance using human surrogates, volunteers and computer simulations to explain more accurately the mechanism of injuries to the human body. This book summarizes current research and conclusions in many areas applicable to injury prevention and is essential knowledge for all those interested in reducing and eliminating many of the preventable deaths and injuries.

San Diego, CA, USA — Alan M. Nahum, MD, FACS

Preface

The book covers the biomechanical and prevention aspects of accidental injuries of the human body in 29 chapters. Research efforts focused on injuries, injury mechanisms and human tolerance are included. Experimental studies on all body regions including their anatomy; collection, analysis and scoring of injuries; computational modeling including finite element and stochastic techniques; motor vehicle safety standards; aviation studies; and ballistic environment research are presented.

Well-known multidisciplinary authors, ranging from practicing physicians in neurosurgery, orthopaedic surgery and trauma surgery, to biomedical engineers have contributed to this book. Many have attained unique statuses such as fellows of multiple scientific organizations including the American Institute of Medical and Biological Engineering, American Society of Mechanical Engineers, Association for the Advancement of Automotive Medicine and Society of Automotive Engineers; Presidents of International Societies; members of the National Academy of Engineers; and members of the Editorial Boards of scientific international clinical and bioengineering peer-review journals. Their collective experience of over 1,000 years, spanning from academia to industry to private organizations, are reflected in this book. I offer my sincere thanks for their timely contributions.

I would like to place on record my deep sense of appreciation and gratitude to all individuals who have helped shape my professional career. While I was a student at the Indian Institute of Science, Bangalore, India, I was fortunate to learn the "art" of conducting research under the guidance and supervision of Professor and former Deputy Director of the Institute, Asuri Sridharan, Ph.D. His mentoring during the budding days of my graduate studies generated lifelong curiosity to pursue academic research. Although his guidance was in structural mechanics and geotechnical engineering, principles continue to remain the same. I owe a debt of gratitude to my Professor.

The Department of Neurosurgery has been an intellectual home for me. I would like to thank Dennis J. Maiman, M.D., Ph.D., Chair, Department of Neurosurgery, Medical College of Wisconsin, for cultivating an excellent research atmosphere spanning decades and extending his unselfish support to researchers like me in our institutions. I thank Frank A. Pintar, PhD, Professor and Vice Chair of Neurosurgery Research in our department, my colleague and a trusted friend for over 30 years for all his valuable and timely suggestions. The encouragement and assistance of Brice Osinski, MBA,

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Jan Schiebenes, Administrative Assistant III, Department of Neurosurgery, Medical College of Wisconsin, deserves special acknowledgment for her tireless work that included weekends and "countless" e-mail communications. While electronic media has helped administrative and editorial aspects compared to my experiences with two books, an effort of this nature still demands diligence and perseverance. She took personal pride and always had a smile on her face in all her work. On behalf of all authors, I offer my heartfelt thanks.

I thank my wife Malini and daughter Asha for their understanding of this profession and help in bringing this work to fruition. My brother Prasad S. Narayan, sister-in-law Ashwini and nephew Nikil Prasad deserve a special note of thanks for their support and taking care of my parents and my family, especially during this period. My parents are always a source of inspiration and hard work, and I am fortunate to have their support to explore my academic curiosities and expand my knowledge. Blessings of the Lord, parents, teachers and individuals such as those mentioned above, are what I need.

Milwaukee, WI, USA

Narayan Yoganandan, PhD, FAAAM, FAIMBE, FASME, FSAE

Contents

Contributors

Kristy B. Arbogast, Ph.D. Children's Hospital of Philadelphia, University of Pennsylvania, Philadelphia, PA, USA

Mike W.J. Arun, Ph.D. Neuroscience Research Lab, Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA

Ann M. Bailey, B.S. Center for Applied Biomechanics, University of Virginia, Charlottesville, VA, USA

Jamie L. Baisden, M.D. Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA

Cameron R. Bass, Ph.D. Department of Biomedical Engineering, Duke University, Durham, NC, USA

Cynthia Bir, Ph.D. Keck School of Medicine, University of Southern California, Los Angeles, CA, USA

Karen Brasel, M.D., M.P.H. Medical College of Wisconsin, Milwaukee, WI, USA

Todd L. Bredbenner, Ph.D. Southwest Research Institute, San Antonio, TX, USA

John M. Cavanaugh, M.D. Department of Biomedical Engineering, Wayne State University, Detroit, MI, USA

Chaoyang Chen, M.D. Department of Biomedical Engineering, Wayne State University, Detroit, MI, USA

Dennis D. Chimich, Ph.D., P.Eng. MEA Forensic Engineers & Scientists, Richmond, BC, Canada

Jeff R. Crandall, Ph.D. Center for Applied Biomechanics, University of Virginia, Charlottesville, VA, USA

Clifford C. Chou, Ph.D. Department of Mechanical Engineering, Wayne State University, Detroit, MI, USA

Nathan D. Crosby, Ph.D. Department of Bioengineering, University of Pennsylvania, Philadelphia, PA, USA

Richard L. DeWeese, B.S. Federal Aviation Administration, Civil Aerospace Medical Institute, Oklahoma City, OK, USA

Benjamin S. Elkin, Ph.D. MEA Forensic Engineers & Scientists, Richmond, BC, Canada

Jason Forman, Ph.D. University of Virginia, Charlottesville, VA, USA

Rikard Fredriksson, Ph.D. Autoliv Research, Vargarda, Sweden

Hampton C. Gabler, Ph.D. Center for Injury Biomechanics, Virginia Tech-Wake Forest University, Blacksburg, VA, USA

Warren N. Hardy, Ph.D. Center for Injury Biomechanics, Virginia Tech – Wake Forest University, Blacksburg, VA, USA

Meghan K. Howes, Ph.D. Center for Injury Biomechanics, Wake Forest University, Blacksburg, VA, USA

Annette L. Irwin, Ph.D. General Motors Company, Troy, MI, USA

Srinivasu Kallakuri, Ph.D. Department of Biomedical Engineering, Wayne State University, Detroit, MI, USA

Andrew R. Kemper, Ph.D. Center for Injury Biomechanics, Virginia Tech – Wake Forest University, Blacksburg, VA, USA

Richard W. Kent, Ph.D. University of Virginia, Charlottesville, VA, USA

Albert I. King, Ph.D. Department of Biomedical Engineering, Wayne State University, Detroit, MI, USA

Kathleen D. Klinich, Ph.D. Transportation Research Institute, University of Michigan, Ann Arbor, MI, USA

W. Brent Lievers, Ph.D., P.Eng. Bharti School of Engineering, Laurentian University, Sudbury, ON, Canada

Dennis J. Maiman, M.D., Ph.D. Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA

Matthew R. Maltese, Ph.D. Children's Hospital of Philadelphia, University of Pennsylvania, Philadelphia, PA, USA

Miriam A. Manary, M.Eng. Transportation Research Institute, University of Michigan, Ann Arbor, MI, USA

David F. Meaney, Ph.D. Departments of Bioengineering and Neurosurgery, University of Pennsylvania, Philadelphia, PA, USA

John W. Melvin, Ph.D. Tandelta Inc., Ann Arbor, MI, USA

Harold J. Mertz, Ph.D. General Motors Corporation, Harper Woods, MI, USA

David M. Moorcroft, M.S. Federal Aviation Administration, Civil Aerospace Medical Institute, Oklahoma City, OK, USA

Barry S. Myers, M.D., Ph.D. Duke University, Durham, NC, USA

Alan M. Nahum, M.D., F.A.C.S. School of Medicine, University of California at San Diego, San Diego, CA, USA

James A. Newman, Ph.D. Newman Biomechanical Engineering Consulting Inc., Edmonton, AB, Canada

Daniel P. Nicolella, Ph.D. Southwest Research Institute, San Antonio, TX, USA

Roger Nightingale, Ph.D. Duke University, Durham, NC, USA

Joseph A. Pellettiere, Ph.D. Federal Aviation Administration, Washington, DC, USA

Jacob R. Peschman, M.D. Department of Surgery, Medical College of Wisconsin, Milwaukee, WI, USA

Audrey Petitjean, M.Sc. CEESAR, Nanterre, France

Frank A. Pintar, Ph.D. Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA

Priyaranjan Prasad, Ph.D. Prasad Consulting, LLC., Plymouth, MI, USA

Raj Rao, M.D. Department of Orthopaedic Surgery, Medical College of Wisconsin, Milwaukee, WI, USA

Stephen W. Rouhana, Ph.D. Ford Motor Company, Dearborn, MI, USA

Jonathan D. Rupp, Ph.D. University of Michigan Transportation Research Institute and the Departments of Emergency Medicine and Biomedical Engineering, University of Michigan, Ann Arbor, MI, USA

Robert S. Salzar, Ph.D. Center for Applied Biomechanics, University of Virginia, Charlottesville, VA, USA

Gunter P. Siegmund, Ph.D. Injury Biomechanics, MEA Forensic Engineers & Scientists, Richmond, BC, Canada

Ciaran Knut Simms, Ph.D. Department of Mechanical and Manufacturing Engineering, Trinity College, Dublin, Ireland

Jenell R. Smith, Ph.D. Department of Bioengineering, University of Pennsylvania, Philadelphia, PA, USA

Brian D. Stemper, Ph.D. Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA

Joel D. Stitzel, Ph.D. Biomedical Engineering, Wake Forest University, Center for Injury Biomechanics, Virginia Tech-Wake Forest University, Winston-Salem, NC, USA

Xavier Trosseille, Ph.D. LAB PSA Peugeot Citroen RENAULT, Nanterre, France

Mei Wang, Ph.D. Department of Orthopaedic Surgery Research, Medical College of Wisconsin, Milwaukee, WI, USA

Ashley A. Weaver, Ph.D. Biomedical Engineering, Wake Forest University, Center for Injury Biomechanics, Virginia Tech-Wake Forest University, Winston-Salem, NC, USA

Beth A. Winkelstein, Ph.D. Departments of Bioengineering and Neurosurgery, University of Pennsylvania, Philadelphia, PA, USA

Denis Wood, Ph.D. Denis Wood Associates, Dublin, Ireland

King H. Yang, Ph.D. Bioengineering Center, Wayne State University, Detroit, MI, USA

Narayan Yoganandan, Ph.D. Department of Neurosurgery, Medical College of Wisconsin, Milwaukee, WI, USA

Introduction to and Applications of Injury Biomechanics

1

Albert I. King

Abstract

The four aims of impact biomechanics are (1) Identification and explanation of injury mechanisms (2) Quantification of mechanical response of body components to impact (3) Determination of tolerance levels to impact and (4) Assessment of safety devices and techniques to evaluate prevention systems. These are briefly described followed by a discussion of the methods used to study injury biomechanics. The rest of the chapter is devoted to injury mechanisms which need to be understood if preventative measures are to be developed and implemented. The mechanisms covered are brain injury and acute subdural hematoma, neck pain due to whiplash, aortic rupture, spinal injury due to vertical acceleration, disc rupture, hip fracture in the elderly, ankle (pilon) fracture and foot fracture. It is concluded that much misinformation exists in the literature regarding these injuries and the statement in Lancet (Issue 9773): "The most entrenched conflict of interest in medicine is a disinclination to reverse a previous opinion" is indeed true.

1.1 Introduction

Injury biomechanics is a huge field and covers many areas of study, involving many parts of the human body. Obviously, it is not possible to cover the subject in a single chapter. This invitation by the Editors asking me to write the first chapter was for me to introduce the reader to this field of endeavor and to encourage him/her to seek out more detailed information from journal papers and books on this subject.

There is no question that we all want to avoid being injured. Severe injuries can be fatal or at least life changing. The fact that we are getting older is another reason why we need to be more careful in our activities, whether we are driving, walking, or even sitting. Generally, the main symptom of injury is pain which is a warning that something bad has happened and we should try to get out of that situation or not to repeat what we have been doing. Of course, when an impact

A.I. King, Ph.D. (✉)
Department of Biomedical Engineering, Wayne State University, Detroit, MI, USA
e-mail: king@rrb.eng.wayne.edu

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occurs, this is not possible. So we have to do the next best thing – to completely avoid the impact or to ameliorate the impact so that the injury would not be as severe. This brings up the question of injury prevention or injury mitigation. So the study of injury biomechanics boils down to a concerted effort to prevent injury.

Of course, there are many ways one can get injured. The two principal categories are intentional and unintentional injury. The former is difficult to prevent because there are so many ways someone can inflict injury on another person or oneself. However, unintentional injuries are preventable and, for biomechanicians, the focus of injury prevention is on unintentional injuries.

There are many forms of unintentional injuries. Perennially, motor vehicle crashes appear to be the leading cause of unintentional injury deaths [1]. In 2010, this was followed by unintentional poisoning and unintentional falls. Sports-related injuries do not rank high as one of the leading causes of nonfatal unintentional injuries in the US but are headline grabbers because of such issues as concussion in American football.

In the automotive safety arena, a very effective method of injury prevention is active safety which aims to avoid the crash. By the use of electronic sensors and other devices, the vehicle can be made to slow down or the driver can be warned to take evasive action to avoid the collision. This is not always possible if there is no time to react or the momentums of the vehicles involved is too large to overcome. An inebriated pedestrian stepping out from behind a parked truck just as a car is passing by would probably not benefit from any currently available active safety systems. Similarly, if a vehicle is suddenly and deliberately made to swerve and cross the centerline of a busy two-lane highway, it will most likely hit an oncoming vehicle before the active safety system of either vehicle can take effective action. That is, despite what the computer can do, crashes will still occur and passive safety in its many forms will still be necessary to protect the occupant. There are also opportunities to combine active and passive safety for more effective injury prevention. One such example on whiplash prevention is discussed in this chapter.

1.2 Definition of Injury Biomechanics

Biomechanics is the application of the principles of mechanics to biological systems and injury biomechanics is a subfield that studies the effect of mechanical impact on biological systems, in particular, the human body. Although the field is made up of a relatively small number of researchers and professionals, it was the first area of biomechanics to be studied. Gurdjian and Lissner [2] published the first paper on head injury in 1944 and initiated skull fracture mechanism research in 1939 at Wayne State University.

1.3 Motivation to Study Injury Biomechanics

The principal motivation to study injury biomechanics is, or at least, it should be that of saving lives and reducing suffering due to injury. Safety professionals have always said that injury is preventable if we only take the necessary steps to protect the body. For the elderly who tend to fall frequently, the obvious countermeasure is to instruct them to walk carefully and watch where they are going. Of course, this does not cover falls due to dizziness, heart and cardiovascular problems or fracture. In the case of automotive related injuries, the Federal government and the automotive industry have worked on improving automotive safety over the last half century. Traffic deaths as measured by the number of fatalities per 100,000 miles driven has been going down steadily with the introduction of belt restraints, padding of interior surfaces of cars and the airbag. These passive safety features are found in a modern vehicle. They constitute the use of environmental control to protect the occupant and do not need occupant participation with the exception of the wearing of the belt restraints. In the US, there are seat belt laws in place in all 50 states and the wearing rate has been going up steadily. Nationally, the average rate in 2010 was 85 % with the rate exceeding 90 % in 15 states and the District of Columbia [3]. Biomechanically, seat belt use is an essential component of passive safety and all occupants

need to be belted when traveling in cars, regardless of the length of the trip.

In sports, the motivation is to reduce brain and joint injuries, especially in contact sports. Much remains to be done in the area of concussion prevention in American football. The football helmet in use currently was designed to prevent skull fracture because the standards governing helmet performance were drawn up for that purpose. There have been many attempts to reduce the incidence of concussion but there is no concerted effort on the part of any national sports organization or the Federal government to initiate the design and fabrication of an anti-concussion helmet. In particular, a computer based design which takes into account the response of the human brain to impact would be advisable because the helmet can be hit in almost every direction and reliance on experimental impact data alone would not be adequate.

1.4 The Four Aims of Injury Biomechanics

Research in injury biomechanics evolved from ad hoc crude experiments and back of the envelope calculations to well organized studies funded by the Federal government and large corporations. The principal aims of this research are [4]:

1. Identification and explanation of injury mechanisms
2. Quantification of mechanical response of body components to impact
3. Determination of tolerance levels to impact
4. Assessment of safety devices and techniques to evaluate prevention systems

1.4.1 Identification and Explanation of Injury Mechanisms

As the saying goes, "you cannot prevent an injury unless you know the cause." In this case, it means the mechanism whereby the injury is caused. In the section below, several lesser known or somewhat controversial mechanisms are discussed. Some mechanisms are obvious. For example, if a bicyclist falls and hits his head on the ground, suffering a skull fracture, the mechanism is impact of the head with a hard surface and the method of prevention would be to wear a helmet. Other injury mechanisms are not immediately obvious and it is necessary to perform detailed studies to ensure that exact cause can be identified before a fix is introduced. A perfect example of this is the headrest mounted atop of automotive seat backs. The headrest was meant to prevent neck pain after a rearend collision (whiplash). It has been installed in cars sold in the US since 1986 but the whiplash problem did not go away. Several mechanisms of injury are discussed in Sect. 1.6 below.

1.4.2 Quantification of Mechanical Response

In engineering, the standard procedure for classifying a material is to define its mechanical response to an applied load. The same can be done for biological materials. For bone and, we can apply a compressive load and obtain its force –deflection response. Similarly, for ligaments, we can apply a tensile load and obtain the same response. However, for more complex body regions, such as the chest, we need to load it in different directions and locations to obtain its response to frontal and lateral loads for the upper, mid and lower thorax. For organs that are within the body, the task is more challenging, especially if we want to test it in situ. Accurate quantification of brain motion inside the skull due to a blunt impact was not achieved until beginning of this century [5].

Mechanical response can take many forms. The traditional engineering approach is to define the response in terms of stress versus strain or load versus deflection. However, for dynamic response, we can define a force-time response or a displacement-time response. Figure 1.1 shows the displacement response of the brain. Targets within the cadaveric brain make motions in the shape of a figure 8 when the head is impacted. The force–deflection response of the thorax is shown in Fig. 1.2. The response data are not as clean cut and predictable as with inanimate manufactured materials. Variations among individuals are large and are affected by age and gender. Reliability of the response data depends heavily

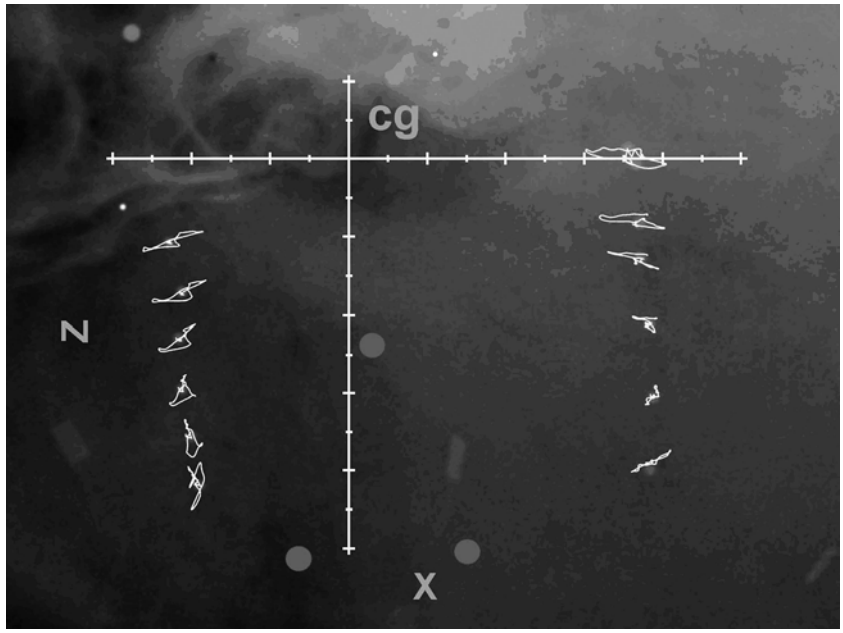

Fig. 1.1 Brain motion due to a blunt impact (Taken from Hardy et al. [5])

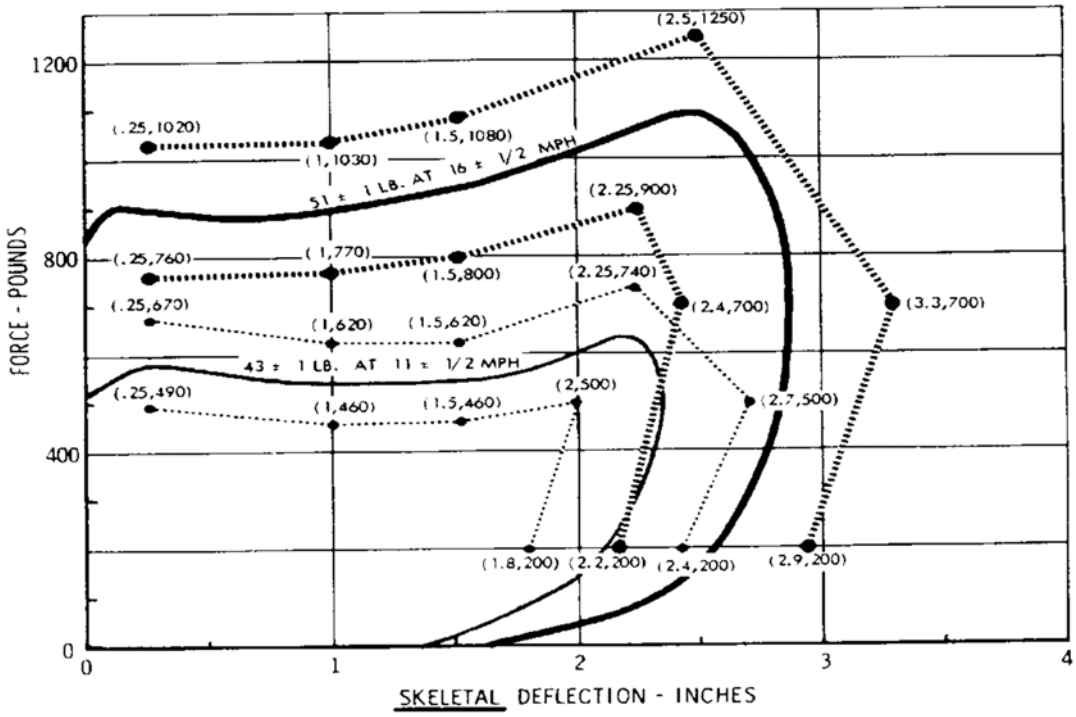

Fig. 1.2 Thoracic response to frontal impact. The two solid curves are for responses by two different impactors

Fig. 1.3 Photograph of a Hybrid III dummy. (Courtesy of Humanetics Innovative Solutions, Inc.

on the number of specimens tested, especially if a response corridor is to be drawn.

To design a human surrogate (crash dummy) for impact testing, a large amount of impact response data is needed so that the dummy can be as human-like as possible. The current dummy used universally by automakers is the Hybrid III. It is shown in Fig. 1.3. Not all mechanical responses of the Hybrid III are human-like because it is very difficult to have a device that is simultaneously biofidelic and behaves in a repeatable and reproducible manner during testing. In general, the dummy is stiffer than the human and its torso is less flexible.

1.4.3 Determination of Human Tolerance to Impact

In the design of safety systems for vehicles of any kind, it is necessary to know how much acceleration or force the body or any part of the body can take before serious injury is incurred. This is the study of human tolerance to impact. To the perennial question: How many g's can I take? The answer is another question: How badly do you want to get hurt? There are several levels of injury and the safety design can focus on one of those levels for a given impact severity. The injury levels are:

1. The "Ouch" level
2. Minor injury
3. Moderate injury
4. Serious injury
5. Critical injury
6. Fatal injury

The "Ouch" level is used when testing human volunteers. If the test causes pain in any way, the test should not proceed beyond that level and the volunteer can withdraw from the program. Minor and moderate injuries are real injuries which may require a visit to the emergency room but should not require prolonged hospitalization. At the serious injury level, the healthy individual will require hospitalization but the injuries are not life threatening. They may be for the elderly and the infirm. For a vehicle to be affordable, the design should result in injuries between the moderate and the serious. Critical injuries are life threatening to the healthy individual and should be avoided. Of course, no design should result in fatal injuries.

Like response, tolerance is also highly variable among subjects and is also dependent on age and gender. Because of this variability, absolute tolerance values are not very meaningful. Instead, a probabilistic approach is taken and, for a given level of injury, the tolerance values are expressed as probability of injury. For example, for head injury at a serious injury level, the probability of injury in terms of angular acceleration is shown Fig. 1.4. In this case, the angular acceleration for 50 % probability of a minor traumatic brain injury is 5,500 rad/s^2. Other parameters can be used as injury predictors, using this Logist analysis. Statistical parameters can be computed to determine which parameter is the best predictor of a particular injury.

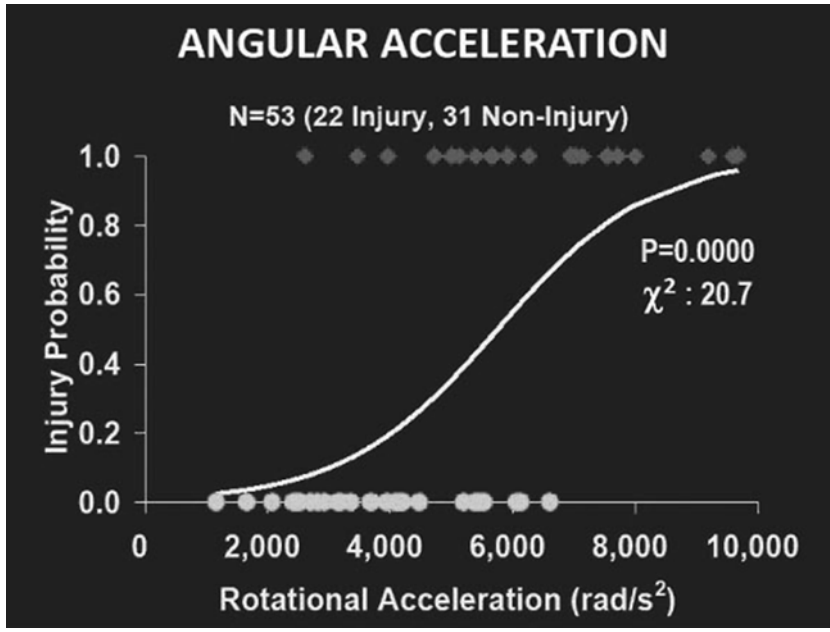

Fig. 1.4 Logist plot of mTBI versus rotational (angular) acceleration. For a 505 probability of injury the rotational acceleration is predicted to be approximately 5,500 rad/s^2

1.4.4 Assessment of Safety Devices and Techniques

The knowledge gained in the three areas of study described above can be used to assess safety devices in vehicles and other systems. The two principal tools are the crash dummy (Hybrid III) and computer models. For the last half century, most of the assessment has been experimental, using the crash dummy. This assessment is made as the design is progressing and calls for many crash tests that are costly and time consuming. The automotive industry has begun to realize that computer models are now sufficiently reliable to be used as an assessment tool. With the aid of models, the designer can skip much of the crash testing and do a final test of the system at the end of the development process. There is now a consortium of model developers, in the US, funded by the automotive manufacturers to develop a single total human body model that can be used in place of the Hybrid III. This is a lofty goal that may not be achievable because all kinds of models can be developed quickly and at low cost whereas a repeatable and reproducible crash dummy takes years of work to come to fruition. Nevertheless, this modeling effort is a laudable goal that will save the industry time and money.

1.5 Methods to Study Injury Biomechanics

Impact biomechanics began as an experimental discipline, much like many other fields of study. From observation, it progressed to testing and organized experimentation. The study began with injuries to particular body region and was gradually extended to the whole body. For example, Gurdjian and Lissner [6] concentrated their efforts on head injury and performed experiments on human skulls initially before venturing to test anesthetized animals. Evans and Lissner [7] also initiated studies on the thoracolumbar spine by testing embalmed whole cadavers on a specially designed vertical accelerator that was housed in an elevator shaft of an eight-story building. Mertz and Patrick [8] studied human neck response using a horizontal sled. Later on, the response and tolerance of the knee was also studied using horizontal sleds.

Mathematical models were developed in conjunction with the experimental studies, usually after some experimental data have become available to test the viability of the model. As computers became faster and capable of storing large amounts of data, the models became more complex and more detailed. A record of model development over half a century can be found in Yang et al. [9]. It goes without saying that most researchers have more faith in experimental data than in the predictions of mathematical or computer models. As a result, it is traditional to expect the model developers to validate their models against appropriate experimental data. The policy of the Stapp Crash Journal is that every model published in the journal must be validated. As a result, improvements in model predictions have been rapid and they are becoming reliable predictors of response and injury. In fact, because of the ready availability of models of many body regions, it is now possible to model the experiment and determine what can be expected to occur in an experiment, where to make the critical measurements and what levels of force or acceleration are to be expected in a given series of experiments. For example, in a current project on blast-related brain injury, the modelers are being asked to determine where to expect the maximum pressures in the brain and what pressure levels will be reached before any testing is done. These predictions will assist with the placement of pressure transducers in the brain to measure the peak pressures.

1.6 Injury Mechanisms from Head to Toe: Application to Design

1.6.1 Head Injury Mechanisms

1.6.1.1 Brain Injury Mechanisms

The existence of two injury mechanisms for the brain is well known. Gurdjian et al. [10] proposed that linear acceleration producing pressure waves within the brain was a mechanism for brain concussion. On the other hand, Ommaya et al. [11] were of the opinion that angular acceleration was the prime cause of concussion. This debate became quite heated for a time in the 1970s but has since died down somewhat when it became apparent that both forms of acceleration usually increased in a monotonic fashion with increased impact severity. However, the injury mechanisms due to these two forms of acceleration are quite different. Based on the work of Hardy et al. [5], we can say that angular acceleration is responsible for the relative motion of the brain within the skull during a blunt impact and that the resulting diffuse axonal injury is due to this motion. Although axonal stretch has not been measured directly, it can be deduced that this relative motion of the brain is essentially the mechanism that can stretch the axons. As for linear acceleration, it causes a pressure wave to be generated, starting as a compressive wave at the site of impact and becoming a reflected tensile wave as it is reflected from the skull at the opposite or contrecoup side. Gurdjian et al. [12] invented the fluid percussion method of causing concussion in dogs after they discovered the existence of a transient pressure wave traversing the brain. Fluid percussion tests that varied in duration from less than 1 ms to 46 ms and in magnitude from 34.5 to 345 kPa (5–50 psi), resulted in concussion in experimental animals. Unfortunately, no biomarkers were identified to describe the injury. Thus, we know that pressure is an injury mechanism but we do not know what cells are injured or what parts of the cell are damaged by pressure. Ongoing research to study the effects of blast overpressure on military personnel may soon reveal one or more mechanisms of injury at the cellular and/or the molecular level. Preliminary data on rodents [13] indicate that blast overpressure is causing the glial cells to go into apoptosis with possible deleterious effects on the neurons they support and that there was no axonal injury associated with blast exposure. This finding is reasonable in that there was very little head motion during a pure (primary) blast.

1.6.1.2 Injury Mechanisms for Acute Subdural Hematoma

The accepted injury mechanism for acute subdural hematoma (ASDH) is bridging vein rupture.

However, from an engineering point of view, the acute formation of a hematoma from a ruptured vein violates the principles of fluid mechanics. The mechanism proposed in this book chapter is a hypothesis with no data to support its veracity. The reader is asked to consider the logic of the hypothesis and decide if it has more merit than the accepted mechanism.

The physiopathogenesis of ASDH formation has been a subject of debate since the early thinking of an organized space between the arachnoid and dura was detailed by Key and Retzius in the late 1800s [14]. This group described the structures of the meninges and experimentally determined that substances injected into the presumed subdural space did not mix with other substances within the tissue. Early researchers believed that fluids within the alleged space could move between compartments of the brain [15]. Thus, authors of this time period believed and offered evidence that a fluid-filled space existed between the dura and arachnoid [15–19]. As Weed continued his studies, he determined that the structures were fused together in embryos, but could be separated in mature animals [19]. These early investigators injected fluids into the subdural area and visualize the distinctive compartmentalization of these fluids. Microscopically, layers of unique cells between the dura and arachnoid tissue were recognized and these cells were thought to produce a fluid which appeared to be present within the 'space'. Leary [20] concluded that the inner dura was lined with fibroblasts and that the cells lining the outer arachnoid were dissimilar. Thus, investigators began examining the dura and arachnoid as two exclusively separate identities.

The Dura Mater. The dura mater appears to be a thick layer of fibroblasts and extracellular collagen [21]. The cells look large and flattened and the collagen is abundant and somewhat organized. Haines [14] summarized the dura-arachnoid organization. The dura is characterized as having an inner and outer portion. The periosteal dura is adherent to the inner skull and the meningeal layer of the dura, contains a specialized layer that Nabeshima et al. [22] named the dural-border cell layer. This layer appears to be continuous with the dural aspect of the arachnoid and the histological aspects of this dural-border cell layer have brought much interest to researchers [22–25]. This amorphous layer appears to have flattened cell processes, varying sizes of extracellular spaces and little collagenous material. The amorphous structure possibly makes this an area of weakness within the tissue. A cross-section of the meninges and cell layers is shown in Fig. 1.5. If an ASDH is to form, the bleed needs to occur in the border cell layers.

The Arachnoids. The arachnoid portion of the meninges also consists of two distinct areas, the arachnoid barrier cell layer, which is attached to the dural-border cell layer, and the arachnoid trabeculae, which is closely attached to the pia mater. Both the cells and the extracellular material are dissimilar as compared to the dura mater. The cells are larger, more densely packed, having numerous mitochondria and filaments within their cytoplasm making the layer distinctive [22, 23, 26]. This closely packed structure of the arachnoid barrier cell layer excludes the presence of extracellular space, making it distinctive from the attached dural-border cell layer. Existing literature supports this idea. The description of the layers above has been verified [14, 27, 28] and testing has shown that the 'space' is not pre-existing. However, the junction between the dural and arachnoid border cells would be an area of weakness in cases of brain impact injury because the loosely organized dural border cell layer is attached to the more rigid arachnoid barrier cell layer. In fact, there is evidence that the space is easily created by a mechanical separation [29–31]. Since the biomechanical properties of the border cell layers have not been investigated, the adhesive properties of the layers in radial traction or in shear need to be quantified. These properties are crucial to the understanding of the formation of ASDH because of the close association of the bridging vein and cortical arteries within these layers. Only when this mechanism is established will preventative and clinical strategies be able to be discovered and tested. This will ultimately decrease morbidity and mortality rates associated with these types of brain injuries.

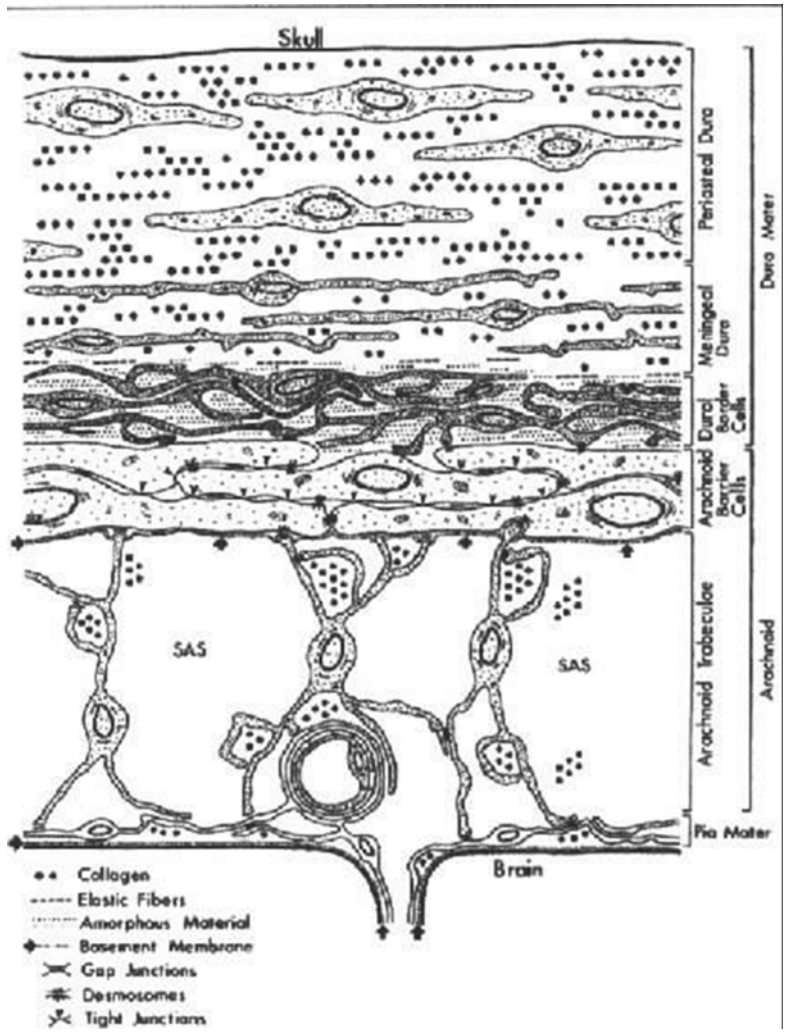

Fig. 1.5 Meningeal structure according to Haines et al. [14]

On the other hand, neurosurgeons are often of the opinion that the dura is attached to the skull and the arachnoid goes with the brain. Thus, even if there is no space in the subdural layer in the young, an actual space maybe created in the elderly should their brain shrink because not all of that space can be accommodated in the CSF layer. Since this is still controversial, we need to consider the mechanism of ASDH with no subdural space as well as in the presence of a subdural space occupied by CSF.

Anatomy of Cortical Vessels. The cortical vessels consist of bridging veins and cortical arteries and veins. The bridging veins traverse the dural/arachnoid complex. Their rupture has been traditionally considered responsible for ASDH and they have been studied extensively by researchers [32, 33]. The number of veins and their range of diameters have all been documented. Yamashima and Friede [34] provided a detailed description of the vessel wall as it traverses the dura/arachnoid complex in a straight course with no tortuosity to allow for the possible displacement of the brain. The cranial end is firmly attached to the rigid dura while the cerebral end is attached to the movable hemisphere. Leary [20] found that the thickness of the bridging vein walls varied

remarkably in the subdural portion, the thinnest part measuring 10 μm with a range of 10–600 μm. In the subarachnoid portion, the walls have a more consistent thickness of 50–200 μm. The collagen fibers in the subdural portion were loosely woven with a pattern that was more resistant to distension while less resistant to traction. That is, bridging veins are vulnerable to leakage in the subdural region. In fact, Yamashima and Friede [34] speculated that the bridging vein can rupture in the dura/arachnoid complex due to a physiological increase in venous pressure or due to cardiac resuscitation as well as due to a head impact. Trotter [35] regarded the rupture of the bridging vein as the cause of chronic subdural hematoma. However, another bleed source is the cortical artery traversing the dura/arachnoid complex. Information on the size, distribution and number of cortical vessels is sparse. Cortical arteries are found in the CSF layer. They run along the surface for a short distance and penetrate the pia to enter the cerebral cortex. However, some of the arteries running under the arachnoid can extend branches into the subdural layer. There is even evidence of a cortical artery forming a kink (knuckle) in the subdural space, as shown in Fig. 1.6 [36]. When the dura separates from the arachnoid, the vessel wall of the knuckle is torn off and bleeding from this tear results in an ASDH.

Acute Subdural Hematomas. Subdural hematoma (SDH) is a clinical condition due to a quickly clotting blood collection amid the dura and arachnoid membranes. ASDH's are most frequently the result of an acute head injury, however they can sometimes occur spontaneously in the elderly. The mechanism behind the separation of the arachnoid from the dura has yet to be determined. ASDH's usually transpire when the brain is subjected to a high energy, short duration force from trauma. It is thought that this shearing force will tear the bridging veins and as a consequence ASDH will form. However, epidemiological studies have shown that injuries other than bridging vein rupture accounted for a significant portion of ASDH cases. Thus, the need to determine the mechanism behind the injury is vital before any effective preventative and therapeutic strategies can be attempted and implemented. Finding the pathogenic mechanism through a more open-minded approach will lead to new innovative treatments for this disabling condition.

Epidemiology. Traumatic ASDH's are among the most lethal of all head injuries, carrying the highest

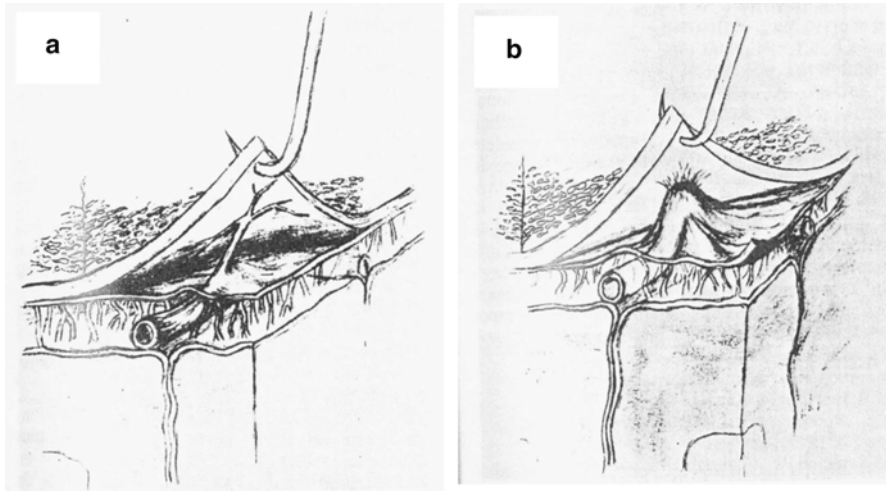

Fig. 1.6 (**a**) Bridging cortical artery connected to the dura. (**b**) Adherence of cortical arterial knuckle to dura and arachnoid (Bongioanni et al. [36])

risk to the patient, with a mortality rate of greater than 50 % in most studies. ASDH kills or severely disables more head injured patients than any other complication of cranial trauma. The main pathological factor involved is ischemic neuronal damage that results from cerebral vascular damage, raising the intracranial pressure. ASDH was found in patients who were involved in motor vehicle crashes, falls and assaults [37]. It is also found in boxers [38]. According to Gennarelli and Thibault [39], ASDH is the most important cause of death in severely head injured patients due to high incidence (30 %), high mortality (60 %) and head injury severity (2/3 with Glasgow Coma scores of 3–5). They also found that the cause of ASDH by falls or assaults was 72 % while that due to motor vehicle crashes was only 24 %. Maxeiner [40] attributed the source to bleeding in ASDH cases to extensive brain surface damage (contusion) and ruptured superficial cerebral vessels, including bridging veins and small arteries of the cortex. However, he also indicated in another publication [41] that rupture of the bridging veins did not lead to the formation of ASDH's. In fact, Maxeiner and Wolff [42] showed that there was an equal probability of ASDH caused by bridging vein rupture and by cortical artery rupture. Moreover, Shenkin [43] reviewed 39 consecutive cases of ASDH and found that there was a high incidence of cortical artery rupture (61.5 %). Bleeds of venous origin constituted 25.6 % of the cases and cerebral contusions were the cause in 7.7 % of the cases. The elderly were found to be more susceptible to ASDH [44, 45]. Since there can be brain shrinkage with age resulting in stretching of the bridging veins, the high incidence among the elderly can be explained by bridging vein rupture. However, the simple rupture of the bridging vein should not lead to ASDH formation unless additional mechanical factors are present, as explained below. Thus, clear mechanisms of ASDH formation need to be formulated before we can claim to understand why there is a high incidence of ASDH among the elderly. Karnath [46] found that ASDH usually occurs in younger adults while chronic SDH usually occurs in older individuals between 60 and 70 years of age. Finally, although the literature is silent in terms of a detailed injury mechanism, there is an implication that ASDH occurs when there is head contact with a rigid surface. However, in their experiments on subhuman primates, Gennarelli and Thibault [39] applied a pure angular acceleration to the head without direct impact to cause ASDH in these animals. More than one injury mechanism is in play in the formation of ASDH.

In terms of the locations of ASDH, not all ASDH's occur along the superior sagittal sinus into which the bridging vein empties the venous blood. Obviously, non-bridging vein related ASDH's are caused by bleeds from other sources, such as cortical vessels and brain contusion or laceration [47]. We will now consider the mechanism of ASDH formation from cortical bleeds for reasons stated in the section below.

Biomechanical Mechanisms for the Formation of ASDH

Based on current thinking, ASDH can arise from one of three sources, the first being the cortical arteries and veins. Laceration or rupture of these vessels can occur with penetrating injuries. Secondly, closed head injuries resulting in large contusions can cause similar bleeding into the adjacent subdural area. Thirdly and the most common type of ASDH is thought to occur from tearing of the veins that bridge the subdural area as they travel from the surface of the brain to the various dural sinuses. This last mechanism assumes rupture of the bridging vein in the subdural space since if it ruptured below the arachnoid, the result would be a subarachnoid hematoma. Ultimate strain to failure of the bridging veins and possibly other tissue components is inversely related to the strain rate [48]. Thus, the threshold for injury decreases as the strain rate or acceleration is increased. Gennarelli and Thibault [39] opined that ASDH is due to the rupture bridging veins during angular acceleration of the head associated with rapid onset rates (high strain rate). They contend that nothing needs to strike the head in order for ASDH to occur. That is, although impact to the head is certainly the most common cause of ASDH, it is the angular acceleration induced by the impact and not the head