Jack W. Tsao *Editor*

Traumatic Brain Injury

A Clinician's Guide to Diagnosis, Management, and Rehabilitation

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Editor
Jack W. Tsao, MD, DPhil, FAAN
Traumatic Brain Injury Programs (M96)
Wounded, Ill, & Injured Directorate
US Navy Bureau of Medicine and Surgery
Washington, DC, USA

Department of Neurology
Uniformed Services University of the Health Sciences
Bethesda, MD, USA

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Traumatic Brain Injury

For Emmanuel and Veronica

Preface

Traumatic Brain Injury: A Clinician's Guide to Diagnosis, Management, and Rehabilitation was written to enable medical professionals to quickly learn about the latest issues and treatments in this evolving clinical field. Traumatic brain injury (TBI) has been labeled one of the "signature injuries" of the military conflicts in Iraq and Afghanistan and in the USA, and the rise in public awareness of combat-related brain injuries has coincided with awareness of the potential long-term consequences of sports concussions. This book was developed as a result of a course on TBI which I directed for the American Academy of Neurology (one of the professional associations of neurologists in the United States) in 2008.

The term "TBI" describes a spectrum of injury ranging from mild (typically called "concussion") to moderate and severe (including penetrating brain injuries). Most TBI cases are of the mild variety, so the book focuses on this particular area. Readers will note that chapters discuss the most common clinical sequelae following TBI. The chapter authors were asked to summarize the key findings, issues, and treatments in their areas of expertise to enable this book to serve as a guide for busy clinicians managing patients with head injuries. To address a wide readership, initial chapters focus on acute clinical management including intensive care, imaging, neurocognitive testing, and sports and battlefield concussions. Later chapters discuss treatment of sleep disturbance, vestibular symptoms, headaches, seizures, and mental health consequences which might be seen after TBI. Finally, the book concludes with chapters on rehabilitation, including cognitive therapy, and gaps in knowledge with future research directions. As an aide to the clinician, an appendix reviewing ICD coding for TBI is also included.

I would like to thank my family for their support in the writing and editing process and Brian Belval, who was my initial publishing editor and who convinced me to take on the role of book editor.

Finally, as many of the authors of this book are United States military officers or government employees, it remains for me to issue a blanket disclaimer:

The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Departments of the Navy or Army, the Department of Defense, or the Department of Veterans Affairs.

Bethesda, MD, USA — Jack W. Tsao

Contributors

Aimee L. Alphonso, B.S. Department of Orthopedics and Rehabilitation, Walter Reed National Military Medical Center, Bethesda, MD, USA

Rocco A. Armonda, M.D. Department of Surgery (Neurosurgery), Uniformed Services University of the Health Sciences, Bethesda, MD, USA

Randy Bell, M.D. Department of Surgery (Neurosurgery), Uniformed Services University of the Health Sciences, Bethesda, MD, USA

John Betjemann, M.D. Department of Clinical Neurology, University of California at San Francisco, San Francisco, CA, USA

Justin S. Campbell, Ph.D. U.S. Navy Space and Naval Warfare Systems Pacific, San Diego, CA, USA

Anthony J.-W. Chen, M.D. Department of Neurology, VA Northern California Health Care System and University of California at San Francisco, San Francisco, CA, USA

Robert L. Collins, Ph.D., A.B.P.P. Neurology Care Line, Michael E. DeBakey VA Medical Center, Houston, TX, USA,

Department of Neurology, Baylor College of Medicine, Houston, TX, USA

Jay C. Erickson, M.D. Neurology Service, Madigan Army Medical Center, Tacoma, WA, USA

Alisa D. Gean, M.D. Department of Radiology, San Francisco General Hospital, University of California, San Francisco, San Francisco, CA, USA

Department of Neurological Surgery, San Francisco General Hospital, University of California, San Francisco, San Francisco, CA, USA

Brain and Spinal Injury Center (BASIC), San Francisco General Hospital, San Francisco, CA, USA

Division of Neuroradiology, 1X55, San Francisco General Hospital, San Francisco, CA, USA

Mel B. Glenn, M.D. Department of Physical Medicine and Rehabilitation, Harvard Medical School, Boston, MA, USA

Outpatient and Community Brain Injury Rehabilitation, Spaulding Rehabilitation Hospital, Boston, MA, USA

NeuroRestorative Massachusetts, Braintree, MA, USA

Community Rehab Care, Newton, MA, USA

Kim Gottshall, Ph.D. Physical Therapy Department, Naval Medical Center San Diego, San Diego, CA, USA

Department of Neurosciences, School of Medicine, University of California-San Diego, San Diego, CA, USA

Jeffrey H. Greenberg, Ph.D. Altarum Institute, Washington, DC, USA

Arthur Guerrero, M.D. Endocrinology and Metabolism Service, Department of Medicine, Tripler Army Medical Center, Honolulu, HI, USA

Michael E. Hoffer, M.D., F.A.C.S Department of Otolaryngology, Naval Medical Center, San Diego, CA, USA

Michael S. Jaffee, M.D., U.S.A.F. Department of Neurology, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

Department of Neurology, University of Virginia, Charlottesville, VA, USA

Department of Neurology, University of Texas Health Sciences Center at San Antonio, San Antonio, TX, USA

Thomas A. Kent, M.D. Neurology Care Line, Michael E. DeBakey VA Medical Center, Houston, TX, USA

Department of Neurology, Baylor College of Medicine, Houston, TX, USA

Department of Psychiatry and Behavioral Sciences, Baylor College of Medicine, Houston, TX, USA

Theresa B. Lattimore, M.S.N., N.P-C. Traumatic Brain Injury Programs (M96), Wounded, Ill, & Injured Directorate, US Navy Bureau of Medicine and Surgery, Washington, DC, USA

Carson Lawall, M.D. Naval Medical Center San Diego, San Diego, CA, USA

Tuong H. Le, M.D., Ph.D. Department of Radiology, San Francisco General Hospital, University of California, San Francisco, San Francisco, CA, USA

Geoffrey S.F. Ling, M.D., Ph.D., F.A.A.N. Neurology and Critical Care, San Antonio Military Medical Center, Brooke Army Medical Center, Fort Sam Houston, TX, USA

Department of Neurology, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

Department of Surgery (Neurosurgery), Uniformed Services University of the Health Sciences, Bethesda, MD, USA

Critical Care Medicine for Neurology, Anesthesiology, and Surgery, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

Donald W. Marion, M.D. The Defense and Veterans Brain Injury Center, Henry M. Jackson Foundation for the Advancement of Military Medicine, Rockville, MD, USA

Scott A. Marshall, M.D. Neurology and Critical Care, San Antonio Military Medical Center, Brooke Army Medical Center, Fort Sam Houston, TX, USA

Department of Neurology, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

David F. Moore, M.D., Ph.D., F.A.A.N. Department of Neurology, Tulane University, New Orleans, LA, USA

Ian C. Murphy, B.A. Department of Orthopedics & Rehabilitation, Walter Reed National Military Medical Center, Bethesda, MD, USA

Tatjana Novakovic-Agopian, Ph.D. Department of Neurology, University of California at San Francisco and Martinez VAMC, San Francisco, CA, USA

Karen Parko, M.D. Department of Clinical Neurology, University of California at San Francisco, San Francisco, CA, USA

VA Epilepsy Centers of Excellence, San Francisco VA Medical Center, San Francisco, CA, USA

Nicholas J. Pastorek, Ph.D., A.B.P.P. Rehabilitation Care Line, Michael E. DeBakey VA Medical Center, Houston, TX, USA

Department of Psychiatry and Behavioral Sciences, Baylor College of Medicine, Houston, TX, USA

Seb Potter, D.Clin.Psych. Lishman Brain Injury Unit, South London & Maudsley NHS Foundation Trust, London, UK

Adam Reuben, M.B.B.S. Emergency Medicine, Royal Devon and Exeter Hospital, Exeter, UK

Michael Russo, M.D. Department of Neurology, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

Helen J. Ryland School of Psychology, University of Exeter, Exeter, UK

Shirley I. Stiver, M.D., Ph.D. Department of Neurological Surgery, San Francisco General Hospital, University of California San Francisco, San Francisco, CA, USA

Brain and Spinal Injury Center (BASIC), San Francisco General Hospital, San Francisco, CA, USA

Victoria Tepe, Ph.D. Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury, Arlington, VA, USA

Survivability/Vulnerability Information Analysis Center, Booz Allen Hamilton, Dayton, OH, USA

Andra T. Tharp, Ph.D. Department of Psychiatry and Behavioral Sciences, Baylor College of Medicine, Houston, TX, USA

Brett J. Theeler, Ph.D. 187th Medical Battalion, Fort Sam Houston, TX, USA

James Tonks, Ph.D., D.Clin.Psych. School of Psychology, University of Exeter, Exeter, UK

Jack W. Tsao, M.D., D.Phil., F.A.A.N. Traumatic Brain Injury Programs (M96), Wounded, Ill, & Injured Directorate, US Navy Bureau of Medicine and Surgery, Washington, DC, USA

Department of Neurology, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

Erik S. Viirre, M.D., Ph.D. Department of Neurosciences, School of Medicine, University of California-San Diego, San Diego, CA, USA

Naval Health Research Center, San Diego, CA, USA

Sharon R. Weeks, B.A. Department of Orthopedics and Rehabilitation, Walter Reed Army Medical Center, Washington, DC, USA

Jennifer M. Weil, Ph.D. Deployment Health Clinical Center, Defense Centers of Excellence for Psychological Health and Traumatic Brain Injury, Arlington, VA, USA

W. Huw Williams, Ph.D. School of Psychology, University of Exeter, Exeter, UK

Philip J. Yates, D.Clin.Psych., C.Psychol School of Psychology, University of Exeter, Exeter, UK

Department of Psychological Medicine, Devon Partnership NHS Trust, Exeter, UK

Contents

Overview of TBI

1

David F. Moore, Michael S. Jaffee, and Geoffrey S.F. Ling

Abstract

The continued significant societal challenge of both civilian and military traumatic brain injury (TBI) makes the development of preventive strategies ranging from primary to secondary to tertiary pressing. The invisible and visible loss of societal productivity further underscores this urgency. The clinical complexity of traumatic brain has resulted in controversy, especially in the appreciation of concussion and its sequelae with the need to clearly define terms such as mild TBI and the persistent post-concussive syndrome or symptom complex. The following overview highlights some of the key areas of the required interdisciplinary approach to TBI.

Keywords

Traumatic brain injury • Concussion • Persistent post-concussive symptoms • Strain-rate continuum • Material properties • Pore viscoelasticity

Wartime Traumatic Cerebral Vasospasm: Recent Review of Combat Casualties

Accounts of neurological trauma are present in the *Iliad* and *Odyssey* of Homer from Greek antiquity, where concepts consistent with interpretation loss of consciousness, penetrating brain injury, spinal cord injury, and brachial plexus and nerve injury are present. These injuries concepts of the nervous system are well summarized with direct translation from ancient Greek in two review articles by Walshe (1997) and Sablas (2001). One important aspect of these oral tradition epics to the ancient Greeks may have been to preserve warrior knowledge about injury vulnerability allowing more formalized military training. It is clear that even in antiquity, traumatic brain injury (TBI) was described both in the military and civilian contexts.

The historical account of concussion is well summarized and described in the paper by McCory and Berkovic (2001). Initial use of the term "concussion" in the modern sense of an alteration or

D.F. Moore, M.D., Ph.D., F.A.A.N. (✉)
Department of Neurology, Tulane University, New Orleans, LA, USA
e-mail: dmoore14@tulane.edu

M.S. Jaffee, M.D., U.S.A.F. • G.S.F. Ling, M.D., Ph.D., F.A.A.N.
Department of Neurology, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

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temporary loss of adaptive brain function or an abnormal brain physiological state as opposed to distinct brain injury was used by medieval Persian physician Rhazes (Muhammad ibn Zakariyā Rāzī, 826–925 A.D.). Subsequent to this and with Chauliac (1300–1368 A.D.), the concept of a brain concussion or "commotio cerebri" with a relatively benign outcome from "contusio cerebri" or brain injury such as a skull fracture with a poor outcome became accepted in Western medicine with some variation. In more recent discussion, the consideration of a structural versus a functional cause of concussion has been considered in light of modern medical advances and technologies but still contains significant indeterminacies depending on the length and time scale of the approach. For example, in acute concussion, neuroimaging is typically negative yet with more extended techniques such as diffusion tensor imaging (DTI) and susceptibility-weighted imaging; previously unrecognized lesions are becoming increasingly appreciated indicating sustainment of structural abnormalities (Niogi et al. 2008; Bazarian et al. 2007). The conception of the length and time scale of injury is fundamental to the subsequent discussion of TBI since at a molecular level membrane disruption may result in alteration in membrane channel physiology or mechanoporation with resultant abnormal ionic fluxes and altered cellular and axonal function. Distinct examples of pathological sensitivity to brain trauma are present in abnormalities of calcium channel subunits *CACNA1A* and CACH (Childhood Ataxia and CNS Hypomyelination) (Kors et al. 2001; Schiffmann and Elroy-Stein 2006).

Complexity of Intracranial Anatomy

The brain is a uniquely anisotropic organ with the gyrencephalic cortical gray matter (GM), broadly orthogonal white matter (WM) fascicles, and subcortical gray matter nuclei together with multiple solid fluid interfaces between the brain parenchyma and the cerebrospinal fluid (CSF) both internally as represented by the ventricles and externally by the subarachnoid space. The entire brain is tethered by the dura together with the bridging veins and other vascular structures surrounded by the CSF cushion of the subarachnoid space. The skull represents a further protective layer of similar complexity with the diploic bone structure, numerous air sinus cavities together with foramina for exit and entrance of various neuro-vascular bundles. The complexity of the intracranial contents is well illustrated in Fig. 1.1, an axial section of the brain from the Visible Human Project (http://www.nlm.nih.gov/research/visible/visible_human.html).

Definition of Traumatic Head Injury

The current definition of TBI is phenomenological. Often, there is confusion in the nosology of TBI especially in relation to mild TBI (mTBI), a term that implicitly refers to the TBI event consistent with acute concussion. TBI is categorized according to the clinical pillars of post-traumatic amnesia (PTA) and/or a disturbance of consciousness [either alteration of consciousness (AOC) or loss of consciousness (LOC)]. Both of these clinical features, although correlated, allow for independent diagnosis of TBI severity. The overall TBI diagnosis is due to the severity of *Primary Traumatic Brain Damage* that is brain injury that results from mechanical forces producing tissue deformation at the moment of injury with direct damage to blood vessels, axons, neurons, and glia. The Glasgow Coma Scale (GCS) is also used as a TBI severity and diagnostic scale with mTBI being a GCS of 13–15, moderate TBI having a GCS range of 9–12, and severe TBI having a GCS of 3–8. *Secondary Traumatic Brain Damage*, on the other hand, is by definition due to the complications of primary damage including brain tissue hypoxia, ischemia, hydrocephalus, raised ICP, and CNS infection. The TBI spectrum definitions for closed head are summarized in Table 1.1. TBI is dichotomized into penetrating (pTBI) and closed head injury (cTBI) with the subclassification of cTBI into mild, moderate, and severe TBI. Although there is variation between epidemiological studies and it is a truism that all epidemiological studies are, in some degree, biased due to a trade-off between the veracity of ascertainment and the extent of the population sampled, rough

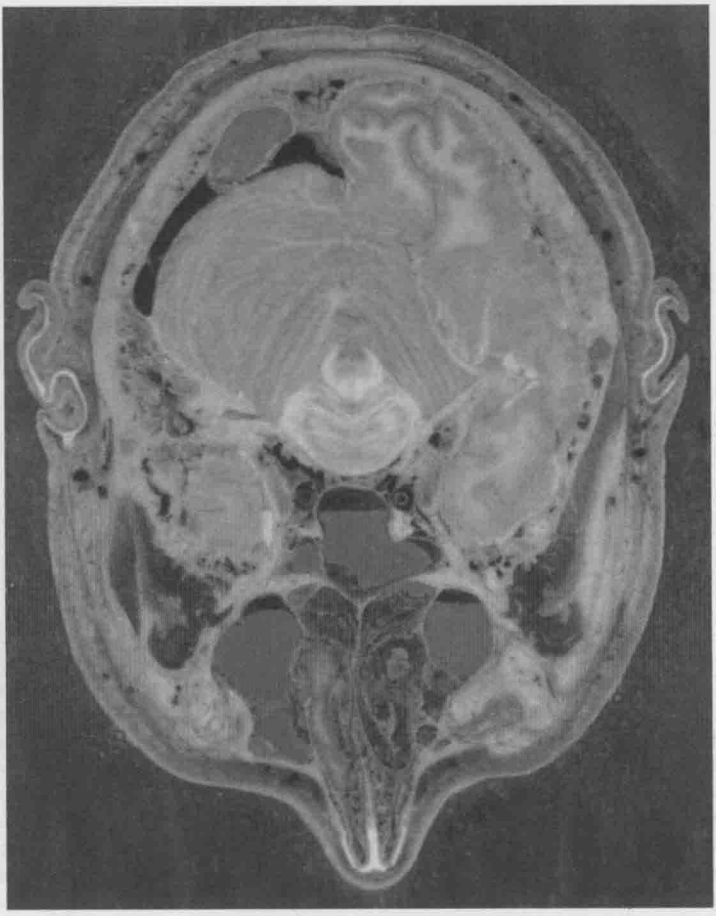

Fig. 1.1 Illustrating the intracranial contents illustrating the diploic nature of the skull bone, the numerous air sinus spaces together with the venous sinuses and dural sheathing. The gyrencephalic quality of the cortical ribbon is well seen in the occipital–temporal region. The complexity of brain anatomy has significant implications for the transmission of mechanical forces that may injure brain tissue. In particular, this is seen in the military context across impact to penetrating to blast brain injury

Table 1.1 Ascertainment of TBI according to the accepted severity scales

Definitions of TBI spectrum			
GCS	LOC	PTA	TBI
13–15	<1 h	<24 h	Mild or mTBI
9–12	>1 h and <24 h	>24 h and <7 days	Moderate
3–8	>24 h	>7 days	Severe

categorization suggests ~17% of cTBI being severe with ~13% being moderate and ~70% being mTBI (Zasler et al. 2007).

The above classification of TBI is inherently clinical and dependent on either direct observation or self-report. The current clinical trend is to attempt to redefine categorization of TBI in a patho-anatomic framework (Saatman et al. 2008). This is motivated, in part, by the recurrent failure of randomized clinical trials (RCTs) in TBI (except initially promising results with progesterone in moderate TBI), but also by a drive for standardization of common data elements (CDEs) to facilitate ongoing and new RCTs (Wright et al. 2007; Beauchamp et al. 2008; Xiao et al. 2008). CDE will also be particularly important in cross-sectional and longitudinal epidemiology studies allowing for "core" datasets to be acquired in studies with undoubted comparative value between study populations. A key epidemiological fact concerning TBI is that ~1.7 million civilian TBIs occur annually in the USA, with a cost to society estimated at 60 billion dollars both in direct medical costs and indirect costs due to lost productivity (Finkelstein et al. 2006; Xu et al. 2010) (http://www.cdc.gov/TraumaticBrainInjury/index.html).

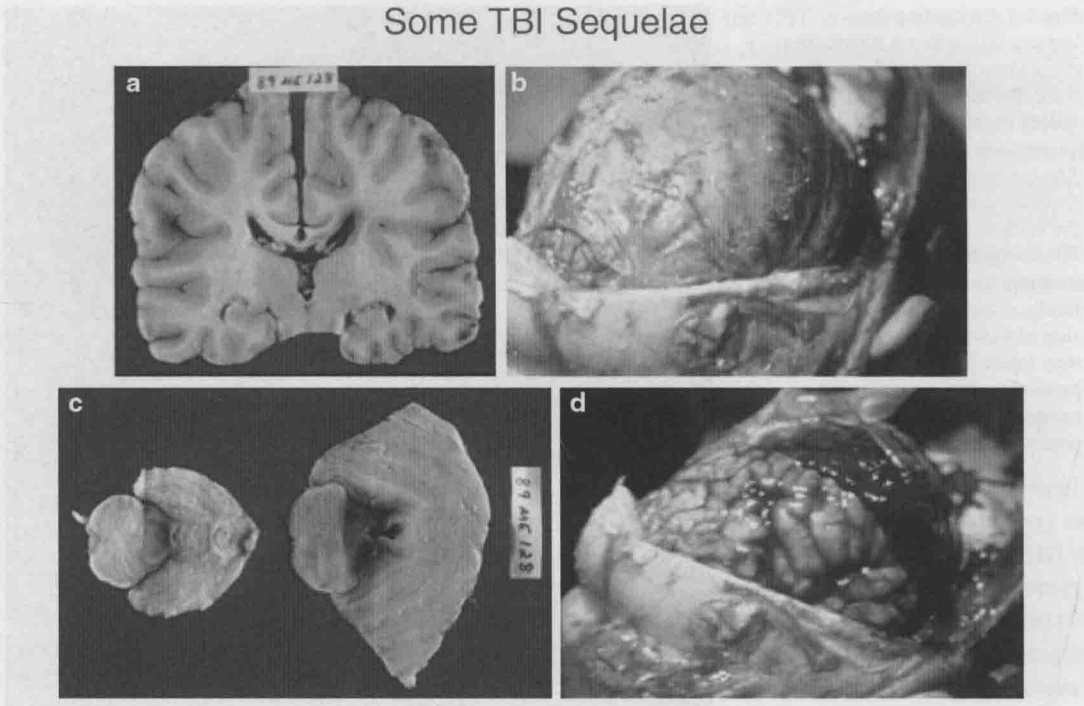

Fig. 1.2 Illustration of the neuropathology of traumatic brain injury. (**a**) and (**c**) illustrate the gross neuropathology of diffuse axonal injury with white matter hemorrhage in the corpus callosum (**a**) in the pontine white matter (**c**). (**b**) and (**d**) illustrate the subdural hematoma with (**b**) showing the dura intact and (**d**) the underlying hematoma with the dura reflected. (**e**) demonstrates cerebral contusion with bifrontal and bitemporal contusions; (**f**) the *left-hand side* of the image shows a coronal section that clearly shows the edematous and swollen brain compared to the normal brain tissue on the *right-hand side* aspect of the image. (**g**) shows a swollen optic nerve head in sagittal section due to chronically raised intracranial pressure. (**h**) illustrates delayed apoptosis of neuronal cells following TBI

TBI Spectrum: Neuropathology, Acute, Subacute, and Chronic Effects

In primary TBI, the spectrum of injury may range from diffuse or multifocal resulting in diffuse axonal injury (DAI) and diffuse vascular injury (DVI) to focal with intracerebral hemorrhage, subdural hemorrhage, epidural hemorrhage, and subarachnoid hemorrhage (Zasler et al. 2007). Other injuries include direct axonal injury, direct brain laceration, and contusion. Injuries from secondary TBI may also be diffuse or focal in the setting hypoxic-ischemic damage and brain swelling. While acute moderate and severe TBI may often require neurosurgical intervention, mTBI or concussion typically requires limited observation and intervention with recuperation occurring over several days to weeks. The prolonged sequelae of TBI are an opportunity for extensive rehabilitation care and therapeutic intervention. Of particular interest is the potential for metabolic abnormalities after concussion that if not adequately resolved may predispose the brain to more extensive damage if a further concussion occurs during the period of vulnerability—the second impact syndrome (Shaw 2002; Henry et al. 2010) (Fig. 1.2).

Concussion Biology and Mechanism

The neurobiology of concussion is incompletely understood and this has resulted in several theories ranging from interference with the reticular activating system or the cholinergic reticular inhibitory system to a paroxysmal depolarization shift of neurons resulting in "kindling" and a potential convulsive episode resulting in concussion

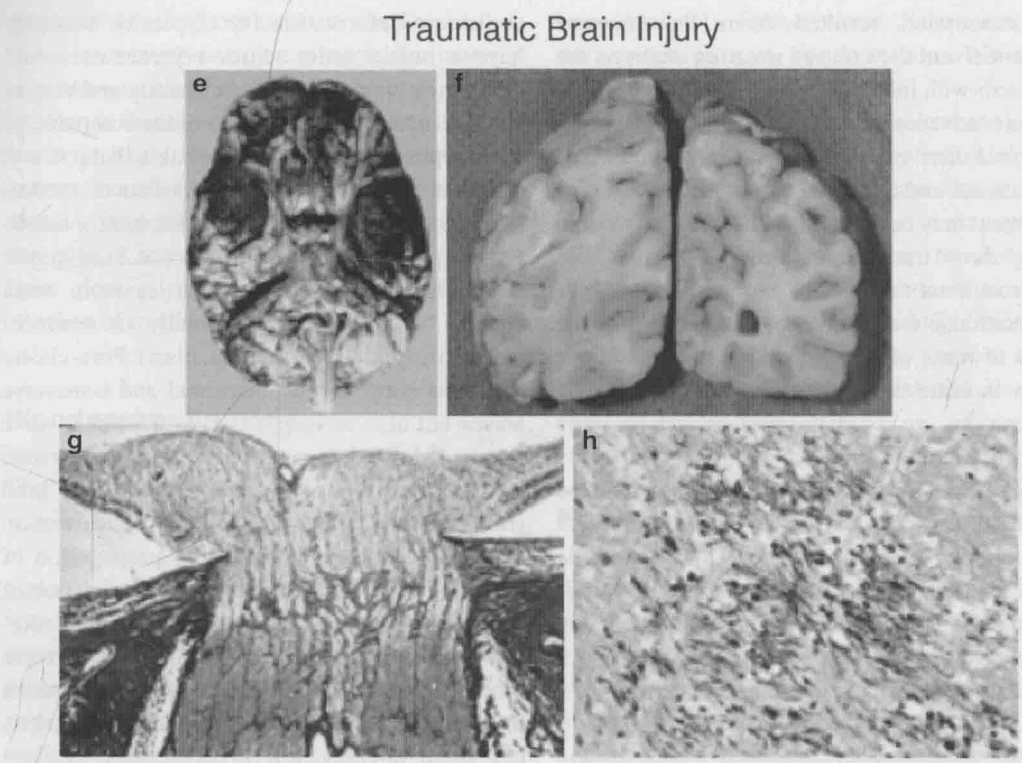

Fig. 1.2 (continued)

(Walker's Convulsive Theory) (Shaw 2002; Casson et al. 2008). From clinical neurology, it is a maxim that an alteration in consciousness results from either a bi-hemispheric process or a process in the posterior fossa. In relation to AOC and LOC, it is probable that most concussive processes result from a bilateral process suggesting more of a convulsive process secondary to a paroxysmal depolarization shift, although this cannot be stated with certainty. Similar reasoning is applicable to PTA with a resulting failure to lay down memory engrams bilaterally—the memory consolidation hypothesis (Shaw 2002).

The mechanical events precipitating concussion have been the subject of debate since the 1940s. Denny-Brown and Ritchie Russell (1940) demonstrated that injury in ketamine-anesthetized cats that were subjected to a concussive blow required that the head was able to undergo acceleration with associated translation and rotational effects. The blow was able to induce death without any rise in intracranial pressure but failed to result in concussion if the head was restrained and did not undergo acceleration. The cause of death appeared to be respiratory depression, but all brainstem reflexes were depressed with the respiratory centers being the most sensitive. Denny-Brown commented that "momentary deformity of the skull and stimulation of superficial structures, therefore appear to play no part" and finishes with "the nervous effect of a blow is thus considered to be due to the physical acceleration directly transmitted to each and every centre" (Denny-Brown and Ritchie Russell 1940). A threshold of 23'/s (angular minutes per second) was found for the cat with a higher value for the Macaque monkey. Subsequent to this, Holbourn in 1943 suggested that due to the incompressible nature of the brain linear acceleration would be unable to result in brain tissue injury; however, angular acceleration would result in shear strain and subsequent brain injury (Holbourn 1943). This was countered by Gurdjian and Lissner (1944) at Wayne State who suggested