

# **Cognitive Rehabilitation Therapy for Traumatic Brain Injury**

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*A Guide for Speech-Language Pathologists*



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Typeset in 10.5/13 Minion Pro by Flanagan's Publishing Services, Inc.  
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#### Library of Congress Cataloging-in-Publication Data

Names: Ostergren, Jennifer A., author.

Title: Cognitive rehabilitation therapy for traumatic brain injury : a guide for speech-language pathologists / Jennifer A. Ostergren.

Description: San Diego, CA : Plural, [2018] | Includes bibliographical references and index.

Identifiers: LCCN 2017047091 | ISBN 9781597567893 (alk. paper) | ISBN 1597567892 (alk. paper)

Subjects: | MESH: Brain Injuries, Traumatic—rehabilitation | Brain Injuries, Traumatic--complications | Language Disorders—therapy | Cognition Disorders--therapy | Cognitive Therapy—methods | Speech-Language Pathology—methods

Classification: LCC RC451.4.B73 | NLM WL 354 | DDC 617.4/81044—dc23  
LC record available at <https://lccn.loc.gov/2017047091>

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# Introduction

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As the title suggests, this book is about cognitive rehabilitation therapy (CRT) after traumatic brain injury (TBI). Its emphasis is on TBI in adults, written specifically for use by speech-language pathologists (SLPs). This is not to suggest that this is the sole area of rehabilitation needs of individuals with TBI. To the contrary, SLPs who have worked with individuals with TBI understand that there are many facets of rehabilitation following TBI (not solely CRT), and several parallel roles of SLPs who work with individuals with TBI. This includes rehabilitation guided by SLPs in the areas of speech, swallowing, and alternative and augmentative communication (AAC), to name a few. There are many helpful resources in each of these areas. This book is specific to CRT, given the growing (but still limited) research evidence in this area, as well as the cardinal role cognition plays in communication, daily activities, and the rehabilitation process after a TBI.

This book is limited to CRT for adults, as the research evidence for CRT in adults and children are distinctly different. Children with TBI and their families are also in need of helpful and evidence-based resources in this area, but collapsing these two distinct evidence foundations is neither simplistic nor conducive to the format of this text, as an applied resource for SLPs. The approaches described in this book are drawn directly from the research studies in the area of CRT for adults following TBI.

Section I of this book (Foundational Knowledge) includes information on the

mechanisms of injury and neuropathology of TBI (Chapter 1: Mechanisms of Injury and Neuropathology), and recovery patterns and applicable classification/outcome measures following TBI (Chapter 2: Classification and Recovery). These two chapters are followed by a description of common compounding and concomitant conditions that impact individuals with TBI and the rehabilitation process (Chapter 3: Compounding and Concomitant Conditions), and of course a description of the nature of cognition and communication following TBI (Chapter 4: Cognition and Communication). The final two chapters in Section I focus on general rehabilitation practices and principles, including the concept of CRT within the broader landscape of interdisciplinary and patient-centered rehabilitation (Chapter 5: Coordinated Care) and general CRT treatment principles (Chapter 6: Cognitive Rehabilitation Therapy Principles), which will serve as the foundation for the CRT approaches discussed in Section II.

Section II (Treatment in Action) is devoted to describing clinically relevant information about CRT approaches with demonstrated efficacy and effectiveness for adults following TBI. There are four chapters in Section II, structured around the categorical nature of descriptions of cognitive impairments following TBI. They include a chapter on approaches for attention and information processing speed impairments (Chapter 7: Attention and Information Processing Speed), one on memory impairments

(Chapter 8: Memory), a chapter on deficits in the area of executive function and awareness (Chapter 9: Executive Function and Awareness), and a chapter devoted specifically to *social* communication (Chapter 10: Social Communication). The format of these Section II chapters includes first an introduction to the landscape of CRT evidence in that specific area, followed by sub-chapters that address a specific evidence-based treatment approach *in detail*, including: (1) recommended candidates, (2) theoretical foundations, (3) relevant background information, and (4) a section titled, "Treatment in Action." In this Treatment in Action section, the research evidence available on a specific approach is distilled into applicable steps, procedures, and needed materials for implementation. In each of these sections, the applied nature

of this text for SLPs was given special attention. Where possible, charts, figures, tables, and appendices are included to facilitate the use of these approaches tailored by an SLP for a wide variety of individuals with TBI. Although this later section is more formulaic in nature, throughout this text the reader is encouraged to view each individual with TBI as unique. The one undeniably true fact about TBI is that there are no two individuals with TBI who are alike. As such, their rehabilitation (and the use of these techniques) is never the same. This text will serve as a guide only, offering descriptions of approaches supported by research evidence. As is consistent with our training, it is then the job of the SLP to individualize each approach to the unique needs and desires of a specific individual with TBI.



# Acknowledgments

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Thank you to my wonderful husband, Scott, and lovely daughter, Gwen, for your tireless support and endless patience. Without your gifts of love and time, this book would have not been possible. Thank you as well to the many individuals with TBI (and their families) whom I have had the great pleasure of serving throughout my many years as a speech-language pathologist. You have

informed the content of this book with your experiences and inspired me with your grace, humor, creativity, and courage in the face of traumatic brain injury (TBI). My deepest wish is that this book will be a helpful resource to speech-language pathologists in offering cognitive rehabilitation therapy that meets the unique needs of each individual with TBI (and his/her family).

# Contributor

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Carley B. Crandall is a graduate student at California State University, Long Beach (CSULB). She earned her BA Communicative Disorders from CSULB and is pursuing her Master's degree with a keen interest in traumatic brain injury-related communicative disorders, as well as the application of treatment within group settings. Carley

has held research assistant positions in the area of evidence-based practice in traumatic brain injury (TBI) and augmentative and alternative communication (AAC). Carley is a licensed speech-language pathology assistant and looks forward to furthering her education of, and contributing to, the field of speech-language pathology.

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Plural Publishing, Inc. and the author would like to thank the following reviewers for taking the time to provide their valuable feedback during the development process:

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# SECTION I

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## **Foundational Knowledge**



# 1 Mechanisms of Injury and Neuropathology

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*In examining disease, we gain wisdom about anatomy and physiology and biology. In examining the person with disease, we gain wisdom about life.*

—Oliver Sacks

Author Stephen Shore once said about individuals with autism, “If you’ve met one person with autism—you’ve met one person with autism” (as cited in Kolarik, 2016, p. 479). The same is true for individuals with traumatic brain injury (TBI). Diversity among individuals who experience a TBI is infinite, as are the ways in which the trauma associated with TBI can occur. No two individuals or their injuries are exactly alike. It is nonetheless important for those who want to be of assistance to individuals with TBI (and their families) to consider both general trends in the nature of TBI, as well as its potential neurologic impact. This chapter will discuss important terms and definitions, the distribution and determinants of TBI (epidemiology), and the neuropathology associated with TBI.

## Defining TBI

To define something is the act of making it clear or distinct. This is an important first step in understanding TBI. The Centers for Disease Control and Prevention (CDC) defines TBI as “a disruption in the normal function of the brain that can be caused by a bump, blow, or jolt to the head or a pene-

trating head injury” (CDC, 2015, p. 15). An explosive blast can also cause a TBI (CDC, 2015). The World Health Organization (WHO) uses the term “neurotrauma” to highlight the acquired and traumatic nature of this type of injury. TBI can result when the head suddenly and violently hits an object, or when an object pierces the skull and enters brain tissue (National Institute of Neurologic Disorders and Stroke [NINDS], n.d.).

The presence of any of the following clinical signs constitutes a disruption in normal brain function after a TBI (Menon, Schwab, Wright, & Maas, 2010):

1. Any period of loss of or decreased consciousness
2. Any loss of memory for events immediately before (retrograde amnesia) or after the injury (posttraumatic amnesia)
3. Neurologic deficits such as muscle weakness, loss of balance and coordination, disruption of vision, change in speech and language, or sensory loss
4. Any alteration in mental state at the time of the injury such as confusion, disorientation, slowed thinking, or difficulty with concentration.

TBI ranges in severity (from mild to severe), but according to the *Diagnostic*

and *Statistical Manual of Mental Disorders*, 5th edition (DSM-5; American Psychiatric Association, 2013), after an injury, the characteristics of TBI include one or more of the following:

1. Changes in levels of consciousness
2. Memory disturbances
3. Confusion associated with deficits in orientation
4. Neurological signs such as brain injury observable on neuroimaging, new onset or worsening of seizure disorder, visual field deficits, hemiparesis, and so forth.

A concussion is also a form of TBI, defined by McCrory et al. (2013, pp. 1–2) as:

1. Caused either by a direct blow to the head, face, neck, or elsewhere on the body with an “impulsive” force transmitted to the head.
2. Typically resulting in the rapid onset of short-lived impairment of neurological function that resolves spontaneously. However, in some cases, symptoms and signs may evolve over a number of minutes to hours.
3. May result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury, and as such no abnormality is seen on standard structural neuroimaging studies.
4. Results in a graded set of clinical symptoms that may or may not involve loss of consciousness.

## Epidemiology of TBI

Incidence is the occurrence, rate, or frequency of a disease, while prevalence is the percentage of a population affected by that disease. These are both important factors

to consider in understanding TBI, particularly since TBI has often been described as a silent epidemic. Worldwide, TBI is a major cause of death, especially among young people, and a significant source of lifelong disability (CDC, 2015; Roozenbeek, Maas, & Menon, 2013). Unfortunately, the incidence of TBI is on the rise globally (CDC, 2015).

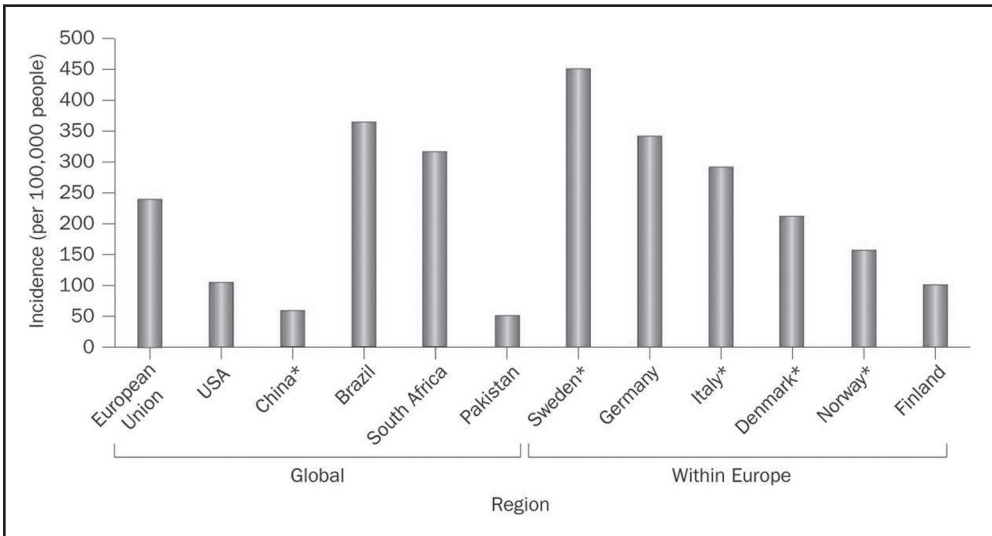
Each year in the United States, 1.7 million people sustain a TBI (CDC, n.d.-a). Every day, 138 people die from injuries related to TBI (CDC, n.d.-c). In the European Union, estimates suggest that approximately 7.7 million people are living with TBI-related disabilities, while in the United States this number is estimated to be 5.3 million people (Roozenbeek, Maas, & Menon, 2013). Globally, the incidence of TBI varies (Figure 1–1). The WHO estimates that TBI will surpass many diseases as the major cause of death and disability by the year 2020 (Hyder, Wunderlich, Pucanachandra, Gururaj, & Kobusingye, 2007).

Common causes of TBI vary globally as well. Figure 1–2 represents the leading causes of TBI in the United States, with falls currently reported as the most prevalent (CDC, n.d.-a). Worldwide, motor vehicle accidents are the most prevalent cause of TBI (Roozenbeek, Maas, & Menon, 2013). Figure 1–3 contains the distribution of types of injury within specific age groups.

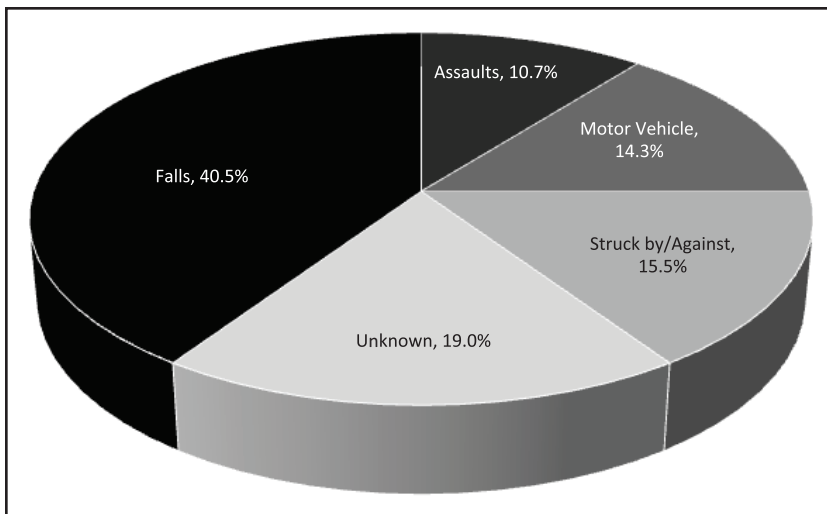
## Risk Factors

Risk factors associated with TBI are multiple, complex, and interdependent (Coronado, McGuire, Faul, Sugerman, & Pearson, 2012). Table 1–1 contains a summary of several risk factors in the United States associated with TBI, including age, sex, race/ethnicity, socioeconomic status, recurrent TBI, comorbid factors, and prescription drug and alcohol use.

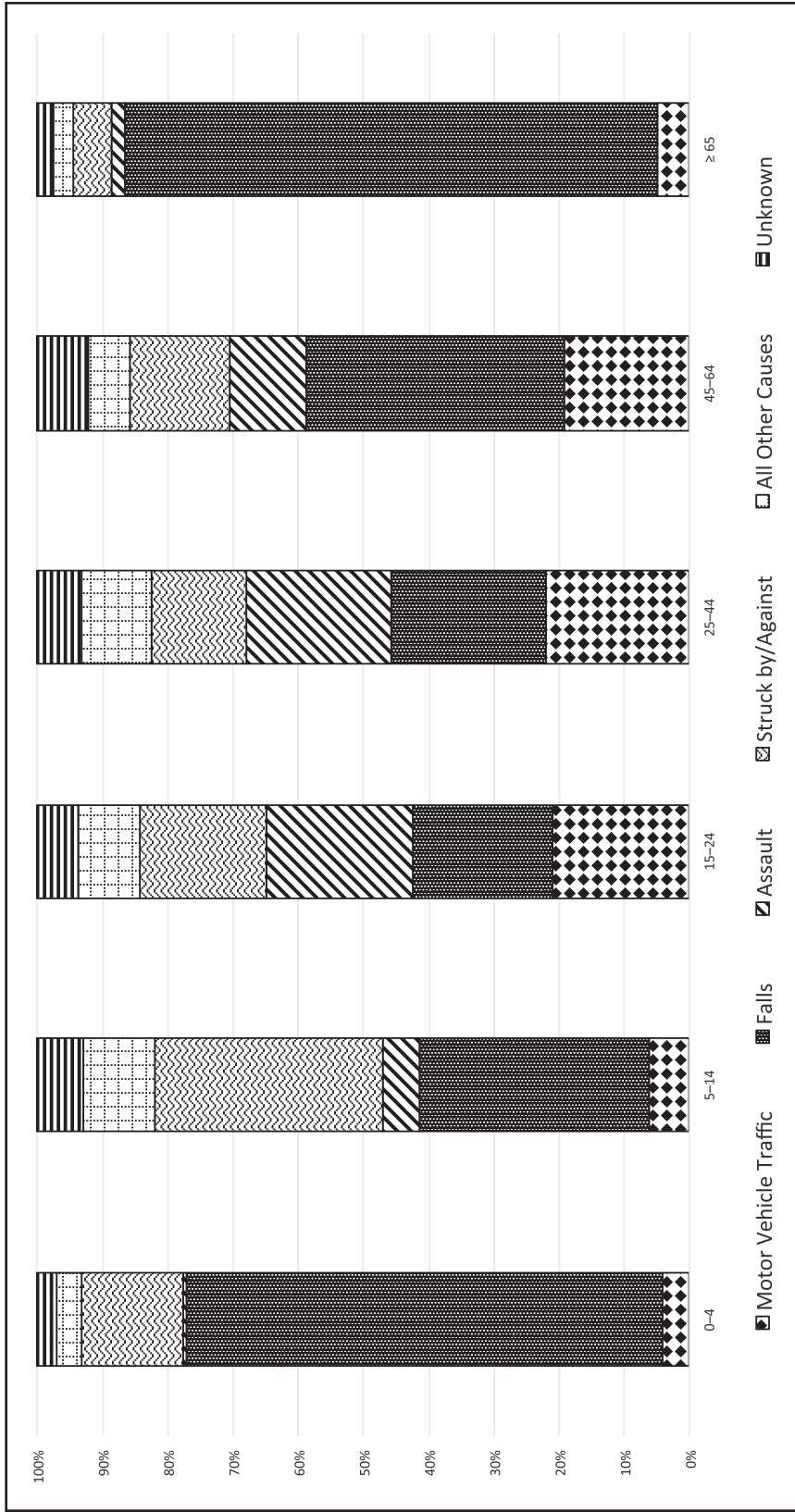




**Figure 1–1.** Estimates of global incidence of TBI. *Source:* Roozenbeek, Maas, and Menon (2013). Copyright © Macmillan Publishers Limited, part of Springer Nature. Used with permission.



**Figure 1–2.** Leading causes of TBI in the United States. *Source:* CDC (n.d.-a, Leading Causes of TBI).



**Figure 1-3.** Percent distributions of TBI-related emergency department visits by age group and injury mechanism. *Source:* CDC (n.d.-c, Percent Distributions).

**Table 1–1.** Risk Factors Associated with TBI

<i>Demographic Feature</i>	<i>Risk Factor</i>
Age	Children ages 0–4, adolescents ages 15–24, and adults ages 65 and older are at greater risk of TBI. This risk of TBI for older adults increases with age.
Sex	TBI rates are higher among males than females (across all age groups).
Ethnicity	Emergency room visits for TBIs are highest among African Americans and Caucasians, followed by American Indian, Alaska Native, Asian, or Pacific Islanders.
Socioeconomic status	Higher rates of TBI are seen in geographical areas with lower mean incomes and in metropolitan areas.
Recurrent TBI	The risk of a TBI progressively increases after a first or second brain injury, with the risk of experiencing a second TBI three times as great in those with a previous TBI than those in the general population.
Comorbidity and prescription drug use	Prescription drug use (especially in older adults) increases the risk of TBI, as does the comorbidities of diabetes mellitus, cardiac arrhythmias, dementias, depression, and Parkinson disease.
Alcohol use	Alcohol use increases the risk of TBI, with one quarter to one half of all adults with acute TBI being intoxicated at the time of injury.

Source: Coronado, McGuire, Faul, Sugerman, & Pearson (2012, pp. 91–92).

## Neuropathology of TBI

When studying how a TBI may impact an individual who experiences one, it is important to understand the neuropathology of the injury. To view TBI from this perspective is to understand how a TBI impacts the brain and its structures and functions. This is a critical foundation for speech-language pathologists providing rehabilitation services, as it shapes an understanding of the nature of recovery and rehabilitation approaches after a TBI.

As was mentioned at the start of this chapter, the ways in which TBI can occur

are infinite. As such, it is helpful to have a method for categorizing TBI according to shared qualities or characteristics. This is also known as classification. To date, there is no one classification system that encompasses all clinical and pathological features of TBI (Koehler & Wilhelm, 2011; Langlois Orman, Kraus, Zaloshnja, & Miller, 2011; Saatman, Duhaime, Bullock, Maas, Valadka, & Manley, 2008). Classification of TBI from a clinical or symptom perspective includes descriptions of TBI given severity and those variables associated with prognosis and outcome following a TBI. These aspects of TBI will be discussed in Chapter 2 (Classification and Recovery). TBI can also be

classified given pathological classifications, including anatomical (diffuse versus focal) or physiological (primary versus secondary injuries). Pathological classifications can be further extended to descriptions of TBI as a result of penetrating versus non-penetrating injuries (primary brain injury) and extracranial and intracranial injuries (secondary brain injury). Figure 1–4 contains a representation of this method of classification. The section that follows will discuss each of these in greater detail.

## Primary Brain Injury

Primary brain injury is the result of the initial mechanical forces of the trauma (Greve & Zink, 2009). Primary injuries are classified into those that are caused by penetrating forces (also known as open-head injuries) and those caused by non-penetrating forces (also known as closed-head injuries). More recently, blast injuries have also been discussed as a unique form of primary brain injury (Kocsis & Tessler, 2009; Magnuson, Leonessa, & Ling, 2012).

### *Penetrating (Open) Head Injury*

A penetrating injury (also known as an open-head injury) occurs when the skull is pierced by an object (NINDS, n.d.). This could be from a bullet, shrapnel, bone fragment, or a weapon such as a baseball bat, hammer, or knife. With this type of injury the meninges are ruptured, with cerebral tissue that is torn or cut (also known as laceration) (Smith, 2011). This type of injury is less common than non-penetrating injuries (Kazim et al., 2011). Brain tissue is damaged along the route of the penetrating object, as well as in surrounding areas (Kazim et al., 2011; Young et al., 2015). The trajectory and location of the wound and

the speed and velocity of the object both play a significant role in the outcome after a penetrating brain injury (Greve & Zink, 2009; Kazim et al., 2010; Young et al., 2015). Penetrating brain injuries are commonly classified as high-velocity or low-velocity, as follows.

### **High-Velocity Penetrating Brain Injury.**

High-velocity penetrating brain injuries are most commonly from high-velocity objects, such as rifle bullets, artillery shells, and shell fragments traveling at a high speed (Kazim et al., 2011; Young et al., 2015). High-velocity penetrating brain injuries result in more complex injuries and a higher mortality rate than low-velocity penetrating brain injuries (Kazim et al., 2011; Young et al., 2015). They are characterized by high levels of kinetic energy and an accompanying shock wave (Young et al., 2015). This shock wave creates three distinct areas of tissue damage: (1) crushing and cutting of brain tissue along the path of the projectile, (2) an adjacent area of tissue damage due to shearing and stretching of brain tissue, and (3) a surrounding area of tissue damage as a result of a lack of filling of small blood vessels and leakage of blood into brain tissue.

### **Low-Velocity Penetrating Brain Injury.**

Low-velocity penetrating brain injuries result from a penetrating object traveling at a lower rate of speed (compared with high velocity projectiles), such as clubs, baseball bats, and knives (Kazim et al., 2011; Young et al., 2015). Low-velocity penetrating brain injuries are rarer and are commonly associated with a better outcome than high-velocity penetrating injuries (Kazim et al., 2011; Young et al., 2015). Low-velocity penetrating wounds cause lacerations to the scalp, depressed skull fractures, and localized brain tissue damage along the object's path within the brain (Smith, 2011; Young et al.,