BRAIN INJURY

Applications from War and Terrorism

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To my mom, Shirley Collins,
who always said
that the best day of her life
was September 16, 1955.
And to my sisters,
Marsha and Suzanne,
who always disagreed.

"Only the dead have seen the end of war"
Plato, 367 BC

". . .and the end of terrorism."
Gean, 2014 AD

CONTENTS

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I would like to thank those colleagues and close friends whose loyal support has enriched my life and eased the ups and downs of my

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PREFACE

On September 11, 2001, the world changed forever. While I had been, for decades, professionally "addicted" to understanding traumatic brain injury (TBI), I had never focused on blast injuries or other mechanisms that are typically encountered in war, terrorist attacks, and natural disasters. Until this tragic event, my interest in TBI had revolved around San Francisco—falls from heights, assaults, motor vehicle and pedestrian accidents on the streets and highways, and the comparatively occasional gunshot injury to the head. Naively, I thought this experience credentialed me to volunteer at Landstuhl Regional Medical Center (LRMC) in Germany, the overseas military hospital operated by the U.S. Army that serves as the nearest treatment center for wounded military personnel returning from Iraq and Afghanistan. While abroad, I quickly realized that I had much to learn. In 1962, John F. Kennedy said, "The greater our knowledge increases, the greater our ignorance unfolds"—such was the motivation behind this book, born of my new personal commitment to understanding the unusual and unique brain injuries sustained in modern warfare.

I have tried to present a clear, concise yet comprehensive, up-to-date, and accurate text

with graphic and realistic illustrations. Given the development and expansion of technology over the two decades since the publication of my textbook on head trauma, this new book should fill a unique void for the physician interested in these new, profound, and emerging head injuries.

The book is divided into six chapters related to specific topics in combat neurotrauma. One of the chapters will also review penetrating head trauma occurring on the home front because gunshot injuries are, sadly, prevalent in both combat and civilian settings. Although the book is primarily neuroradiologic in focus, relevant clinical correlation is included. In addition, the radiologist, neurologist, neurosurgeon, maxillofacial surgeon, emergency physician, pediatrician, ophthalmologist, psychiatrist and psychologist, and member of the rehabilitation team should find this book useful. Not only is the characteristic imaging appearance of various injuries illustrated, but pathophysiologic correlation and clinical considerations are also incorporated into the discussion. To this end, I have made liberal use of illustrations to facilitate assimilation of the written text and to emphasize key concepts. I have also included ample references that will provide a guide for further study as this field of medicine expands and is progressively understood over time.

Finally, my primary goal in writing this textbook is to get the message out about this type of trauma. The threat of war is not going away anytime soon, and terrorist bombings have been increasing worldwide. Natural disasters also appear to be increasing. I would like to disseminate to the many providers, from all disciplines, the unique dimensions of blast injury incurred in these settings. May they not be needed, but should future struggles of the world lead to injury of this nature, may the lessons from my experiences be shared to improve the care for the victims.

Alisa D. Gean, MD

FOREWORD

In 2006, shortly after being named co-anchor of ABC's World News Tonight and while embedded with the U.S. 4th Infantry Division in Iraq, I sustained a traumatic brain injury (TBI). An improvised explosive device (IED) detonated near the tank I was riding in and nearly killed me. As is true for so many who experience TBI, my life changed in an instant. For some, the agent of change is an unexpected fall, motor vehicle or bicycle accident, or sports injury; for others, it is an unpredictable act of violence or a natural disaster, each of which is increasingly common in today's complex world.

TBI is a global epidemic. In America alone, over 1.5 million people fall victim to a TBI each year, and currently, 5.4 million live with the consequences of TBI for the rest of their lives. These available statistics do not account for the recent wars and therefore clearly underestimate the burden of TBI. Owing to improvements in body armor and advances in medical technology and treatment, soldiers are surviving more often than in previous conflicts, and they commonly survive with brain injury. TBI is acknowledged as the "signature injury" of the wars in Iraq and Afghanistan. Hundreds of thousands of troops returning home have some form of brain injury, with or without concomitant post-traumatic stress disorder (PTSD). It is critical that TBI be recognized as a major public health problem, whether caused by trauma suffered in war, terrorism, natural disasters, or in the civilian setting.

Severe TBI is usually obvious: there are skull deformities, partially shaved heads, or the wearing of helmets to protect them from injury during post-traumatic seizures. However, the full extent of brain injury is often hidden from plain view, yet may remain inside the "walking wounded" long after the outside visible injury has healed. If there is any "good" news from these wars, it is that they are helping us to rewrite what we know about many aspects of TBI. The tragedy of TBI in victims of war and terrorism has fueled public support for research funding, clinical trials, emergency response and disaster planning, accessible psychiatric and psychology services, and government-sponsored employment and recovery planning programs to improve the quality of life for all TBI survivors. After I survived, we realized that the veterans were not getting enough care and started the Bob Woodruff Foundation (bobwoodrufffoundation.org), an initiative that provides resources to injured service members and their families. We still have a long way to go, but Dr. Gean's book will help to get us there.

I am honored to write the foreword to *Brain Injury: Lessons from War and Terrorism* for my friend and colleague, Dr. Alisa Gean. Alisa is an outstanding neuroradiologist, known throughout the world for her expertise in TBI neuroimaging. She is a Professor of Radiology, Neurology, and Neurosurgery at the University of California, San Francisco (UCSF), having spent nearly 25 years studying TBI at a major Level I trauma center, San Francisco General Hospital. Almost 20 years ago, Alisa wrote the seminal textbook on imaging TBI suffered in civilian society: *Imaging of Head Trauma*. After September 11, 2001, she realized the significant void in our understanding of brain injury caused by war and terrorism. In 2008, Dr. Gean volunteered her clinical expertise at Landstuhl Regional Medical Center

in Germany to study combat TBI suffered in the Iraq and Afghanistan conflicts. Through this experience, she was motivated to devote the last 4 years of her academic pursuits to understanding the similarities and differences between civilian TBI and TBI suffered in war, terrorism, and natural disasters. She discusses these issues from the points of view of a clinician and a radiologist, and she illustrates her points with her trademark zeal for meticulous detail. The chapters bring together fundamental pathophysiology, clinical applications, and neuroimaging nuances to all facets of blast injury, ballistic principles, how different weapons of war and terrorism determine the nature of the resultant TBI, the role of polytrauma and burn trauma, vascular considerations, lessons highlighting distinguishing features of wartime TBI, and application of triage and emergency response systems to future conflicts and natural disasters. This extraordinary, magnificently illustrated and unique single-authored textbook, *Brain Injury: Lessons from War and Terrorism,* is the culmination of Dr. Gean's dedication and experience. It's really not just a book—it is a telegraphed documentary of a lifelong conviction to recognizing and responding to TBI by an acknowledged global expert.

Bob Woodruff
TBI survivor
ABC News CORRESPONDENT

CHAPTER 1

Introduction

Trauma from war has been part of the human condition since the beginning of civilization. However, classic conflicts between opposing armies in open fields, man against man, belong to the past. The intent is no longer the destruction of another man, or even the whole army. Rather, the goal is the global destabilization of the opponent's political, social, cultural, and psychological infrastructure. Modern conflict hits on all fronts. In historical conflicts, gunshot injuries were responsible for most of the damage. Approximately 85% of injured World War II (WWII) soldiers suffered from gunshot wounds.[1] Today's enemy is using a new weapon to kill, defeat, and demoralize: the **improvised explosive device (IED)**.[2] Indeed, the vast majority of current battlefield trauma is caused by whole-body blast trauma rather than gunshot wounds. We are only now beginning to appreciate the effect of a blast force on the brain. This book aims to review what should be considered neuroradiologically relevant to this extremely important, yet relatively enigmatic, injury.

Because of the different weapons used, each war tends to be associated with its own "signature" injury; examples are "shell shock" in World War I (WWI), "Agent Orange" exposure in Vietnam, and "Gulf War Syndrome" in the first Iraq War.[3,4] In the current conflict in Iraq and Afghanistan, traumatic brain injury (TBI) has been termed the "*signature injury*."[5] The percentage of soldiers suffering from TBI is significantly greater in this war than in prior conflicts.[6] This increased incidence of brain injury in today's battlefield may result from the significantly higher likelihood, as compared to any other war in American history, that the victim will survive his or her other battle injuries. Via a "personal pictorial tutorial," I will try to illustrate why this is the case, as well as how combat trauma differs from civilian trauma.

This book will also specifically review "penetrating" head trauma occurring both at home, in combat, in terrorist attacks, and in natural disasters. Gunshot injuries are prevalent in all four settings and are increasingly

problematic on our city streets. Until the current wars in Iraq and Afghanistan, penetrating TBI caused by bullets and shrapnel had received far more attention than the "closed" concussion from the blast wave. This skewed focus was likely because penetrating trauma is physically obvious, whereas concussions are typically devoid of an obvious wound. However, over the last decade, the military, and society in general, has become more cognizant of the devastating injury that can result from the brain "rattling" within the skull, leaving the patient physically unscathed and the initial radiologic images unrevealing. Another type of brain injury that can leave the patient looking "physically" normal is post-traumatic stress disorder (PTSD). This book will briefly address this "war inside."

To date, over 2,500,000 U.S. troops have served in Operation Iraqi Freedom (OIF) in Iraq, Operation Enduring Freedom (OEF) in Afghanistan, and Operation New Dawn (OND), the latter of which includes troops serving in Iraq after September 1, 2010. According to the Pentagon, there have been 6,760 deaths recorded as of September 24, 2013.[7] Due to conflicting statistics and differing diagnostic criteria, we cannot as yet tell how many of these 2,500,000 troops have suffered TBI.[8] According to the Department of Defense (DoD) and the Defense and Veterans Brain Injury Center (DVBIC), 233,425 service personnel have been diagnosed with TBI as of 2011. These data, however, underestimate the true prevalence of brain injury in this population because many military personnel with mild TBI likely never seek medical treatment or come to the attention of health care providers. An independent study by the RAND (Research and Development) Corporation has reported that up to *20% of all returning soldiers suffer TBI* while in combat,[9,10] and of these, only 43% reported having been evaluated by a physician for that specific injury. Fortunately, the majority of brain injuries sustained by military personnel are mild in severity.[11,12] Unfortunately, of those wounded in action, over half returned to duty within 72 hours, thereby incurring risk of serious cognitive damage from another TBI due to the detrimental cumulative effects of repetitive TBI and the "second-impact syndrome."[13,14]

Another alarming statistic is that there has been an increase in the incidence of mild TBI among active duty service members during the last decade, and the rate has risen dramatically in the most recent years.[15] The observed increase in the incidence of mild TBI toward the end of the conflict is thought to be due to a combination of factors, including an increase in IED attacks against coalition forces, the implementation of screening measures, and an increase in operational temp within the U.S. military. But what really is "mild" TBI? The current definition is based solely on the Glasgow Coma Scale (GCS). However, should a TBI patient with a normal GCS who subsequently loses his job; gets divorced; and struggles with depression, chronic headaches, and short-term memory deficits really be classified as having mild TBI? Unfortunately, this is the current reality.

Finally, note that the recorded casualty toll does not include injuries to the estimated 200,000 private civilian contractors working in Iraq, coalition forces, nor collateral damage to the Iraqi citizens. These statistics also do not include soldiers suffering from PTSD or the nearly 25 million veterans that the U.S. military health care system is already serving. Although the Veterans Affairs (VA) system is advanced in its treatment of lost limbs, it now has to deal with an unanticipated volume of TBI and PTSD cases. This book will illustrate how brain-injured soldiers are among the most catastrophically wounded, and recovery can be painfully slow and, often, elusive.

The heavy financial burden to our health care system during this conflict was recently

indirectly estimated by calculating the difference between the total health care delivered to military members during wartime and that which would have been delivered if participation in the war had been averted.[16] Overall, there were estimated excesses of 17,023,491 ambulatory visits, 66,768 hospitalizations, and 634,720 hospital bed days during the war period relative to that expected based on prewar experience. The category of mental disorders was the single largest contributor to the total estimated excesses. The authors of the study emphasized that the total health care burden associated with the wars in Afghanistan and Iraq is undoubtedly greater than that in their report because their analysis did not address care delivered in deployment locations or at sea, care rendered by civilian providers to reserve component members in their home communities, care of veterans by the DoD and VA, preventive care for the sake of force health protection, and future health care associated with combat injuries and illnesses, which will increase for decades after the cessation of war fighting. Another study by a leading Harvard economist, Linda Bilmes, and a Nobel Laureate in Economics, Joseph Stiglitz, conservatively estimated the cost of caring for TBI from the war in Iraq and Afghanistan at $14 billion over 20 years.[17] The long-term cost is impossible to calculate, but it is no doubt enormous. For example, did we lose an Einstein to war? No one can tell.

Lessons learned in combat should be incorporated into the armamentarium of providers on the home front. There is no doubt that victims of future terrorist attacks will suffer a combination of blast, blunt, penetrating, and thermal injuries that fall outside the routine experience of most health care providers. Indeed, most current health care providers believe that caring for victims of explosions and bombings is a remote possibility.[18] Explosives will likely continue to be the most common weapon of terrorism because of their potential to quickly inflict considerable harm to large groups of people.[19] Such explosives are relatively inexpensive, easy to make with instructions readily available online, and inconspicuous. One recent example took place at the 2013 Boston Marathon bombing, in which a simple kitchen pressure cooker IED that was hidden in a trash can on the side of the road became a devastatingly effective weapon. In addition, future accidental or "Mother Nature" mass casualty incidents, such as those encountered during the 2011 earthquake/tsunami in Japan, will also challenge the home front with large-scale injuries similar to those suffered on the battlefield. This is especially the case for low- and middle-income countries, which have suffered disproportionally more economic and human losses from disasters.[20]

During the 50 years prior to September 11, 2001, there were approximately 2,064 major disaster declarations made in the United States alone.[21] The vast majority of these declarations were due to severe storms, flooding, tornadoes, fires, earthquakes, and hurricanes. Indeed, the United States has sustained a historic number of hurricane-related disasters in the last decade.[22] For example, Hurricane Katrina in 2005 was the costliest natural disaster, as well as one of the five deadliest hurricanes, in the history of the United States.

After 9/11, however, terrorism became a major impetus to improve disaster preparedness. Whereas traumatic injuries suffered in natural disasters are somewhat similar to civilian traumatic injuries, blast injuries are a different beast. As discussed in Chapter 3, the supersonic nature of military grade explosives ensures this. Finally, a terrorist attack could actually trigger a natural disaster—for example, detonating enough explosive on a fault line could trigger a massive earthquake. To deal with future disaster scenarios, there

have been several iterations of a national response plan, the most recent of which is the National Response Framework (NRF), which was enacted in 2008. The NRF provides policy guidance regarding how the federal government responds to all hazards that occur within the United States and works in concert with recently developed state and local guidelines.[23] The final section of the book will address the physician's role in the response team.

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CHAPTER 2

In Comparison to Prior Wars, More Troops Are Surviving

Firepower has increased, but lethality has decreased. The wars in Iraq and Afghanistan have an unprecedented ratio of wounded to deaths on the order of 8:1 (**Table 2.1**). In contrast, the Vietnam War had an injury to death ratio of 3:1. In World War II (WWII) and in the Civil War, nearly half of all casualties died.[1] Even in the relatively bloodless 1991 Gulf War, 383 were killed in battle and 467 were wounded in action. In the Iraq and Afghanistan conflicts, however, nearly 90% of those wounded have survived.[2] This high wounded-to-death ratio can be attributed to a more urban battlefield (with immediate access to medical treatment) as well as a radically updated military with improvements in soldier protection, injury management, and patient evacuation.[3,4] While improvements in body armor and helmet technology have clearly saved lives, other factors have been instrumental in effecting the dramatic reduction in mortality from recent wartime. Significant advances in combat medic training, miniaturization of medical equipment, better designed tourniquet and hemostat bandages, and rapid access to fresh whole blood and the procoagulant recombinant factor VII all give frontline physicians powerful new tools for controlling hemorrhage.[5] Applied in combination, these measures help prevent severely injured soldiers from bleeding to death. In addition, there is an overall improved understanding of how to care for the polytrauma victim (with particular attention to avoiding hypotension and hypoxia), more effective methods of resuscitation, early brain imaging, prompt surgical or endovascular intervention, and fastidious intensive care, including the monitoring and control of intracranial pressure.

The implementation of the military's Joint Theater Trauma System (JTTS) initiatives and the Joint Theater Trauma Registry (JTTR) during the last decade has additionally led to improved patient outcomes.[6,7] Modeled after the success of civilian trauma systems, but modified to account for the realities of the battlefield, the JTTS was developed to better organize and

TABLE 2.1 Record-Breaking Lifesaving Statistics Are Seen in the Current War.[a]

Casualty Statistics in American Conflicts[a]

	Participants	% Deaths	% Wounded	Casualties	# Deaths	# Wounded	Survival % of Total Casualties
Civil War	2,213,363	16.47%	12.73%	646,392	364,511	281,881	44%
WW1	4,734,991	2.46%	4.31%	320,518	116,516	204,002	64%
WW2	16,112,566	2.52%	4.16%	1,076,245	405,399	670,846	62%
KW	5,720,000	0.64%	1.81%	139,858	36,574	103,284	74%
Vietnam	8,744,000	0.66%	1.75%	211,523	58,220	153,303	72%
Gulf War 1	2,225,000	0.02%	0.02%	850	383	467	55%
OIF/OEF/ OND	2,500,000	0.27%	2.07%	58,540	6,777	51,763	88%

[a]As of December 23, 2012.

WW1, World War I; WW2, World War II; KW, Korean War; OIF, Operation Iraqi Freedom; OEF, Operation Enduring Freedom; OND, Operation New Dawn.

coordinate modern combat casualty care using contemporary systems-based methodologies. Strict adherence to the JTTS evidence-based clinical practice guidelines (CPGs) has helped "deliver the right care, to the right patient, at the right place, at the right time."[8] Advances in medical imaging have likely improved the survival rate. And finally, speed saves! The Air Force has evolved a system to move thousands of casualties from the war zone quickly. This has been a crucial innovation that has allowed not only expeditious transportation of the wounded but also ongoing intensive medical care during the evacuation process in the early critical phases after injury. Surgeons can now leave wounds open and vacuum-sealed with plastic, and the most critical of the wounded patients can be air-evacuated with rapidity. Such technology simply did not exist during prior wars. Transport from the battlefield to the United States has decreased from 3 months in WWII to 30 days in the Vietnam War to now 30 hours from Iraq and Afghanistan. This carefully choreographed continuum of care will be discussed further in Chapter 5, Lesson 2. More troops surviving translates into more troops living with traumatic brain injury (TBI). Similarly, because more are surviving, more are remembering—thus, posttraumatic stress disorder (PTSD) has become a long-term sequela in war, terrorist attacks, natural disasters, and also civilian trauma.

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CHAPTER 3

Blast Injury Basics

WHAT HAPPENS DURING AN EXPLOSION?

The moment an improvised explosive device (IED) detonates, liquid and/or solid chemical material is instantly converted into a gas (**Fig. 3.1**). It is this *instantaneous* conversion that results in a release of a massive amount of energy.[1] As predicted by the law of conservation of energy in physics, the chemical energy that is released must be converted into other forms of energy. In a typical IED blast, this includes electromagnetic energy (i.e., light), acoustic energy (i.e., noise), thermal energy (i.e., heat), and a supersonic high-pressure blast wave (i.e., pressure). The supersonic shock wave compresses the surrounding air in an accelerated wave-front of superheated air molecules. It lasts a fraction of a second, exerts pressures of up to 700 tons, and, depending on the strength and amount of explosive, travels at initial velocities of 1,600 ft per second and beyond. Depending on the composition of the IED, projectile fragments can be propelled at velocities up to 3,000 mph (approximately 5,000 kph).[2] Interestingly, the amount of potential energy in the gas tank of a car is enormous, but the slower rate of combustion makes all the difference.

Sequence of Events in an IED Explosion

Blast waves follow complex nonlinear physics (**Fig. 3.2**). After the explosion, there is an initial positive pressure wave (also known as the primary *blast wind*). The peak of the overpressure wave is partially determined by the type and amount of explosive material. The wave exponentially decays back to atmospheric pressure (ATM) and is followed by a longer negative pressure phase. Because negative pressure creates a vacuum, and Aristotle taught us that "nature abhors a vacuum," a large volume of displaced air floods back into the vacuum, again under intense pressure, leading to the secondary blast wind. Technically, it

Sequence of Events in an IED Explosion

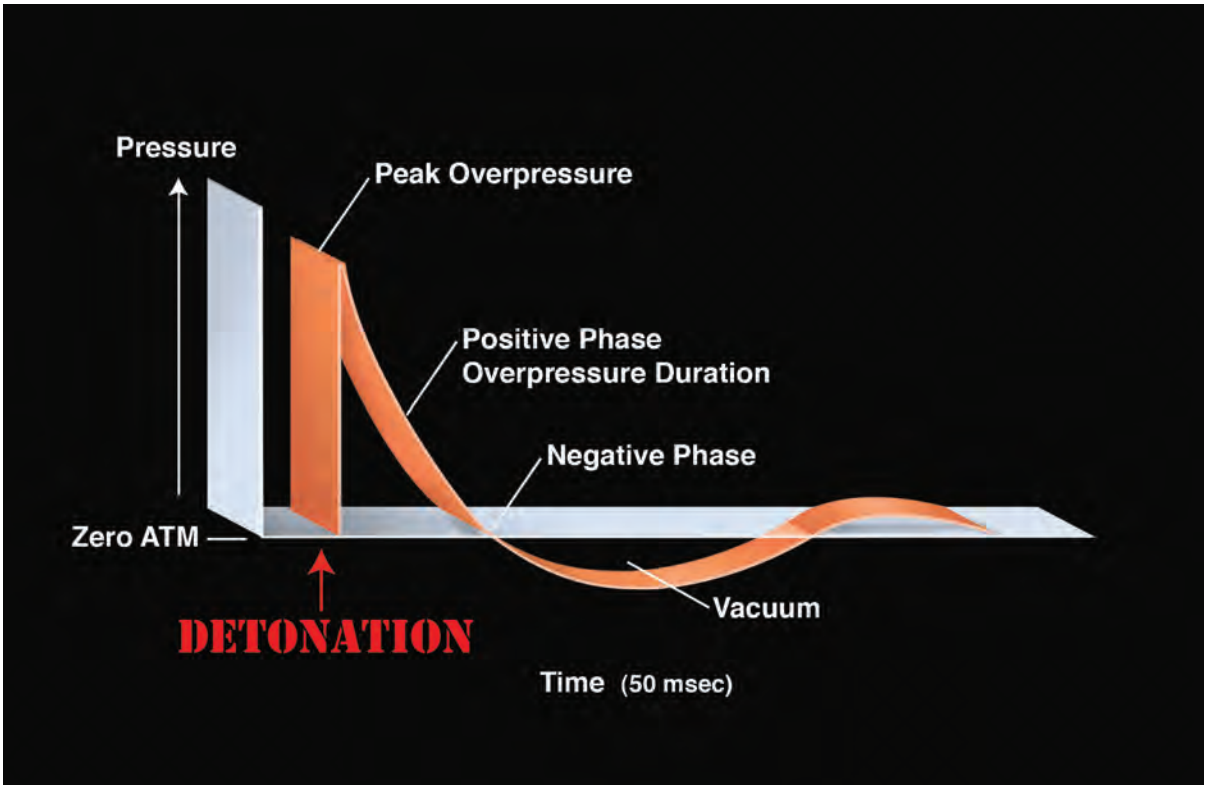


Figure 3.2. **Idealized Blast Pressure Waveform.** Explosions consist of a blast wave that has two components: an initial high-pressure shock wave front that compresses the surrounding air and a subsequent negative pressure phase. This latter drop in atmospheric pressure (ATM) below normal creates a relative vacuum that causes air to be drawn back toward the point of detonation. The blast waveform shown is called *idealized* because explosions usually occur in complex spaces like that found in an urban environment. As a result, shock waves reflect off surfaces and interact with each other in highly variable ways.

Figure 3.1. **High-Speed Video Showing the Sequence of Events during Experimental Detonation of an IED.** In this experiment, the IED was made of a 155-mm howitzer shell (see **Fig. 4.2**) and was placed on the surface of the ground. The detonation sequence was filmed at 100,000 frames per second. The entire explosion lasted approximately 10 milliseconds. **A.** The first thing that occurs in an explosion is a loud luminous *fireball* (*asterisk)*. The bright light results from conversion of chemical energy within the explosive into electromagnetic pulse energy. The heat results from the conversion of the chemical energy within the explosive into thermal energy. The noise results from conversion of the chemical energy into acoustic energy. Because light travels much faster than sound, explosions are seen before they are heard. This also explains why we see lightning before we hear thunder during a storm. **B.** After the fireball, innumerable small projectiles are identified within the air (*circle*). The projectiles arise from the exploded metal casing of the IED. If the IED was buried underground, the projectile fragments would also include dirt and rocks. Note how the expulsion of the fragments is in advance of the shock wave. **C.** The sky is now full of projectiles, and the supersonic shock wave front is identified (*arrows*). The shock wave is responsible for propelling the fragments. (Courtesy of Tim Imholt, PhD.)

★**KEY POINT** This experiment illustrates how chemical energy in a bomb is converted to other forms of energy, including electromagnetic pulse energy (light), thermal energy (heat), kinetic energy (from the moving projectile fragments), and acoustic energy (sound).

isn't actually "negative" pressure, but it is less than the normally felt 1 ATM, which is equal to 14.5 psi. The body needs a certain amount of pressure on it to help keep tissues intact, and an abnormal decrease in pressure may allow normal gases within tissue (e.g., oxygen, nitrogen, carbon dioxide) to expand and potentially burst. As will be discussed later, this is termed a *cavitation* injury. The subatmospheric pressure phase dissipates as the pressure returns to a steady state. Blast winds from both the positive and negative pressure phases can propel objects and people considerable distances.

In reality, this idealized behavior of a blast wave is uncommon because explosions tend to have more complex 3D flow characteristics that are altered by ambient conditions and environmental boundaries, which can result in wave reflections and amplification.[3] A more realistic waveform signature, encountered in combat and civilian trauma, has multiple positive and negative phases arising from the initial wave reflecting off of surfaces such as buildings or other structures. Shock waves can bounce around inside a vehicle and hit the person many times over; although the energy dissipates a bit each time, it isn't much if it reflects off a metal surface. Finally, while rare in current combat situations, underground and underwater explosions propagate shock waves further and with more force than do those transmitted in air. In these situations, significant injuries occur at a greater distance from the primary blast site. This is because pressure waves travel faster in solids and liquids than in a gas.[4] By analogy, pressure waves travel faster within the skull than in brain tissue, a feature that will be discussed more in the following text.

Idealized Blast Pressure Waveform

There are four types of blast injury: *primary*, *secondary*, *tertiary*, and *quaternary*. **Primary** blast injury (aka *barotrauma*) is caused by the initial overpressure and underpressure shock wave propagation through the tissue (**Figs. 3.1** to **3.3**). It is the least understood mechanism of blast trauma. **Secondary** blast injury refers to penetrating trauma caused by the projectiles arising from both the explosive device and the surrounding environment. **Tertiary** blast injury results from acceleration and deceleration of the human body and its impact with other objects. Secondary and tertiary blast injury mechanisms are commonly encountered in civilian trauma. **Quaternary** blast injury results from thermal injury (i.e., burns) and the inhalation of toxic gases. Most explosions cause injury via a complex mix of all four mechanisms (**Figs. 3.3** to **3.5**).[5,6]

There Are Four Mechanisms of Blast Injury

Figure 3.3. Blast Injury Mechanisms. The drawing illustrates the potential mechanisms of injury to a soldier during a tank explosion from a roadside IED. Similar mechanisms would apply to a blast in any enclosed space. Most explosions cause injury via a combination of *(primary)* **1°** (barotrauma), *(secondary)* **2°** (projectiles), *(tertiary)* **3°** (translational blast injury, i.e., acceleration and deceleration of the body and its impact with other objects), and *(quaternary)* **4°** (burns and toxic inhalation) mechanisms. A blast under a tank can blow the vehicle hatch open and propel the soldier under a high temperature pressure wave. The explosive jet causes catastrophic burns, and *spall* fragments can scab off the inside of the tank and injure troops within, resulting in small but deeply localized penetrating thermal injuries (see **Fig. 3.13**).

★**KEY POINT** Injuries to troops *within* armored vehicles represent a distinct subset of combat casualties. Compared to the infantry, injuries to personnel inside vehicles are associated with increased mortality and an increased incidence of burns and traumatic amputations.[9] This increased risk of injury also applies to individuals residing inside buses or buildings during a civilian terrorist attack explosion. In contrast, open air explosions dissipate the blast energy and are not influenced by reflective surfaces, thereby resulting in less severe trauma.

Armored Vehicle Capsized by a Roadside IED Explosion

Figure 3.4. IED Blast Injury. Note the huge hole in the road where the IED was buried prior to detonation (*asterisk*). Ejecta from the crater can deliver a significant blast load and is more destructive than a simple air blast injury. Troops within the vehicle are exposed to a complex combination of the four types of blast trauma described in the previous text. Note that the *Stryker* vehicle shown here weighs roughly 20 tons (including troops, gas, slat armor, etc.), and it was capsized by an IED that was probably the size of a small duffle bag. Imagine both the deafening sound and the intensity of the impact that the troops experienced at the moment of detonation.

Blast Injury Terminology

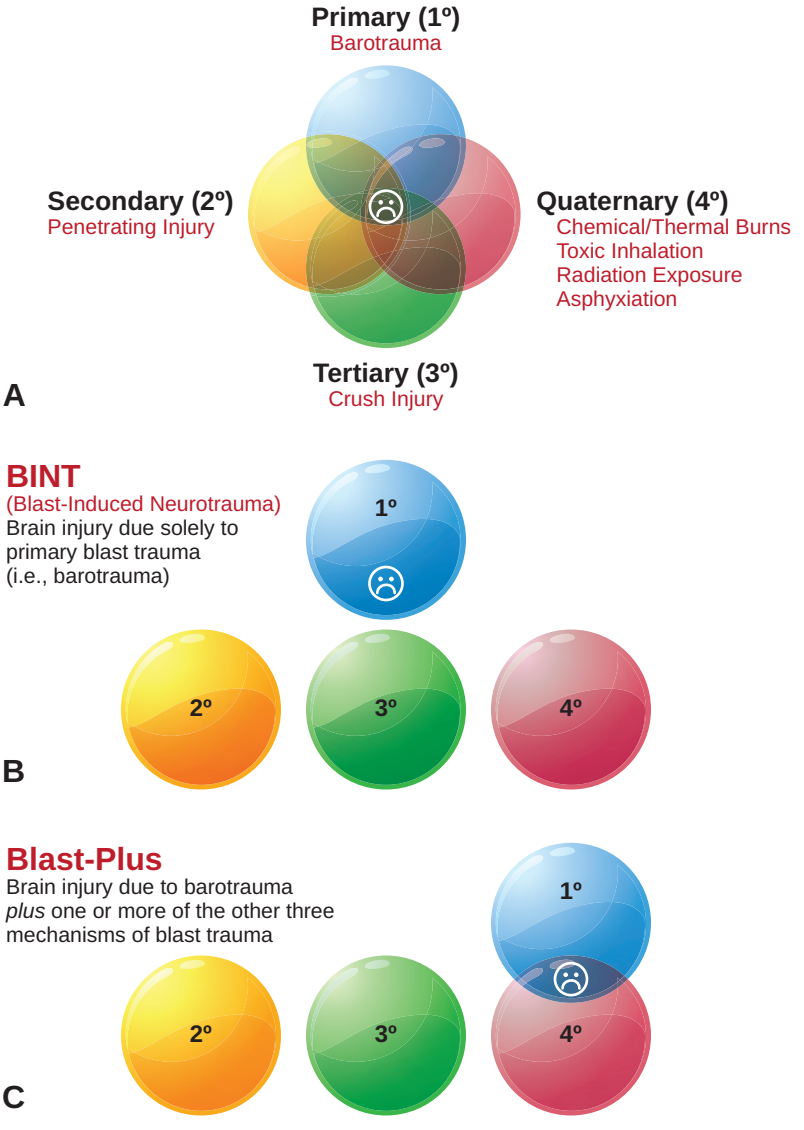


Figure 3.5. Basic Blast Injury Terminology. A. The extent of blast trauma is due to a complex mix of the four mechanisms described in the following text. It can be likened (simplistically) to a Venn diagram showing overlapping primary, secondary, tertiary, and quaternary injuries (indicated by ☹). In the example shown in **(A)**, the blast injury suffered by the patient is an *equal* mix of all four mechanisms. In reality, however, depending on the specific circumstances of the explosion and the patient, one or more of the mechanisms predominate. If isolated primary blast injury to the brain occurs, it is termed *blast-induced neurotrauma,* or *BINT,* illustrated in **(B)**. Most blast injuries to the brain, however, are thought to be caused by a combination of mechanisms and are therefore termed *blast-plus traumatic brain injury* (*TBI*). In the example shown in **(C)**, the patient suffered a combination of primary blast neurotrauma and toxic inhalation/thermal injury (4° blast trauma). He did not suffer penetrating blast injury (i.e., 2° blast trauma) nor was he thrown or crushed by the blast force (i.e., 3° blast trauma).

Primary Blast Injury

Primary (1°) blast injuries result from direct shock wave–induced changes in ATM. This type of injury is also termed barotrauma. Although advances in helmet technology and combat body armor have provided soldiers with considerable protection against penetrating and blunt trauma, they may not protect against the effects of the 1° blast overpressure and underpressure waves. The new pads placed within the helmet help somewhat, but the helmet does not cover the areas that are most problematic, namely, the eyes, ears, and nose. Bone, and the calvarium in particular, appears to be a very good mitigator against a blast force. This may be due to the skull's sandwich-like anatomic configuration consisting of an inner table and an outer table filled with intervening diploic marrow. Such a configuration is somewhat analogous to the pads in a helmet. Traditionally, the primary blast wave has been thought to affect primarily air-filled organs such as the lung, colon, and tympanic membrane of the middle ear.[10] For example, of the 242 deaths that occurred in the Madrid train explosion, tympanic rupture was found in 40%, lung injury in 40%, fractures in 25%, burns in 25%, and eye injuries in 25%. Perforation of the tympanum is the most common physical sign following blast trauma, and some believe that it is a sensitive screening test for the presence of 1° blast brain injury.[11]

HOW DO SHOCK WAVES INJURE TISSUE?

Over 2,000 years ago, the Greek philosopher Socrates said, "The beginning of wisdom is the definition of terms." Thus, this section will briefly define the vocabulary used in primary blast trauma. A blast wave is a combination of both *compressive* and *tensile* forces. The **compressive** force results from the blast's leading edge, a high-frequency supersonic wave front that hits the surface of the body. This wave front can propagate at velocities up to 185 mph into the underlying tissue. These forces can thereby cause *tensile shear strain injury*. The term **tensile** refers to a *stretching* force, and the degree of injury is related to the elasticity of the impacted tissue. **Elasticity** refers to the properties of a material as it undergoes stress, deforms in response to that stress, and then returns to its original shape after the stress dissipates. Elasticity is basically a measure of the stiffness of the tissue. The term **strain** refers to the extent of tissue deformation. More specifically, the term **shear strain** refers to *a change in shape without a change in volume,* such as changing a square into a parallelogram. Shear strain injuries are typically found in incompressible tissues, such as the brain.

There are three main mechanisms of injury caused by the primary blast force: *spalling, implosion,* and *inertia.*

1) **Spalling** occurs when a blast wave moves from a dense medium such as water to a less dense medium such as air. It refers to fragments that are ejected from the target substance (either human tissue or an inanimate object such as the inside of a tank) due to impact or stress caused by the shock wave. This often is compared to the effect of striking the outside of a rusty bucket with a hammer and watching the flakes of rust fly off the inside of the bucket. In human tissue, the alveolar walls of the lungs from such a gas–liquid interface with the air in the alveoli. In addition, the transfer of reflected blast injury through the dense substrates such as muscle and liver into the less dense material of the gastrointestinal (GI) tract may cause spalling. Particles of the denser tissue are spalled (thrown) into the less dense tissue. Spalling also is believed to occur when the blast wave transits from the rib cage into the lung.

2) **Implosion** occurs when the high-pressure blast wave compresses gas-filled spaces. For this reason, organs containing air are considered most vulnerable to primary blast injury. The typical injuries include tympanic membrane perforation, pulmonary contusions, pneumothorax, and GI hemorrhage. The opposite of explosion, implosion injures the tissue via compression. In some cases, the pressure wave can be so forceful as to impel air directly into a blood vessel, creating air emboli. Shock waves also generate transient pressure gradients from the rapid depressurization from overpressure to underpressure, as shown in **Figure 3.2**. In this setting, inert gases like nitrogen come out of solution and result in tiny bubbles that, when they pop, leave a cavity in the tissue. When a cavitation bubble forms in a liquid (e.g., by a high-speed water propeller), the bubble rapidly collapses—implodes—by the surrounding liquid. In brief, *tissue cavitation is a process of bubble generation, bubble implosion, and vaporization that occurs as a result of a decrease and subsequent increase in pressure*. This mechanism is identical to that suffered by scuba divers who ascend too quickly. The formation and collapse of the cavitation bubbles is dependent on the properties of the tissue; the higher the number of nuclei in the targeted medium, the greater the number of cavitation bubbles.

3) **Inertia** is perhaps the most important mechanism of primary blast injury. The inertial effect of a blast is related to its effect on adjacent tissues that differ in density. Tissues of different density accelerate at different rates in response to a blast force. Thus, differences in inertia lead to different rates of acceleration between neighboring tissues. This results in stretching and shearing forces between two adjacent tissues, and if these forces exceed the tensile strength of either tissue, macroscopic and microscopic tears occur at the tissue interface, an effect similar to TAI in head injury. As shock waves travel through the different organs, stress waves focus and defocus the entrant shock wave, leading to nanoshearing of cellular membranes. At transition sites, energy is released and there is focal mechanical disruption of the tissue (in essence, the basic mechanism underlying extracorporeal lithotripsy). Reflected and/or transmitted shock waves through the tissue cause shearing of the vasculature, cardiopulmonary contusions, and mesenteric shear injury.[5]

The causes of *extracranial* primary blast injury described previously are relatively well understood. By contrast, mechanisms of *intracranial* primary blast injury, also termed *blast-induced neurotrauma* (*BINT*), are only beginning to be understood. Indeed, the effect of shock waves travelling through the brain is currently hotly debated. For example, with respect to the implosion mechanism, it is unclear if the cerebrospinal fluid (CSF) itself cavitates due to the presence of impurities and dissolved gas or if the interface between the CSF and the subarachnoid membrane is unable to support tensile stresses from the shock wave. Additionally, the "bubbles in the brain" concept of implosion microcavitation injury remains controversial. For the general mechanisms of blast injury discussed previously, however, it is agreed that different parts of the brain will focus and defocus the entrant shock wave differently and, depending of the different inertia effects between adjacent brain structures, the blast force will lead to shearing at those interfaces. That is to say, the brain doesn't act as a single homogenous tissue in response to the blast. Further, tensile shear strain forces behave quite differently in fluids (e.g., the CSF) than in solids (e.g., the brain). Transmission of nonlinear blast wave energy into brain tissue can result in energy deposition at high strain rates in millionths of a second. The shearing stress wave may be particularly harmful to the anisotropic white matter tissue of the brain because the

directionality of white matter fiber tracts may render them highly vulnerable to propagation of shear forces and tearing injury.[12] The amount of energy deposition (and therefore incurred blast brain injury) is determined by several factors that are discussed later in this chapter.

SEVERAL THEORIES HAVE BEEN ADVANCED TO EXPLAIN PRIMARY BLAST-INDUCED NEUROTRAUMA (BINT)

The numerous theories to explain intracranial blast injury, many of which are based on the concepts of spalling, implosion, and inertia mechanisms of extracranial blast injury described earlier, are outlined in the following text.[13–44]

1) **Pressure waves propagating through the body via major vessels, cerebrospinal fluid, and soft tissue.** The blast from a positive pressure wave moving approximately 3,000 m per second is equivalent to about 40,000 kg being thrust against the chest and abdomen. The blast wave abruptly compresses the inferior vena cava and transmits the sudden increase in venous pressure to the brain via the jugular veins. In this *vascular surge* theory, the more central brain structures such as the hippocampus are more vulnerable to injury than the cerebral cortex. Support for this theory comes from recent animal studies demonstrating that shielding of the torso from the blast force ameliorated the severity of the brain injury. A related mechanism suggests that sudden compression of the thecal sac within the spinal canal can retrogradely transmit a pressure wave via the CSF to the cerebral ventricles and brain tissue. This much pressure can cause vaporization and tissue cavitation. It has also been postulated that shock waves are transmitted to the brain via viscera, muscles, and bones. Injury to the brain by ballistic pressure waves may occur following a high-energy bullet injury to an extremity. This mechanism of injury is called **hydrostatic shock**. It is based on the theory that a blast force can produce remote wounding via a hydraulic effect transmitted through liquid-filled tissues, in addition to the local effects caused by direct tissue impact. Such studies have shown that the hypothalamus and hippocampus are particularly vulnerable to damage after high-energy missile impact to an extremity.

2) **Pressure waves entering orifices of the skull.** Unlike the brain (which is protected by both the skull and combat helmet), the face remains directly exposed to the pressure wave. The nasal cavity, external auditory canals, and the orbits are potential entrance sites for blast waves to enter the cranium.

*Note that theories 1 and 2 suggest that wearing a helmet would be superfluous in protecting against BINT; these mechanisms dictate that the brain damage is caused by the shock wave propagating through other tissues to reach the brain.

3) **Skull flexure**. The skull is more elastic than expected. In the skull flexure theory, deformity of the calvarium by the shock wave results in "ringing" of the skull, which causes secondary brain tissue distortion. Recall that the brain is essentially an incompressible (but deformable) viscoelastic fluid with varying viscosity and shear wave velocity under strain deformation. An effective mitigation strategy would be to prevent the blast wave from entering the airspace between the head and the helmet. Indeed, studies have shown that helmets without padding increase the gap between the head and the helmet and allow the entering blast force to amplify pressures directly on the skull. There is also pressure *enhancement* within the skull, which is believed to

be caused by shock wave reflection at the interface of tissues with distinct wave impedances (e.g., dura). Because of the skull's elasticity, its transmission properties following a blast exposure will change.[45] Thus, one blast exposure may reduce the ability of the cranium to protect itself from a subsequent blast injury.

4) **Bulk acceleration of the head.** The abrupt positive pressure phase of an explosion first accelerates the rigid skull, but because the brain is unattached and suspended in CSF, it lags behind. The brain thus suffers an initial blow on the blast-facing surface of the skull. When the subsequent negative pressure phase of the blast wind pulls the head in the opposite direction, the brain is struck again, this time on the opposite, non–blast-facing surface. This acceleration–deceleration injury is essentially equivalent to the coup–contrecoup mechanism present in civilian trauma.

5) **Air emboli.** Air is compressible; fluid is not. Implosion of the pulmonary alveoli can cause them to rupture, and if microbubbles of air are forced into the pulmonary vasculature by the force of the blast wave, they may circulate as air emboli to the cerebral vasculature. The resultant air emboli can then become lodged in the smaller cerebral microvessels, leading to microvascular occlusion and stroke.

6) **Impaired cerebrovascular reactivity.** The inability to autoregulate and maintain a stable cerebral blood flow in the face of low or high blood pressure can be catastrophic. This particular problem is common in the polytrauma patient. Impairment in autoregulation is also more frequently seen in younger patients, as typifies those serving duty. The inability to vasodilate in the setting of reduced intravascular pressure renders the brain vulnerable to secondary ischemic injury. At the other extreme, the inability to vasoconstrict in the setting of elevated blood pressure increases cerebral blood volume, resulting in increased intracranial pressure (ICP). Impaired cerebrovascular reactivity may also manifest as hypercontractility (i.e., vasospasm). Recent research has shown that forces exerted on arteries are different during an explosive blast versus blunt force trauma. In addition, vasospasm can occur even without concomitant subarachnoid hemorrhage.

7) **Electromagnetic pulse (EMP).** If the EMP from a blast is powerful enough, it can interfere with local regional electronic devices. Because the brain is an electrical organ, this theory proposes that the blast energy could "short-circuit the brain." It has been shown that energy generated within the first 15 m of an explosion is high enough to be called *ionizing energy* and is thus capable of stripping electrons off of neurons and causing them to become positively charged (Tim Imholt, PhD, personal communication, January 2013). This mechanism could relate to possible risk of seizures from blast injury. However, little is known about the incidence of seizures or cortical spreading depression events when the brain experiences a blast force.

8) **Vagal reflex.** Blast studies have demonstrated apnea and transient depolarization of cortical activity. This depolarization may explain transient paralysis (flat electroencephalogram [EEG]), vasovagal reactions, and cardiac arrhythmias that can follow a blast wave. These secondary insults to the brain may be perilesional, widespread, or cause focal hippocampal injury. One early study suggested that vagal afferentation from lung injury, rather than energy transfer to the brain, may be the cause of medullary injury. Cardiac injury can also occur from a rapid increase in intrathoracic hydrostatic pressure or via direct external pressure *commotio cordis* by the blast wave. If the trauma takes place during

systole when the atrioventricular (AV) valves are closed, atrial rupture may occur; whereas if the insult occurs during end diastole, ventricular rupture may occur.

*Note that none of the previously described mechanisms is mutually exclusive, and, indeed, they probably work together to cause BINT. Additionally, one should not underestimate the possible systemic physiologic consequences of an explosion, which can exacerbate the central manifestations of the injury. This latter contribution to BINT is addressed in Chapter 5.

Regardless of the initial macro mechanisms of injury, numerous distinctive pathoanatomic findings of BINT have been identified:

- Gross pathologic findings of BINT in animal studies have revealed small hemorrhages in the white matter in a pattern similar to traumatic axonal injury (TAI), subdural hemorrhage, subpial hemorrhage, congested cortex, perivascular space enlargement, diffuse brain injury, and venous engorgement.[46] Indeed, a frequent observation among operating neurosurgeons is the presence of hyperemic cerebral swelling (**Figs. 3.6**, **3.7**, **5.12**, **5.20**, and **5.48**). Although speculative, one aspect of normal anatomy that may be relevant to the development of BINT is the "suspension" of the brain within the skull by the arachnoid granulations and cranial nerves. Just as a suspension bridge is vulnerable to large torsional oscillations, the brain with its relative tethering to the skull by the arachnoid granulations and cranial nerves may be similarly affected when exposed to a blast force. If true, then the longer central nervous system (CNS) axons would be particularly vulnerable, and this was indeed the case in a recent animal study.[47] Another aspect of normal anatomy that may affect the development of BINT is the tentorium, which may amplify the blast wave to the cerebellum, which is often victimized by BINT.[48]
- Histologic and ultrastructural findings of BINT include white matter hemorrhage into the myelin sheath and perivascular spaces, chromatolytic changes in neurons, and widespread microglial activation (hypertrophic astrogliosis), particularly in the superficial layers of the cerebral and cerebellar cortices.[49] The hippocampus is another favorite target for BINT: One animal study confirmed that exposure to blast overpressure was not only associated with ultrastructural and biochemical impairment in the hippocampus but also the development of cognitive deficits.[22] Abnormalities of the pineal gland can also be seen.[50] Additional histologic markers of neuronal injury include expanded perineuronal spaces, cytoplasmic vacuoles, myelin deformation, and axoplasmic shrinkage, and neuronal apoptosis.[22,51,52] The change in localization of neurofilament protein staining from axons and dendrites to the cell body in cortical neurons following blast exposure is thought to be a result of disturbed anterograde axonal transport and redistribution in cytoskeletal proteins.[53] As mentioned earlier, a very recent study of the cytoskeletal injury revealed overstimulation of integrins, causing axons to contract and blood vessels to constrict when abruptly stretched.[54] The researchers hypothesized that this might explain the high incidence of vasospasm in blast victims (discussed in Chapter 5, Lesson 10). Vasospasm can also be affected by an alteration in nitric oxide synthetase, which is known to occur with blast trauma.[55,56] Interestingly, unlike blunt TBI, one study of BINT neuropathology demonstrated widespread traumatic axonal fiber degeneration *without* obvious cell loss, suggesting that the axonal injury from BINT may not be tightly coupled to irreversible neuronal cell death.[43]

TABLE 3.1 Mechanisms of Primary Blast Brain Injury

Current Theories for Primary Blast Brain Injury (BINT)
1. Pressure waves propagating through the body via major vessels, CSF, and soft tissue such as bone, muscle, and viscera
2. Pressure waves entering orifices of the skull, particularly the orbit
3. Skull flexure
4. Bulk acceleration of the head
5. Air emboli
6. Impaired cerebrovascular reactivity
7. Electromagnetic pulse (EMP)
8. Vagal reflex

CSF, cerebrospinal fluid.

■ Metabolic consequences of BINT are similar to non-blast TBI. In brief, BINT causes a deleterious cascade in a number of biochemical processes, leading to hyperglycemia, ischemia, inflammation, hormonal change, oxidative stress, and mitochondrial dysfunction. Mitochondrial injury results in free radical oxidative stress, causing membrane lipid peroxidation that can be seen as an alteration in nitric oxide synthetase.[56] Ruptured erythrocytes release hemoglobin, which further amplifies the oxidative stress. Hyperglycemia usually develops within minutes of the injury and exaggerates ischemic brain damage by enhancing formation of tissue lactic acid (metabolic acidosis) and impairing normal phosphorus metabolism. Inflammation also plays an important role in blast TBI.[56] Leukocytosis is found in trauma and stress, and both clearly occur on the battlefield. Indeed, a significant elevation in the white blood cell (WBC) count, even with minimal signs of injury initially, should heighten suspicion for the possibility of an underlying occult injury.[57] Further, the stressful insult from blast trauma causes a sudden release of catecholamines, which may also lead to a leukocytosis. Pro-inflammatory molecules such as nitric oxide, prostaglandins, oxygen free radicals, glutamate, and inflammatory cytokines break down the blood–brain barrier and contribute to brain edema, as will be emphasized later.[58] Finally, multiple organ systems are affected due to activation of the hypothalamic-pituitary-adrenal axis and significant hormonal changes in the brain.[59] Needless to say, this multisystem metabolic mayhem creates a very hostile environment for the injured brain.

"HERE EDEMA, THERE EDEMA, EVERYWHERE EDEMA, EDEMA!"

Primary blast injuries are characterized by tissue swelling and edema. In the extremities, fasciotomies are often performed to prophylax against

or relieve impaired perfusion from a compartment syndrome (**Figs. 3.8** and **3.13**). The GI tract is notable for intestinal edema, and an *open abdomen* may be necessary to relieve abdominal compartment syndrome and to protect against bowel ischemia (**Figs. 3.8** and **5.36**).[60,61] To reduce edema, hasten primary closure, promote healing, and reduce infection, sequential fascial closure in conjunction with the wound vacuum-assisted closure (VAC) has been very helpful.[62,63] This technique was first described in 1997 and entails applying subatmospheric pressure to a wound through a porous dressing in a controlled manner. The wound VAC has revolutionized the treatment of combat wound care. It should be remembered that increased abdominal pressure/compartment syndrome can increase ICP and exacerbate secondary injury from TBI.

In the lungs, pulmonary edema (*blast lung*) can occur (**Fig. 3.9**). Pulmonary neutrophils undergo diapedesis from the lung microvasculature and release enzymes and oxygen free radicals into the surrounding perivascular space and epithelial membranes of the lung alveoli. Enzymes and free radicals disrupt endothelial junctions of the microvasculature and epithelial integrity of the lung alveoli, thus permitting an exudate of protein-rich fluid to enter the lungs. Inhalation of toxic smoke (4° blast injury) leads to the release of inflammatory mediators, which through vasoactive mechanisms increase pulmonary artery pressures. Increased pulmonary artery pressures in turn cause further secondary damage to the respiratory epithelium and the release of additional mediators, such as tumor necrosis factor (TNF). Release of inflammatory molecules into the systemic vasculature causes injury to other organs, including the brain, particularly if there is breakdown of the blood–brain barrier.[44,64]

The brain injuries following blast exposure are also characterized by a disproportionate amount of cerebral edema relative to other injuries such as hemorrhage (**Figs. 3.10** to **3.12**). Indeed, early radical decompression via a large (>12 cm) craniectomy is a common procedure in TBI patients with refractory intracranial hypertension following blast trauma (**Figs. 3.6**, **3.10B**, **3.12**, **3.18**, **5.11**, **5.12**, **5.15**, and **5.20**). Over 30% of wartime cranial surgeries have been decompressive craniectomies (DCs).[65] The DC better enables the safe transfer of neurologically unstable patients from the battlefield to tertiary military hospitals, which may be located 10 to 18 hours from the war zone. This aeromedical transfer occurs without the aid of a neurosurgeon or neurocritical care specialist. Unpredictable elevations in ICP that would otherwise entail intensive bedside management by physicians may be circumvented by a DC. Without a DC, the edema and swelling of blast injury would require casualties to be placed in barbiturate-induced coma, which, together with the attendant, need for continuous EEG monitoring would be difficult to execute during aeromedical transport in a battlefield setting. It is not known why cerebral edema and cerebral hyperemia appear to be more common in combat compared to civilian trauma. Theories for increased brain edema include (1) venous hypertension because veins may be particularly vulnerable to the blast force, (2) breakdown of the blood–brain barrier (note that blast-induced oxidative stress activates matrix metalloproteinases and aquaporins, promoting loosening of the vascular unit, enhanced leakiness of the blood–brain barrier, and progression of neuroinflammation), (3) cerebral dysautoregulation, (4) cytotoxicity from the previously mentioned ischemic and inflammatory mediators, and (5) increased susceptibility to aggressive, or even normal, fluid resuscitation.[44,66] Hyperemic cerebral swelling is a known manifestation of cerebral dysautoregulation, and it is more common in younger patients than older patients.[67] This may partially explain why cerebral edema and hyperemia are more common in the younger combat population than in the older civilian TBI population. Additional neuroimaging manifestations of BINT are discussed in Chapter 5, Lesson 4.

Hyperemic Cerebral Swelling and Scalp Edema (IED Blast Injury)

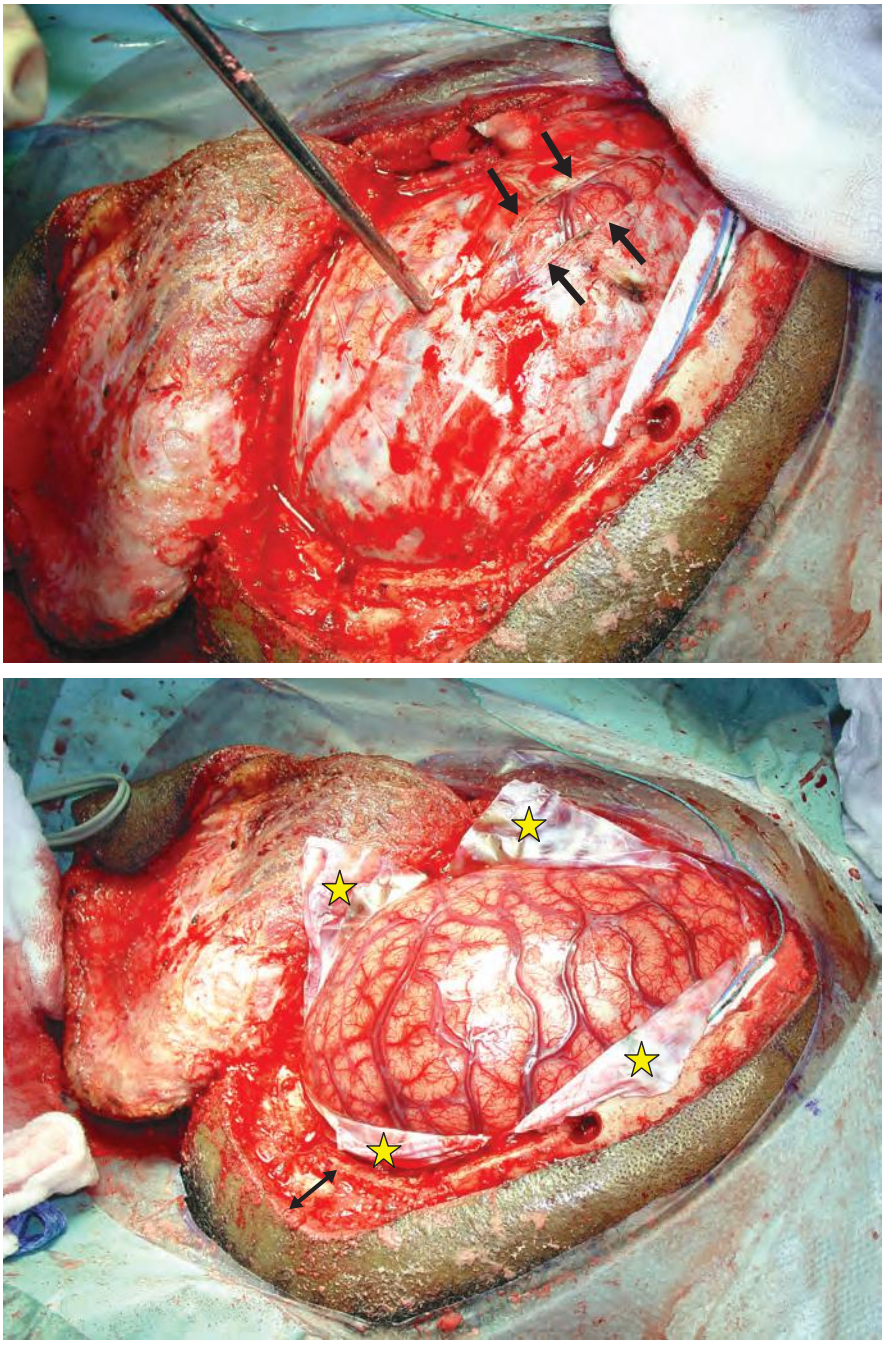


Figure 3.6. Hyperemic Cerebral Swelling (IED Blast Trauma). A. Intraoperative photograph showing oozing from numerous dural vessels in the epidural space. At this stage in the decompressive hemicraniectomy, the dura has a small slit-like opening (*arrows*), and swelling of the underlying brain is evidenced by splaying of this slit incision. **B.** After complete opening of the dura, severe hyperemic holohemispheric cerebral swelling is noted with external herniation through the durotomy defect. Note how the brain surface appears flat. This is due to diffuse sulcal effacement and loss of normal cortical convolutions. The reflected stellate dural leaflets (*stars*) and marked swelling of the scalp soft tissues (*double-headed arrow*) are also noted.

Cerebral Edema and Scalp Swelling Are Common in Blast Trauma

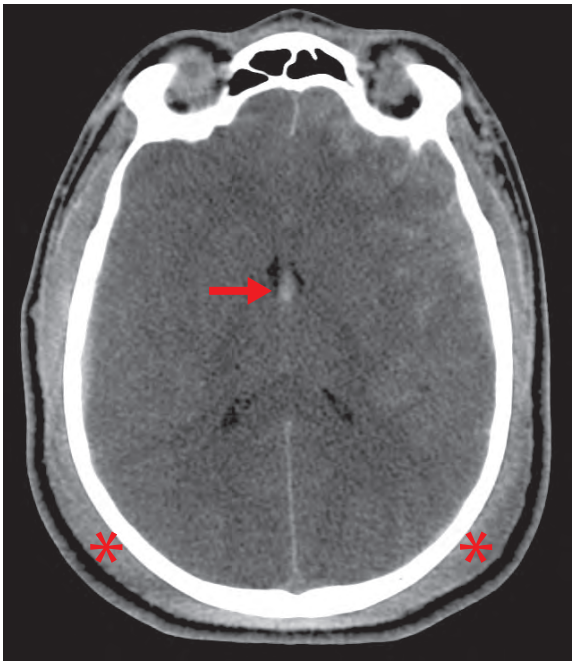


Figure 3.7. IED Blast Trauma with Diffuse Cerebral Edema and Scalp Swelling. Noncontrast axial computed tomography (CT) image demonstrates complete effacement of the cerebral sulci and partial effacement of the ventricular system. There is subtle loss of the gray–white matter differentiation, consistent with cerebral *edema* (as opposed to *hyperemia*). In cerebral hyperemia, there is relative preservation of the gray–white matter differentiation in addition sulcal effacement. Subtle heterogeneity throughout the left frontotemporal region is present, consistent with a small amount of subarachnoid hemorrhage and early cortical contusions. Focal high density is seen in the fornix, consistent with hemorrhagic TAI (*arrow*). Note also extensive scalp soft tissue swelling (*asterisk*).

★**KEY POINTS** Cerebral swelling (both edema and hyperemia) are manifestations of cerebrovascular dysautoregulation. It is more common in younger patients than older patients. This may partially explain why it appears to be more common in combat than in civilian TBI.

Soft Tissue Edema Is Common in Blast Trauma

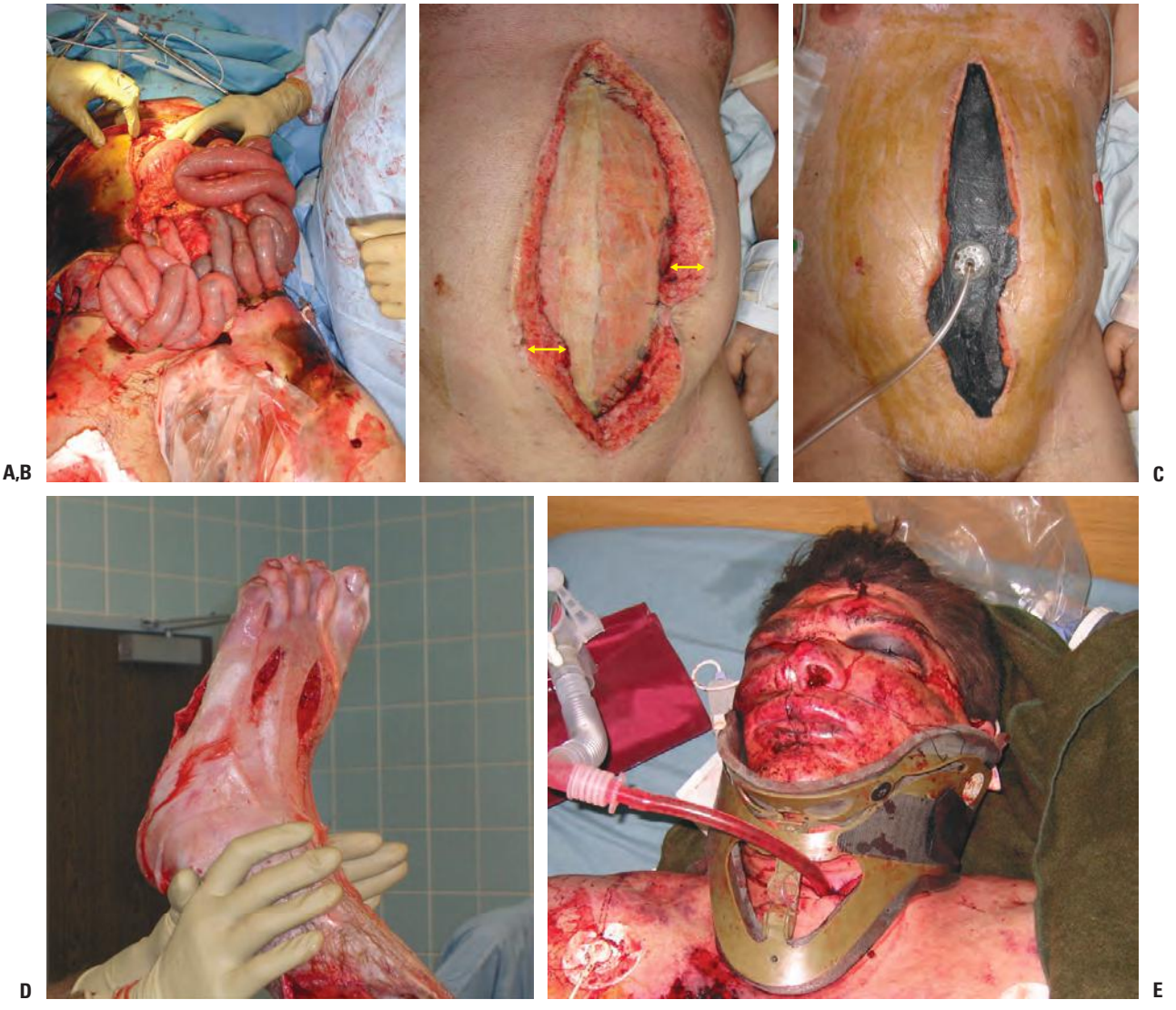


Figure 3.8. IED Blast Trauma and Tissue Edema. All of the patients in the figure were victims of an IED explosion. **A.** Intestinal hemorrhagic edema (note also cutaneous thermal injury). His abdomen could not be closed due the severe swelling and potential risk for an abdominal compartment syndrome due to the inelastic rectus abdominis sheath. **B.** A different patient status-post partial closure of his edematous abdomen showing an *open abdomen* before and **(C)** after VAC. In addition to intra-abdominal swelling, there is marked edema of the anterior abdominal wall (*arrows*) that decreases following application of the wound VAC. Please note that this soldier is *not* overweight. **D.** Fasciotomies in an edematous foot were performed to reduce the chance of a lower extremity compartment syndrome. **E.** Facial hemorrhagic edema necessitating an emergency tracheostomy. Note also cervical spine injury immobilization, making patient management even more difficult.

★**KEY POINT** Elevated intra-abdominal pressure can increase ICP in patients with TBI.

Blast Lung and Foreign Body Embolus

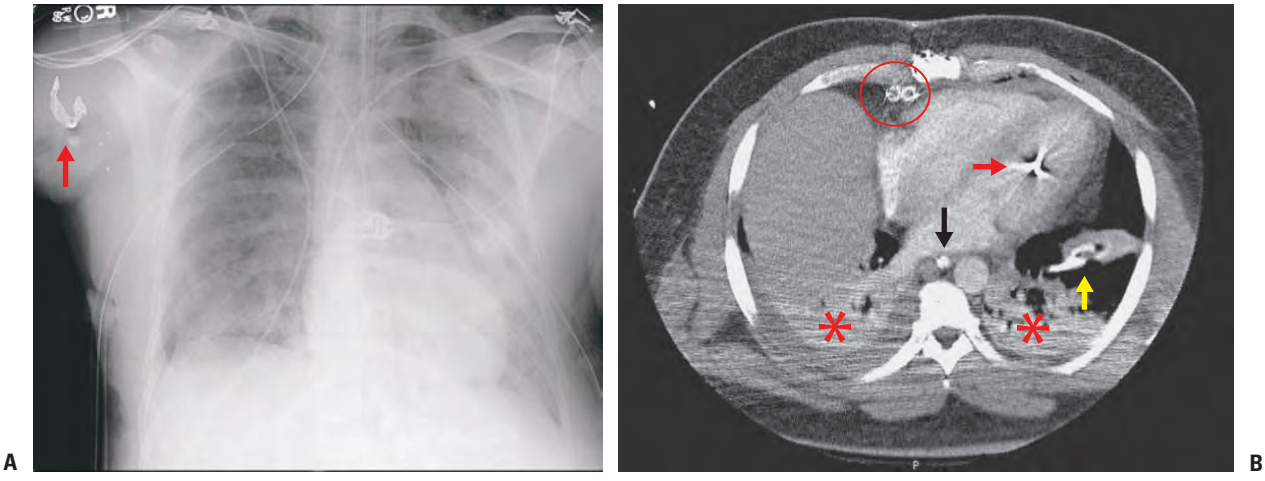


Figure 3.9. **Blast Lung. A.** Frontal chest radiograph shows bilateral air-space opacities within the lungs, bilateral thoracostomy tubes, endotracheal tube (ET) tube, pneumomediastinum, traumatic right upper extremity amputation (*arrow*), left central venous pressure catheter, normal heart size, and normal mediastinum. These imaging findings are typical of *capillary leak* found in blast lung. **B.** Axial contrast–enhanced thoracic CT through the heart shows a metallic foreign body within the left ventricle, consistent with a foreign body embolus (*red arrow*). Metallic artifact is also seen within the sternum, consistent with median sternotomy wires. Mediastinal thoracostomy tubes are present in the right cardiophrenic space (*circle*) and a pleural thoracostomy tube courses through the left major fissure (*yellow arrow*). An enteric tube is present within the esophagus (*black arrow*). Extensive basal consolidation (*asterisk*) and small bilateral pleural effusions are seen, and a small amount of pericardial fluid is present.

★**KEY POINT** Blast lung is a combination of pulmonary contusion, hemorrhage, and edema with damage to alveoli and blood vessels. It is the primary cause of death among people who initially survive an explosion.

Cerebral Edema and Hyperemia Are Common in Blast Brain Injury

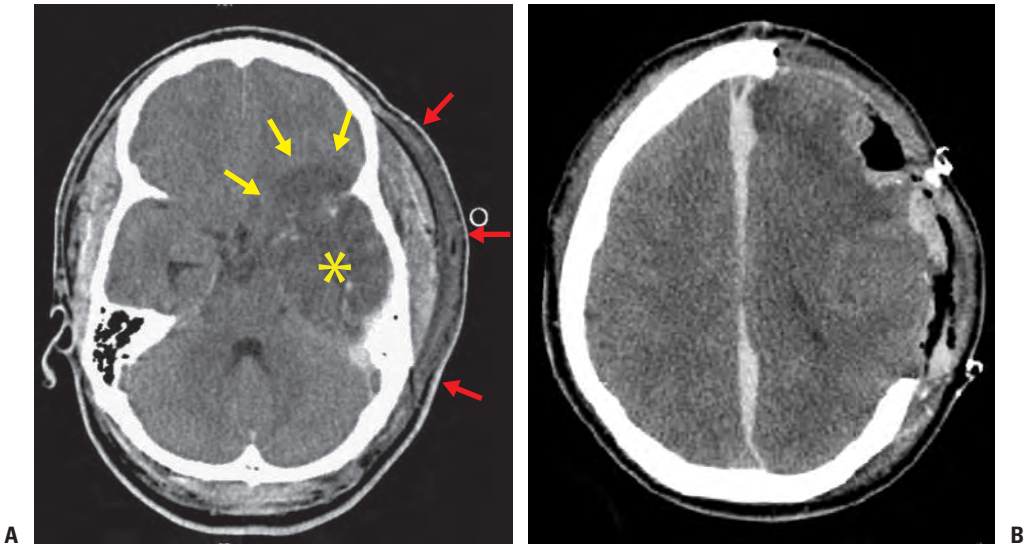


Figure 3.10. Left Temporal Lobe Edema and Decompressive Craniectomy. A. Noncontrast axial CT shows low density within the left temporal lobe (*asterisk*) and posterior orbitofrontal lobe (*yellow arrows*), consistent with cerebral edema. Extensive overlying subgaleal scalp soft tissue swelling is present (*red arrows*). Note the relative paucity of intra-axial hemorrhage and disproportionate cerebral edema. Given the proximity of the blast exposure to the left temporal region, the temporal and orbitofrontal edema may be attributable to venous hypertension from injury to the left vein of Labbé. **B.** Noncontrast postoperative CT demonstrates an interval left decompressive craniectomy with effaced cerebral sulci, loss of gray–white matter differentiation in the left hemisphere, and a small interhemispheric subdural hematoma.

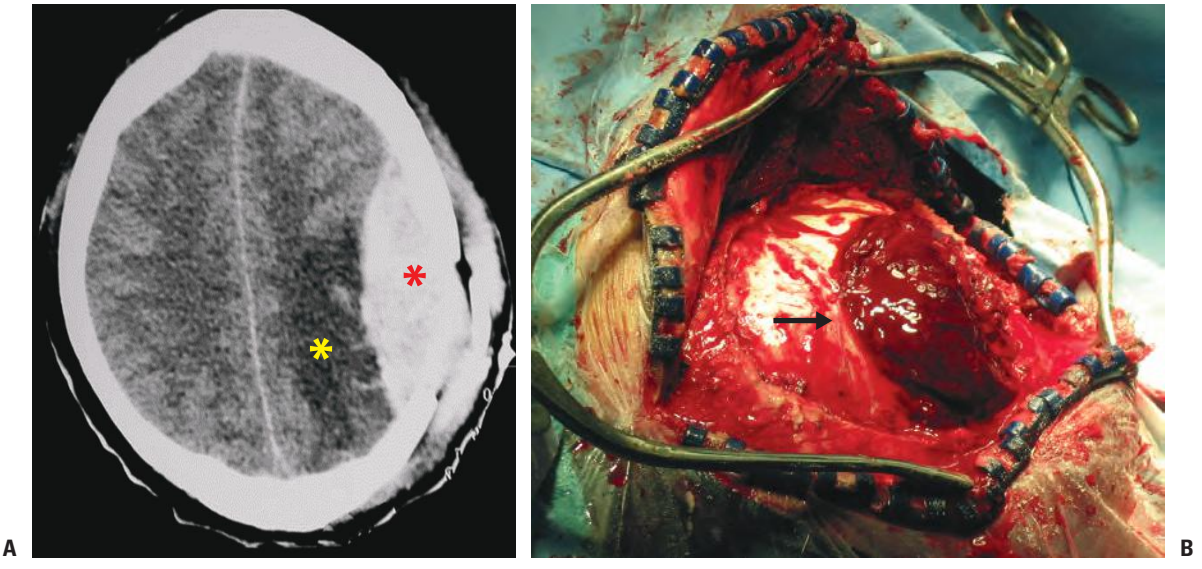


Figure 3.11. Epidural Hematoma (EDH) with Disproportionate Cerebral Edema. A. Noncontrast CT demonstrates an epidural hematoma (*red asterisk*). Unlike an epidural hematoma that is encountered in civilian TBI, this EDH is associated with prominent parenchymal edema (*yellow asterisk*). **B.** Intraoperative photograph shows the large acute clot (*arrow*) located on top of the dura.

Cerebral Edema and Hyperemia Are Common in Blast Brain Injury

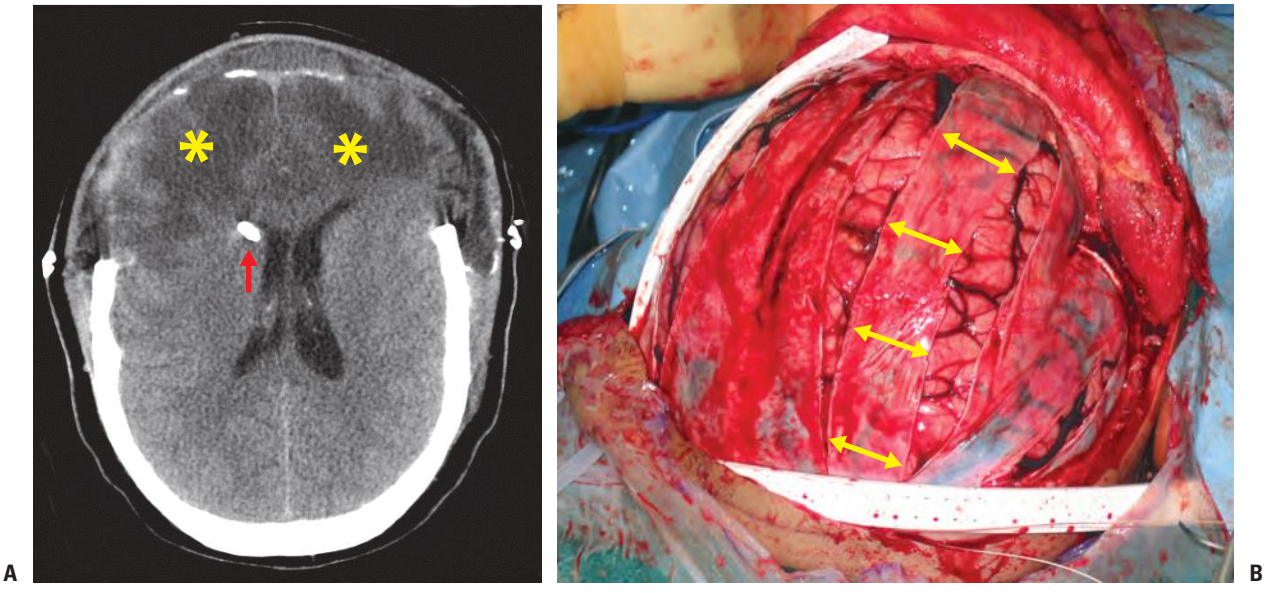


Figure 3.12. **Decompressive Craniectomy. A.** Postoperative noncontrast axial CT shows a bifrontal decompressive craniectomy with extensive subjacent frontal lobe edema (*asterisk*) and external herniation. A right frontal external ventricular drain (EVD) is present (*arrow*). The preoperative CT exam showed bifrontal subdural hemorrhage. **B.** Intraoperative photograph shows parallel surgical slits in the dura (*lattice durotomy*) designed to expose and facilitate aspiration of an acute subdural hematoma while preventing malignant brain swelling (*arrows*).

Secondary Blast Injury

Secondary blast injuries are penetrating and/or perforating injuries that result from flying debris propelled by the intense release of energy from the explosion. Penetrating trauma is found in nearly 70% of battlefield wounds, in contrast to 11% in civilian trauma.[68] The propelled foreign bodies are often loosely referred to as shrapnel. More precisely, however, shrapnel should be reserved for the obsolete high-explosive shell fragmentation named after the English artillery officer, Major General Henry Shrapnel (1761–1842). Because of the explosive characteristics of shrapnel-producing IEDs, multiple penetrating injuries are the rule. The projectiles can arise from the exploded container, objects deliberately placed in the container, and from material surrounding the detonator and target. Patients injured in suicide bomb attacks are at risk of not only penetrating injury from the blast fragments but also transmission of biologic contaminants from "body part missiles."

On physical examination, shrapnel injury appears as innumerable wounds peppering the patient's skin surface (**Figs. 3.13**, **5.64**, and **5.77**). Unlike the neck and face, the brain is fairly well protected from penetrating injuries by the presence of two "helmets" (i.e., the skull and combat helmet). However, focal impact by the projectile can sometimes cause the inner layer of the helmet to delaminate and impact the underlying scalp and skull. This creates a piston-like, high-energy impact that can reverberate through the brain tissue and cranial vault and cause a cerebral concussion. If the patient is not wearing protective headgear at the time of the injury (e.g., terrorist attack or natural disaster), the skull alone may not afford adequate protection (**Figs. 3.14** and **3.15**). Penetrating TBI is discussed in detail in Chapter 4.

In general, the low-velocity wounding characteristics of shrapnel, as opposed to the high velocity characteristics of military rifle wounds, help to explain the comparatively high survival rates of patients with penetrating shrapnel injuries. Shrapnel travels at about 185 m per second, handgun munitions travel at about 245 to 425 m per second, and modern rifle rounds have velocities exceeding 760 m per second. Some warheads can cause damage without penetration. For example, the subsonic detonation of a large "blob" of plastic explosive that smashes against a tank can create a shock wave that passes through the vehicle wall and throws off large scabs of metal spall when the shock wave reaches the far wall. This shock wave can also cause secondary explosions and fires involving the munitions inside the vehicle. As a general rule, all wounds incurred on the battlefield are grossly contaminated. Such wounds should not be closed primarily because they will become infected unless appropriate treatment (including debridement and antibiotic administration) is rapidly initiated.

Secondary Blast Injury (Typical Fragmentation Wounds)

Figure 3.13. **IED Penetrating Wounds. A.** Intraoperative photograph demonstrating innumerable penetrating lesions to the lower extremities with several fasciotomies (*arrows*). Note characteristic sparing of the torso due to protection by body armor. **B.** The edematous nature of the injury is illustrated by multiple fasciotomies and "weeping" wounds (*black arrows*). Note the handwriting that is seen directly on the wound dressing (*red arrows*). Because of the rapid transport of patients from many treatment sites in a very short time (described in Chapter 5, Lesson 2), this is a safe and effective way to communicate combat care.

★**KEY POINT** Blast injuries are typically contaminated, and multiple episodes of debriding and wound washouts are necessary to prevent infection.

Secondary Blast Brain Injury

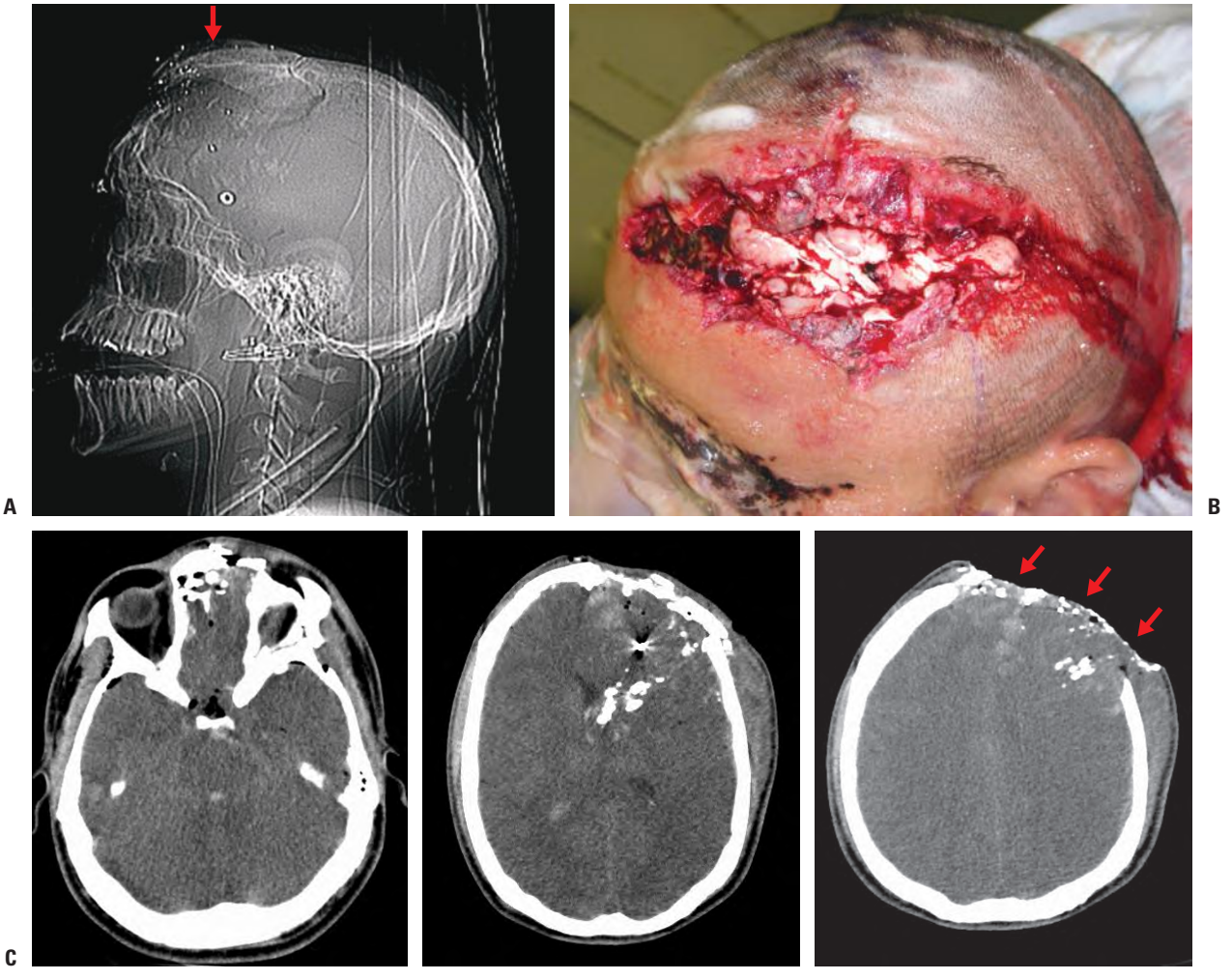


Figure 3.14. Secondary Blast TBI due to IED Explosion. A. Lateral scout view demonstrates a comminuted, superiorly displaced frontal fracture with multiple radiopaque foreign bodies. The appearance literally looks as if the affected region exploded off the top of the head. **B.** Intraoperative photograph shows an extensively comminuted skull fracture with exposed bone. **C.** Axial CT imaging reveals complete effacement of the perimesencephalic cisterns and cerebral sulci. Bone, air, and metallic fragments are displaced into the left frontal lobe, and focal scalp degloving is present (*arrows*). Ominous loss of gray–white matter differentiation is noted, consistent with diffuse cerebral edema. This case is similar to the *keyhole* fracture (see **Fig. 4.37**), in which a projectile strikes the calvarium in a tangential fashion, causing perpendicular implosion of fragments into the brain and severe brain injury.

Intracranial Shrapnel

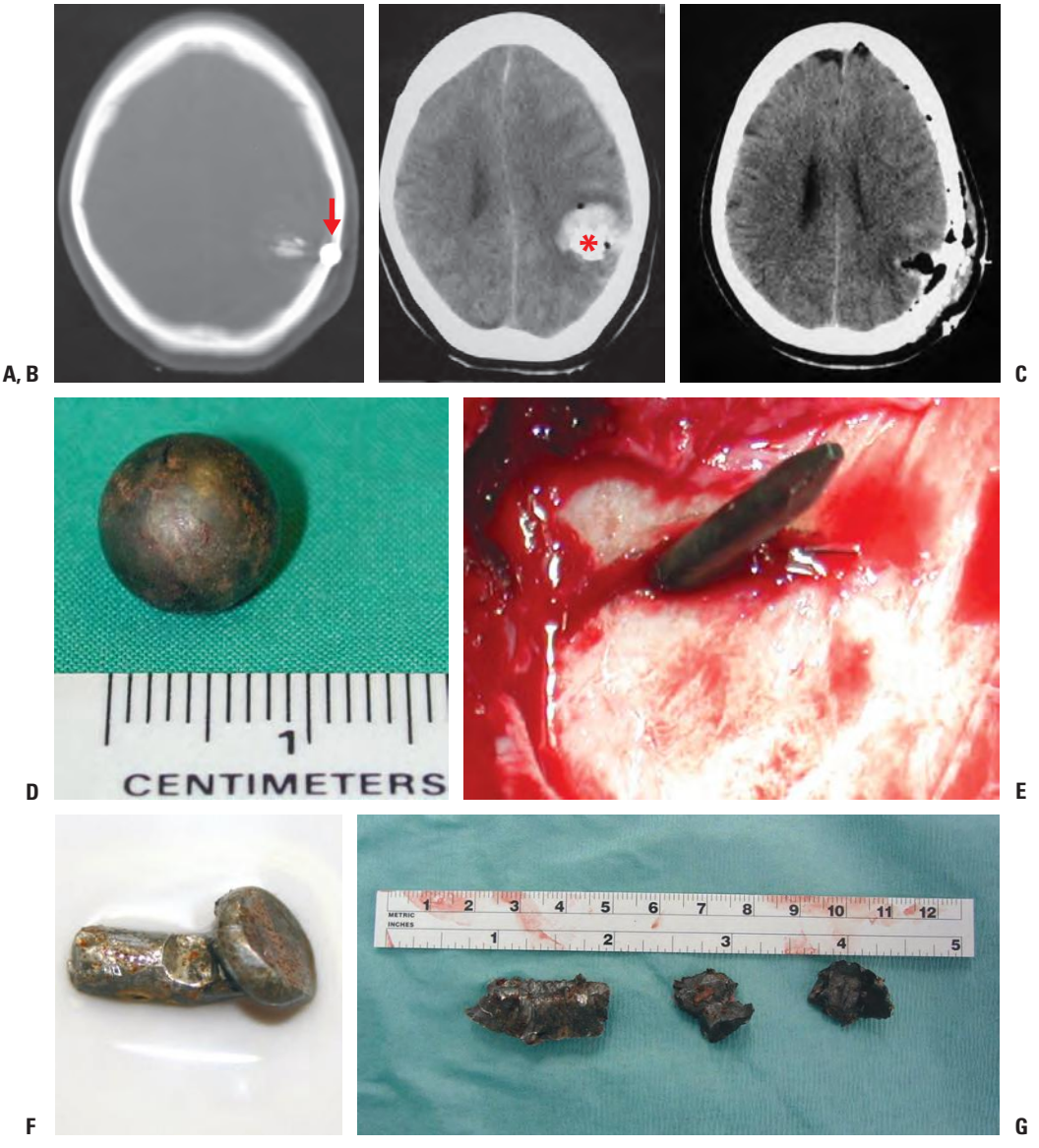


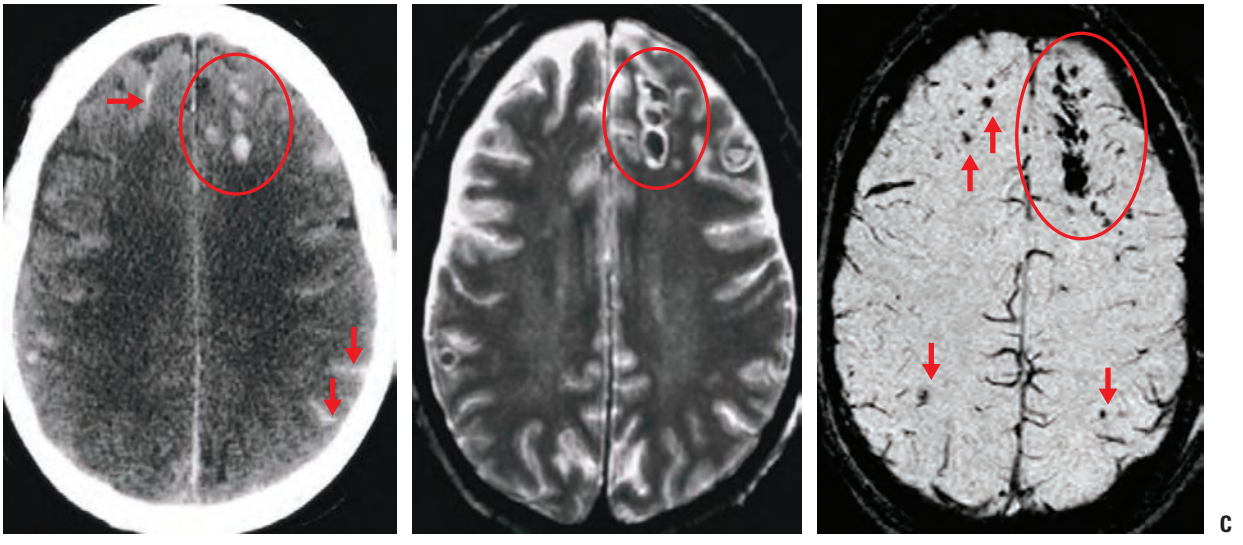
Figure 3.15. **Intracranial Foreign Bodies.** This young victim of a terrorist bomb blast presented to the emergency department with a Glasgow Coma Scale (GCS) of 14. Nobody suspected that she had suffered a head injury until she had a seizure. **A, B.** Preoperative noncontrast images demonstrate a ball bearing (*red arrow*) embedded in the left parietal calvarium with subjacent hemorrhage and imploded bone fragments (*asterisk*). **C.** Postoperative CT shows removal of the foreign body, evacuation of accessible bone fragments, and a small amount of parenchymal edema and pneumocephalus. **D.** Photograph of the foreign body. Other objects placed in terrorist bombs include **(E)** nails (pictured here embedded in the inner table of the skull), **(F)** metal bolt, and **(G)** sharp metal fragments and rocks.

★**KEY POINT** Contaminated wounds are characteristic, and the entry wound on clinical exam can be surprisingly subtle (see also **Figs. 5.65** and **5.72**).

Tertiary Blast Injury

Tertiary blast injuries result from the victim being thrown by the blast force and/or crushed by collapse of a structure (**Figs. 3.16** to **3.20**). These injuries resemble the typical blunt traumatic injuries found in urban civilian trauma, such as that following a motor vehicle accident (MVA), fall, or assault. It should be remembered that, in addition to the more esoteric blast injury, troops are susceptible to the same types of rapid acceleration/deceleration injuries found in civilian TBI, such as TAI and coup–contrecoup hemorrhage. While helmets reduce the likelihood of TBI from direct contact, they may not protect against the blast wave force nor do they significantly reduce rotational acceleration/deceleration injuries.[69] Additional examples detailing imaging findings found in blast brain injury are illustrated in Chapter 5, Lessons 4 to 12.

Traumatic Axonal Injury



A, B C

Figure 3.16. Traumatic Axonal Injury. A 33-year-old male status-post-vehicle rollover. **A.** Noncontrast CT scan on admission demonstrates multifocal subarachnoid hemorrhage (*arrows*) and several small intra-axial hemorrhages within the left frontal subcortical white matter (*circle*), consistent with hemorrhagic TAI. **B.** Axial T2-weighted image obtained 4 days after injury shows hypointensity within the lesions, consistent with acute deoxyhemoglobin (*circle*). The thin halo of hyperintensity surrounding the lesions represents vasogenic edema. **C.** Susceptibility-weighted imaging (SWI) at a slightly higher level better demonstrates the extent of TAI (*circle*). Note additional foci (*arrows*) not evident on the T2-weighted sequence shown in **(B)**.

★**KEY POINT** As in civilian TBI, SWI is currently the MRI sequence of choice for identifying recent and/or remote intracranial hemorrhage. SWI is three to six times more sensitive than conventional gradient-recalled echo (GRE) imaging in detecting hemorrhagic lesions and reveals about twice the aggregate hemorrhage volume.[70]

Tertiary Blast Trauma (Crush Injury)

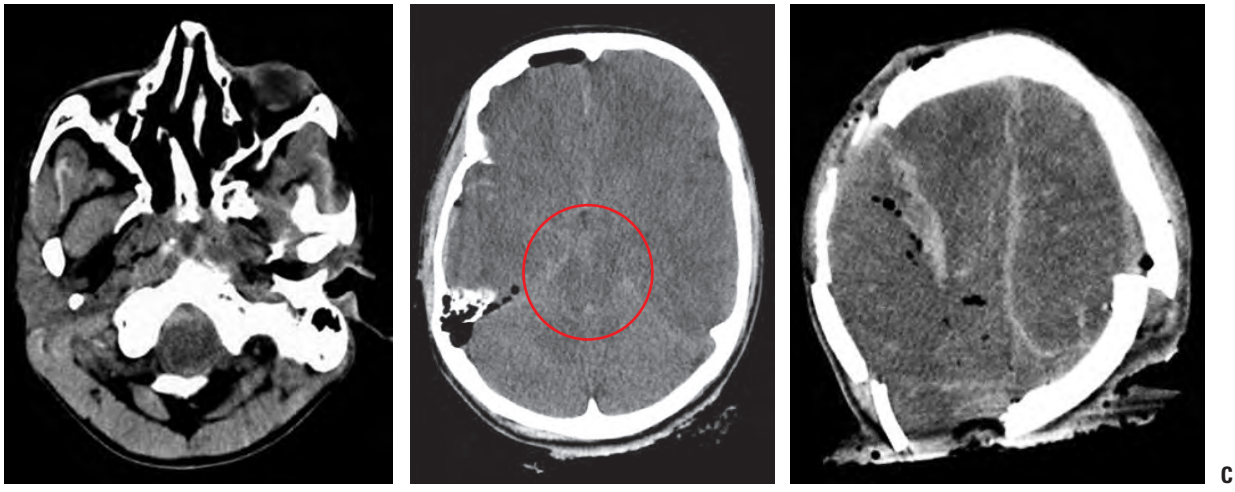
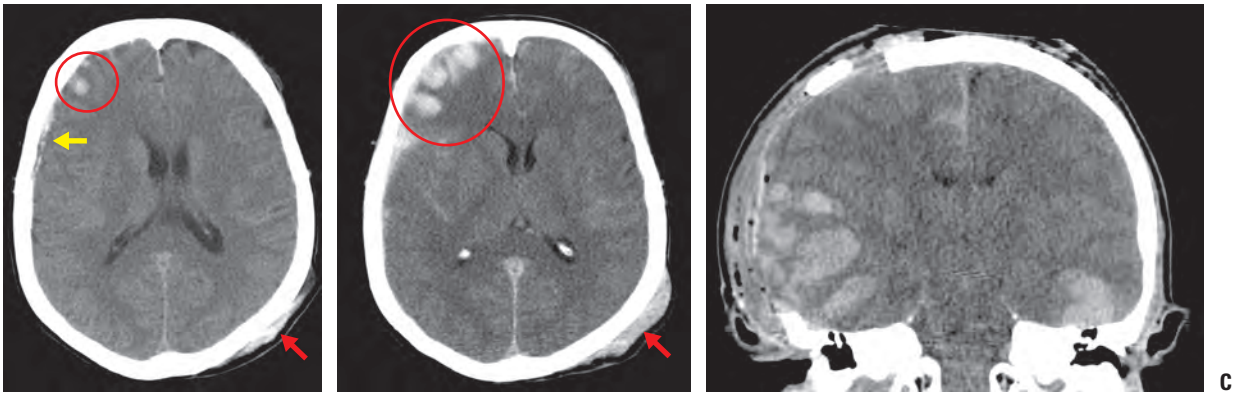


Figure 3.17. IED Blast Injury with Cranial Crush Trauma. Noncontrast axial CT images show a comminuted compound fracture of the calvarial convexity. The sagittal sinus is likely disrupted as judged by the location of the displaced fractures and severity of injury. In other less severe cases, a computed tomography venogram (CTV) may be helpful to exclude dural sinus injury. Note loss of gray–white matter differentiation (i.e., diffuse cerebral edema), complete effacement of basal cisterns (*circle*) and cerebral sulci, multifocal pneumocephalus, and subarachnoid and subdural hemorrhage. He quickly succumbed to his injuries. (Courtesy of Roni Rooks, MD, LTC, MC.)

★**KEY POINT** Disruption of a major dural venous sinus can result in venous hypertension and exacerbate intracranial hypertension.

Tertiary Blast Trauma (Enlarging Contusions)



A, B C

Figure 3.18. Enlarging Contusions. A. A 28-year-old soldier whose high mobility multipurpose wheeled vehicle (HMMWV), commonly known as a *Humvee*, was hit by a roadside bomb. His presenting GCS was 14, and his CT scan was normal. Noncontrast CT on admission shows a small right frontal contrecoup contusion (*circle*) and subdural hematoma (*yellow arrow*), causing minimal mass effect. The *coup* site (i.e., impact location) manifests as mild subgaleal scalp soft tissue swelling in the left occipital region (*red arrow*). **B.** The 2-hour follow-up CT shows interval increase in both the size of the intracranial and extracranial hemorrhages. Because of medically refractory intracranial hypertension, the soldier underwent a decompressive hemicraniectomy. **C.** Postoperative coronal reformatted CT shows the right hemicraniectomy and development of new hemorrhages within both temporal lobes, causing mild right external herniation.

★**KEY POINT** The number of decompressive craniectomies performed during this conflict is unprecedented.

Tertiary Blast Trauma (Crush Injury)

Compartment syndrome and rhabdomyolysis can complicate musculoskeletal crush injuries, especially in the setting of a collapsed building and/or prolonged extrication.[71,72] Being trapped in the rubble for long periods of time is less common in modern war zones than in prior conflicts, but it is not uncommon in natural disasters such as an earthquake. A fasciotomy can sometimes prevent compartment syndrome, but amputation may ultimately be necessary. Crush injuries cause muscle breakdown, which releases potassium and creatine kinase/myoglobin into the blood. The electrolyte, metabolic, and viscosity abnormalities result in cardiac arrhythmias and impaired renal function, both of which can exacerbate secondary mechanisms of injury in the wounded brain. Furthermore, cardiac and kidney dysfunction add significant complexity to the management of the severe TBI patient. Crush injuries often lead to hypothermia from exposure of open wounds and from the volume of intravenous (IV) fluids required for the resuscitation. Hypothermia has a favorable effect on ICP, but it may also contribute to other pathophysiologic events, such as coagulopathy, that may worsen secondary brain injury. The most devastating systemic effects can occur when the crushing pressure is suddenly released without proper preparation of the patient, causing *reperfusion syndrome*.[73] In this setting, the patient, with pain control, may be cheerful before extrication but die shortly thereafter.

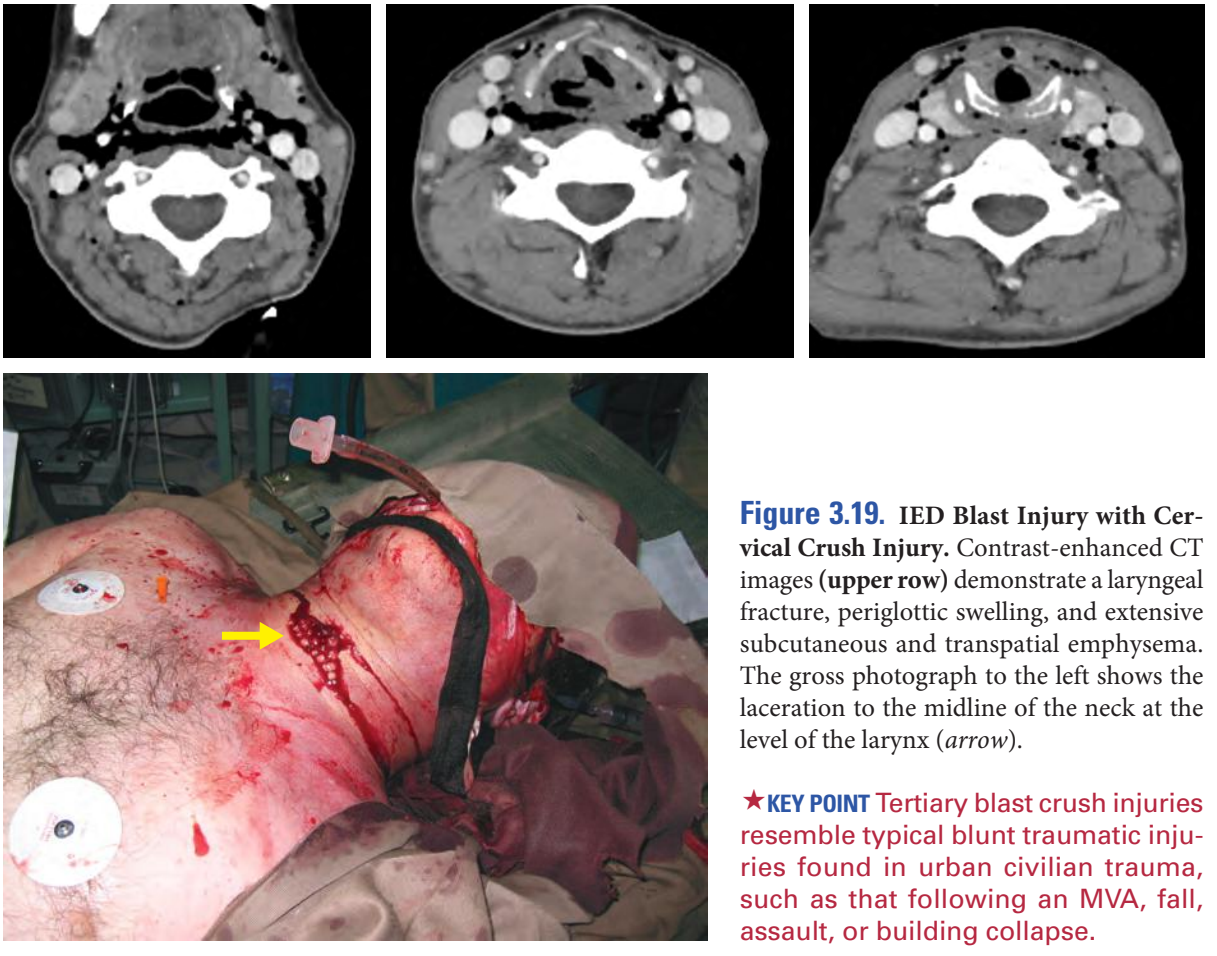


Figure 3.19. IED Blast Injury with Cervical Crush Injury. Contrast-enhanced CT images **(upper row)** demonstrate a laryngeal fracture, periglottic swelling, and extensive subcutaneous and transpatial emphysema. The gross photograph to the left shows the laceration to the midline of the neck at the level of the larynx (*arrow*).

★**KEY POINT** Tertiary blast crush injuries resemble typical blunt traumatic injuries found in urban civilian trauma, such as that following an MVA, fall, assault, or building collapse.

Tertiary Blast Trauma (Remote Traumatic Axonal Injury)

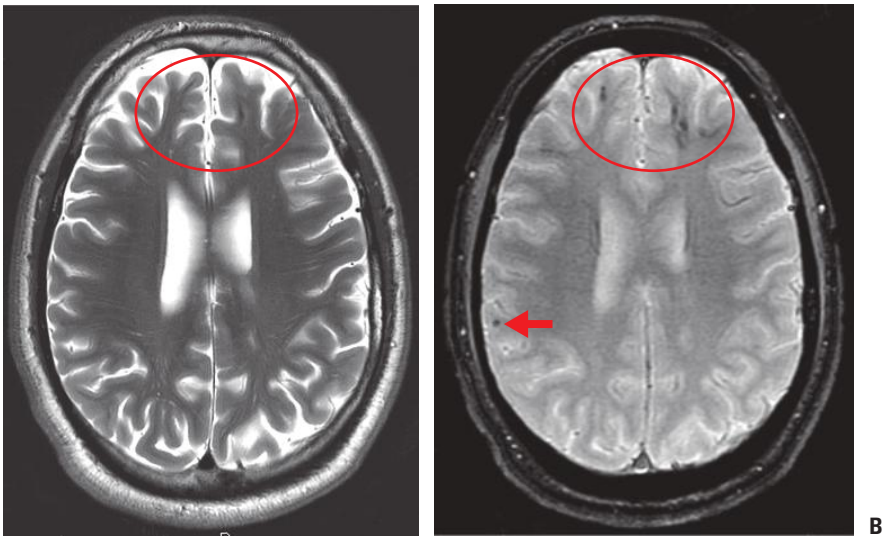


Figure 3.20. Remote Traumatic Axonal Injury not Evident on Admission CT and Only Visualized with T2* Gradient MRI. A. Axial T2-weighted and **(B)** T2* gradient MR images obtained 1 year after the injury demonstrate numerous bilateral microhemorrhages within the subcortical white matter of the frontal lobes (*circle*) and a single focus within the right parietal subcortical white matter (*arrow*). Diffuse cerebral volume loss is also evident as judged by abnormal prominence of ventricles and sulci for a patient this age. As expected, the TAI lesions are more conspicuous on the T2* gradient sequence than on the T2 spin-echo sequence.

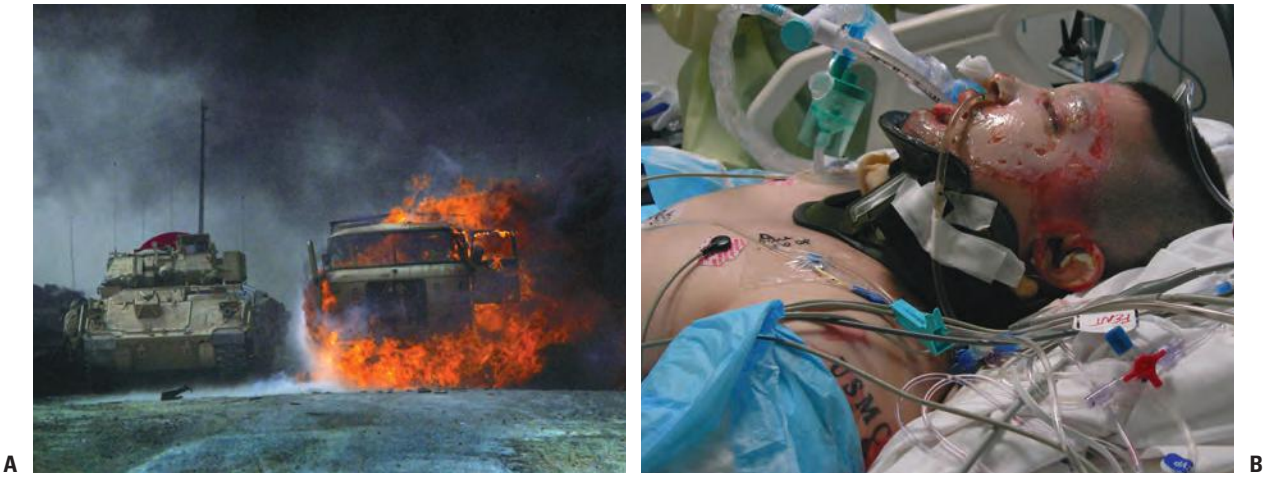
★**KEY POINT** The imaging appearance of TAI suffered in combat is similar to the imaging appearance of TAI suffered in civilian blunt TBI.

Quaternary Blast Injury

Quaternary blast trauma refers to all other explosion-related injuries, including chemical or thermal burns, toxic inhalation, radiation exposure, and asphyxiation. Most quaternary blast injury is an incendiary thermal injury resulting in varying degrees of burn severity. At the epicenter of the detonation, the temperature rises precipitously, heating the surrounding air and creating a highly luminous spherical mass of air and gaseous weapon residues, termed the **fireball** (**Figs. 3.1**, **3.21**, and **3.23**). Victims close to the detonation sustain third-degree burns that can be fatal. The majority of survivable thermal injuries are due to exposure to the shock wave's heated air, not to the fireball itself. As with 2° blast injury, 4° blast injury typically involves the exposed parts of the body, including the face, neck, and hands (**Figs. 3.21B**, **4.7B**, **5.47**, **5.53**, and **5.77**). Mortality from major burns has significantly decreased during the past several decades. In earlier years, over half of all victims died if the burn involved >50% total body surface area (TBSA). Today, burns involving >90% TBSA are survivable.[74] Inhalation injury, however, still constitutes one of the most critical adverse factors after thermal insult. Inhalation injury is associated with a mortality rate of 25% to 50% when patients require ventilator support for more than 1 week after injury.[75]

Burns associated with IED explosions behave differently from typical domestic burns, possibly due to a compounding effect of the thermal injury superimposed on the 1°, 2°, and 3° blast injuries. Inhalation injury is caused by steam or toxic inhalants generated from the bomb contents that result in increased pulmonary microvascular hyperpermeability and edema formation, atelectasis, and tracheobronchitis.[76] In particular, phosgene-like combustion by-products from the Teflon-coated interiors of armored vehicles may cause devastating pulmonary injury. In addition, a recent article in the *New England Journal of Medicine* described how soldiers returning from Iraq and Afghanistan have developed a "diffuse constrictive bronchiolitis" thought attributable to inhalational exposure.[77] It is well known that pulmonary insufficiency contributes to poor outcomes after severe TBI.[78] Burn injury and how it contributes to brain injury are discussed further in Chapter 5, Lesson 7.

Quaternary Blast Trauma (Thermal and Toxic Inhalation Injury)



A B

Figure 3.21. Quaternary Blast Injury. A. Military cargo vehicle engulfed in flames following an IED explosion. **B.** Soldier injured in a similar attack. Note severe burn injuries to the face and ear with relative sparing of the scalp and torso. The soldier is intubated because of blast inhalational injury and he is wearing a cervical collar because of cervical spine injury suffered at the time of impact.

★**KEY POINT** Burns associated with IED explosions behave differently from typical domestic burns, possibly due to a compounding effect of the thermal injury superimposed on the 1°, 2°, and 3° blast injuries.

TABLE 3.2 Factors that Determine the Severity of Blast Injury

1. Protective gear
2. Outdoor versus indoor explosion
3. Type of tissue injured
4. Distance from the explosion
5. Mass, velocity, and shape of the secondary blast fragments
6. Peak blast pressure
7. Genetic predisposition

What Determines the Severity of a Blast Injury?

1) Protective Gear (*more is not necessarily better*)

■ Body Armor

On the battlefield, soldiers must balance their need for protective coverage with the need for mobility. In addition, head gear must preserve the soldier's ability to determine distance and direction of incoming sound. Impaired mobility can prove just as fatal as ineffective armor. Current soldiers can carry up to 120 lb of gear, depending on the mission (**Fig. 3.22**). Excess weight not only decreases maneuverability and reaction times but it also increases fatigue and leads to musculoskeletal injuries that can render highly trained soldiers nondeployable. From a historic perspective, recall that knights wore rigid metallic suits of armor during the Middle Ages. Soldiers in the civil war wore bulletproof vests that were made of cast iron. In World War II, the Air Force issued *flak jackets* that consisted of steel plates sewn into cloth and hung over the chest and stomach like a baseball player's catcher chest protector. After the war, research continued to make flak jackets lighter and better. During the Vietnam War, soldiers wore flak vests that would stop shrapnel but not a bullet. They were hot, uncomfortable, heavy, and bulky, and it was extremely hard to move around while wearing them.

The development of Kevlar (DuPont, Wilmington, Delaware) and ceramic material in the 1970s made real bulletproof vests possible. In addition, Kevlar is flame resistant and provides thermal protection from blasts and fire. Today's vest is a big improvement over prior systems and consists of a very fine Kevlar weave that insulates a soldier from shrapnel and 9-mm pistol rounds. It is a modular system that can be fitted with neck, axillary, deltoid, and groin protection attachments. Protective inserts from small arms (e.g., handguns, rifles, machine guns) can be added for additional protection and are capable of stopping an AK-47 round at muzzle velocity. **Muzzle velocity** is defined as the speed a bullet has at the moment it leaves the muzzle (i.e., end) of the gun. The velocity of a bullet is always highest

Protective Body Armor

Figure 3.22. Body Armor and Vulnerable Areas following an Explosion. A. Soldiers are protected by advanced individual body armor (IBA), but certain exposed body areas remain. **B.** Humvee gunner illustrates the vulnerability of the face and extremities (*arrows*). **C.** Drawing showing the typical transtemporal and transorbital trajectory of blast wave fragments (Courtesy of Rocco Armonda, MD, Col [ret] MC, USA). **D.** Antero-posterior (AP) plain radiograph of a soldier's hand and wrist shows marked deformity and partial amputation of the digits. (*Continued*)

E

F

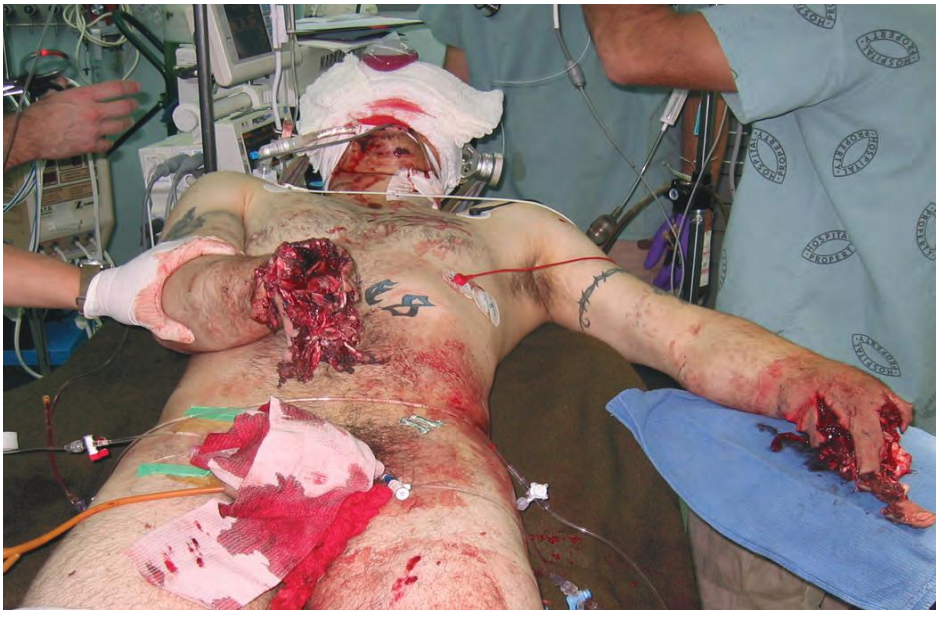


Figure 3.22. (*Continued*) **E, F.** Photographs of mutilating upper extremity blast injuries. Concomitant facial injury is also noted.

★**KEY POINT** In IED explosions, there is disproportionate injury to the face and extremities with relative sparing of the torso.

at the muzzle and drops off steadily because of air resistance. Muzzle velocities range from approximately 400 ft per second (120 m per second) to more than 4,000 ft per second (1,200 m per second) in modern rifles with high-performance cartridges, all the way to 5,700 ft per second (1,700 m per second) for tank gun ammunition (bullet nomenclature and behavior is discussed later in Chapter 4). Webbing on the front and back of the Kevlar vest permits attaching such equipment as grenades, walkie-talkies, and pistols. The vest has a quick release feature, so if the soldier needs to drop the plates, they can be instantly released.

The basic flak vest weighs approximately 25 lb but is tailored to fit the mission. If a soldier is on a peacekeeping patrol, the vest alone will work. If the mission is more dangerous, then the soldier can add plates and attachments for additional ballistic protection. There is some controversy about the ability of the vest to diffuse the blast wave. Namely, computer modeling studies done by Raul Radovitzky and colleagues at the Massachusetts Institute of Technology (MIT) Institute for Soldier Nanotechnology have shown that the vest may actually amplify, rather than deflect, the blast force. Current research is designing a sensor-activated vest that can detect a blast wave and deploy a countermeasure, much like how an air bag functions in MVAs.

Helmet

In 2003, the military introduced the Advanced Combat Helmet (ACH).[79] It currently delivers the world's most advanced ballistic, thermal, and impact head protection with adequate comfort for extended periods of use. The helmet's low-profile design reduces the risk of interference during target acquisition, and it is compatible with night-vision goggles, gas masks, and communication devices. An innovative suspension system of movable comfort pads provides customized sizing. The pads prevent sound from reverberating within the helmet, so it does not impair a soldier's ability to quickly identify the direction from which a sound is coming. In older helmets, sounds would bounce around inside the hard surface of the helmet. The ACH is a composite helmet with natural dampening. In addition, because it is lighter (it weighs roughly 3 lbs) than prior models, it improves mobility and reduces fatigue. The current helmet protects against submachine gun bullets. It should be noted, however, that although helmets are great defense against penetration injuries, they do not provide complete protection from concussion injuries, and their ability to protect against the primary blast wave is currently unknown. Recent work has shown that adding a simple face shield to the helmet can significantly reduce the incidence of TBI.[80]

Tactical Vehicles

Armored vehicles such as the HMMWV, affectionately referred to as the Humvee, have also evolved significantly over the last decade. It was originally designed as an administrative vehicle, not a combat vehicle, but because there was no urban combat vehicle at that time, it morphed into the role. The *Stryker* was later quickly fielded to fit that function. As is the case with body armor, more is not necessarily better. The heavier the vehicle, the more impermeable it is, but the weight decreases

maneuverability and reaction times. Early on in the conflict, troops were far more vulnerable to vehicle penetration by IED blasts and less connected to combat communication infrastructure. Currently, there is a new breed of Internet-centric, ruggedized tactical vehicles that are deployed and monitored during mission operations.

2) Outdoor Versus Indoor Explosion

Outside is better.[41,81] Blast injury is worse if it occurs in a confined space such as a tent, room, or Humvee than if it occurs in the open air (see **Fig. 3.3**). This is attributed to reflected, and therefore potentially enhanced, shock waves that occur indoors or within enclosed spaces. Once the initial blast wave strikes a stationary object, it can rebound with an amplification of two to nine times its original magnitude due to wave reflection and the phenomenon known as *Mach stem*. In contrast, open-air bombings allow rapid dissipation of the shock wave. Blast explosions inside buildings can also create foreign-body injuries from exploded concrete, metal, wood, and other building materials. In open air explosions, the predominant injury is penetrating soft tissue injury caused by shrapnel.

3) Type of Tissue Injured

Air-filled structures are particularly vulnerable. Because air is compressible and water is not, more distortion occurs in air-filled tissue. Thus, the lungs, intestines, and middle ear are most commonly injured. Other characteristics that modify the extent of blast injury include the "shear strain" properties of the tissue—these include its *bulk modulus* (tissue compressibility), *shear modulus* (response to shear force), and *Young's modulus* (response to linear strain force). In addition, if a projectile from the explosion hits bone, multiple secondary projectiles can arise from both the bone and shrapnel, which increases the injury. Lastly, blast or gunshot injuries to the abdomen can cause a transient increase in abdominal pressure, which can propagate a pressure wave via the inferior vena cava to the internal jugular veins and thereby cause a sudden increase in ICP. The detailed effect of the blast force on brain tissue was discussed previously.

4) Distance from the Explosion

*The closer you are, the worse the injury (see **Fig.3.23**).*[82] The intensity of a pressure wave from an explosion declines with the cube of the distance from the explosion. Therefore, a person 3 m (10 ft) from an explosion experiences nine times more overpressure than a person 6 m (20 ft) away from the blast. When the soldier is close to the explosion, thermal radiation from the fireball predominates, and devastating burns occur. At further distances from the epicenter, the thermal effects decrease and the soldier is more likely to be injured by the blast overpressure force. With even greater distance from the explosion, impact and crush injuries are more common. Individuals who are furthest from the epicenter are more likely to suffer ballistic-type penetrating injuries.

5) Mass, Velocity, and Shape of the Secondary Blast Fragments

Heavy, fast-moving, jagged objects inflict the greatest harm (**Figs. 3.15**, **4.1**, and **4.2C**). This is why IEDs are often filled with nails and ball-bearings. The injury follows basic ballistic physics (kinetic energy = $\frac{1}{2}mV^2$), which is described in greater detail in the section on gunshot injuries. Note that munitions such as grenades, artillery shells, and the "pressure cooker" IED used in the 2013 Boston

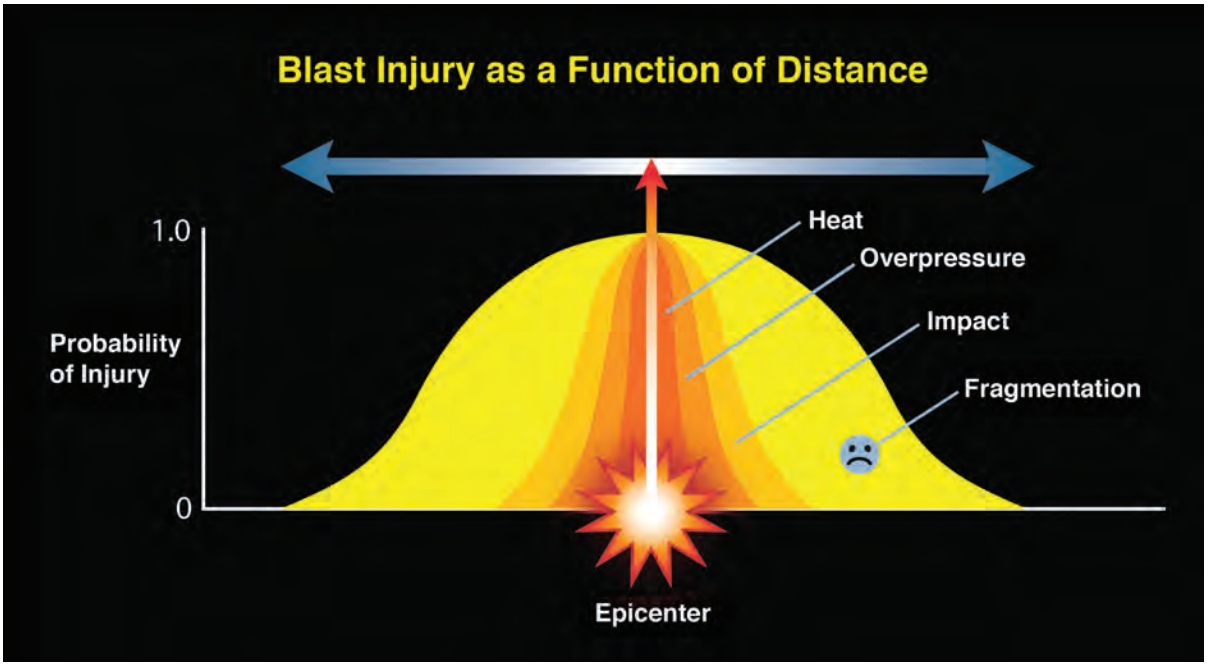


Figure 3.23. Blast Injury as a Function of Distance. This illustration shows the relationship of injuries incurred versus distance from the blast source. Casualties close to the epicenter of the explosion are likely to suffer from all four wound-causing factors. These victims are not likely to survive. Casualties within the inner zones surrounding the epicenter are more likely to suffer thermal injury and those farther away from the epicenter are more likely to experience penetrating trauma from missile fragments (☹).

bombing can dissipate a significant fraction of their potential explosive energy during the process of bursting the case and accelerating secondary fragments. These weapons likely do most of their damage via secondary blast trauma because the fragments they generate are able to cause injuries far beyond the effective range of the blast wave.

6) Peak Blast Pressure

The higher the peak pressure, the worse the injury.[83] The type and amount of combustible agent within the bomb determines the peak blast pressure and therefore the potential to inflict injury. Typically, the larger the IED, the more severe the injury. Therefore, a vehicle-borne improvised explosive device (VB-IED) (car bomb) tends to cause more damage than an isolated IED. The primary blast injury results from the initial supersonic shock wave with peak pressures up to 1,000 times greater than ATM.[84] In practice, no soldier will experience this unless he or she is an explosive ordnance disposal (EOD) specialist diffusing a bomb that suddenly detonates. One has to be either really close to the detonation or the bomb has to be massive to experience such peak pressures. The use of fertilizer (ammonium nitrate) or isolated trinitrotoluene (TNT) without an accelerant is *subsonic*; therefore, the explosive force will not be nearly as extreme as military grade supersonic explosions containing C4. Indeed, the common use of homemade fertilizer bombs by Afghan insurgents help explain why blast injuries tend to be less severe in Operation Enduring Freedom (OEF) than in Operation Iraqi Freedom (OIF).

7) Genetic Predisposition

Patients with apolipoprotein e4 (ApoE4) genotype and neprilysin polymorphism have increased deposition of amyloid-β plaques following in TBI.[85–87] There is a well-documented role of this genotype in increasing the risk of Alzheimer disease, but studies are currently mixed regarding whether these genotypes lead to worsened TBI outcome. Other genotypic variations that appear to play a role in cognitive decline after TBI include the genes for catechol-O-methyltransferase (COMT), which is essential for the metabolic degradation of dopamine in the prefrontal cortex; brain-derived neurotrophic factor (BDNF); dopamine beta-hydroxylase (DBH); and glutamic acid decarboxylase (GAD). Genetic abnormalities of aquaporins (proteins responsible for regulating brain water homeostasis) may also predispose an individual to the development of brain edema.[88] In one large study of Vietnam veterans, pre-injury intelligence was the most consistent predictor of cognitive outcome after penetrating head trauma.[89] Although speculative, several studies suggest a potential genetic screening test for individuals at risk for TBI, such as participants in contact sports and military personnel.[90–92]

How Common Is Blast Brain Injury?

We don't know the true incidence of blast injury to the brain. This often invisible injury is emblematic of modern warfare and is the subject of much research and controversy. Yes, we can see injuries resulting from the soldier being thrown through the air by the force of a bomb's blast wave and/or crushed by an object—this tertiary type of blunt trauma from a blast injury is physically obvious and resembles civilian blunt trauma. As defined earlier, this is often referred to as *blast-plus* TBI, meaning that the victim was exposed to a primary blast force *plus* blunt force impact. However, as in civilian sports injuries, minor concussive injuries may be overlooked, especially when the patient is being treated for other more obvious injuries that might require immediate medical care, such as an amputation. I vividly remember talking to a young soldier at Landstuhl Regional Medical Center (LRMC) who had just lost three of his four limbs, and he was able to describe the explosion to me in amazing detail, without any obvious evidence of TBI—so, was his brain truly spared? Did we fail to diagnose the injury? Or is he one of the soldiers whose functional deficits won't manifest until later? In other patients, there are no external wounds to accompany the brain injury. These more subtle cases of blast brain injury may manifest only as persistent cognitive deficits, and they may not appear until after the soldier returns home.[93]

Penetrating injuries from bomb fragments (i.e., secondary blast injuries) are also physically evident, and in these cases, there is no question that the soldier was exposed to a blast injury. Penetrating combat injuries are somewhat different from penetrating civilian injuries in that they are more likely to be multiple, more likely to be contaminated, and more likely to have superimposed thermal injury. Whether the primary blast wave exacerbates these lesions is uncertain. Kevlar helmets have significantly reduced the frequency of penetrating TBI, but they may provide little protection from the primary blast overpressure and, as mentioned earlier, do not prevent closed-brain concussive injuries produced by the blast.[94] Indeed, Kevlar body armor and helmets are one reason for the higher proportion of closed-brain injuries among soldiers in OIF/OEF as compared to earlier conflicts.

In contrast to penetrating (2°) and blunt (3°) blast TBI mentioned before, the prevalence of

1° blast TBI (i.e., BINT) remains uncertain. If the soldier shows physical evidence of having been exposed to a blast force, does exposure equal injury? This is the billion dollar question. Traditionally, BINT has been thought to be uncommon, partly because the primary blast force drops off exponentially with distance and most explosions occur in open space. However, the current conflict is characterized by more urban warfare, which places our troops in closer proximity to the explosion (e.g., IED detonated under the Humvee). Recent studies have shown that BINT may actually be as high as 50% in soldiers surviving combat blast injuries.[95] One explanation for this perceived increase in BINT is that improved body armor has resulted in a relative decrease in thoracic and abdominal blast injury and, therefore, increased soldier survival. As is the case with concussions suffered in sports, soldiers on the battlefield may appear fine after an explosion, but may in fact need rest, treatment, or rehabilitation. Despite the anatomical and scaling differences in animals compared to humans, numerous studies suggest that undiagnosed BINT occurs in combat veterans following shock wave exposure.[41,64,96–100] Unfortunately, many of these brain injuries may simply be wounds we currently can't see. BINT is discussed further in Chapter 5, Lesson 4.

Another point worth making here is the deep-rooted tradition of soldiers (and athletes) masking their physical pain and emotional turmoil for the good of the mission. Soldiers often believe that getting knocked around is part of the job, but over time, the effects of each mild successive TBI can become more severe. This machismo mentality makes the wounded warrior less likely to leave his or her buddies after he or she has been exposed to a blast force. The soldier tries to tough it out, which sets him or her up for more damaging cumulative brain injury. Indeed, it has been shown that the incidence of concussion increases with multiple tours of duty and repeated exposure to explosions.[101] It has also been shown that a single mild TBI increases the brain's vulnerability to a second insult for a period of time, during which a subsequent mild TBI will worsen outcome.[102,103] Thus, troops who sustain brain injury might return to the battlefield too early, complicate their recovery, and potentially compromise the success of their mission. One study found that over half of the troops exposed to a blast injury returned to duty within 72 hours.[104] In rare cases, subsequent head injury can lead to the *second-impact syndrome*, which, in younger patients (e.g., soldiers), has up to a 50% mortality rate (**Fig. 5.48**).[105,106]

Screening for TBI

The military has dramatically improved its screening policies for TBI. In 2008, the U.S. Department of Defense (DoD) officially mandated a screening program designed to identify deployment-related mild TBI and associated residual symptoms. On the battlefield, the Military Acute Concussion Evaluation (MACE) is used, and if the soldier fails the test, he or she is not allowed to return to combat.[107] Embedded within MACE is the Standardized Assessment of Concussion (SAC), a validated tool used extensively in the sports realm to assess neurocognitive functioning.[108] Because not all soldiers admit to having suffered TBI, or even realize that they suffered TBI, a policy enacted in 2010 mandates the medical evaluation for any soldier exposed within 50 m of a blast. In addition, routine predeployment TBI screening is being combined with postdeployment TBI screening when the soldier arrives at LRMC (**Fig. 3.24**). This data is being compiled in a TBI registry. Further, the Veterans Affairs (VA) has

3 Question DVBIC TBI Screening Tool

1. Did you have any injury(ies) during your deployment from any of the following? (check all that apply):

A. ☐ Fragment
B. ☐ Bullet
C. ☐ Vehicular (any type of vehicle, including airplane)
D. ☐ Fall
E. ☐ Blast (Improvised Explosive Device, RPG, Land mine, Grenade, etc.)
F. ☐ Other specify: _____

2. Did any injury received while you were deployed result in any of the following? (check all that apply):

A. ☐ Being dazed, confused or "seeing stars"
B. ☐ Not remembering the injury
C. ☐ Losing consciousness (knocked out) for less than a minute
D. ☐ Losing consciousness for 1-20 minutes
E. ☐ Losing consciousness for longer than 20 minutes

NOTE: Endorsement of A-E meets criteria for positive TBI Screen

F. ☐ Having any symptoms of concussion afterward (such as headache, dizziness, irritability, etc.)
G. ☐ Head Injury

NOTE: Confirm F and G through clinical interview

H. ☐ None of the above

3. Are you currently experiencing any of the following problems that you think might be related to a possible head injury or concussion? (check all that apply):

A. ☐ Headaches
B. ☐ Dizziness
C. ☐ Memory problems
D. ☐ Balance problems
E. ☐ Ringing in the ears
F. ☐ Irritability
G. ☐ Sleep problems
H. ☐ Other specify:_____

Figure 3.24. Brief TBI screening tool used to detect mild TBI in soldiers returning from combat.[114]

mandated TBI screening for all veterans who present to a VA hospital for any reason, and it has established a polytrauma/TBI system of care nationwide to deal with unidentified cases.[109] Preliminary data suggests that >20% of service members (i.e., >400,000 soldiers) have screened positive for a *probable traumatic brain injury*, including 45,000 to 90,000 veterans with persistent symptoms requiring specialized care.[95,110] Furthermore, at least 20,000 U.S. troops who were not classified as wounded during combat in Iraq and Afghanistan have been found to have signs of brain injury (i.e., nearly five times as many as the 4,471 officially listed by The Pentagon). The U.S. Defense and Veterans Brain Injury Center (DVBIC) claims that approximately 225,000 U.S. military service members have been given the diagnosis of TBI during the period of 2001 to 2010.[111] Of these soldiers, more than 50% of those sustaining TBI have been the result of blasts.[112] However, because many servicemen with possible TBI remain undiagnosed or have delayed diagnosis, this number is likely higher. Service members who incurred mild TBI during deployment are now reporting postconcussive symptoms many months or even years postinjury. Therefore, it is currently recommended that all service members who sustain mild TBI in the context of polytrauma, regardless of the absence of symptom reporting in the acute stage, be followed up for postconcussive symptoms.[113] As you can see from the variety of statistics, we really do not know the true prevalence of blast TBI in war. As yet, there has not been a scientifically rigorous, evidence-based epidemiologic study to answer this question. In 2011, the Defense Advanced Research Projects Agency (DARPA) began a study to monitor soldier blast injuries by having them wear sensors on their chest, helmet, and shoulders to detect overpressure data. The blast dosimeter will provide medical personnel with a quantitative measurement of a warfighter's exposure level, allowing better assessment of the potential for blast-related injuries and the treatment required for each individual soldier. The results of this initiative have not yet been released.

Serum and Cerebrospinal Fluid Biomarkers of Brain Injury Have Been Shown To Correlate with Patient Outcome[115–121]

Recent advances in genomics, proteomics, and biotechnology have provided unprecedented opportunities for surrogate markers of TBI. Molecular data can be generated to detect and classify injury and to identify mechanisms for therapeutic targets. As mentioned earlier in Chapter 3, acute head injury sets off a series of biochemical events that cause cellular disruption and tissue breakdown resulting in the release of proteins into the blood—it is these breakdown products that serve as surrogate brain injury biomarkers. The most studied examples include glial fibrillary acidic protein (GFAP), neuron-specific enolase (NSE), S-100B, ubiquitin C-terminal hydrolase, microtubule-associated protein tau, lactate dehydrogenase, glutamic oxaloacetic transaminase, myelin basic protein (MBP), creatine phosphokinase, cyclic adenosine monophosphate, interleukin-10, serum catecholamines, secretogogue, and αII-spectrin generated by calpain proteolysis. For example, S100B, NSE, and GFAP levels are significantly higher in patients who die or have worse outcomes. Concentrations of interleukin-10, a molecular biomarker of the inflammatory response in the early acute phase of intracranial hemorrhage, is independently associated with an increased probability of rebleeding. Elevated

serum troponin is frequently observed after severe TBI, and the level of troponin correlates with the severity of head injury and is an independent predictor of adverse outcomes; in these patients, beta-blocker therapy is associated with a survival advantage.[122]

As surrogate markers of TBI, it is hoped that such biomarkers will help determine injury severity, predict patient outcome, and expedite the diagnosis, triage, and management of the TBI patient, especially in those patients who are sedated, unconscious, or polytraumatized. Biomarkers could also help determine which patients need imaging (e.g., concussion) and provide successful early pharmacotherapy in altered biochemistry following head injury, thus advancing early administration of drug treatment in human TBI.[123] Unfortunately, although animal and clinical research has boomed in this field over the past 10 years, no single brain-specific biomarker has yet been unanimously established for TBI in routine clinical practice. Significant limitations that must be overcome include false positives (especially in the polytrauma patient), short biomarker half-lives, and patient age- and gender-related differences. In the future, it is likely that clinicians will identify and use panels of biomarkers containing optimal combinations of different biomarkers that reflect the multiple mechanisms of TBI.

BIOMARKERS OF BINT, IN PARTICULAR, REMAIN ELUSIVE[124,125]

The DoD is currently funding a major study of brain injury biomarkers in more than 1,000 human patients at 20 hospitals in the United States and overseas. This first-of-its-kind study, which is expected to start in 2011, will explore whether biomarkers can reliably assess the extent of brain injury and help doctors decide on treatment. The Army's goal is to one day have a portable blood test and/or device that a medic could carry onto the battlefield, analogous to the current cardiac biomarker, troponin, that is used to diagnose heart attacks. Improved prognostication, stratification of severity of injury by serum biomarker concentration, and identification of specific intracranial pathologies remain the goal in these biomarker studies.

Another recent effort by the military to screen for blast injury is the outfitting of 1,145 soldiers with helmets that contain a tiny sensor to monitor the pressure wave from the bomb blast on the cranium. These new high-tech helmets will gather data that will be downloaded to establish a database on the effects of blasts, and the data will be applied in the development of safer helmets.

New advances to detect BINT are also occurring in the area of neuroimaging. Conventional neuroimaging has been notoriously poor at identifying 1° blast TBI, and more advanced imaging techniques are beginning to provide evidence of abnormalities, such as metabolic derangements not evident on macroscopic anatomic imaging.[126] It is hoped that the results of these newer techniques, such as diffusion tensor imaging (DTI), functional magnetic resonance imaging (fMRI), higher field (3T-7T) MRI, single photon emission computed tomography (SPECT), positron emission tomography (PET) scanning, magnetoencephalography (MEG), and magnetic source imaging (MSI), will be validated and become predictive of patient outcome. These advanced imaging technologies are discussed in Chapter 5, Lessons 4 and 12.

In summary, a blast injury is a combination of pressure, fragmentation, and incendiary effects.

These effects cause injury via a combination of at least five biodynamic variables: direct blast overpressure (tissue strain), bodily displacement, penetrating trauma, thermal injury, and cardiopulmonary insufficiency. The precise cause and prevalence of primary blast TBI (BINT) is not known. The ***external*** *variables that determine the amount of brain damage include the peak blast pressure, duration, medium in which the explosion occurs, distance from the explosion, and body protection. The* ***internal*** *variables that determine the development and extent of brain injury, including preexisting genetic factors and the mechanism(s) of blast brain injury, are only beginning to be understood. Predicting injury is further complicated by the fact that such variables may interact, not in a simply additive fashion but in a synergistic manner with one another. Some of the injuries look similar to civilian impact and penetrating injuries, but they tend to involve more edema and to be more severe partly because of the superimposition of thermal injury, tissue contamination, and the influence of polytrauma. Primary blast TBI is difficult to diagnose and under-reported. Serum and imaging biomarkers are needed to aid identification of this injury in soldiers exposed to blast trauma.*

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CHAPTER 4

The Weapons of War and Terrorism

As in previous conflicts, the type of weapon used relates directly to the type of injury sustained. Therefore, a brief review of the current weapons is useful for understanding the nature of the injuries. A breakdown of the weapons of modern conventional and asymmetric (i.e., terrorist) warfare as they relate to the cause of traumatic brain injury (TBI) reveals the following: improvised explosive device (IED) (70%), gunshot wound (GSW) (9%), mortar blast (8%), rocket-propelled grenade (RPG) (3%), landmine (5%), grenade (1%), and other/unknown (4%).[1] The pattern of the injuries varies considerably depending upon the type of the weapon, protective gear, terrain, and type of military operations. In brief, the primary weapons of the Iraq–Afghanistan conflict include the following:

1) **Improvised Explosive Device.** The IED is the signature weapon of the war in Iraq and Afghanistan. It is the most commonly used explosive munition by the insurgents.[2] In 2010, insurgents planted nearly 15,000 IEDs in Afghanistan, an alarming 62% increase over the previous year. IEDs are triggered by a number of different means, including electrical, timed fuse, trip wire, cell phones, digital camera, or copper wire. In addition to chemical explosive, IEDs contain a variety of objects, such as nails, ball bearings, metal bolts, rocks, glass, and segments of metal rods, intended to maximize damage (**Figs. 3.15, 4.1, 4.2, and 5.73**). Some contain human feces to increase the likelihood of secondary infection. In the case of "suicide bombers," the body parts of the bomber can become contaminated secondary projectiles. The IED is particularly dangerous because it can be disguised as virtually anything and hidden anywhere. To date, these bombs have been concealed as refuse in the roadway, in burlap bags, in potholes, and even inside animal carcasses. As a result, the devices are very difficult to spot at convoy speeds. This is compounded by the fact that Iraqi

roadsides are already littered with regular trash. IED attacks are frequently followed by an ambush using small arms and RPGs. IEDs are usually free standing, but they are also placed within cars and buses (also known as vehicle-borne improvised explosive devices [VB-IEDs] or car bombs) and buildings (also known as house-borne improvised explosive devices [HB-IEDs]). In these cases, exploded pieces of the home or car become additional secondary projectiles.[3] VB-IEDs often contain tanks of propane or other toxic chemicals to enhance the burn injury (**Figs. 4.3 and 4.4**). Despite being a relatively crude weapon, the indiscriminate and unpredictable nature of the IED is the essence of its effectiveness in 21st century terrorism. The 2013 Boston Marathon bombing used a simple kitchen pressure cooker and was hidden in a street side trashcan.

"Homemade" Improvised Explosive Device (IED)

Figure 4.1. Improvised Explosive Device. Insurgents fabricate these homemade bombs with penetrating objects—in this case, nails are attached to a bottle containing explosive material. The dispersal of these fragments results in innumerable penetrating wounds such as those illustrated in **Figure 3.13**. (With permission from ABC News, American Broadcasting Company, a subsidiary of Walt Disney Company, Los Angeles, CA).

Typical IED and Ballistic Fragment

Figure 4.2. IED. A. The 155-mm artillery shell (howitzer round) is commonly used to make IED bombs. It consists of an 80-lb forged steel case filled with explosive material. **B.** Curbside camouflage of the artillery shell-based IED. **C.** Example of one explosive remnant of the artillery casing shown in **(A)**. *This fragment was recovered from the experiment shown in* ***Figure 3.1***. Note the irregular shape and the sharp edges of the fragment, capable of inflicting maximal tissue damage. (Courtesy of Tim Imholt, PhD, Raytheon Co.).

★**KEY POINT** The IED is the signature weapon of the war in Iraq and Afghanistan. It is the most commonly used explosive munition by insurgents and terrorists worldwide.

Vehicle-Borne IED ("Car Bomb" Injury)

Figure 4.3. Vehicle-Borne IED (Bus Bomb). Photograph of the remnants of a terrorist bus explosion. Note extensive thermal blast injury. Vehicle bombs can carry a large amount of explosive without attracting suspicion. The gasoline in the vehicle's fuel tank makes the explosion of the bomb even more powerful. The VB-IED also produces a lot of shrapnel, causing secondary damage to bystanders. It is widely used by suicide bombers. The bomb can also be activated by opening of the vehicle's door, starting the engine, depressing the accelerator, or setting a timing device. (Courtesy of Guy Rosenthal, MD, Hadassah Hospital, Jerusalem, Israel.)

★**KEY POINT** Since ancient times, when the Greeks deployed a Trojan horse to enter the city of Troy, the idea of taking an object that can breach a security system because it is familiar or desirable and using it to deliver a payload to enemy forces has been a highly effective tactic. The VB-IED is but the latest incarnation of this very old theme—but with a deadly force. The VB-IED typically carries from 110 lb up to 1,100 lb of explosives but is not limited and can even exceed 12,000 lb, many times more powerful than the lone suicide bomber.

Vehicle-Borne IED ("Car Bomb" Injury)

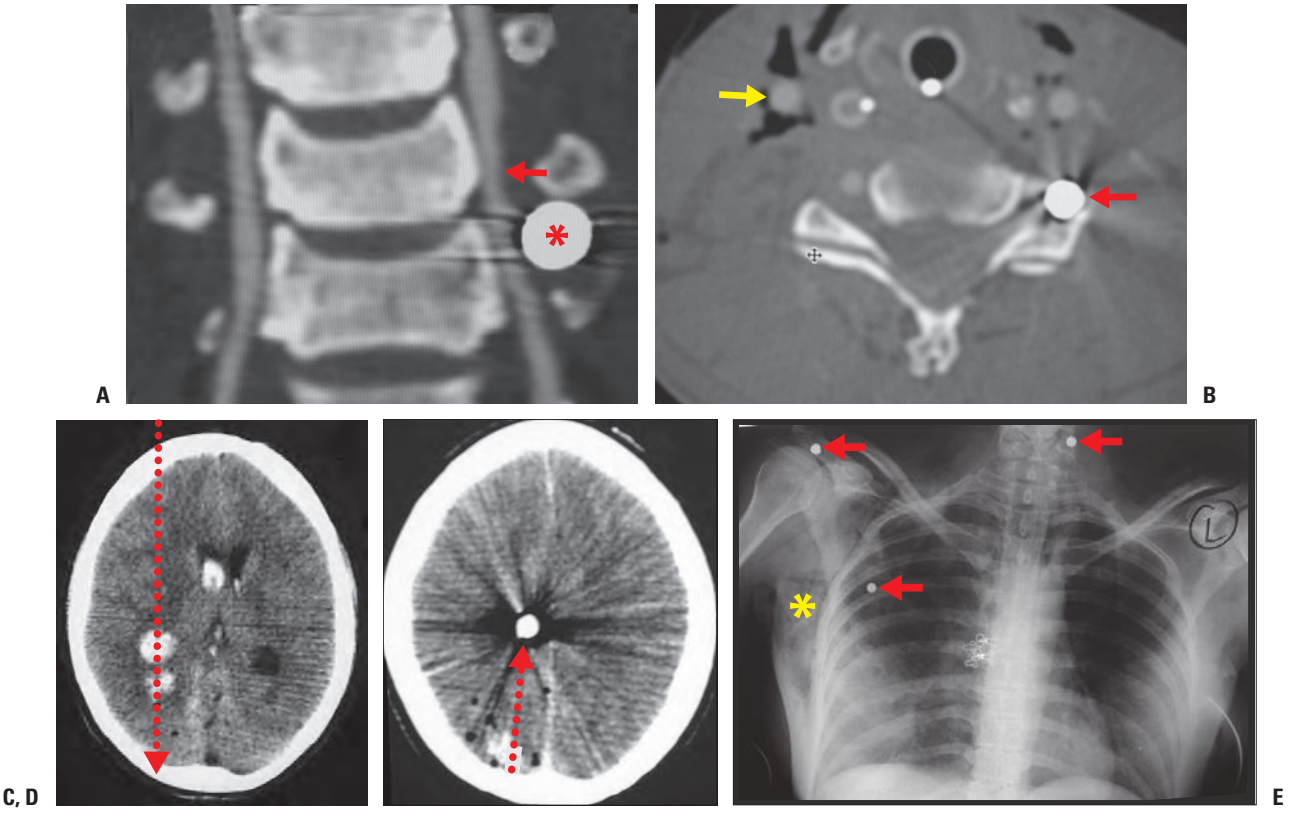


Figure 4.4. Vehicle-Borne Improvised Explosive Device (VB-IED) Blast Trauma. A. Coronal maximum intensity projection (MIP) image from the computed tomography angiography (CTA) of the cervical region shows the left neck ball bearing (*asterisk*) located adjacent to the vertebral artery. Mild caliber irregularity (*arrow*) is present, consistent with vasospasm, dissection, and/or perivascular hemorrhage. **B.** Contrast-enhanced axial computed tomography (CT) image viewed at wide windowing shows interval intubation of the patient, subcutaneous emphysema with air surrounding the right common carotid artery (*yellow arrow*), and the ball bearing within the left foramen transversarium (*red arrow*). **C, D.** Admission CT of the brain shows the unihemispheric "front-to-back" penetrating injury. The projectile entered the victim's right forehead, ricocheted off the inner table of the right parietal calvarium, and terminated in the right centrum semiovale (*arrows* outline the missile trajectory). See also **Figs. 4.21**, **4.22**, and **4.23** for additional ricochet examples. **E.** Anteroposterior (AP) chest radiograph demonstrates a pneumomediastinum, subcutaneous emphysema overlying the right pectoral region (*asterisk*), and three ball bearings (*arrows*). (*Continued*)

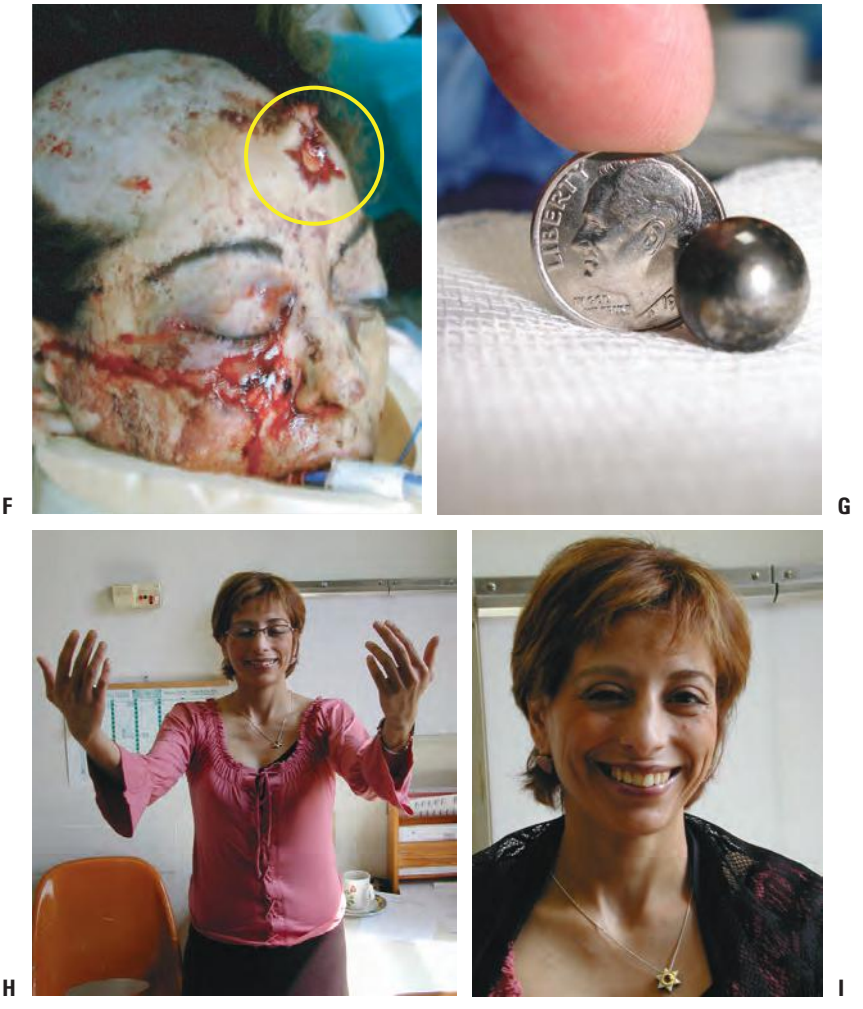


Figure 4.4. (*Continued*) **F.** Intraoperative photo showing the entry wound (*circle*) and extensive facial lacerations. **G.** Photograph of the foreign body ball bearing. **H, I.** Follow-up photographs several months later. (Courtesy of Guy Rosenthal, MD, Hadassah Hospital, Jerusalem, Israel.)

2) Explosively Formed Projectile. The explosively formed projectile (EFP; also known as explosively formed penetrator) is a self-forging warhead. It consists of a metal pipe or cylinder that contains explosive material and a copper liner in the shape of a shallow dish (**Fig. 4.5**). The force of the detonation molds the copper plate into a giant aerodynamic slug. As this projectile hits the tank/Humvee, it creates a shock wave that travels through the vehicle as a compression wave. The compression wave has sufficient force to break metal on the inside of the vehicle, and the resulting spall is dangerous to the crew and equipment. The slug itself can also penetrate the tank and spray fragments of the copper liner material, from which it was formed, throughout the inside of the vehicle. The impact can also partially disintegrate the EFP and generate a large number of small fragments from the impact surface. EFP injuries tend to be less blast-like than the typical IED and resemble more of a giant bullet wound. The EFP is quite lethal, even to the new generation of mine-resistant vehicles and to many tanks.

Explosively Formed Projectile (EFP)

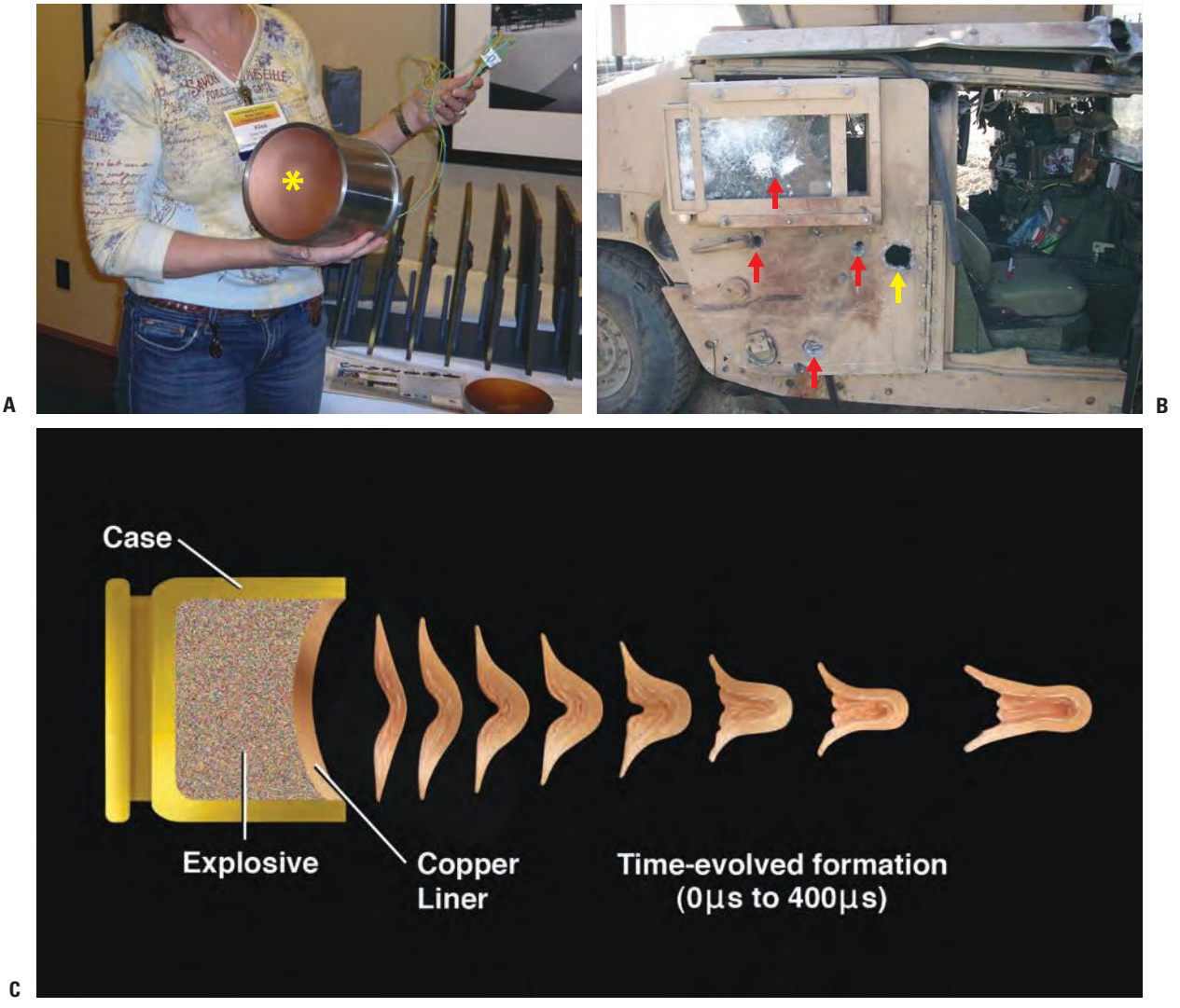


Figure 4.5. **Explosively Formed Projectile (EFP). A.** Predetonated EFP and its wire fuse. Note the copper dish, which ultimately forms the warhead (*asterisk*). Explosive material fills the cylindrical metal case. **B.** Photograph of my friend's Humvee attacked with a remotely detonated EFP. Note the large hole in the reinforced metal door (*yellow arrow*) corresponding to the entry site of the slug. Smaller spall fragmentation damage to the door and window are also present (*red arrows*). **C.** Schematic illustration of the formation of an EFP. Note how the copper liner morphs into a giant slug.

★**KEY POINT** As with the IED and VB-IED, detonation of the EFP can be controlled by a simple wire cable, radio control, cellphone, TV or infrared (IR) remote controls, or remote arming with a passive IR motion detector.

1) **Rocket-Propelled Grenade.** The RPG is a portable, handheld, shoulder-launched, anti-tank weapon that fires an unguided rocket containing an explosive warhead (**Figs. 4.6A and 4.7**). It is essentially a handheld grenade launcher and is sometimes referred to as a *bazooka*. The RPG contains two parts: the *launcher* and the *rocket* (which contains the grenade). The rocket consists of explosives packed around a cone of metal. Due to the lack of a guidance system in the RPG rocket, the operator must fire relatively close to the intended target, increasing the chances of being spotted. Most modern armies deploy anti-tank guided missiles as their primary infantry anti-tank weapon, but the RPG still remains a potent threat to armored vehicles, especially in situations such as urban warfare or jungle warfare, where they are favored by guerrillas. The RPG is a favorite weapon of the insurgents in Iraq and Afghanistan.

2) **Hand Grenade.** The hand grenade is a handheld bomb that is thrown by the individual (**Figs. 4.6B and 5.73**). When a grenade explodes, it is designed to spew shrapnel (derived from its casing and incendiary contents) in all directions. Grenades come in different shapes and sizes, but all have two things in common: They are hollow so that they can be filled with explosive, and they contain a fuse. Most grenades have a hard shell that allows them to ricochet off hard surfaces (e.g., a wall) before exploding. Grenades have a 5-m kill zone and a 20-m wound zone. In addition to the "antipersonnel fragmentation grenade" described earlier, there are also chemical and gas grenades that include smoke grenades and incendiary grenades. Unlike fragmentation grenades, chemical and gas grenades are designed to burn or to release gas, not to explode.

3) **Mortar Fire.** Mortar rounds refer to a muzzle-loading weapon with a short barrel (10 cm) (**Fig. 4.6C**). The shells are fired at a much lower velocity and higher ballistic arc than other ballistic ordnances. It can be carried by two soldiers and can fire up to 20 rounds per minute. Its recoil is directed into the ground, rendering it simple to use. Mortar fire (and most field artillery) is an example of *indirect* fire, that is, it is not aimed directly at the target, but the parabolic trajectory is estimated, allowing for differences in distance, altitude, atmospheric conditions, the velocity of the projectile, and other factors. The original purpose of indirect fire was to enable fire from a *covered position*, one where gunners could not be seen by their enemies. Until the introduction of *smart munitions*, the trajectory of the projectile could not be altered once fired.

Typical Combat Munitions

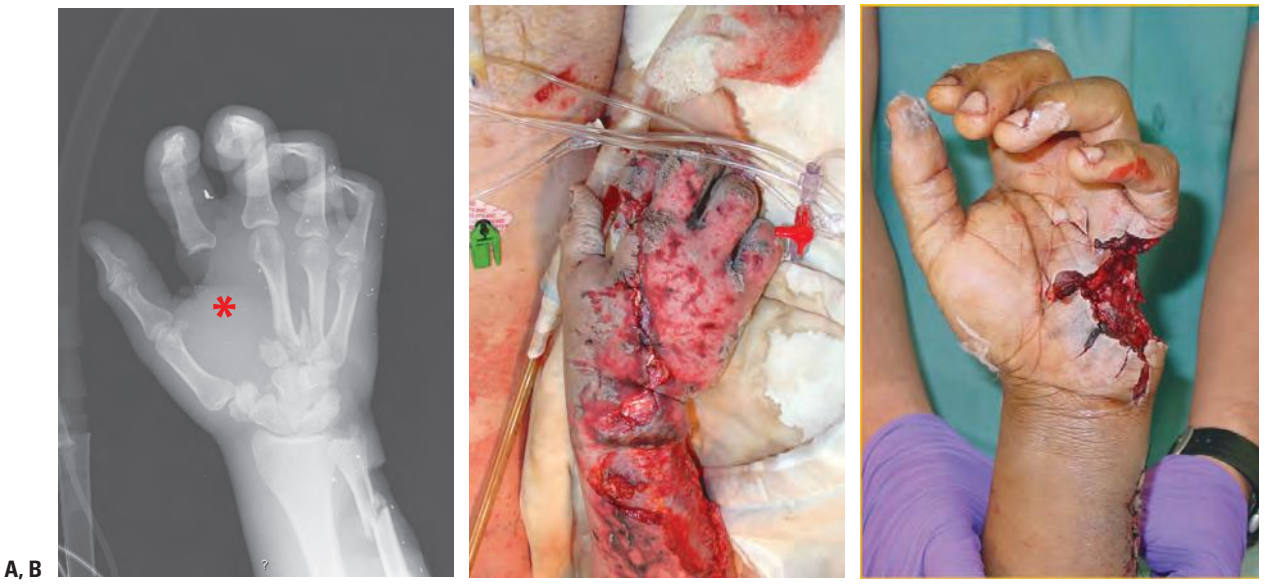
A

B

C

Figure 4.6. Common Combat Munitions. A. RPG. Note the resemblance to the bazooka. The RPG is an example of *direct* fire because it is aimed in the direct line of sight between the weapon and the target. **B. Hand grenade.** Photograph of a modern hand grenade with the outer casing cut away to reveal the innumerable small ball bearings embedded within the inner case. The hard casing allows it to ricochet off surfaces before exploding. When the grenade explodes, these ball bearings and fragments of the casing become projectiles. **C. Mortar fire.** Note the actual warhead (*yellow arrows*) emerging from the barrel of the weapon accompanied by heat and a surrounding spray of powder (*red arrows*). Mortar round artillery is an example of *indirect* fire as the warhead is not aimed directly at the target. Instead, the parabolic projectile trajectory is estimated, allowing for differences in distance, altitude, atmospheric conditions, and other factors. The blast is extremely loud (note the soldier covering his ears).

Rocket-Propelled Grenade (RPG) Injury



A, B C

Figure 4.7. Injury from a Rocket-Propelled Grenade. A,B. Traumatic amputation of the second metacarpal (*asterisk*), with comminution of the wrist and severe burn injury. Multiple small residual radiopaque foreign bodies are also noted. **C.** Different patient with a typical focal RPG "bite" wound of the hypothenar eminence.

4) Booby Traps, Landmines, and Unexploded Ordnances. The **booby trap** is a disguised device that explodes when a nearby harmless-looking object is touched. The initiating object is fairly obvious, as it is the object that the enemy hopes the victim will touch in order to set off the trap (e.g., a war souvenir).

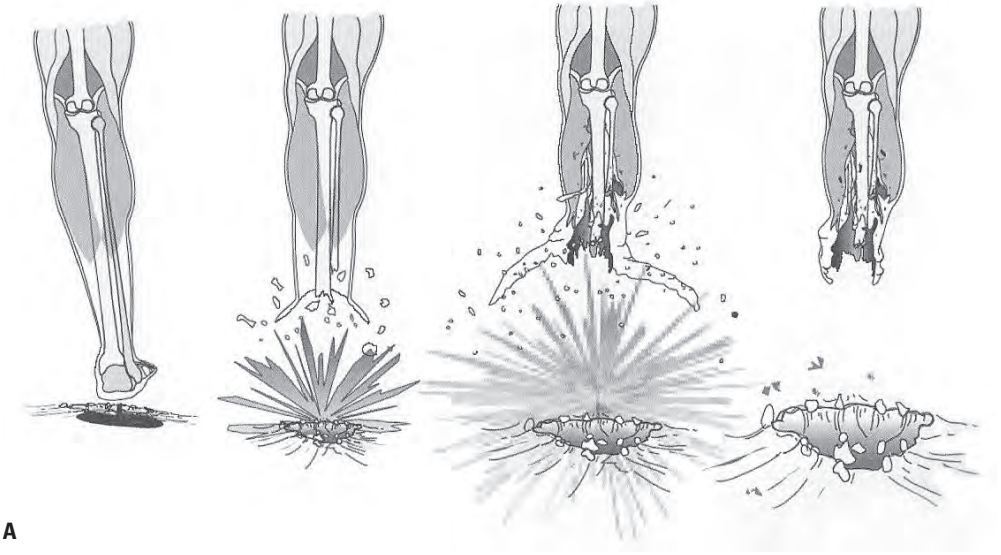
The **landmine** is an explosive munition that lies in wait for its target and is triggered by a pressure fuse. It comes in different sizes and shapes with varying pressure thresholds for detonation (**Fig. 4.8**). An anti-tank mine, for example, contains 4 to 5 kg of explosive filler and will not be triggered by someone merely stepping on it. Most types of landmines are small (100 to 200 g of explosive), inexpensive, and cause partial or complete traumatic amputation, most commonly at the midfoot or distal tibia. More proximally, debris and tissue are driven up along fascial planes with tissue stripped from the bone, resulting in the so-called *umbrella effect*. Landmine and booby trap injuries are essentially focal blast injuries that create high-velocity secondary missiles emanating from the underlying ground in which they are buried. As a result, the wounds are filled with dirt, pebbles, and chunks of plants. The extent of injury is determined primarily by the amount of explosive filler in the mine, the depth to which it was buried, and the amount of debris overlying the landmine. The type of footwear worn, the point of contact with the foot, and the size and shape of the limb are additional factors contributing to the severity of the injury.

According to the International Committee of the Red Cross, landmines continue to

kill or maim over 26,000 victims each year. Scattered in some 78 countries, they are a reminder of conflicts that have been ongoing for years or even decades. Afghanistan remains one of the countries most heavily contaminated with landmines. It has been estimated that over 640,000 mines have been laid since 1979, threatening not only our troops but over 4 million Afghans as well. During a 6-month period in 2008, 1,445 victims of mines were reported, and 50% of these were children.[4] Many Afghan farmers have also been injured and lost their farms, and thus their livelihoods, as 75% of the land in which mines are buried is also used for agriculture. Nearly 3% of Afghanistan's population has been labeled as "severely disabled," and 9% of these disabilities have been attributed to landmines. Iraq is also one of the most mine-infested nations in the world because it has liberally used landmines in multiple conflicts, including protecting its borders during the lengthy war with Iran (1980 to 1988), warding off invasion during the Persian Gulf War (1990 to 1991), and subduing the Kurdish people in northern Iraq. Most landmines contain mainly explosive, with only small amounts of metal and plastic (to evade mine detectors), thus making radiologic identification of foreign bodies difficult. This also thwarts efforts to clean up and eradicate these devices. Nitrogen-sensing robotic drones may be helpful in the future.

Unexploded ordnances (UXOs) are rockets, grenades, and mortar rounds that were fired but didn't explode. UXOs are usually found on the ground and have contaminated many areas during recent fighting between the Taliban and Allied forces. Because the weapon typically lies on the surface of the ground, it is more visible and easier to avoid than landmines. However, because of its visibility, UXOs pose a particular threat to children and adolescents, who are drawn to unfamiliar, curiosity-provoking objects. When embedded in the casualty, the vast majority of victims live after removal.[5] However, surgical removal of UXOs is accompanied by potential threat of detonation with risk of blast injury to both the patient and the surgeon.

Landmine Blast Injury



A

B

C

Figure 4.8. **Landmine Trauma. A.** Schematic illustration of the *umbrella effect*, arising from an antipersonnel landmine. Upon stepping on the mine, tissue destruction of the distal extremity occurs, but in addition, the injury propagates proximally as the blast tears muscle and soft tissue off the bone. This results in an injury worse than the devastating destruction to the distal extremity alone, with additional damage more proximally that is not so clinically apparent. (Reproduced from Szul AC, Davis LB. Weapons effects. In Emergency War Surgery, Third United States Revision. Washington, DC: Office of the Surgeon General, Borden Institute, Walter Reed Army Medical Center; 2004.) **B.** Macerated and contaminated lower extremity wounds are typical of landmine injuries. **C.** Photograph of a roadside anti-tank mine partially concealed in concrete and detonated via remote control. These larger mines often cause blunt trauma rather than blast injury as crew members are thrown around inside the vehicle after it detonates the mine; closed fractures of the upper and lower extremities and spine are common.

5) **Gunshot Injuries and Other Penetrating Head Trauma.** From the streets of San Francisco to the battlefields of Baghdad, GSWs continue to plague both civilian and military life. In contrast to civilian trauma, which has an 11% rate of penetrating injury, the rate of penetrating injury on the battlefield exceeds 68%.[6] Nevertheless, gang violence in American urban areas and civilian firearm injuries has become epidemic in the United States, and the incidence exceeds that of other developed countries.[7] Homicide and suicide firearm injuries are also on the rise in developing countries.[8] In the United States, 500,000 GSWs occur annually, resulting in 50,000 deaths. In several states, the mortality rate from GSW exceeds the mortality rate from motor vehicle accidents.[9] When one focuses on all TBI-related deaths alone, firearm-related injuries are the leading cause of mortality.[10] The majority of GSW deaths are due to assaults and suicide attempts. As is seen with other forms of trauma, males are affected in excess of 80% of cases, and the mean age of presentation is about 30 years.[11] The annual financial burden of firearm-related injuries in the United States is estimated to be $2.3 billion.[12]

Ballistics is the science of projectiles in motion. It is divided into three categories: *internal*, *external*, and *terminal* ballistics. Internal ballistics pertains to the bullet within the firearm. External ballistics concerns the bullet in the air. Terminal ballistics relates to the bullet when it hits its target and is clearly the most important category to clinicians.[13,14]

- **Internal ballistics** (within the gun): GSW and missiles associated with secondary blast trauma follow the rules of basic ballistics: kinetic energy (KE) = ½ mV^2, where m is the mass of the projectile and V is the velocity of the projectile. Anything that moves has energy. Note how the equation predicts that the velocity of the projectile is more important than its mass in terms of wounding power. Doubling the mass of the bullet doubles the KE, but doubling the velocity quadruples the KE. In addition, the velocity of the bullet as it exits the weapon depends in part on the length of the barrel (the longer the barrel, the longer time for acceleration of the bullet, and the greater the KE). Exit velocities at the muzzle for revolvers are around 150 m per second; pistols, 300 to 360 m per second; shotguns, 400 m per second; rifles, 400 to over 1,500 m per second; and muzzle velocities from an AK-47 are approximately 900 m per second. However, it is important to understand that the science of wounding power is more than simple physics.[15,16] The ability to *transfer* the energy from the projectile to the tissue ultimately determines the extent of injury. From a ballistics standpoint, the shotgun is quite different from the pistol and rifle. In the case of the latter, the ideal characteristics of a single projectile—good ballistic profile, high sectional density, high velocity, and deep penetration with controlled expansion—are met. But in the shotgun, all these ideal properties are sacrificed in order to obtain target area saturation, therefore making it possible to hit a small, fast-moving target.
- **External ballistics** (from gun to target): External ballistics considers what happens to the bullet after it leaves the end of the weapon muzzle. The bullet doesn't exit in a straight line from the barrel. As it travels through the air, it rotates away from the axis of its trajectory rather than maintaining a direct unwavering trajectory. This deviation in its longitudinal axis from the straight line of flight is termed bullet *yaw*. **Yaw** is basically a measure of the tendency of the bullet to veer off target. It is very important

for high-velocity missiles at short range. Yaw of the projectile is a significant determinant of the amount of damage to the target tissue. Increased yaw increases rotation of the bullet upon entering the tissue and thereby increases the size of the wound tract (**Figs. 4.9, 4.10, 4.12 to 4.14, 4.21**). In addition to yaw, movement of the bullet as it travels through air also arises from *precession* and *nutation*. **Precession**, the rotation of the bullet about its center of mass, and **nutation**, small circular motions of the bullet tip, are not as important, as compared to yaw, in causing tissue destruction.

Other factors important to external ballistics include the wind velocity, air density, air temperature, the mass of the bullet and the effects of gravity, and even the earth gravitation. Taking all these factors into account, a strike to a specific target requires sophisticated mathematics.

- **Terminal ballistics** (within the target): Yaw increases the presented area of the bullet to the tissue and increases its drag coefficient. This results in a dramatic increase in the rate of energy transfer to tissue. Yaw within tissue partially explains why an entry wound (at small yaw) may be quite small while the exit wound is gaping and large; the large exit wound is caused by the missile exiting at high yaw (**Figs. 4.10, 4.13 to 4.14, 4.31C**). Similarly, *deformity* (and therefore increased surface area) of the projectile as it passes through the tissue will also increase the severity of injury. In addition, the size and shape of the projectile also play a role in the extent of injury. In contrast to a simple sphere, blunt or irregular projectiles present a larger surface area for contact with the tissue, thereby increasing the wounding potential. Larger projectiles also increase the surface area for wounding.

The transfer of KE to the tissue depends on several factors. One of these is the *viscoelastic* properties of the target tissue (i.e., skin vs. fat vs. bone vs. muscle vs. brain). The skin is elastic and absorbs cavitation well (i.e., it can stretch and return to its original shape without necrosis). Muscle is less elastic and is directly crushed. By comparison, bone is inelastic and transmits energy to the adjacent muscle, causing necrosis. Finally, the extent of tissue damage would be directly proportional to the transfer of KE, if it were not for the rate dependence of tissue damage. This is perhaps best illustrated by Silly Putty (Crayola, LLC, division of Hallmark Cards, Kansas City, Missouri): Stretch it slowly and it will expand indefinitely, but stretch it rapidly and it will tear. Tissue, of course, is much less rate dependent than Silly Putty, but this *rate effect* helps explain the greater injury with high-velocity weapons.[17]

In comparison to low-velocity bullets associated with civilian trauma, high-velocity military rounds and high-energy explosive devices tend to impart a greater degree of trauma to tissue. However, a bullet fired at high velocity from a distance may cause much less damage than a low-velocity GSW from close range. The injury from a high-velocity round fired from a distance can be likened to that of a stabbing injury if the velocity is so high that the bullet simply passes through the tissue without dissipating its KE. To effect maximum tissue damage, the ideal bullet is designed to deliver all of its KE upon impact with the target tissue. This is exemplified by the so-called *devastator* bullet described in the following text, which is designed to explode on impact with the target.

Bullet Behavior in Tissue

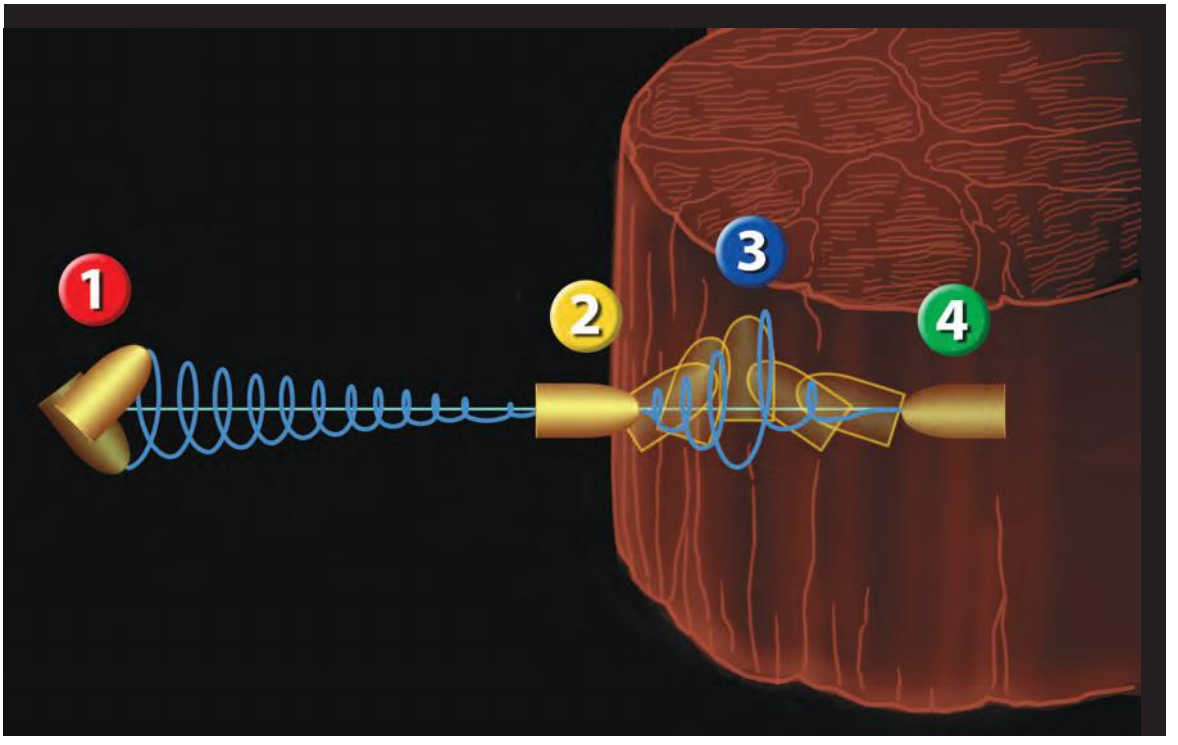


Figure 4.9. Idealized Behavior of a Fired Bullet into Soft Tissue. A bullet emerges from the gun with a significant angle of yaw. (*1*). In the case of rifle bullets, gyroscopic stabilization from *rifling* grooves within the gun barrel tends to align the long axis of the bullet with the line of flight. (*2*). This is analogous to a spinning football. In the case of handguns, however, tumbling of the bullet in the air is a significant determinant of the amount of damage to the target tissue. Increased yaw increases rotation of the bullet upon entering the tissue, and thereby increases the size of the wound tract. After the bullet enters the more dense target tissue, it rapidly destabilizes and begins to wobble. (*3*). Its yaw increases the cross-sectional area of its path. This thereby enhances the drag force, which results in transfer of more KE to the tissue and consequently more tissue injury. The final appearance is often parallel again (because the wound canal collapses around it) but reversed in direction. (*4*). It is this behavior that explains why the final direction of the bullet often "points to" the entry site.

★**KEY POINT** Bullet yaw is the rotation of the projectile to the left or right of its direction of motion. Yaw increases the extent of injury because of increased cross-sectional area of the bullet on impact with the target tissue. Sudden drag force and/or tumbling within the tissue puts strain on the bullet and can cause it to fragment.

Basic Firearm Vocabulary

A rudimentary review of firearm terminology is helpful for understanding GSWs.[18] In general, firearms are classified as handguns, rifles, and shotguns. Handguns are also referred to as *pistols* and *revolvers*, depending on their mechanical actions. Ballistic injuries from these weapons are divided into *low-energy and high-energy* wounds. **Low-energy** wounds are caused by bullets travelling at velocities <1,200 ft per second (i.e., below the speed of sound in air). Most handguns are low velocity and usually cause tissue damage only along the bullet tract when wounding extracranial tissue. Handgun injuries to the cranium are more complex and will be described later. Rifles are **high energy** (>2,500 ft per second) weapons and cause damage along the bullet tract as well as to the surrounding tissue in both intracranial and extracranial tissue. Unlike smoothbore firearms such as handguns and shotguns, rifles have spiral grooves in the gun barrel (i.e., *rifling*) that impart a gyroscopic spin to the projectile and stabilize it in flight. This does two things: First, it increases the accuracy of the projectile by eliminating random drift, and second, it allows a longer, heavier bullet to be fired from the same caliber barrel, increasing range and power. Rifles and handguns carry single bullets, and each bullet produces a single wound. Shotgun ammunition, by comparison, consists of a shell filled with multiple metal pellets (i.e., *shot*) of various sizes. Most shotgun injuries are characterized by numerous wound canals.[19] The shotgun is a very short-range weapon. Moving targets over about 30 yd away are in little danger when fired on with a shotgun in average hands. At 40 yd, even the best shooters are limited by the fact that the cloud of pellets is spreading out and slowing down very rapidly, thus limiting its effectiveness. Shotguns were originally designed for targeting small, fast-moving game, such as wild fowl. *Birdshot* was typically fired as small pellets that dispersed in flight to form a fan-like pattern, thereby increasing the chance of hitting the target. Early American colonists used shotgun ammunition because the general accuracy of the smoothbore (i.e., not *rifled*) shotgun barrel was poor, and the dispersed projectiles made it easier to hit moving targets or those in brush. Although shotguns are classified as low-velocity weapons, close-range injuries can be devastating, especially when larger lead shot, such as buckshot, is used.

Rifles and handguns are classified by caliber. The **caliber** of a weapon is the diameter of the muzzle bore (inside of the barrel), which is the same as the diameter of the bullet. Measurements for American firearms are typically in inches or in millimeters. For example, the .45 caliber pistol bullet is 0.45 in. (1.14 cm) in diameter. Shotguns are also classified by size (i.e., *gauge* or *bore*), the most common being 12, 16, and 20 gauge. The **gauge** is determined from what fraction of a pound a lead ball, with the same diameter as the barrel, weighs. Thus, a 12-gauge shotgun accommodates a lead ball weighing 1/12 of a pound. As the gauge is the denominator of the fractional weight, small gauge shotguns have larger diameter barrels as compared with larger gauge shotguns; that is, the smaller the gauge number, the larger the bore of the shotgun.

The Entry Site of a Gunshot Wound Is Always Smaller Than the Exit Site

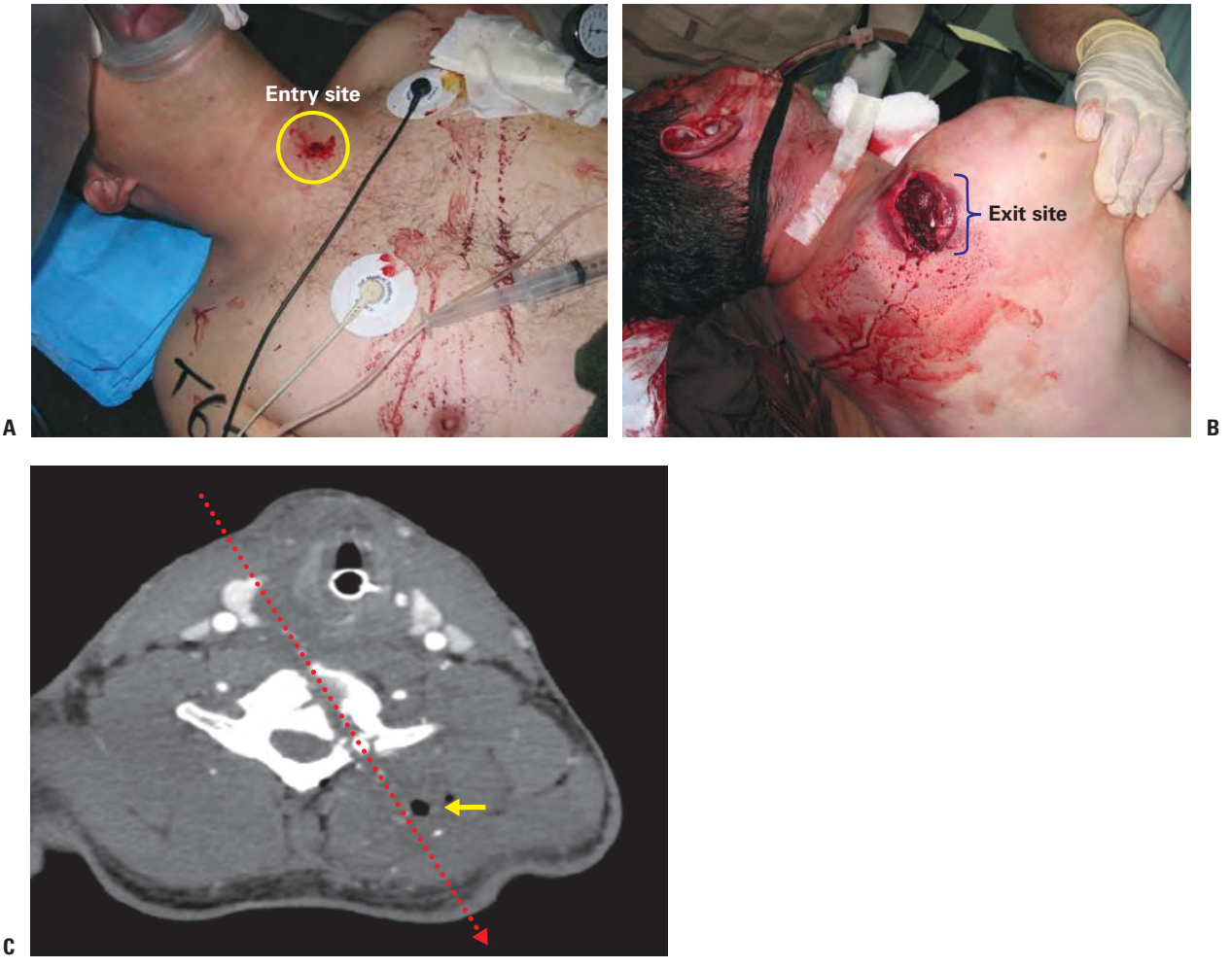


Figure 4.10. Gunshot Wound to the Neck. This 20-year-old Humvee turret gunner was shot in the neck by sniper fire. He presented with an initial Glasgow Coma Scale (GCS) of 10T = E4VTM6 and quadriplegia. **A.** Anterior and **(B)** posterior photographs of the patient's neck and chest show the classic small entry and larger exit wounds. **C.** Axial CTA image at the level of the cervicothoracic junction demonstrates an anterior to posterior missile tract fracture through the C7 vertebral body. Although the major vessels are patent, the spinal cord is in close proximity to the bullet tract, rendering it susceptible to injury from the percussive blast force. Note the small amount of air in the posterior left paraspinal muscles (*yellow arrow*). The bullet trajectory in this case can be deduced from the entry and exit wounds, but in other GSW cases where the entry and exit sites are not so obvious, the presence of air scattered in the soft tissue can be a helpful clue to the wound trajectory.

★**KEY POINT** Note that magnetic resonance imaging (MRI) could not be safely performed in this patient because of retained, potentially ferromagnetic foreign bodies.

"Ammo Anatomy"

Figure 4.11 **Illustration of the Basic Components of Modern Firearm Ammunition. A.** Rifle and handgun ammunition is called a *round* (also known as a *shell* and a *cartridge*). Each round consists of a *casing*, containing the primer and propellant, together with the bullet. The propellant is also known as *charge* or *gunpowder*. The *rim* is the part of the casing that is used for loading. At the center of the rim is the *primer*, which ignites the propellant. **B.** Shotgun shells typically consist of a plastic case with a base cup covered in brass. Gunpowder and primer are compressed into the brass cup at the base of the shell. Wadding material is placed between the powder and the shot pellets. **C.** Photograph of several common types of rounds. The casing, which holds all the parts together, is seen here as the large brass portion of the round. The actual bullet is shown in copper.

★**KEY POINT** Note that the actual projectile (i.e., bullet) is located at the tip of the casing and that the bullet is usually jacketed with copper. Law enforcement agents, hunters, and most criminals use semi-jacketed ammunition—it is forbidden in combat.

"Ammo Anatomy"

Modern firearm ammunition consists of a prefabricated firing cartridge, commonly referred to as a *shell* or *round* (**Figs. 4.11 and 4.12**). In the case of handguns and rifles, each round consists of two parts: the *casing* and the *projectile* (i.e., bullet). The first component of a round, the casing, is usually made of brass and less commonly of steel. It is automatically extracted from the gun chamber upon firing. The casing contains an ignition system comprising a *primer*, most often composed of nitrocellulose, which is positioned at the base of the casing, and the *gunpowder*. The gunpowder occupies most of the casing and acts as a flammable propellant or *charge*. The second component of a round is the actual bullet, located at the tip of the round. The word *bullet* is sometimes colloquially used to refer to ammunition in general, or to a cartridge, which is a combination of the bullet, case/shell, powder, and primer. The word is derived from the French word *boulette*, which means "little ball." Bullets are usually made of lead. Lead is the preferred metal used in bullets because it is very dense, thus providing substantial mass and, therefore, KE per unit volume. Lead is also used because it is cheap, easy to obtain, and melts at a low temperature, thus making it easy to mold and fabricate into varying bullet shapes.

Bullets are sometimes covered or "jacketed" with a partial or complete layer of copper or other metal. If the bullet is entirely encased, it is referred to as a *full metal jacketed* (*FMJ*) bullet. High-velocity weapons often use bullets that are jacketed to protect them from fragmenting and melting while they are in the gun barrel. If the bullet is partially encased, it is termed a *semi-jacketed* or *partial metal-jacketed* bullet. The jacket in semi-jacketed bullets does not extend to the tip, usually leaving lead exposed at the tip. These types of semi-jacketed bullets are called *soft-nosed* bullets. Those that have an open cavity in their tip are called *hollow-point* or *expansion* bullets. Hollow-point bullets are designed to deform (*mushroom*) upon impact because of a collapsible space within the projectile tip. In general, all semi-jacketed bullets are designed to deform or break up on impact. This increases the effective surface area of the projectile and causes it to tumble in the tissue, both of which result in increased energy transfer to the tissue. The semi-jacketed bullet not only expands as it traverses tissue, but it also sheds fragments from its core, causing even more extensive tissue damage. Semi-jacketed high-velocity bullets often leave a trail of fragments, which widen along the wound tract as the distance from the entry site increases. In contrast, semi-jacketed lower-velocity bullets often deform by mushrooming with minimal fragmentation.

The First Hague Convention of 1899 prohibited the use of semi-jacketed bullets in combat. The greater the expansion from deformation, the more the bullet is slowed and the less the penetration of the bullet. Thus, semi-jacketed bullets are routinely used in law enforcement because they provide reduced risk to bystanders being hit by overpenetrating or ricocheted bullets and because of the increased speed of incapacitation of the designated target by these bullets. Semi-jacketed bullets are also favored by hunters and criminals for the desired rapid incapacitation. Arguably, the epitome of a bullet designed to deliver all its KE to the target tissue is the so-called devastator bullet, which is designed to explode on impact. The most infamous use of this bullet was in the attempted assassination of President Reagan in 1981 by John Hinckley. In that case, a small-caliber (.22) handgun bullet with low KE imparted significant tissue damage because it gave up all of its KE to the target and because it was fired at short range. The devastator bullet is composed of a non-jacketed hollow-point aluminum tip

containing an additional detonating compound that greatly increases its deformity, stopping power and wounding potential. The devastator bullet was developed in the 1970s for use by sky marshals to minimize the risk of penetration of the plane fuselage when incapacitating a hijacker, a concept that appears to be returning in light of recent world events.

An FMJ bullet is much more likely to pass right through the body than a semi-jacketed bullet; thus, only part of the KE of the bullet is transferred to tissue damage and used in wound formation. Unlike semi-jacketed bullets, an FMJ bullet usually stays intact when it strikes the body (**Figs. 4.19**, **4.25**, and **4.30**). However, if it strikes bone, the lead component of the bullet may become deformed and separated from its fragmented copper jacket (**Figs. 4.12C** and **4.22** to **4.24**). If an FMJ bullet does not strike bone, its tendency to fragment depends on its velocity and yaw. If it yaws significantly, its cross-sectional area increases, leading to a resultant increase in the drag on the bullet. These drag forces put great strain on the bullet, causing fragmentation. Further, the lead core of the bullet can be squeezed out the base in this situation. The jacket and core of the bullet may separate from each other due to the greater mass of the core. The jacket, because of its light weight, rapidly loses velocity and veers off at an angle from the path of the bullet core.

Shotgun ammunition is called a shell (**Fig. 4.11B**). Modern shotgun shells typically consist of a plastic case with a brass base cup. Gunpowder and primer are compressed into the brass cup at the base of the shell. Wadding material is located on top of the powder. Wadding is usually made of plastic or paper and is placed between the powder and the shot pellets. The primary purpose of a wad is to prevent the shot and powder from mixing and to provide a seal that prevents gas from blowing through the shot rather than propelling it. Shot pellets are located on top of the wadding. There are a variety of different shot pellets (usually referred to simply as shot) available, including lead, steel, bismuth, tin, tungsten-iron, and tungsten-matrix. Lead shot is not permitted for hunting waterfowl in North America because lead is a toxic substance that contaminates soils as well as surface and ground waters and can cause lead poisoning in different species as well as damage to the habitat. Shotguns used for hunting big game can fire shells that contain a single solid metal projectile referred to as a *slug*. **Slugs** are molded chunks of metal, nylon, or plastic. In effect, they turn a shotgun into a crude rifle. Slugs are fired individually, like bullets, instead of in bunches like buckshot and birdshot. The shot pellets or slug are held within the shell casing by another thin wad of paper or plastic. The whole system is then compressed, and the top edge of the casing crimped. When a shotgun is fired, it is the shot, along with the wadding, that leaves the barrel. Commonly encountered shot sizes range from 8 shot (0.23 cm), with approximately 500 pellets in a 12-gauge shell, to number 00 buckshot (0.83 cm), with 9 to 15 pellets in a 12-gauge shell. The rule of thumb for shot size is the higher the number, the smaller the diameter of the shot. Shotgun shells are usually color coded by gauge. For example, 20-gauge shells are generally yellow in color. Most 12-gauge shells are red, but shells of all gauges at times come in red, green, purple, black, and other colors. The shell size is stamped on its brass base, regardless of color. Note that caliber and gauge do not address the amount of charge within the shell, another important variable in determining KE and wounding power.

Bullet Deformity and Fragmentation Increase the Injury

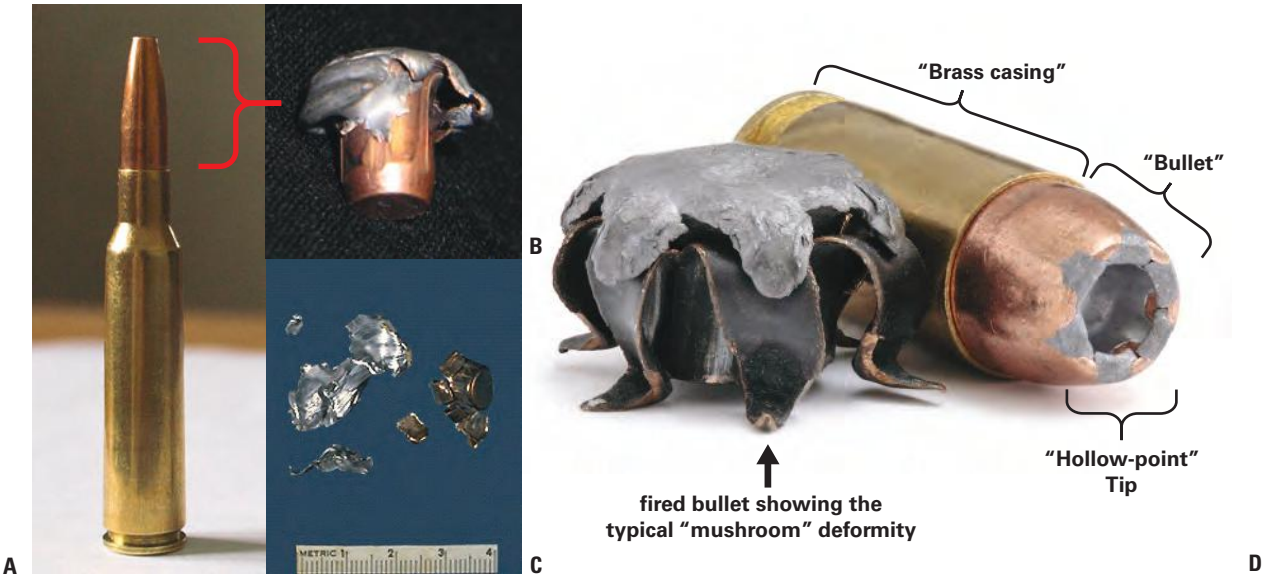


Figure 4.12. Bullet Deformity after Firing. A. Unfired rifle round with a copper semi-jacketed hollow-point bullet at its tip (*bracket*). **B.** Typical mushroom deformity of the fired bullet showing its tip peeled back to expose the lead core. This not only increases or expands the effective surface area of the projectile but it also causes the bullet to tumble in the tissue, both of which result in increased tissue damage. In addition to expansion as they traverse the tissue, deformation of semi-jacketed bullets also leads them to shed and disperse fragments, causing even more extensive tissue damage. **C.** Another example of a fired bullet showing multiple lead fragments from the bullet core and remnants of the copper jacket; these are the fragments that are readily identified on CT and plain film imaging. **D.** A copper semi-jacketed handgun round with a hollow-point tip is shown lying on its side in an unfired state next to an example of its fired and deformed counterpart: Note in the fired bullet that the cylindrical black copper base is topped with a deformed lead tip that has been flattened and splayed back to expose a larger surface area.

★**KEY POINT** Bullet deformity increases the wounding capacity of the projectile in that almost the entire weight is retained but the impact surface area of the bullet is increased over 50%.

What happens when you "pull the trigger"?

First, the firing pin of the gun strikes the primer at the base of the cartridge. This causes the primer to ignite. The ignition flame, in turn, detonates the gunpowder charge within the main chamber of the cartridge casing. This transition from solid to gaseous phase involves a rapid volume expansion of combustion gases. The buildup of pressure exerts force on the base of the bullet and the sides of the cartridge case. Ultimately, the increase in pressure propels the bullet from the tip of the casing through the gun barrel. As the bullet emerges from the barrel of the gun, it is accompanied by a jet of flame, gas, powder, soot, primer residue, metallic particles stripped from the bullet, and vaporized metal from the bullet and cartridge case, all of which can be important in forensic pathology. In rifles, metal grooves within the barrel (rifling) impart a rotational spin to the bullet along its longitudinal axis. This gyroscopic effect stabilizes the bullet's flight through the air, thereby preventing it from tumbling end over end. In other firearms, such as most handguns, the bullet emerges from the barrel and immediately yaws as described earlier.

How do bullets injure tissue?

There are two main mechanisms whereby tissue is damaged: (1) temporary cavitation and (2) direct shredding of tissue in its path (**Figs. 4.13 and 4.14**). The **temporary cavity** is formed by the sonic shock wave that precedes and coincides with the passage of the bullet through the tissue. Abrupt outward (radial) movement of the tissue from the bullet results in stretching, tearing, and shearing of the surrounding tissue. This vacuum cavity lasts a few milliseconds from its initial rapid growth until its collapse. Following the passing of this initial radial force, the tissue cavity undergoes a series of repetitive expansions and contractions that become progressively smaller in magnitude until they gradually dissipate (i.e., it pulsates until the energy is completely transferred to the surrounding tissue). The tissue snaps back to the bullet track vacuum, and this collapse is affected by the elasticity and weight of the surrounding brain tissue. Almost all bullets will be recovered either nose forward or base forward because they will realign with the wound track as the temporary cavity collapses (see **Figs. 4.9** and **4.30**). The rapid expansion of the temporary cavity creates a negative pressure gradient, pushing air inside and sweeping debris, including parts of the scalp, bone, clothing, and dirt that were dislodged by the projectile's impact into the temporary cavity. Of course, whatever bacteria included in this debris will also enter, leading to a contaminated tissue cavity with the brain. In cranial GSWs, bone, hair, and scalp tissue (and clothing if the victim is wearing a hat) will be introduced into the wound canal. It is the organic material, not the inorganic metallic bullet fragments, which is responsible for the majority of infections following penetrating injury.[20] High-velocity bullets frequently cause backward ejection of injured tissue from the entry site, a phenomenon known as *tail splash* (see **Fig. 4.13**).

The size of the temporary cavity is influenced by the size, tumbling, and velocity of the bullet as well as by the properties of the tissue encountered.[21] Because of the sonic nature of the temporary cavity, the volume and diameter of the wound is greater with larger caliber and faster bullets. In general, *the amount of tissue injury is inversely proportional to the elasticity of the target tissue*. In inelastic tissues such as brain and liver, a temporary cavity results from even low-velocity bullets. For extracranial injuries, low-velocity handgun bullets result in little temporary cavity wounding. This is because the temporary stretching of elastic tissues such as muscle and lung allow them to more effectively recover from the brief outward displacement of the tissue. Think of it as water splashing: It is displaced but not destroyed. In contrast, extracranial injuries caused by high-velocity rifle bullets can generate shock waves up to 200 atmospheres of pressure and cause profound destruction.[19] In contrast, high-velocity extracranial injuries caused by rifle bullets in which there is a tail splash, or backward hurling of injured tissue ejected from the entry site, can result in large temporary cavitation within muscle and other tissues. In both intracranial and extracranial GSWs, the maximal diameter of the temporary cavity usually occurs where there is the maximum loss of KE—this is usually where the bullet is at maximum yaw, that is, turned sideways to the trajectory path and/or when it fragments (see **Fig. 4.9**). Similarly, deformity of the bullet also increases the size of the temporary cavity (see **Fig. 4.12**). Thus, while the depth of penetration of a hollow-point bullet is less than half that of a similar nonexpanding bullet, the resulting wound cavity is much wider.

The final wound path left by the projectile is called the **permanent cavity**. The permanent cavity represents the combination of tissue that is injured from the radial stretching and shearing forces that acted during temporary cavitation and that which is permanently injured by the direct action of the projectile on the tissue. Bone and/or bullet fragments dispersed and radiating from the primary path of a bullet after impact weaken the surrounding tissue and cause secondary cavitation

events that work synergistically to further increase the final cavity dimensions. The size of the permanent bullet tract is usually larger near the entry site, but it can also be larger near the exit site or even in the middle. The most common appearance is that of a cone-shaped lesion with the base of the cone at the entry site (**Figs. 4.15G**, **4.31B**, and **4.36F**). There are several compounding influences acting on the narrowing area of damage along the path of the bullet, as defined by a conical model recently described by Folio and colleagues[22]:

> *Energy transfer to the brain is highest immediately following impact and it lessens as the fragment comes to rest. Part of the energy transfer could include lessening shock wave of the projectile. Following entry, the external ballistics of spinning on axis (with some precession) converts to tumbling of various sorts, depending on many factors. The damaging effects of the fragment likely decrease near the point of termination. Although interconnecting neural pathways are acutely damaged, chronic effects of the injury resulting in eventual encephalomalacia may not be acutely visible. Neurosurgical debridement near point of entry could also contribute to the overall conical shape.*

In addition to the above two mechanisms of injury, *contact* GSWs, such as those commonly found in suicide and homicide injuries, have an additional wounding capacity because of the associated muzzle blast, that is, the surge of hot air and gas that exits the gun barrel along with the bullet. Weapons fired against the skin can transmit thousands of pounds per inch squared of escaping powder gases and typically result in devastating wounds.

Ballistic Brain Injury Analogy

Figure 4.13. High-Speed Photography of a Bullet Fired through a Can of Cola. Note: (*1*) backfire at the entry site (analogous to the tail splash of tissue that gets ejected from the entry site), (*2*) abrupt expansion and rupture of the can (analogous to the concentric heaving fractures described later), and (*3*) a disproportionately larger exit injury versus entry site. The fired bullet emerges minimally deformed (*4*). (Courtesy of Andrew Davidhazy.)

Behavior of a Bullet through the Brain

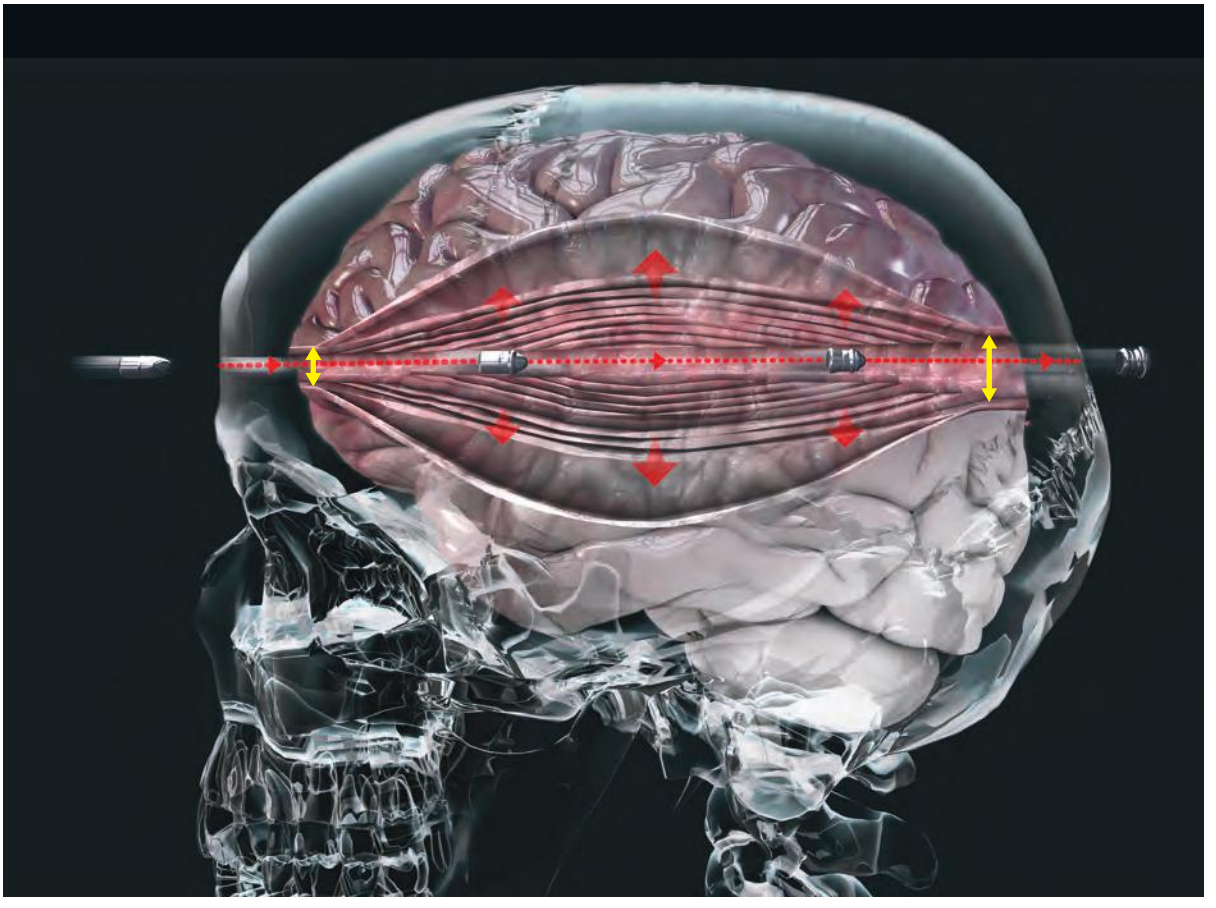


Figure 4.14. Idealized Behavior of a Bullet through the Brain. As the bullet moves through the brain, it crushes and shreds tissue in its path, forming the so-called permanent cavity. At the same time, it transiently displaces outward the surrounding tissue from its path, forming the temporary cavity. In reality, the temporary cavitation is more asymmetric, spreading out in different tissue planes. The size of the final wound track is a combination of these two mechanisms. The energy loss along the wound track is not uniform, and the entry site is always smaller than the exit site (*yellow arrows*). This is partially due to deformation, expansion, and yaw of the projectile as it traverses the tissue. Also note that the temporary cavity is always larger than the permanent cavity. This is particularly the case in high-velocity injuries. As the wound cavity expands within the tissue, a negative pressure gradient arises, and there is aspiration of foreign material into the cavity. In this way, contaminated dead tissue comes to line the final wound tract. (Courtesy of Graham Hutchings, Sinelab, UK.)

★**KEY POINT** Unlike extracranial GSWs, the skull restricts outward movement of the tissue. Unfortunately, this potential protection from temporary cavitation is more than offset by the added curvilinear movement of brain tissue that causes severe strain injury. In summary, it is the transfer of KE from the projectile to the tissue and the ability of that tissue to accommodate that energy that ultimately determines the size and morphology of the final permanent cavity.

Finally, ballistic pressure waves from a bullet impact to the chest can cause remote wounding to the spinal cord, and a GSW to an extremity can cause a concussive-like effect in the brain. Ballistic pressure waves can even break bones.[23] This type of remote injury by a penetrating projectile is called *hydrostatic shock* and is attributed to a hydraulic effect in liquid-filled tissues. Human autopsy studies by Krajsa and colleagues have demonstrated *cerebral* hemorrhage following GSWs to the chest, including cases with handgun bullets[24]:

> *In such meticulously selected cases brain tissue was examined histologically; samples were taken from brain hemispheres, basal ganglia, the pons, the oblongate and from the cerebellum. Cufflike pattern haemorrhages around small brain vessels were found in all specimens. These haemorrhages are caused by sudden changes of the intravascular blood pressure as a result of a compression of intrathoracic great vessels by a shock wave caused by a penetrating bullet.*

TABLE 4.1 Key Points in Wound Ballistics

1. The entry wound is always smaller than the exit wound.
2. The inner table of the skull is beveled at the entry site; the outer table is beveled at exit site.
3. The size of the final wound is a combination of both the temporary and the permanent cavity.
4. Aspiration of foreign material occurs into the temporary cavity, and contaminated dead tissue lines the final wound tract.
5. The diameter of the wound canal always exceeds the diameter of the projectile.
6. The *transfer* of KE from the projectile to the tissue and the ability of the tissue to *accommodate* that energy determine the size and morphology of the final permanent cavity.
7. The *wounding capacity* of a bullet is dependent on its caliber (larger is more destructive), composition (lead vs. alloy), configuration (soft vs. hollow nose), velocity (amount of charge), and the presence/extent of a jacket (e.g., high-caliber, high-velocity, semi-jacketed bullets cause the most severe injury; streamlined FMJ bullets experience less drag and deceleration as they pass through the tissue than expanding semi-jacketed bullets).
8. Bullet deformity, fragmentation, and yaw increase the size of the wound.
9. If a bullet exits the tissue, it transfers less energy than a bullet that embeds and releases its kinetic energy within the tissue.
10. The viscoelastic properties of the target tissue partially determine the extent of injury. Unlike extracranial tissues, the brain is not elastic, and its containment within the skull magnifies the size of the temporary cavity.
11. The denser the target tissue, the greater the drag on the bullet, and thus the greater the loss of KE and the more the tissue damage and wounding potential. Increased tissue density also acts to increase the yaw of the bullet and shorten the period of gyration, which leads to greater slowing of the bullet and an increased loss (transfer) of KE, resulting in more tissue damage.

KE, kinetic energy; FMJ, full metal jacketed.

Fatal Gunshot Wound Showing Typical Poor Prognostic Imaging Findings

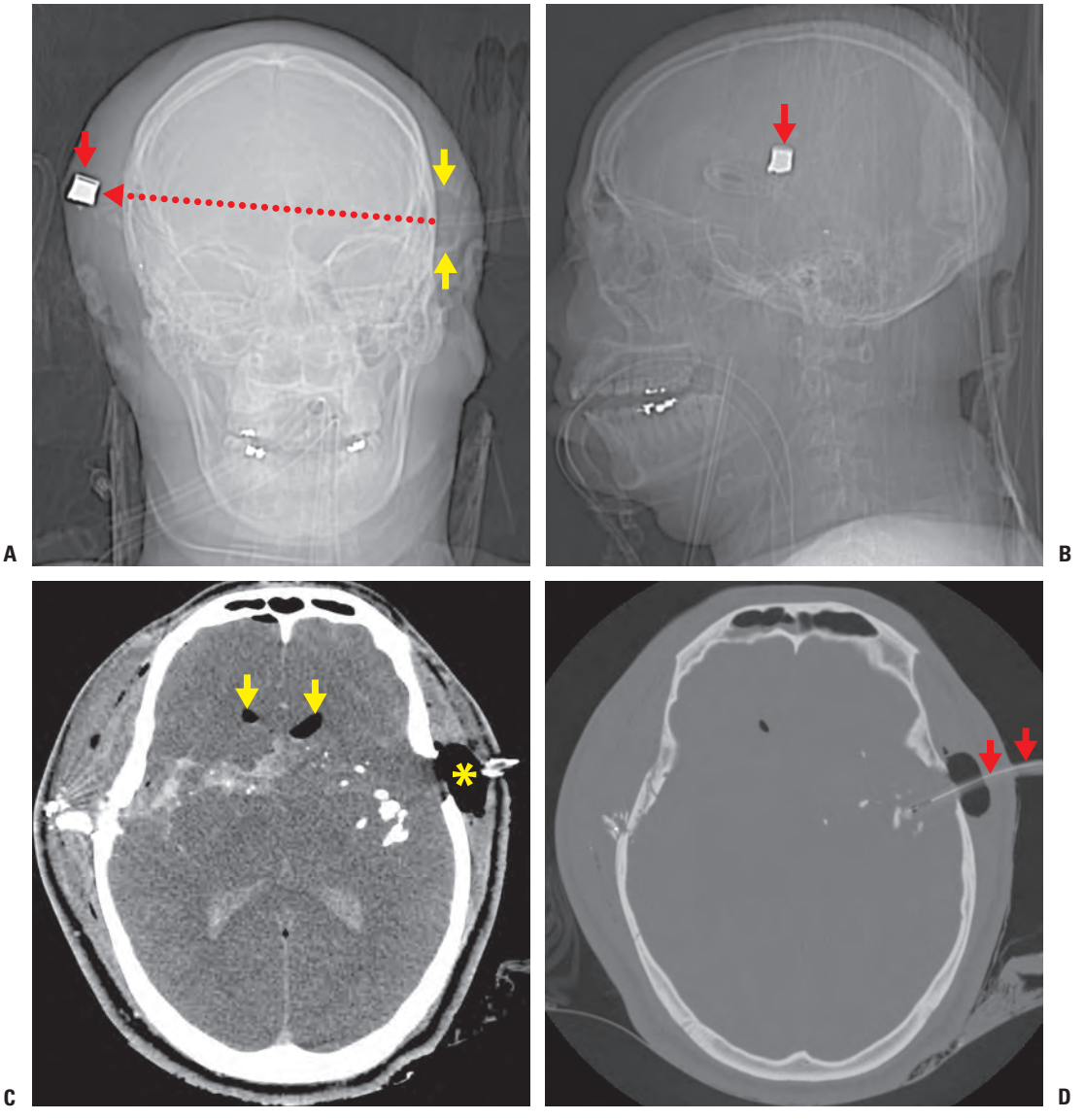


Figure 4.15. **Fatal Gunshot TBI. A.** AP and **(B)** lateral scout views demonstrate the direction of GSW trajectory (*dotted arrow*), the major bullet fragment lodged within the scalp soft tissues (*red arrow*), and the shadow of a Foley catheter at the entrance site (*yellow arrows*). The Foley catheter was inserted because of massive hemorrhaging from the entry site. **C.** Axial CT image demonstrates multiple bone and bullet fragments traversing diagonally across the midline. There is diffuse cerebral edema and bifrontal intraventricular air (*arrows*). The Foley catheter is again noted (*asterisk*). **D.** Bone windowing reveals characteristic beveling of the inner table of the skull at the entry site and the Foley catheter (*arrows*). (*Continued*)

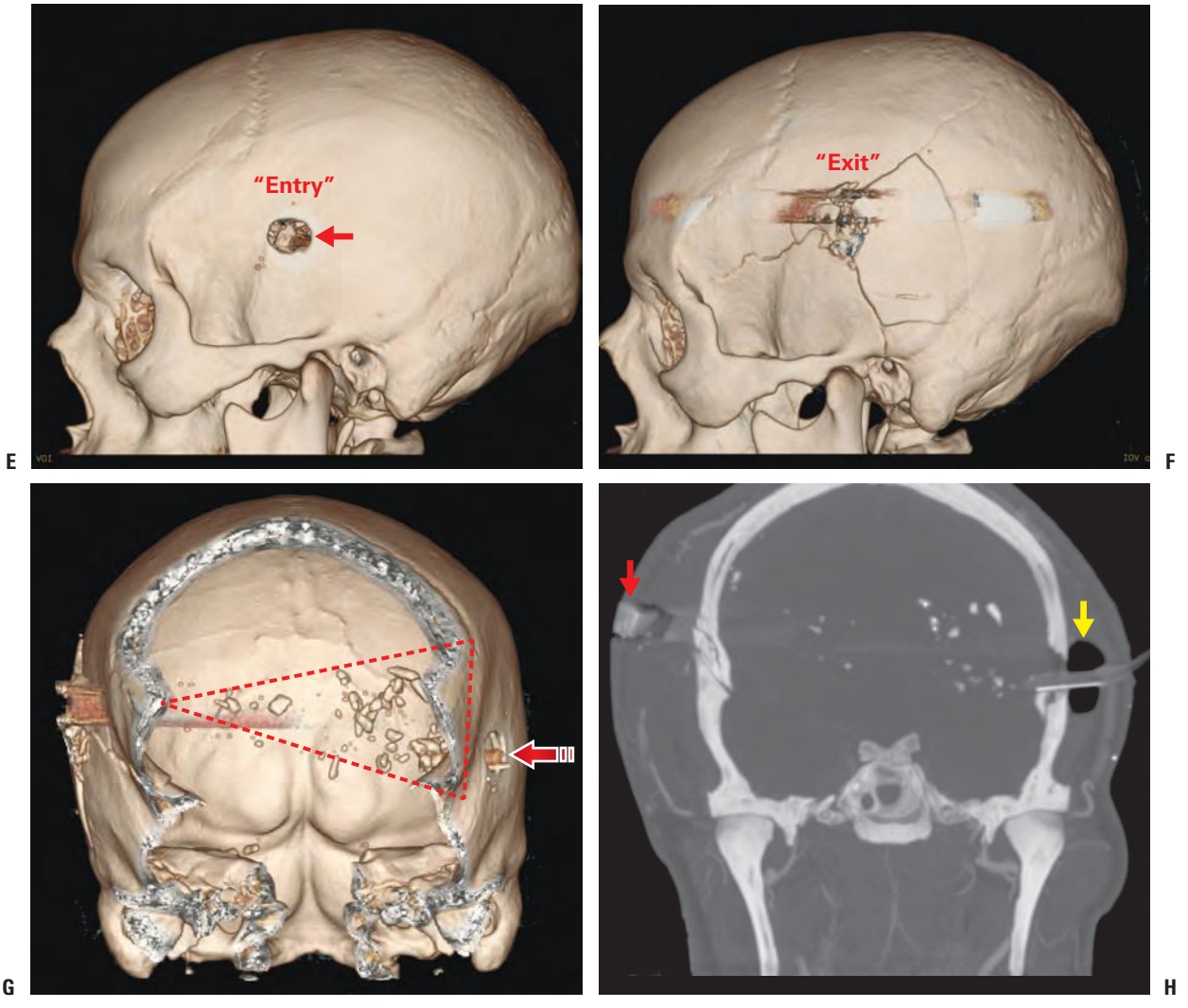


Figure 4.15. (*Continued*) **E, F.** Volume-rendered 3D CT images demonstrate the well-defined entry site (*arrow*) and the comminuted fracture at the exit site. This is a classic example of how a bullet punches out a circular wound at the entrance in the skull, driving fragments of bone into the brain. These bone chips create secondary tracks that deviate from the main path and destroy additional tissue. **G.** Coronal 3D cutaway CT image demonstrates the left-to-right trajectory of the GSW with innumerable fragments scattered throughout the brain, most of which are located toward the entry site (*arrow*). Note the cone-shaped distribution of intracranial fragments with the base of the cone centered at the entry site (*triangle*). **H.** Coronal MIP image from the CTA shows many of the above-mentioned findings, including the Foley catheter occluding the entry site (*yellow arrow*), beveling of the inner table of the skull, multiple bone fragments along the GSW trajectory, a comminuted fracture at the exit site, and the major ballistic fragment lodged with the right frontotemporal scalp soft tissues (*red arrow*).

★**KEY POINT** Imaging findings that predict a poor outcome following GSW and other penetrating brain injuries include (1) multiple lobes involved, (2) bilateral trajectory, (3) transventricular trajectory, and (4) bullets that traverse the diencephalon or brain stem.

Shotgun Injuries

Shotgun ammunition consists of multiple pellets and thereby produces numerous wound canals. Buckshot pellets tend to stick together as they proceed downrange, transferring all of their collective energy into the target. Smaller pellets, such as birdshot, on the other hand, have an inherent tendency to spread out more widely. The clinical characteristics of shotgun wounds are distinctly different from other bullet wounds. In fact, the scope of possible injuries is so great that only a few general observations can be made. A person struck by a single pellet that is smaller than BB size is seldom injured unless hit in the eye. Most human wounds occur at ranges closer than 20 m, wherein most of the total pellet charge hits the victim. A shotgun injury is typified by a relatively large entrance wound, especially if such an injury occurs at close range.[25] Within the target tissue, shotgun pellets produce a cone-shaped temporary cavity with the base of the cone at the entry site, and the diameter of the cavity gradually lessens as the velocity of the pellet decreases. Once within tissue, shotgun pellets decelerate more rapidly than bullets. This is because of the unfavorable ballistic properties of shotgun pellets, that is, they have a large cross-sectional area in relation to their mass.

Shotgun pellets have significant aerodynamic resistance and give up substantial amounts of KE during flight. For this reason, *range* is the most important determinant of the amount of damage inflicted by a given shotgun charge. Therefore, shotgun injuries are often classified based on the distance from the gun barrel to the victim.[26] In type I shotgun injuries (<5 m), the pellets strike the head as a single mass, resulting in massive KE transfer, tissue avulsion, and a high mortality rate (85% to 90%). Type II injuries (5 to 12 m) usually result in less tissue destruction. At these distances, there is significant dispersal of the pellets and loss of energy. Penetration occurs through the scalp soft tissues, but skull fractures are uncommon. Ocular injuries are common, as well as embolization of pellets, but mortality is less than that of type I injuries (15% to 20%). In type III injuries, at distances >12 m, the pellets usually only penetrate the skin, and mortality is rare (0% to 3%). The clinician generally does not have access to specific information on the distance the target (i.e., patient) was from the shotgun weapon. To compensate for this difficulty, a radiologic system, based on the maximum diameter of pellet scatter, was developed to classify shotgun injuries.[27]

On CT, the innumerable shotgun pellets show a characteristic *billiard-ball* dispersal pattern with extensive metallic beam-hardening artifact radiating from each pellet.[28] At very close range, a victim is hit not only by pellets but also by the wadding and plastic casing debris of the shotgun shell, both of which may also be identified on imaging. The diameter of the dispersed pellets at the target site can easily be measured on CT. In brief, type I injuries have >25 cm of pellet scatter. Type II injuries have 10 to 25 cm of scatter (**Fig. 4.16**). Type III injuries have <10 cm of scatter. The diameter of pellet scatter is inversely proportional to the distance between the shotgun and target, and thus, a type III injury in this radiologic classification scheme would roughly correspond to the severe type I injury described in the previous system that relies on knowing the range (**Fig. 4.17**). This radiologic grading scale has also been shown to correlate well with morbidity and mortality. The loss of velocity is much more rapid for shotgun pellets than bullets because of their unfavorable ballistic properties, that is, they have a large cross-sectional area in relation to their mass. In contrast, high-velocity rifle wounds may create a temporary cavity that is many times larger than the bullet caliber.

Type II Shotgun Injury to the Face (Acute)

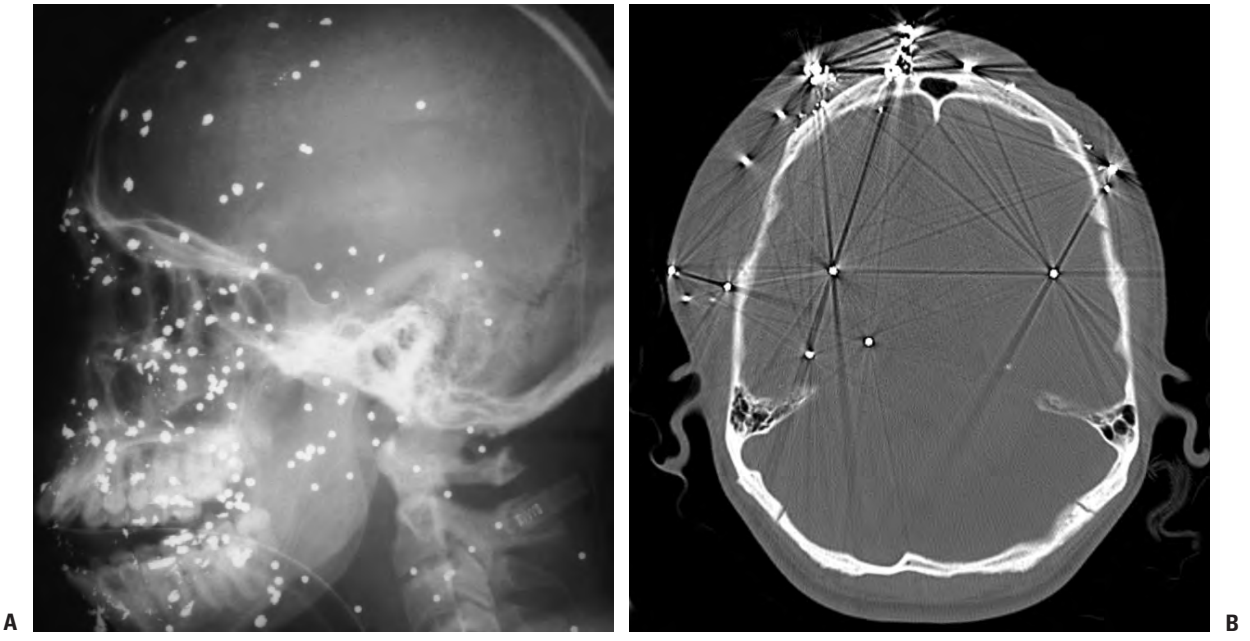
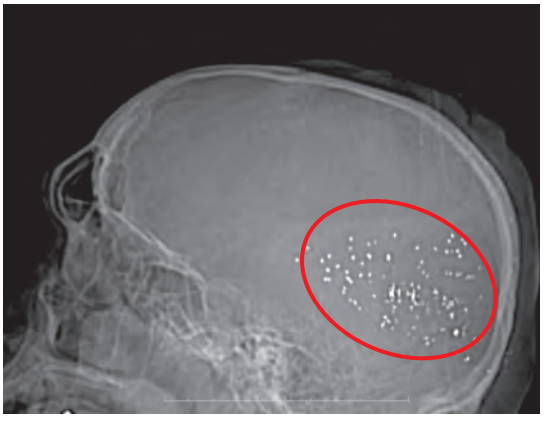


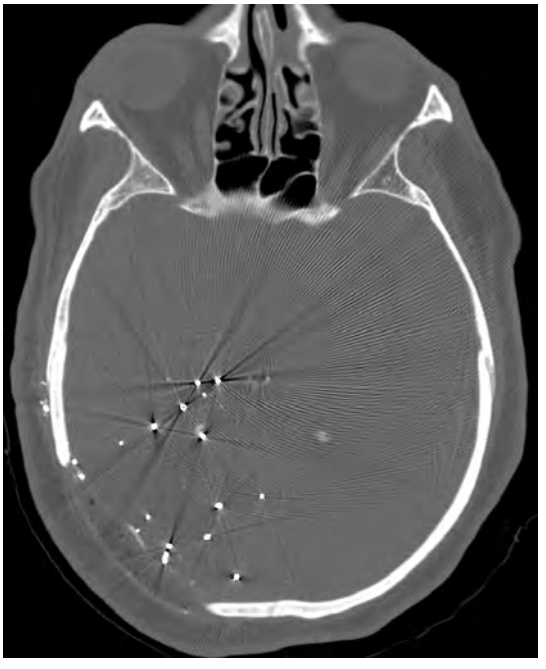
Figure 4.16. **Shotgun TBI. A.** Lateral radiograph of the skull reveals innumerable small metallic foreign bodies overlying the brain and face. **B.** Axial CT image in a similarly injured patient viewed at bone windowing clarifies the intracranial and extracranial location and the typical *billiard-ball* appearance of pellets. The imaging findings are consistent with a type II shotgun injury (i.e., 10 to 25 cm of pellet scatter). The diameter of pellet scatter is inversely proportional to the distance between the shotgun and target. (Courtesy of Curtis Offiah, Royal London Hospital, UK.)

★**KEY POINT** Lead shot deforms on impact; steel shot remains spherical. In this case, note how the metal fragments are slightly irregular in shape (best appreciated on the plain radiograph), consistent with the fact that the shot pellets are not made exclusively of steel.

Type III Shotgun Injury to the Occiput (Chronic)



A



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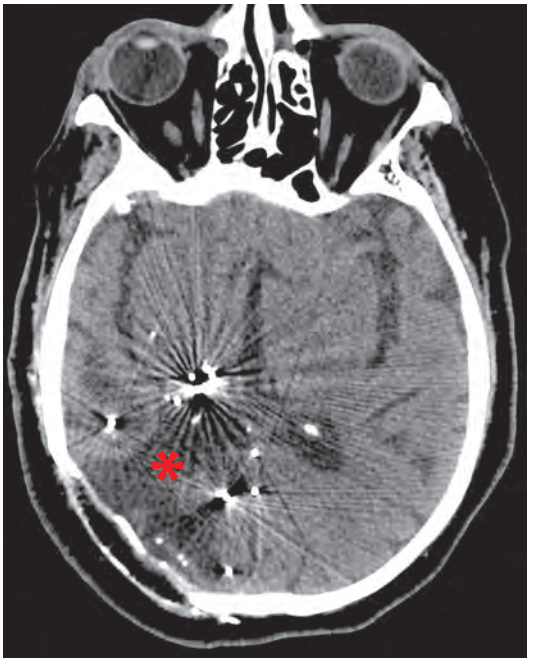


C

Figure 4.17. Shotgun TBI. A. Lateral CT scout view demonstrates a relatively well-localized collection of punctate foreign bodies overlying the occipital region (*circle*). **B.** Axial CT viewed at bone-windowing shows the prior right occipital cranioplasty and retained shot. **C.** Corresponding brain window reveals right occipital encephalomalacia (*asterisk*) that is partially obscured by metallic artifact from the retained foreign bodies. Note the tight pellet scatter pattern (i.e., <10 cm), consistent with a type III shotgun injury. Remarkably, the patient survived this close-range injury.

Imaging approach to gunshot wounds

One of the unfortunate limitations of CT and CTA in the evaluation of GSW (and IED explosions) is the presence of streak artifacts from retained metal fragments. Therefore, reviewing first the AP and lateral CT digital topogram (i.e., scout view) can be helpful to appreciate the location of the metal fragments, assess the wound trajectory, and help identify the type of bullet used (see **Figs. 4.15** to 4.17 and 4.23 to 4.25).[14] Similarly, a dedicated AP and lateral plain radiograph can be helpful (see **Figs. 4.19** and **4.30**). A 3D CT can also be useful to optimally demonstrate the path of the bone and metal fragments (**Figs. 4.15** and **4.36**). Beyond the immediate effects of patient care and prognosis, GSW trajectory analyses can also be important in forensics.[29,30] MRI is not an option in these patients unless one knows for certain that the projectile is non-ferromagnetic (e.g., lead). Although the majority of police,

commercial sporting, and criminal bullets manufactured in the United States are non-ferromagnetic, military and paramilitary ammunition commonly contain ferromagnetic material, typically in the jacket covering the lead or antimony core. On MRI, non-ferromagnetic bullets cause minimal image-degrading artifacts unlike the marked streak artifacts seen with CT.[31] However, new CT technology such as dual-energy computed tomography (DECT) (i.e., spectral CT) is able to decrease metallic streak artifacts and at a lower radiation dose (**Fig. 4.18**).[32] DECT uses two x-ray tubes and two data acquisition systems mounted on the same gantry. Each source has an independent high-voltage generator and is therefore twice as fast. Three-dimensional imaging is more automated, accurate, and faster (20 images per second on 64 slice scan vs. 40 images per second) with DECT. Future applications of DECT in head injury are likely to include (1) providing information about tissue composition, (2) predicting the evolution of soft tissue injuries, (3) generating virtual unenhanced images, (4) helping to determine ferromagnetic properties of foreign bodies, and (5) improving detection of iodine-containing substances on low-energy images, thereby improving CTA and computed tomography perfusion (CTP) in head injury at a reduced radiation dose.

Dual-Energy CT Decreases Artifacts from Metallic Foreign Bodies

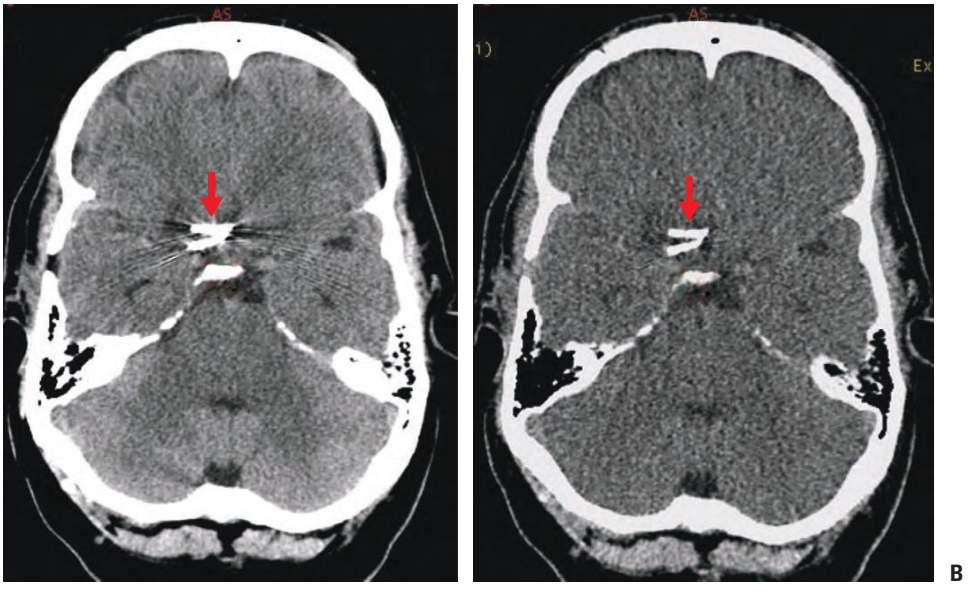


Figure 4.18. **Dual-Energy CT Improves Foreign Body Visualization. A.** Routine noncontrast CT scan performed at 110 keV in a patient with two anterior communicating aneurysm clips (*arrow*). Note metallic streak artifact obscuring the regional anatomy. **B.** Same patient scanned at 70 keV demonstrates decreased metallic artifact (*arrow*). However, the gray–white matter differentiation of the brain parenchyma is not demarcated as clearly in DECT images.

★**KEY POINT** DECT imaging decreases imaging artifacts associated with intracranial metallic projectiles, resulting from ballistic TBI.

If one is imaging a shotgun injury that clearly contains lead pellets, MRI can be safely performed, and it will be far superior to CT for demonstrating the brain injury. A simple analysis of whether the shot is lead versus steel can be made with plain radiographs—steel shot is round, whereas lead shot is deformed. However, this generalization for shotgun pellets does not apply to jacketed bullets, because the type of metal used for the jacket cannot be determined from plain films. Moreover, when a nondeformed bullet is seen on a radiograph, it is not possible to state with certainty whether or not the bullet is fully jacketed. It is possible that deformation might not occur if the bullet, on reaching the target, has insufficient velocity or does not strike a structure hard enough to deform it. Thus, the presence or absence of deformation does not help to determine whether the bullet is fully or partially jacketed, and one cannot use bullet deformation to differentiate which patients are safe to undergo an MRI. Of course, fragments surgically removed from the patient can be helpful for bullet identification, but this is a rare opportunity.

If the trajectory of the wound passes near or through the Sylvian fissure, supraclinoid region, cavernous sinus, or a major venous sinus, further imaging with multislice CTA is warranted. For cervical GSWs, the presence of an artery in association with periarterial gas, a periarterial hematoma, adjacent fat stranding, or a nearby (within 5 mm) missile fragment

raises concern for possible vessel injury, even if the artery itself appears normal and uninjured on the CTA study. In these circumstances, a conventional angiogram should be performed to more closely evaluate the possibility of vessel injury.[33] If conventional angiography cannot be performed, follow-up CTA should be done, generally early within the next few days to 1 week and possibly again at a delayed time point within the first month, to assess for any interim changes in vessel morphology. Over time, traumatic pseudoaneurysms may develop from injured vessels. A cerebral angiogram is absolutely mandated by the occurrence of substantial or otherwise unexplained subarachnoid hemorrhage (SAH) or delayed intracranial hemorrhage in order to exclude evolution and rupture of a traumatic pseudoaneurysm. While MR angiography has been valuable in screening patients at risk for cerebral aneurysms, such as those with polycystic kidney disease and close relatives with aneurysms, this noninvasive screening is not adequate for patients with delayed traumatic hemorrhage. Traumatic pseudoaneurysms are often small, and unlike their nontraumatic counterparts, which localize to the circle of Willis, traumatic pseudoaneurysms are typically situated more peripherally in the vascular tree. Cerebral angiography is required to achieve the sensitivity and specificity necessary to reliably detect the characteristic small size and peripheral nature of traumatic pseudoaneurysms. Additional imaging recommendations for penetrating injuries are discussed later in Chapter 5, Lessons 3, 4, and 10.

Gunshot injuries to the head differ significantly from gunshot wounds to other parts of the body

The rigid skull helps protect the brain from most blunt trauma as well as some penetrating injuries. However, when penetrating trauma does pass through the skull, the closed cranial compartment renders the brain the most susceptible organ of the body to damage from penetrating ballistic injury. Because the skull is a closed-pressure, closed-volume system, the degree of expansion from radial forces during temporary cavitation that is permitted in other tissues is restricted in the brain. Therefore, in comparison to extracranial GSWs, the restricted pulsatile movements of brain tissue, emanating from passage of the bullet, lead to less temporary cavitation. Unfortunately, this restriction does not decrease the amount of damage but rather amplifies the transfer of ballistic KE to the brain. The only way that the radial pressure experienced during temporary cavitation can be released is by "bursting" of the brain tissue and calvarium. Displacement of the cavity-forming forces on the tissue causes significant shear-strain injury to the brain (which is less compliant than most soft tissue found elsewhere in the body). These same forces are also exerted by bone and bullet fragments that serve as secondary missiles, adding to or compounding fractures and bursting injuries.

The role of the temporary cavity in the production of the final permanent wound canal can be appreciated by firing a high-velocity bullet at an empty skull. In this setting, there is a small entrance and exit hole with no fractures. The same bullet fired through a skull containing brain tissue causes extensive fracturing and bursting brain injury.[34] Close-range injuries are also intensified by gas entering and rapidly expanding within the closed confines of the skull, adding further to the pressure that is exerted upon the skull cavity. Wounds from hunting and homicide/suicide bullets tend to be more devastating to the head than wounds produced by military rounds because, even though both bullets possess the same amount of KE on impact, the deformable, nonmilitary bullet will

transfer more energy to the head. Military jacketed bullets may pass right through the head.

Cranial GSWs are influenced by the fact that the brain is not only encased by the skull but is also compartmentalized by the tentorium and falx cerebri. Therefore, brain injury occurs not only in the vicinity of the wound track but also at a considerable distance away from the projectile path where the brain may be thrust against the dura, falx, or tentorium in a contrecoup-type injury. Reports have described remote contusions of the inferior orbital gyri, mammillary bodies, uncus, cerebellar tonsils, and medulla.

In addition to the penetrating mechanisms described previously, craniocerebral ballistic injuries can also result from the basic coup–contrecoup mechanism seen in nonpenetrating TBI. Sudden impact of the projectile to the head can result in abrupt acceleration of the skull, which results in shear strain forces and contusive brain injury. Shear patterns inside the nonspherical compartments of the dural-partitioned cranium change with every different direction of rotation depending on the orientation of the body and the trajectory of the projectile. These shear forces can cause injury in unexpected sites far from the point of impact. Injury can be caused by close-range impact of blunt weapons, such as rubber bullets, or large debris from explosions. Most often, the rotational injury is produced in combination with cranial fractures and penetration. This makes the shearing injury easy to miss because one tends to concentrate on the obvious fracture and local injury to the brain.

Craniocerebral GSWs are classified as *superficial*, *penetrating*, and *perforating* types depending on the depth of injury. In general, there is an increasing severity of injury from superficial to penetrating to perforating injuries.[35,36]

1) **Superficial Missile Injury.** In a superficial or *tangential* injury, the bullet trajectory is extracranial and the skull remains intact. The one exception is the so-called concentric heaving fracture, discussed later (**Figs. 4.33** and **4.34**). Because of the skin's elastic properties, a bullet may be arrested subcutaneously by the trampoline-like action of the skin and caught within the soft tissues of the scalp. However, even when the bullet trajectory is extracranial, sparing the skull, damage can still occur within the underlying brain as a result of the force of the bullet's impact in the overlying scalp soft tissues (see **Fig. 4.19**). This occurs more often with low-velocity, as compared to high-velocity, bullets, which tend to flatten out and are recovered within the scalp. Because the destructive KE from a bullet equals half its mass times its velocity squared, a large low-velocity bullet may not penetrate the skull but may still result in an underlying cerebral hematoma and death. Similarly, pellets from a long-distance shotgun injury can be localized to the superficial scalp region, but the underlying brain may suffer a significant shock wave contusion, and with increasing velocity of the pellets, depressed skull fractures and subjacent parenchymal injury may be observed.

Superficial Gunshot Wound

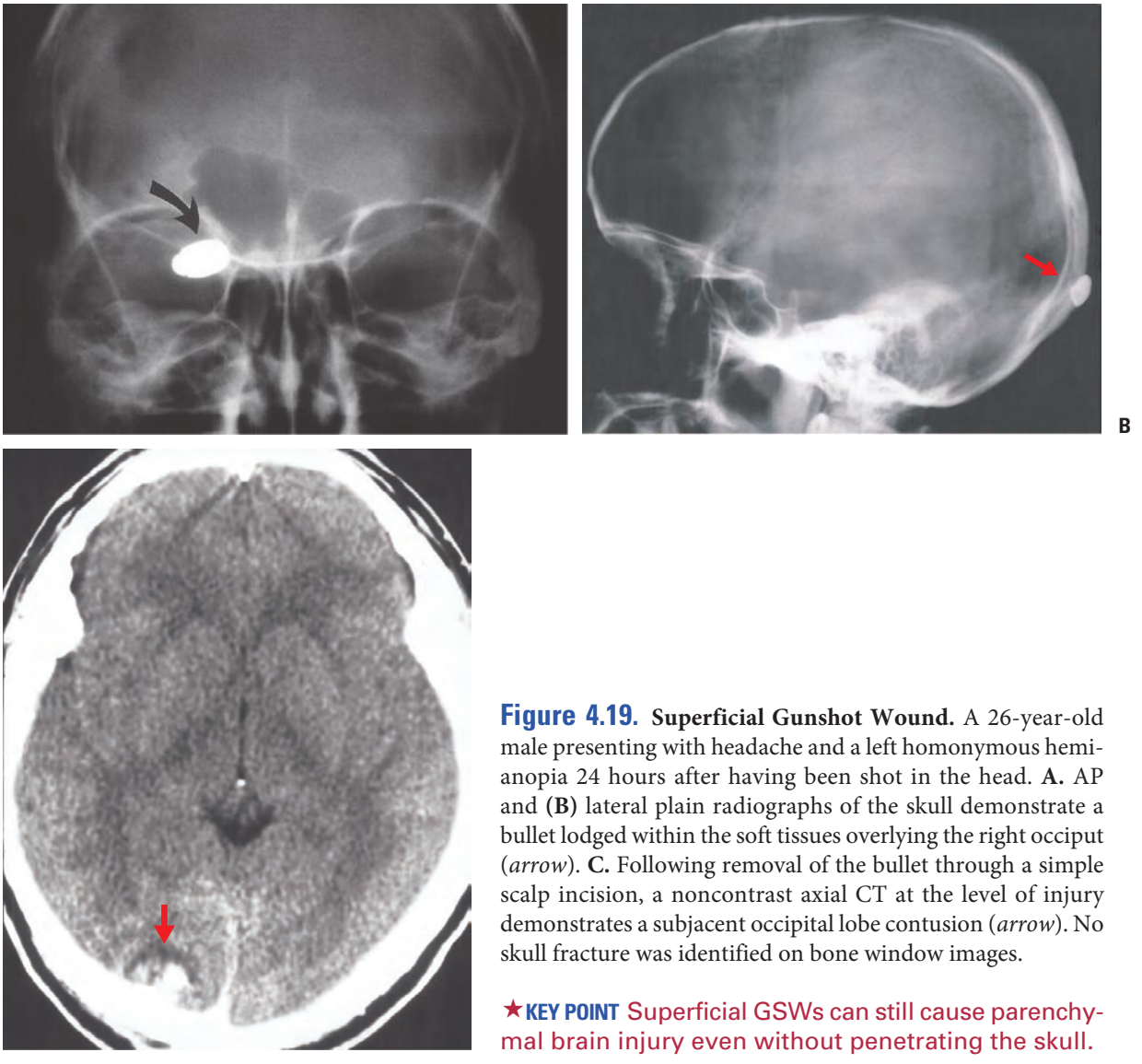


Figure 4.19. Superficial Gunshot Wound. A 26-year-old male presenting with headache and a left homonymous hemianopia 24 hours after having been shot in the head. **A.** AP and **(B)** lateral plain radiographs of the skull demonstrate a bullet lodged within the soft tissues overlying the right occiput (*arrow*). **C.** Following removal of the bullet through a simple scalp incision, a noncontrast axial CT at the level of injury demonstrates a subjacent occipital lobe contusion (*arrow*). No skull fracture was identified on bone window images.

★**KEY POINT** Superficial GSWs can still cause parenchymal brain injury even without penetrating the skull.

Penetrating Gunshot Wound

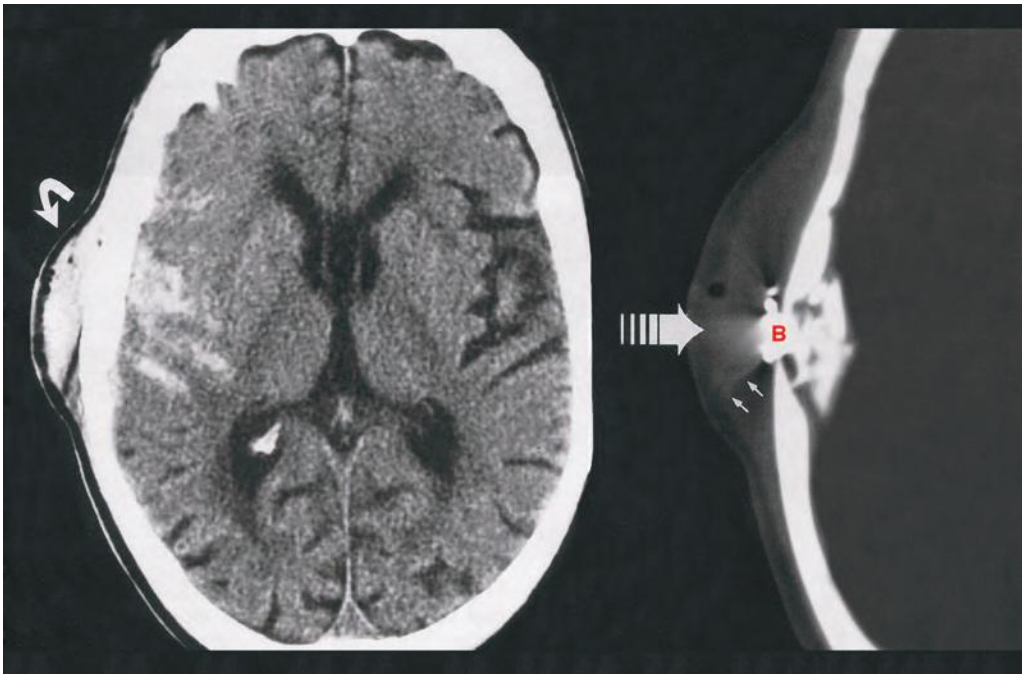


Figure 4.20. Penetrating Gunshot Wound. A. Noncontrast axial CT reveals posterior right frontotemporal scalp soft tissue swelling (*arrow*) together with a subjacent temporal lobe contusion, SAH, and effacement of the right frontotemporal sulci (compare with the normal left sylvian fissure). **B.** Magnified bone window CT technique through the injury demonstrates multiple small bone fragments that are derived primarily from the inner table of the skull. A large bullet fragment (*B*) is lodged within the outer table of the skull. It can be distinguished from the bone fragments by its ability to cause significant streak artifact (*small white arrows*). (Reproduced with permission from Gean AD. Imaging of Head Trauma. Philadelphia, PA: Lippincott Williams and Wilkins; 1994).

2) **Penetrating Missile Injury.** The majority of ballistic injuries penetrate the skull, meninges, and brain, thus resulting in a **penetrating** missile injury, in which the projectile enters without exiting the skull (**Figs. 4.20** to **4.26** and **4.30**). Scalp soft tissue, including hair, is usually embedded into the brain. Impact of projectile with the skull modifies its behavior, slowing it down and increasing its deformity and fragmentation. Once the bullet has entered the skull, its remaining velocity allows it to travel deeper into the brain in direct proportion to the density and square of the velocity of the bullet. As the projectile traverses the brain, tissue is compressed into the walls of the missile tract and is ejected out of both the entrance and exit wounds. As mentioned earlier, the temporary cavitation creates a permanent tract that is several times larger than the projectile diameter. As a general rule, after striking the skull, bullets are not deflected from their original trajectory. However, as discussed in the following text, they may ricochet off the inner table of the skull and terminate in unexpected locations. After coming to rest, projectiles can also change their position within the brain over time.

3) **Perforating Missile Injury.** With still greater velocity, a missile that has penetrated the skull may exit the contralateral side of the skull, resulting in a perforating injury (**Figs. 4.14**, **4.15**, **4.27** to **4.29**, and **4.31** to **4.36**). Such a through-and-through injury leaves only one track through the brain. The entry and exit sites can be distinguished by the direction of beveling of the skull defect: The *inner* table defect of the skull is beveled at the entry site. The larger, *outer* table bevel of the skull is characteristic of the exit site.[37] In addition, the exit site is always larger than the entry site, both in extracranial and intracranial GSWs.[14] Whether a bullet penetrates or perforates the skull is dependent on several factors: close range versus distant range, high velocity versus low velocity, contact area between the projectile and bone (e.g., deformable or jacketed bullet), the mass and caliber of the bullet, the angle of interaction with the skull, and the thickness of bone at the site of impact. Large caliber, jacketed bullets fired at close range are more likely to penetrate/perforate. Both penetrating and perforating injuries are characterized by the presence of intact or distorted projectiles along the trajectory, admixed with skull fragments. The inward driven chips of bone or missile fragments result in additional brain damage because they serve as secondary missiles that transmit additional radial force to the tissue. They can also embolize to the heart, lungs, and brain vasculature (**Figs. 3.9B** and 5.76).[38,39]

Intracranial projectiles can ricochet and migrate. The final resting site of a projectile in the brain may not necessarily be located along the primary wound trajectory. Because the brain is relatively soft (Jell-O consistency), once a bullet penetrates the skull, up to 25% will ricochet within the cranial cavity.[19] Depending on the residual KE of the bullet, it can be deflected from the contralateral surface of the skull and continue to move along the curvature of the inner surface of the skull or ricochet back into the brain parenchyma (see **Figs. 4.4** and **4.21** to **4.23**).[40] When a bullet follows an inner tangential course, the wound may appear superficial and give a misleading message as to the degree of injury. As a bullet follows a tangential path, the damage to the brain may be surprisingly extensive because of laceration of arteries and veins in the subarachnoid space. The bullet can even bounce off the inner table of the skull one or more times, with the production of two or more bullet paths at various angles to one another.[41] Bullets ricocheting off bone generate secondary bone/bullet fragments that can cause additional injury. Intracranial bullet ricochet is not restricted to bony surfaces; it may also occur from other firm structures such as the falx and tentorium. As a general rule, internal ricochet is more common with lead and small-caliber bullets (e.g., .22 lead bullets).

Gunshot Wound Ricochet Injury

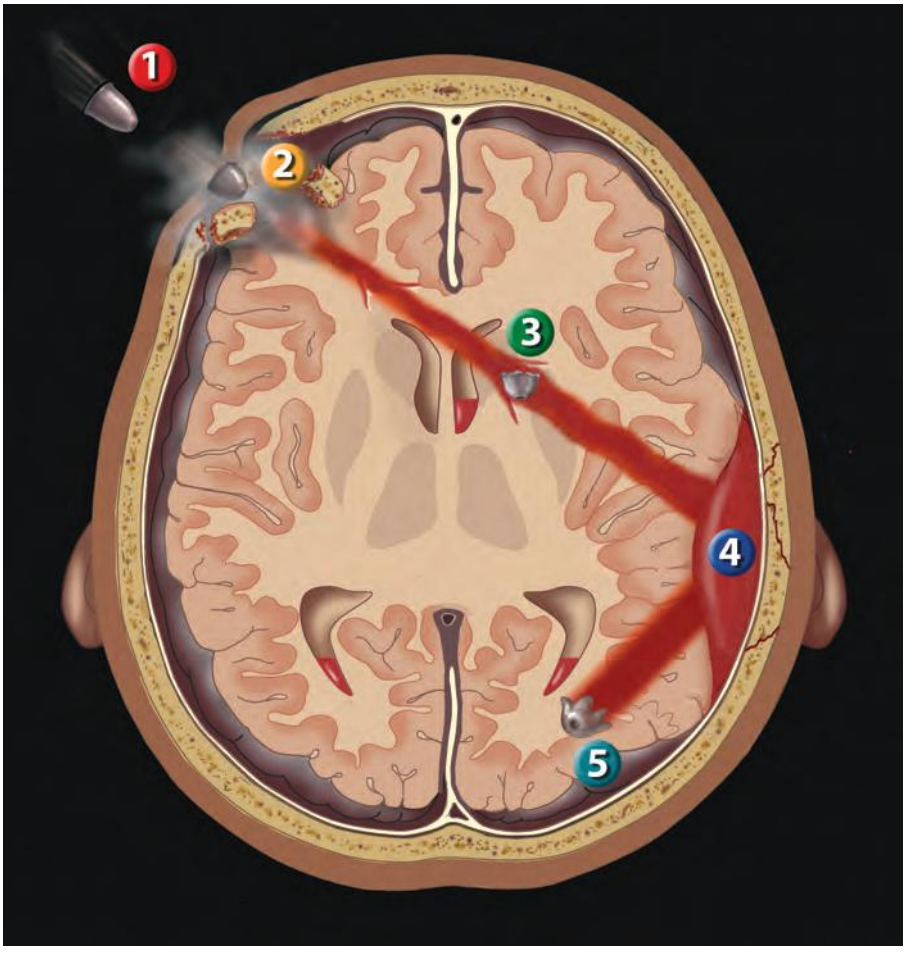


Figure 4.21. Gunshot Wound Ricochet Injury. Illustration of a typical GSW ricochet injury to the head. Note how the final location of the projectile is out of the expected wound tract trajectory. In this drawing, the bullet entered the right frontal calvarium, traversed across the midline, impacted the left temporal calvarium, and then ricocheted into the left occipital lobe. A small amount of intraventricular hemorrhage is noted within the left frontal horn and dependently within the occipital horns.

(1) Extracranial bullet
(2) Impact with the right frontal calvarium causes mild deformity of the projectile and formation of secondary projectiles, arising from comminuted skull fracture fragments
(3) Deformity of the bullet increases as it passes through the brain tissue
(4) Epidural hemorrhage and a left temporal skull fracture resulting from the impact
(5) After ricocheting off the temporal bone, the bullet is maximally deformed and has lost all of its kinetic energy

★**KEY POINT** Note how the size of the bullet tract becomes slightly larger as it traverses the brain tissue. This is because of increased deformity and tumbling of the projectile as it moves through the brain (see also **Fig. 4.9**). In other cases, however, the wound canal may assume a more conical shape with the base of the cone at the entry site (see also **Figs. 4.15G**, **4.31B**, and **4.36F**). The conical shape is likely attributed to maximal energy deposition at the entry site by a combination of bone and bullet fragments.

Gunshot Wound Ricochet Injury

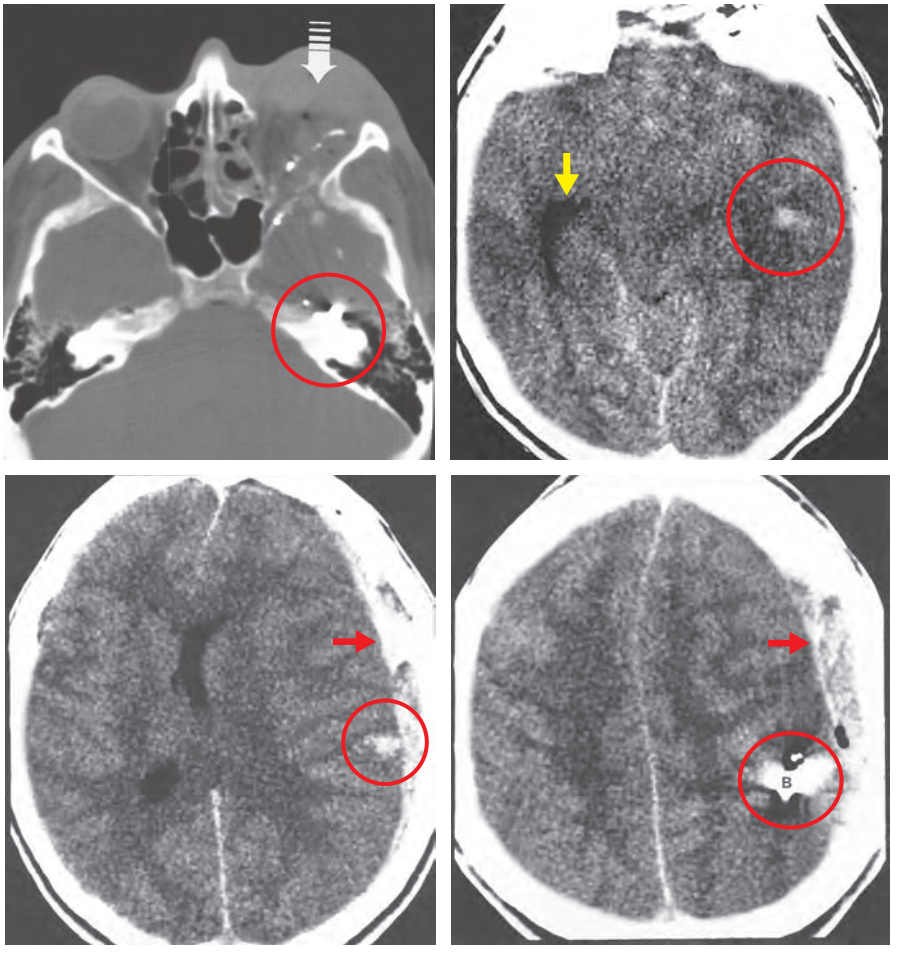


Figure 4.22. Gunshot Wound Ricochet. The bullet entered the cranium via the left orbit (*white arrow*), travelled directly posteriorly through the greater sphenoid wing, and impacted the left petrous ridge (*circle*). It then ricocheted directly superiorly through the left temporal lobe (*circles*) to terminate in the posterior frontal lobe (*B*, bullet). Note several metallic fragments adjacent to the left petrous bone representing part of the projectiles casing that was dislodged from the bullet on impact. Note also a left frontal subdural hematoma (*red arrows*), midline shift, and effacement of the perimesencephalic cisterns. Acute obstructive hydrocephalus is identified by the presence of a disproportionately dilated right temporal horn (*yellow arrow*).

★**KEY POINT** Depending on the residual KE of the bullet after it enters the brain, it can be deflected from the contralateral surface of the skull and continue to move along the curvature of the inner surface of the skull or ricochet back into the brain parenchyma.

Gunshot Wound Ricochet Injury

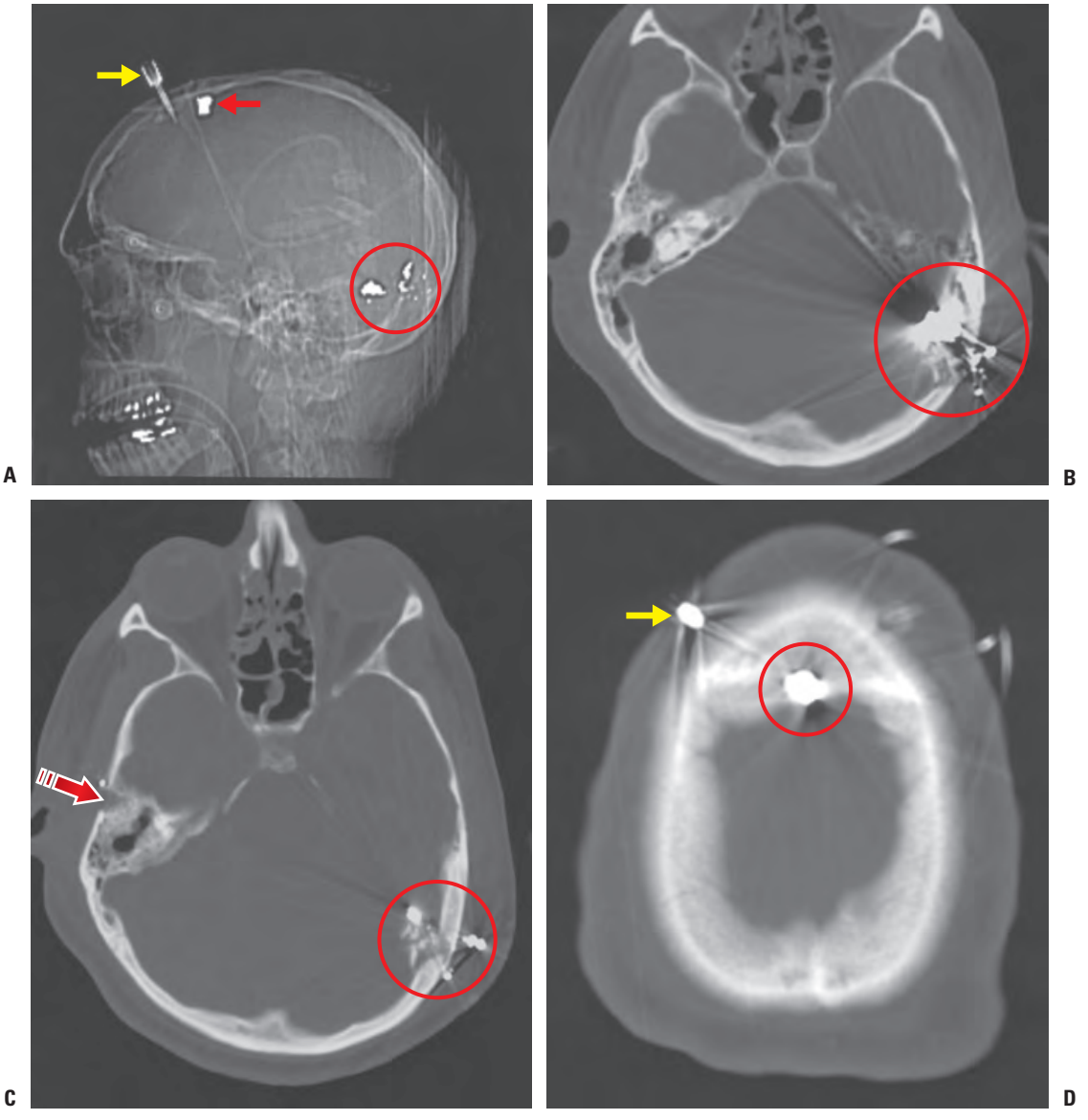
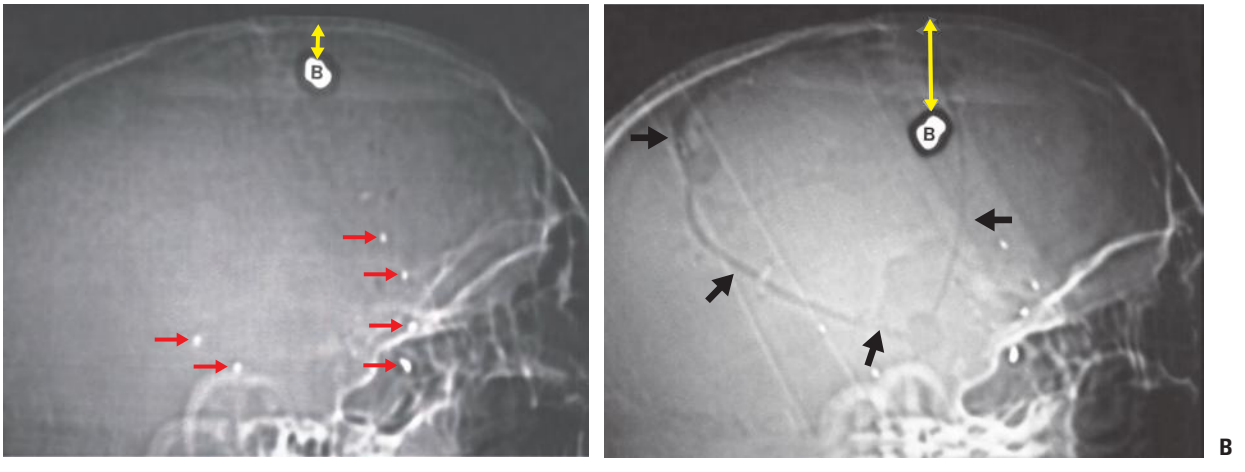


Figure 4.23. Gunshot Wound Ricochet Injury. A. Lateral CT scout view shows multiple irregular metallic fragments overlying the posterior fossa (*circle*). These represent fragments from the bullet's casing. The actual bullet is located immediately beneath the inner table of the frontal calvarium (*red arrow*), just posterior to the intracranial pressure (ICP) monitor (*yellow arrow*). **B, C.** Axial CT images show the entry site (*arrow*) and metallic casing fragments at the site of impact (*circle*). **D.** Axial image through the convexity demonstrates the actual bullet located adjacent to the inner table of the skull (*circle*). A right frontal ICP monitor is also noted (*arrow*).

★**KEY POINT** Bullets frequently separate from their metal casing when they impact a hard surface such as occipital and petrous bone (see also **Figs. 4.12C** and **4.22**). As a result, metallic fragments are often identified at the site of impact.

Besides ricocheting, ballistic fragments sometimes change their location within the brain after the initial injury. Therefore, in addition to documenting the presence, size, and shape of ballistic fragments on CT, the precise location of all bullets and bullet fragments should be determined. Accurate localization is critical for evaluating bullet migration over time, which is an important cause of delayed morbidity in GSWs to the head. Intracranial bullet migration has been estimated to occur in 4% of cases.[42] Migration of bullet fragments from soft tissues into vessels, with subsequent embolization to distal organs, can occur years after the initial injury. Bullet localization also aids in determining trajectory, which, when correlated with clinical information and patient outcome, may provide a valuable means to better understand mechanisms of injury and offer prognostic information.[29,43] Bullet migration usually occurs if the projectile is located within the ventricular system or if there is an adjacent necrotic/liquefied region of brain, such as a brain abscess (**Figs. 4.24**, **4.25**, and **5.72**).[44] Migration of a missile/projectile is often considered an indication for surgical removal. Ventricular projectiles can migrate and come to rest at sites blocking cerebrospinal fluid (CSF) outflow pathways, thereby causing acute obstructive hydrocephalus. Bullets can also fall from the posterior fossa into the spinal canal with sufficient momentum to cause an acute cord injury.[45] Therefore, consideration for craniotomy and bullet retrieval should be given to large bullets lying in the CSF space of the posterior fossa as they pose risk for acute spinal cord injury (see **Fig. 4.25**).

Bullet Migration



A B

Figure 4.24. Bullet Migration. A. Lateral scout view from the admission CT study demonstrates several intracranial metallic fragments (*arrows*), the largest of which is located adjacent to the vertex (*double-headed arrow*). **B.** Follow-up image obtained 3 weeks after the injury reveals a frontoparietal craniotomy (*arrows*). The bullet fragment was not removed at surgery, and an increase in distance between the bullet fragment and the calvarium is noted.

★**KEY POINT** It is not uncommon for ballistic fragments to change their location within the brain if they gain access to the ventricular system or a necrotic/liquefied region of the brain. Follow-up imaging exams should always be assessed for a change in location of all fragments.

Delayed Bullet Migration

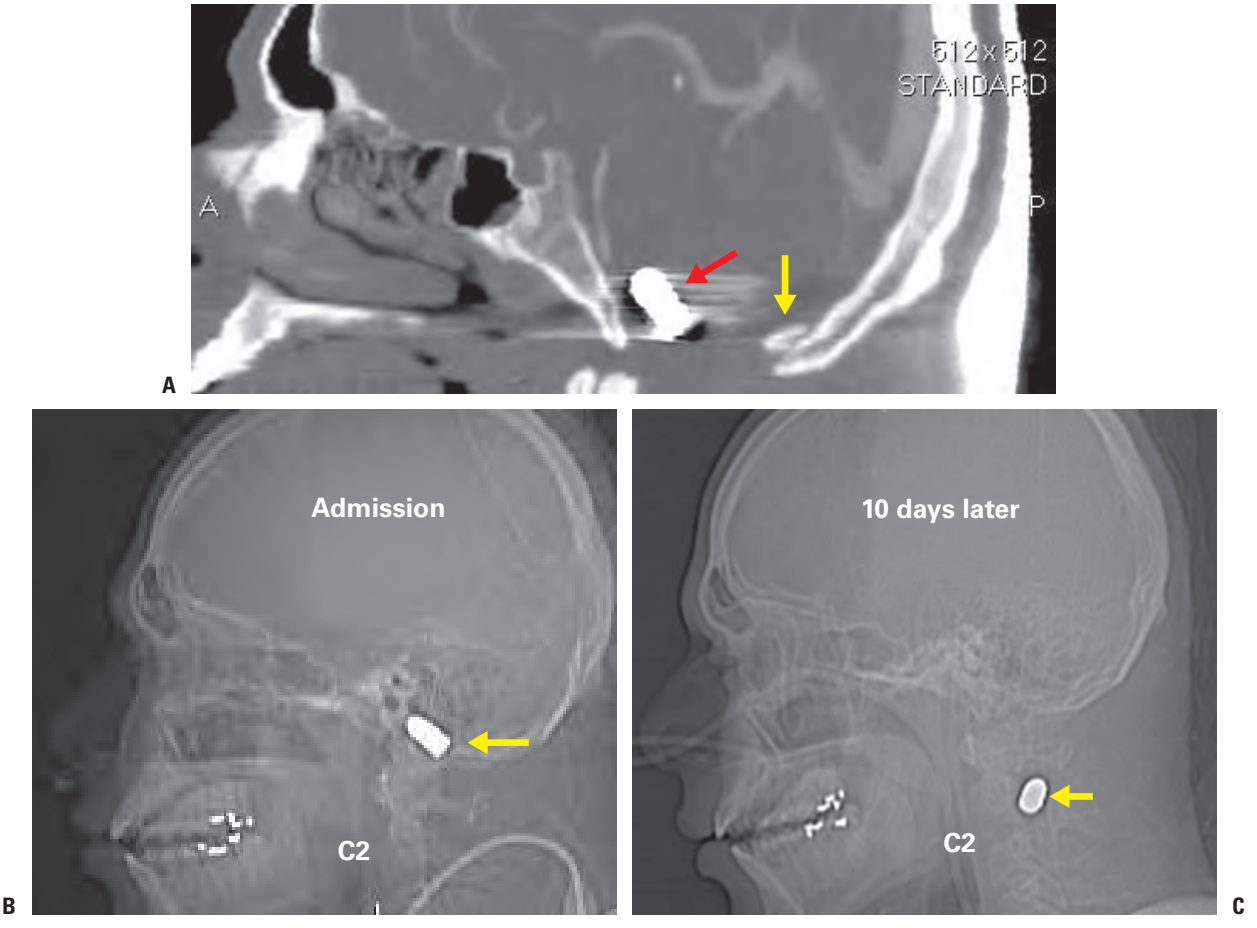


Figure 4.25. **Bullet Migration.** This 21-year-old male presented with a left hemiparesis after an acute GSW. The bullet entered the left scapula, traversed the squamous segment of the occiput, and came to rest in the right lateral medullary cistern. His hemiparesis recovered, but 10 days later, he developed abrupt quadriparesis coincident with fall of the bullet into the anterior spinal canal. The bullet was retrieved following a C2–C3 laminectomy, and a postoperative MRI confirmed signal change in the cord at the level where the bullet had resided. The patient made a good neurologic recovery. **A.** Midsagittal reformatted CTA on admission shows a bullet posterior to the inferior aspect of the clivus (*red arrow*). Metallic artifact makes it difficult to assess its precise margins. A small fracture of the occiput is also noted (*yellow arrow*). **B.** Corresponding lateral scout view. Note the lack of deformity of the bullet, confirming its jacketed nature. **C.** Ten-day follow-up scout image shows interval migration of the bullet into the upper cervical spine (*arrow*). (*Continued*)

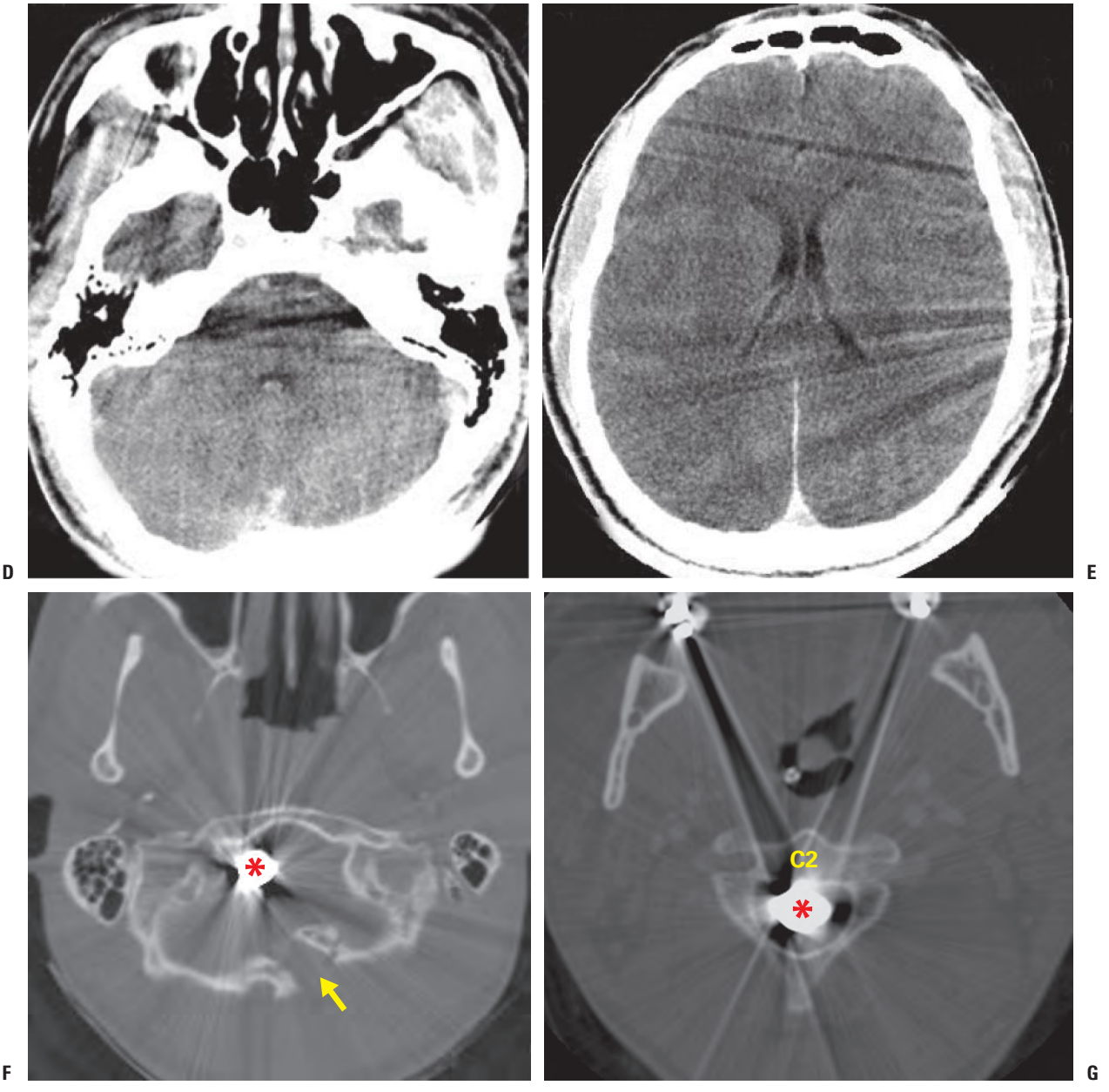
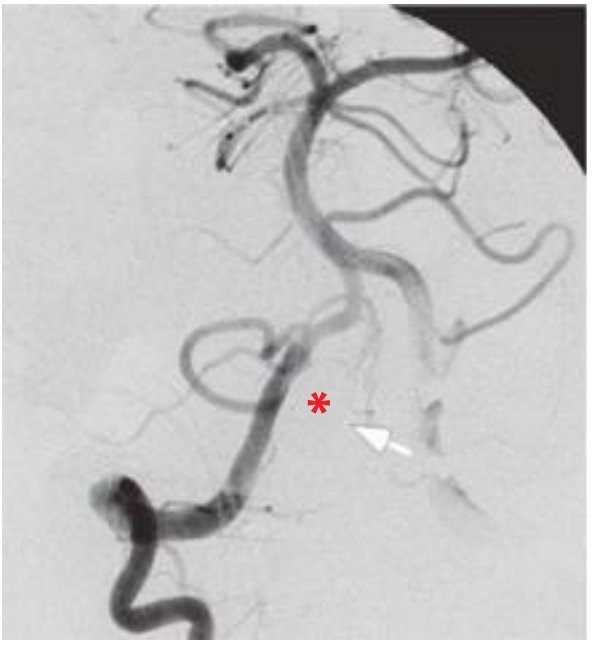
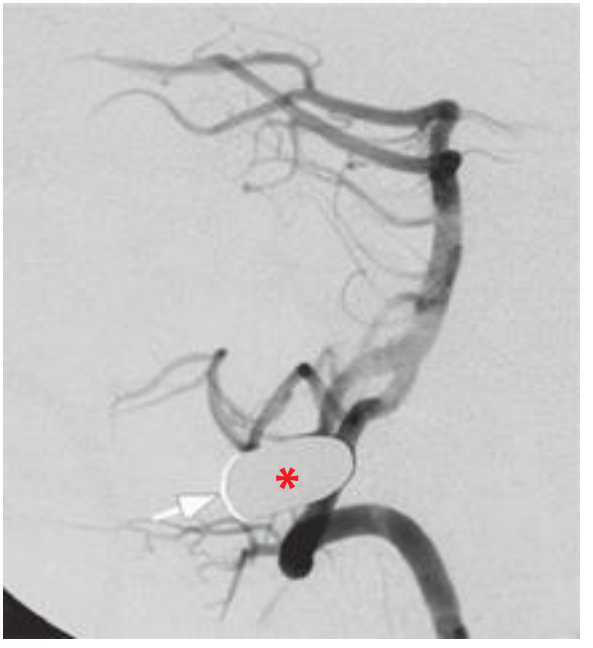


Figure 4.25. (*Continued*) **D, E.** Admission contrast-enhanced CT images show a relatively unremarkable brain. Streak artifact limits optimal visualization of the left cerebral hemisphere. **F.** Axial CT image on admission shows the entry site at the left occiput (*yellow arrow*) and the bullet (*asterisk*) located above the foramen magnum. **G.** Follow-up CT image 10 days later, immediately after the abrupt onset of his quadriparesis, demonstrates the bullet's descent to the level of C2 (*asterisk*). (*Continued*)



H



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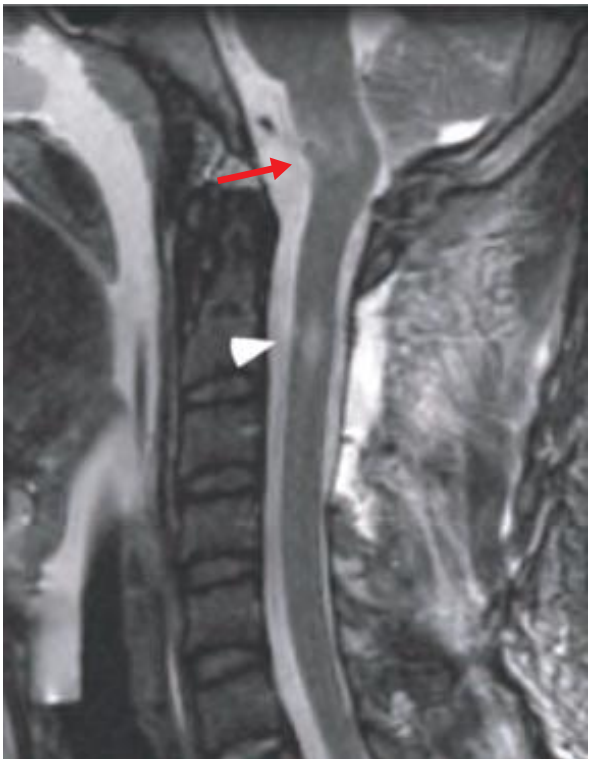


J

Figure 4.25. (*Continued*) **H, I.** Preoperative AP and lateral subtracted views from a catheter angiogram show the bullet (*asterisk*) adjacent to the right vertebral artery but no evidence of vascular injury. **J.** Postoperative T2-weighted midsagittal MRI shows abnormal T2 hyperintensity within the right cervicomedullary junction (*arrow*) where the bullet first rested and within the upper spinal cord, the site to which the bullet migrated (*arrowhead*). (*Continued*)

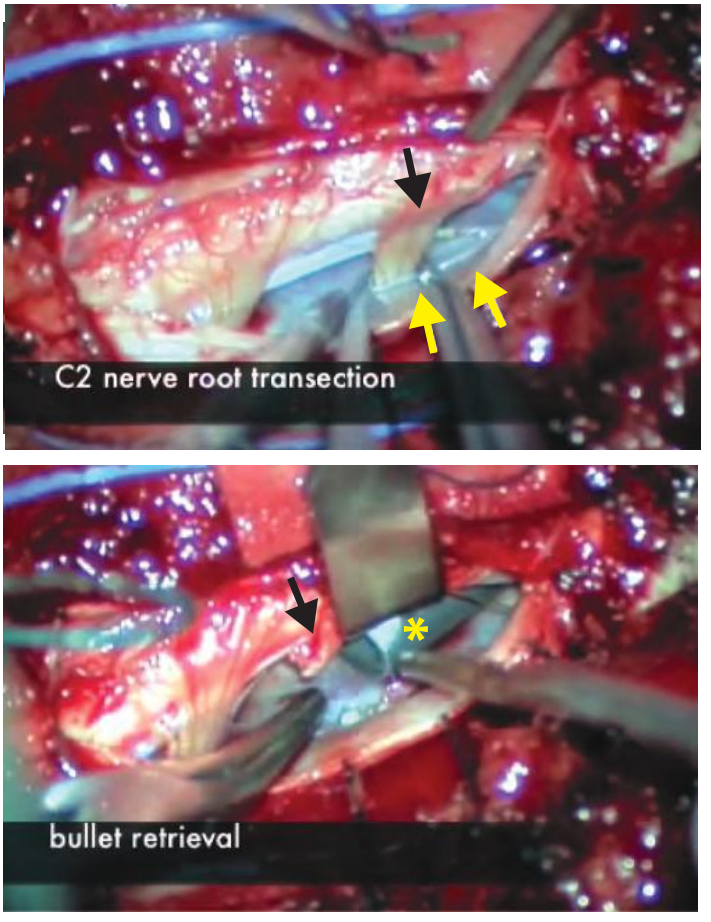


Figure 4.25. (*Continued*) **K.** Intraoperative photograph prior to extraction of the bullet. The dura has been opened, exposing the spinal cord and the C2 posterior nerve root (*black arrow*). A glimpse of the bullet can be seen lying beneath and anterior to the spinal cord (*yellow arrows*). **L.** Intraoperative photograph during extraction of the bullet (*asterisk*) from the intradural-extramedullary space. The C2 nerve root has been cut (*arrow*), allowing visualization and retrieval of the bullet. (Adapted with permission from Cheng J, Richardson R, Gean AD, Stiver S. Delayed acute spinal cord injury following intracranial gunshot injury. *Journal of Neurosurgery* 2012;116(4):921–5.)

★**KEY POINT** Migration of a projectile is often considered an indication for its surgical removal.

Unihemispheric, Nonlethal, Penetrating Gunshot Wound

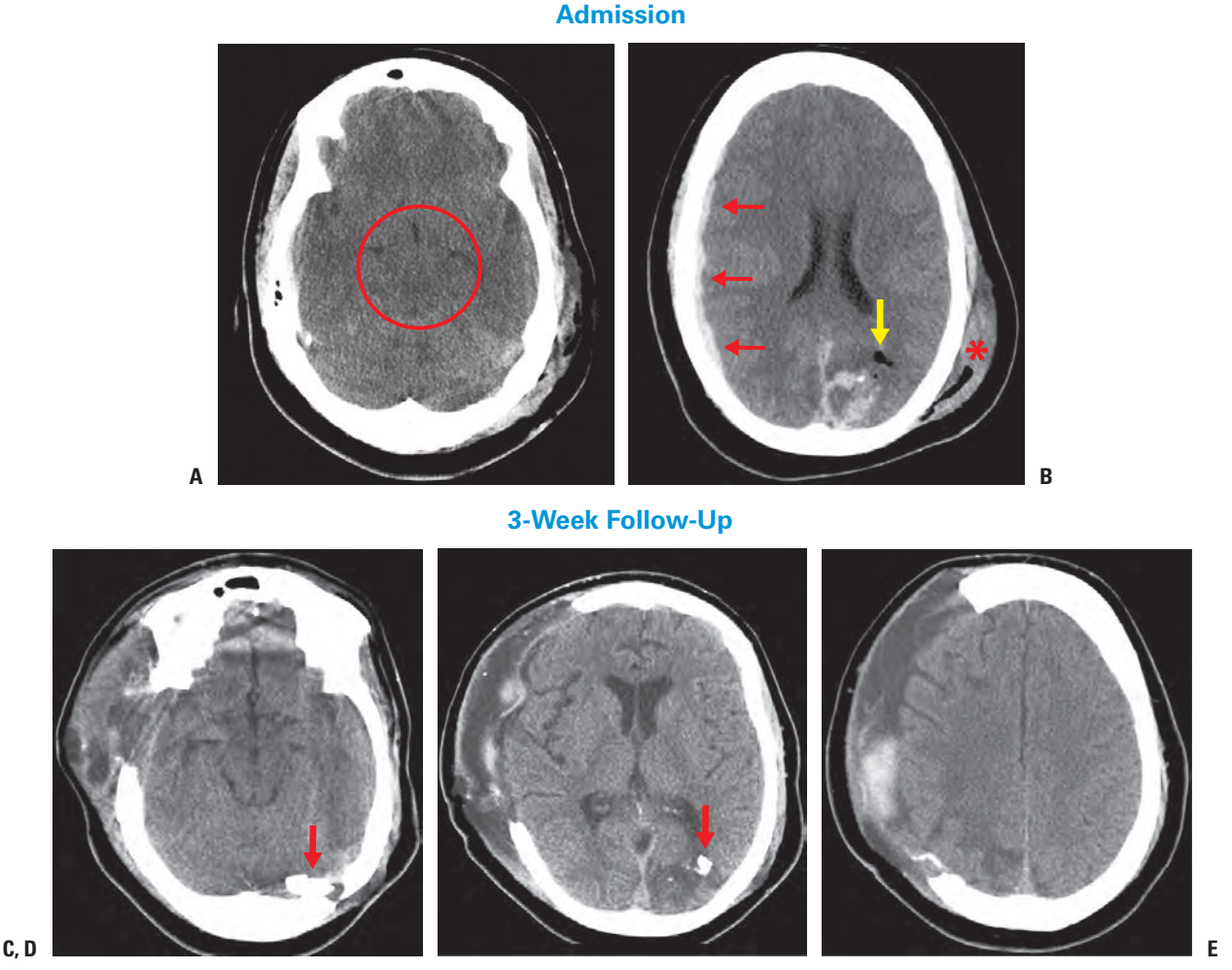


Figure 4.26. **Nonlethal Gunshot Wound.** *History: This 37-year-old woman presented with a GCS = 3 following a GSW to the back of the head. Initial treatment in the operating room (OR) included removal of devitalized brain and accessible foreign bodies, irrigation with antibiotic solution, watertight closure of the dura, and placement of an external ventricular drainage catheter. She improved to localizing the pain the next morning, but her ICP continued to rise despite maximal medical therapy. Therefore, she underwent a decompressive hemicraniectomy. Her 6-month Glasgow Outcome Score (GOS) = 4.* **A, B.** Admission CT images reveal ominous effacement of the cerebral sulci and basal cisterns (*red circle*), left occipital hemorrhage, right subdural hematoma (*red arrows*), a small focus of pneumocephalus (*yellow arrow*), and extensive scalp soft tissue injury at the entry site (*asterisk*). Because of the proximity of the injury to the left transverse sinus, a CT venogram was performed and was negative for venous injury. **C–E.** Postoperative CT images obtained 3 weeks after the injury demonstrate a right decompressive hemicraniectomy, left occipital encephalomalacia, and global atrophy. Note residual bone and metal fragments displaced into the left occipital lobe (*arrows*). Unlike prior war surgery, today's approach to treating penetrating TBI emphasizes the preservation of viable brain tissue and removal only of accessible fragments. Delayed removal of large metallic fragments after recovery may be considered as an option in select cases.

★**KEY POINT** Reasons to remove an intracranial foreign body can include fragment migration, infection, epilepsy, potential lead toxicity, and the future need for MRI in a young patient.

Unihemispheric, Nonlethal, Perforating Gunshot Wound (Cerebral Vasopasm + External Herniation)

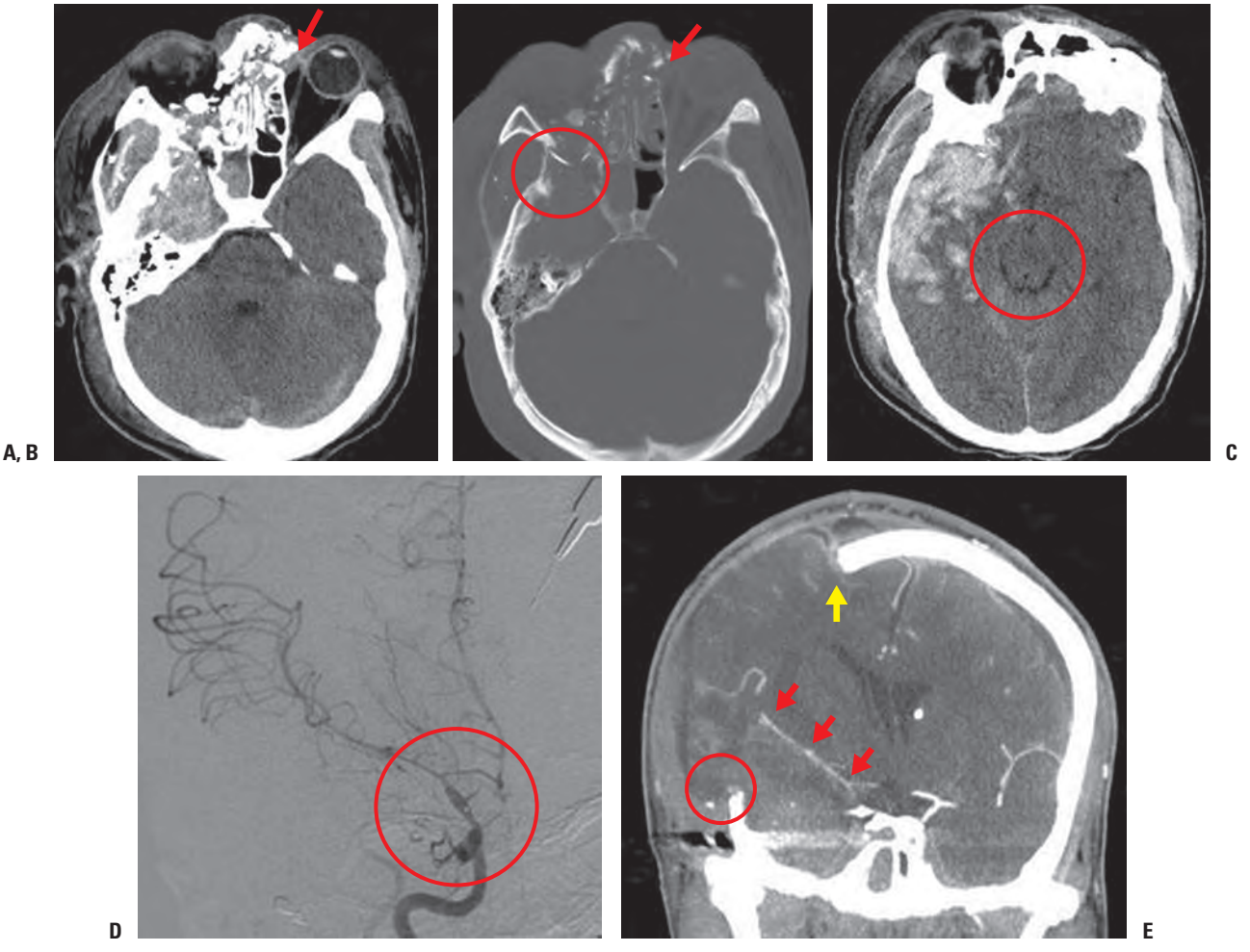


Figure 4.27. **Nonlethal, Perforating Gunshot Wound with Vasospasm, Cerebral Swelling, and External Herniation.** **A.** Brain window and **(B)** bone window CT images on admission reveal a comminuted ballistic fracture with the entry site at the left naso-orbital junction (*arrow*). The bullet trajectory traversed the ethmoid air cells, right orbit, and right sphenotemporal buttress (*circle*) and exited the inferior temporal fossa (not shown). **C.** Admission CT at the level of the upper brain stem shows extensive intra-axial hemorrhage but preservation of the perimesencephalic cisterns (*circle*). The patient subsequently developed refractory intracranial hypertension and underwent an emergent decompressive hemicraniectomy. **D.** AP view from the arterial phase of a catheter angiogram performed 5 days later demonstrates severe cerebral vasospasm of the supraclinoid internal carotid artery and proximal anterior and middle cerebral arteries (MCAs) (*circle*). **E.** Coronal reformatted CTA shows correlative narrowing, straightening, and superior displacement of the M1 segment of the right MCA (*red arrows*). Note also external herniation and compression of the brain parenchyma against the margins of the craniectomy defect (*yellow arrow* and *red circle*). (*Continued*)

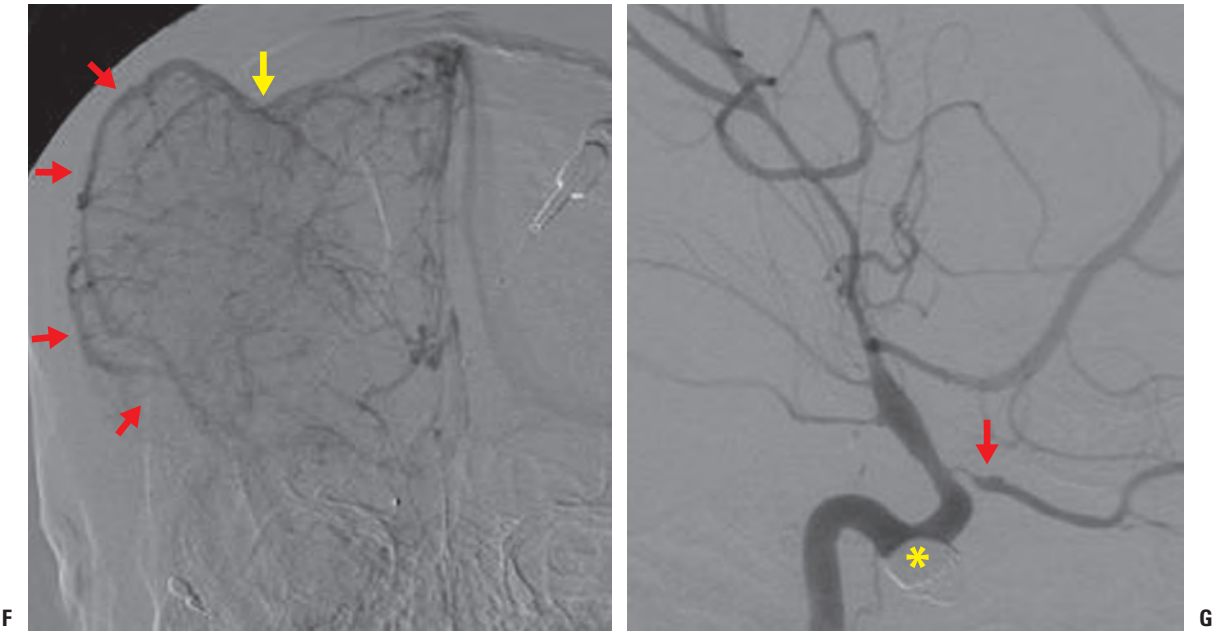


Figure 4.27. (*Continued*) **F.** AP view from the venous phase of the same catheter angiogram shown in **(D)** demonstrates displacement of the cortical veins through the craniectomy defect (*red arrows*), consistent with external herniation. Note kinking of the veins against the craniectomy edge that corresponds to the CTA findings (*yellow arrow*). **G.** Magnified lateral view from the early arterial phase shows severe caliber narrowing of the proximal ophthalmic artery and a small pseudoaneurysm (*arrow*). The apparent defect in the carotid siphon is an artifact from an overlying subtracted bullet fragment (*asterisk*).

★**KEY POINT** Vasospasm is a common complication of blast trauma found in both combat and civilian TBI.

Bihemispheric, Nonlethal, Perforating Gunshot Wound

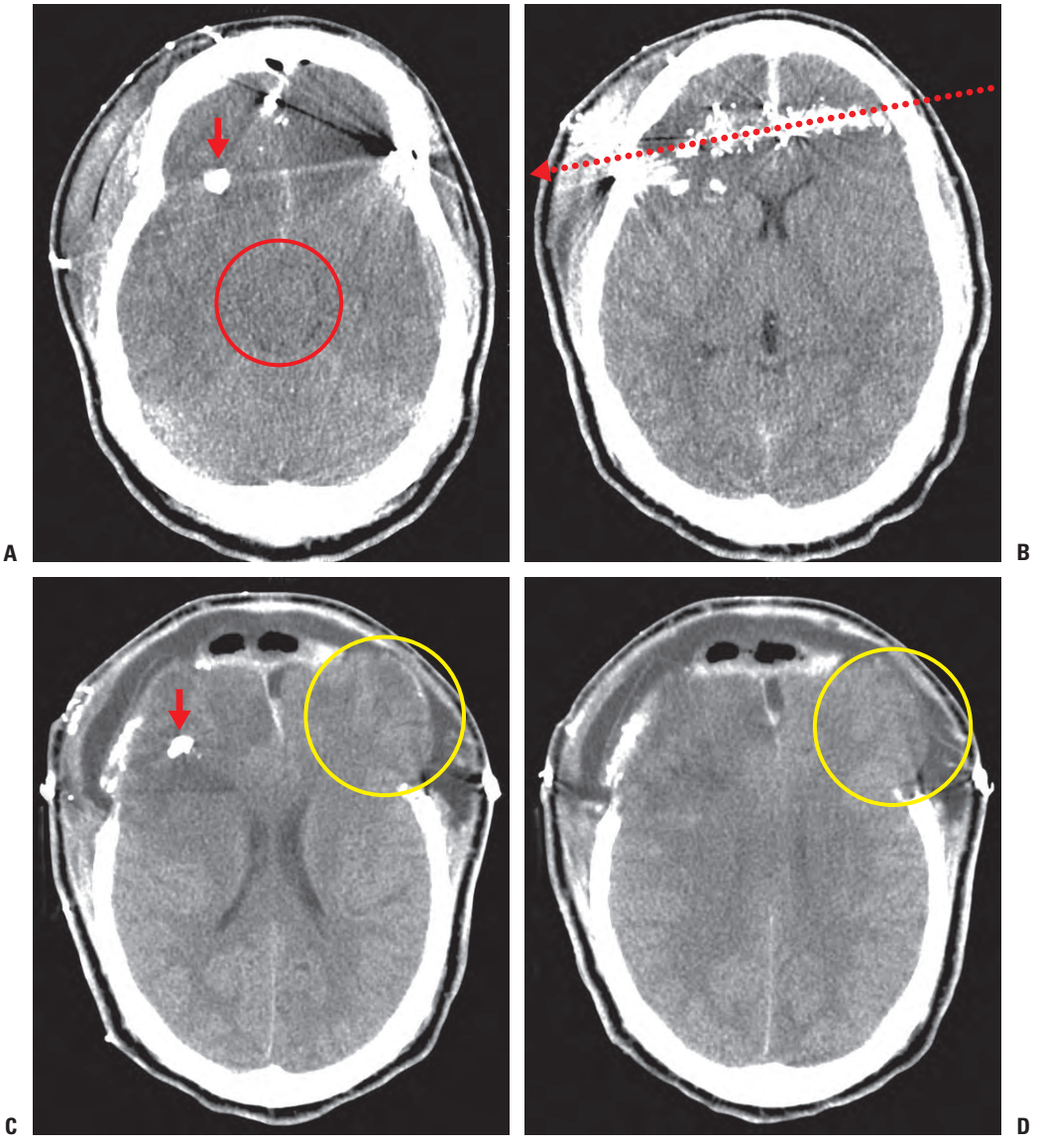


Figure 4.28. Bihemispheric, Nonlethal, Perforating Gunshot Wound. ***History:*** *An 18-year-old man was found with a GCS = 3 following a gang fight. When he arrived at the hospital, his GCS improved to 6 (E1VTM4). He was initially taken to the OR for wound debridement and external ventricular drain (EVD) placement. His ICP at that time was 18 to 25. However, because his ICP elevated further and became refractory to medical therapy, he underwent a bifrontal decompressive craniectomy.* **A, B.** Admission noncontrast CT images reveal a bilateral inferior frontal GSW. There is near-complete effacement of the basal cisterns (*circle*) and sulci. Multiple bone and bullet fragments are noted along the path of the wound canal (*arrows*), and there is a small amount of subdural and subarachnoid hemorrhage. **C,D.** Status-post bifrontal decompressive craniectomy with external herniation through the calvarial defect (*circle*). Also note the residual metallic fragment that was purposely not removed (*arrow*). His GOS = 5 and he is now back at school.

★**KEY POINT** By definition, a perforating GSW is a through-and-through injury in which the bullet exits the skull. In contrast, a penetrating GSW enters the skull but does not exit. The skull remains intact in a superficial GSW.

Bihemispheric, Nonlethal, Perforating Gunshot Wound

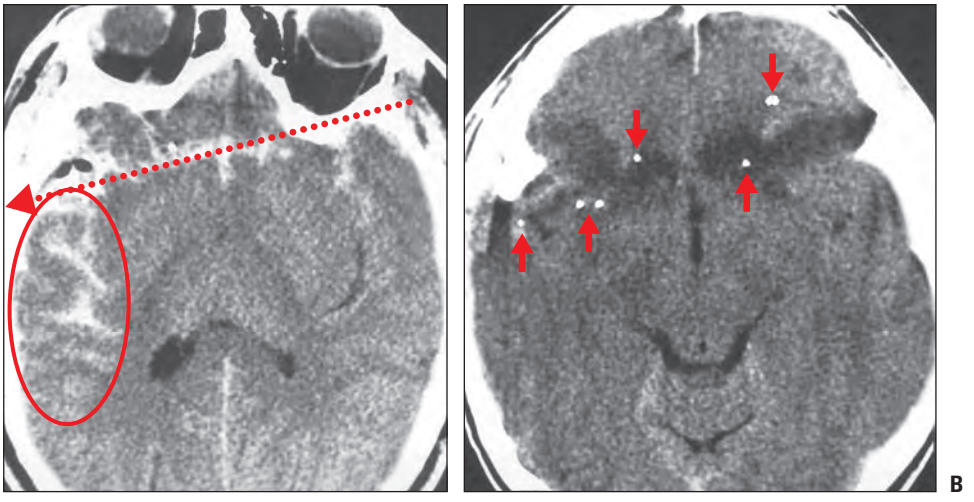


Figure 4.29. **Bihemispheric, Nonlethal, Perforating Gunshot Wound. A.** Admission noncontrast CT of a patient status-post acute bilateral inferior frontal GSW reveals right perisylvian subarachnoid hemorrhage (*circle*). The bullet trajectory is outlined in *red*. **B.** One-year follow-up CT shows a linear tract of low attenuation, representing encephalomalacia along the wound canal. Several punctate metallic foreign bodies (*arrows*) remain along the course of the wound tract.

Bihemispheric, Lethal, AK-47 Sniper Rifle, Penetrating Gunshot Wound (Full Metal Jacket Bullet)

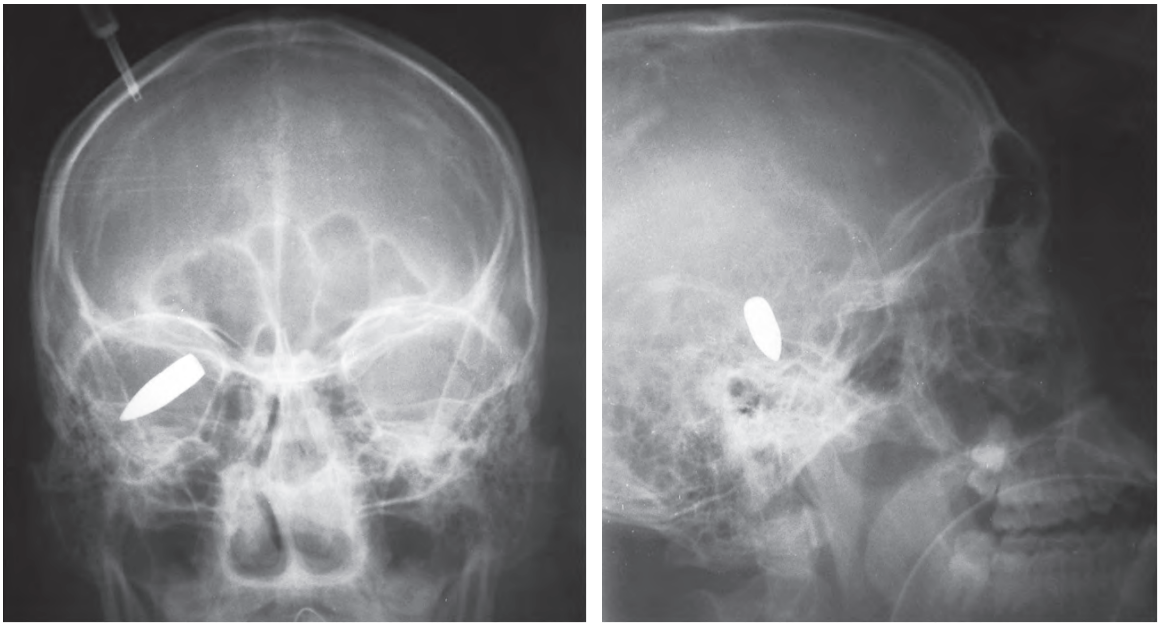


Figure 4.30. Bihemispheric, Lethal Gunshot Wound. A. AP and **(B)** lateral plain radiographs show a bihemispheric transventricular GSW. A right frontal ICP monitor is noted, and the soldier is intubated. This case is notable for the fact that the bullet is retained within the cranium. In most instances, the combination of the nondeformable nature of a jacketed bullet, coupled with the high velocity of a rifle firearm, results in a through-and-through (i.e., perforating) injury. (Courtesy of Rocco Armonda, MD, Col [ret], MC, USA.)

★**KEY POINT** If a bullet is entirely encased, it is called an FMJ bullet. High-velocity weapons often use bullets that are jacketed to protect them from fragmenting and melting while they are in the gun barrel. Unlike semi-jacketed bullets, an FMJ bullet usually stays intact. However, if it strikes bone, the lead component of the bullet may sometimes become deformed and separated from its fragmented copper jacket.

Skull Fractures in Gunshot Wounds

There are several distinct fracture patterns seen with GSWs. In penetrating and perforating GSWs, the entry site appears as a round, punched-out fracture with a small, funnel-shaped, beveled defect involving the inner table of the skull (**Figs. 4.31A,C, 4.36B,** and **4.37**). There is an exit wound in perforating but not penetrating GSW. The bony defect in the exit wound is distinct in appearance from the entrance defect. An exiting bullet causes a small defect in the inner table and a larger beveled defect in the outer table of the skull (**Figs. 4.31C, 4.32,** and **4.36B**). The beveling effect is explained by the fact that bone is weaker under tensile stress than under compressive stress. At the entry site, the outer table of the skull is under *compressive* stress while the inner table is under *tensile* stress; therefore, the inner table defect is larger than the outer table defect. At the exit site, the inner table of the skull is under table compressive stress while the outer table is under tensile stress; therefore, the outer table defect is larger than the inner table defect. For larger, slower projectiles, the fracture is more likely to be a cantilever fracture of

roughly wedge-shaped pieces of cranium, somewhat like the fracture pattern in a crust of ice atop liquid water when a person steps on it. In young patients with a thin skull, or certain parts of the adult skull (e.g., squamous segment of the temporal bone), however, it can be impossible to make this distinction—the skull is too thin for the creation of the funnel-shaped wound that makes differentiation of the entrance versus exit wounds possible.

Lethal Gunshot Wounds (Penetrating versus Perforating)

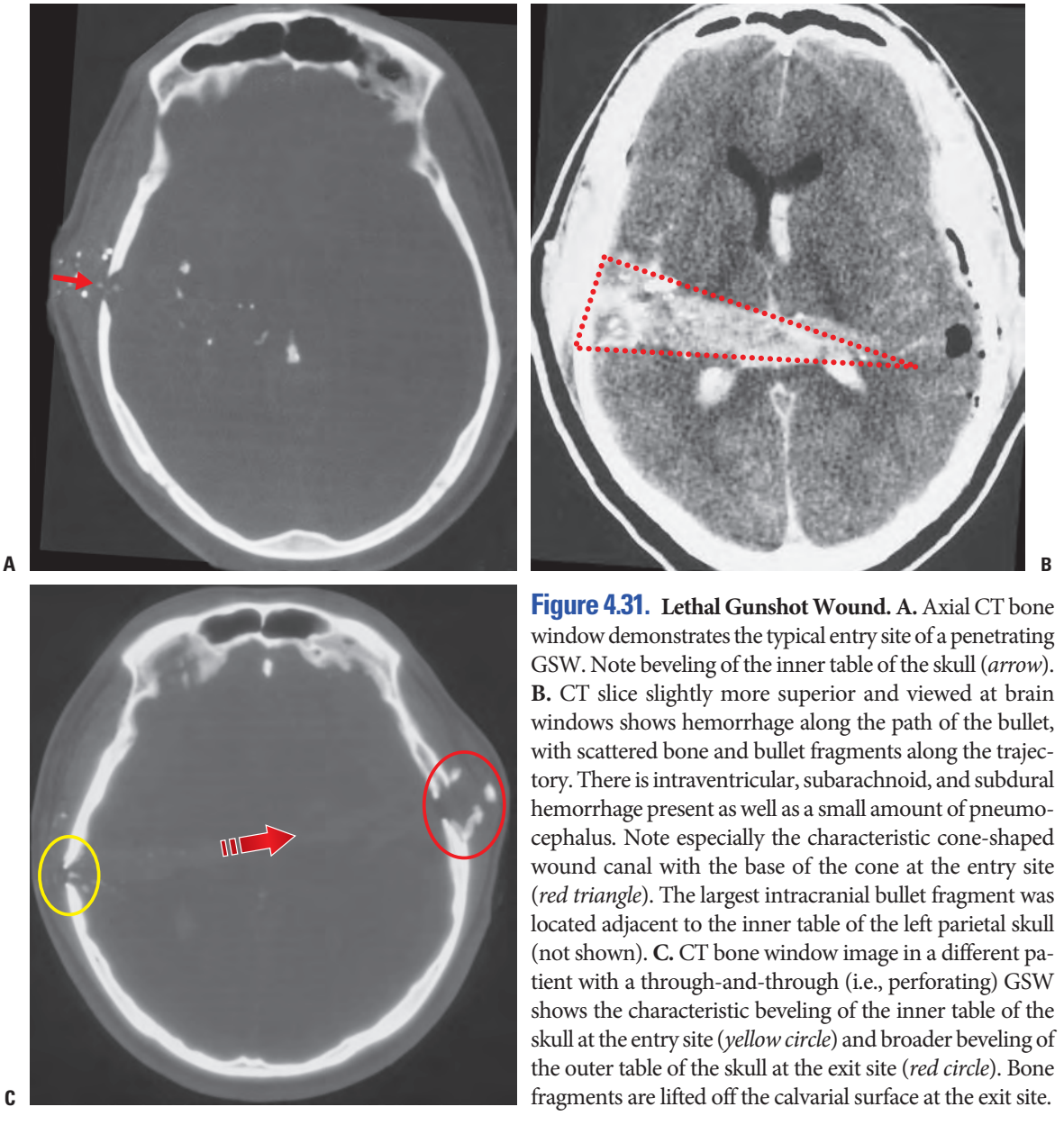


Figure 4.31. Lethal Gunshot Wound. A. Axial CT bone window demonstrates the typical entry site of a penetrating GSW. Note beveling of the inner table of the skull (*arrow*). **B.** CT slice slightly more superior and viewed at brain windows shows hemorrhage along the path of the bullet, with scattered bone and bullet fragments along the trajectory. There is intraventricular, subarachnoid, and subdural hemorrhage present as well as a small amount of pneumocephalus. Note especially the characteristic cone-shaped wound canal with the base of the cone at the entry site (*red triangle*). The largest intracranial bullet fragment was located adjacent to the inner table of the left parietal skull (not shown). **C.** CT bone window image in a different patient with a through-and-through (i.e., perforating) GSW shows the characteristic beveling of the inner table of the skull at the entry site (*yellow circle*) and broader beveling of the outer table of the skull at the exit site (*red circle*). Bone fragments are lifted off the calvarial surface at the exit site.

★**KEY POINT** A bihemispheric GSW trajectory at the level of the diencephalon is a lethal combination.

Fracture patterns of the skull can help determine both the sequence and the direction of fire in GSWs. For example, because fractures propagate through the skull faster than the bullet passes through the brain, the pattern of intersecting fractures can predict which came first. Specifically, radial fractures emanate from the entry hole and begin to wrap the skull. If the bullet exits the skull, fractures extend backward from the exit hole and may intersect those from the entrance hole. The energy dissipates along the preexisting entrance fractures, and the exit fractures go no further, that is, exit fractures stop at entrance fractures. Therefore, linear fractures associated with typical exit sites terminate at the preexisting linear fractures produced by the entering bullet. In addition, radiating fractures associated with the entrance wound are longer and, because they form in advance of exit fractures, they tend not to be arrested by intersection with any other fracture. They can be, however, arrested by suture lines. Likewise, *heaving fractures*, discussed later, have more generations and longer radii than exit-associated fractures. The absence of secondary radiating fractures at the exit site suggests either a low-energy bullet or that the energy was dissipated along a suture or preexisting fracture(s). Because fracture lines do not cross preexisting fracture lines, the sequential timing of the fractures (and sequence of fire) can be determined when more than one bullet is fired. Fractures that originate from the second bullet entrance site are arrested by the radiating linear fractures from the first bullet entry hole. All of these characteristics can be useful in forensic neuropathology.

Typical Perforating Gunshot Wound Exit Site

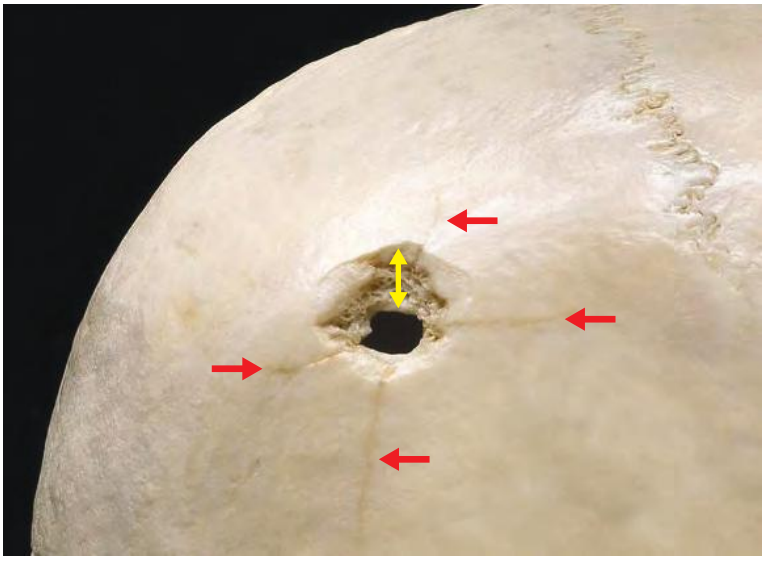


Figure 4.32. Typical Gunshot Wound Exit Site. This autopsy specimen demonstrates the characteristic crater defect found at the exit site of a perforating GSW. This appearance is due to beveling of the outer table of the skull, which always occurs at the exit site of a GSW (*yellow arrow*). Multiple linear fractures are seen radiating away from the wound defect (*red arrows*). Compare with **Figure 4.31**.

★**KEY POINT** In perforating GSWs, the beveling appearance of the inner and outer tables of the skull can be used to differentiate entry and exit sites and thereby determine the direction of fire.

A fracture unique to the GSW is the *concentric heaving fracture*. As its name suggests, this type of GSW fracture appears as arcs connecting the linear fractures that radiate from the entrance and exit holes. The fracture occurs only if sufficient temporary cavitation forces and elevation in ICP are generated in the brain (see **Figs. 4.33** and **4.34**). The concentric heaving fracture appears quite literally as a shattered or exploded skull. A portion of the skull appears lifted off from the skull, hence the *heaving* terminology.

The peak ICP created as the bullet traverses the brain varies in direct proportion to the projected cross-sectional area of the bullet and the square of its velocity and inversely proportional to the distance of the fired weapon from the target. Therefore, heaving fractures are most often seen with close-range injuries, rifles, or large-caliber handguns. Unlike most ballistic fractures, the radiating fractures associated with the entry site tend not to be arrested by the cranial sutures.

Concentric Heaving Skull Fracture in Gunshot Wounds

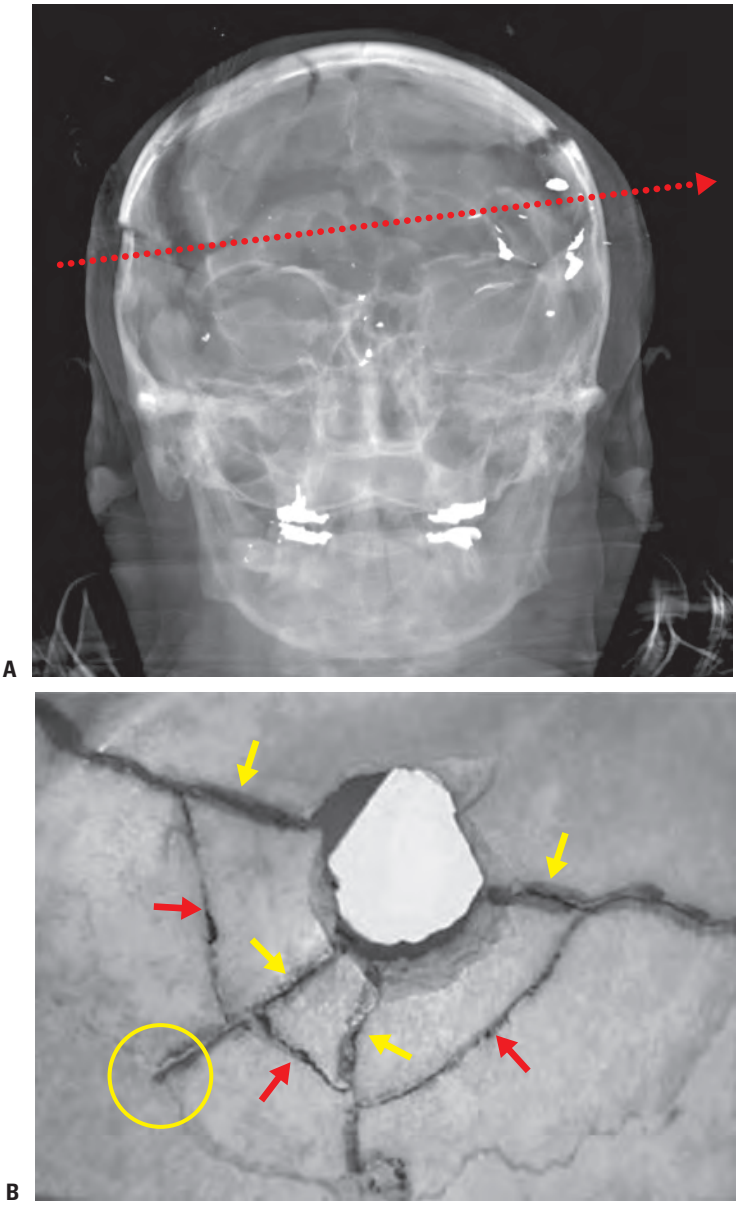


Figure 4.33. Concentric Heaving Fracture. A. AP radiograph demonstrates extensive, bilateral, comminuted fractures of the cranial vault. The skull literally looks exploded. Numerous intracranial bullet fragments are noted, most of which are located in the vicinity of the exit site. **B.** Autopsy specimen in a different patient illustrates the characteristic concentric heaving fracture lines (*red arrows*) around an exit wound. Note how the concentric heaving fractures develop perpendicular to the radiating fractures (*yellow lines*). Note also how they abruptly terminate at their intersection with the radiating fractures. One of the radiating fractures terminates at a suture line (*circle*).

★**KEY POINT** The concentric heaving fracture is produced indirectly by the sudden massive increase in ICP, whereas the radial fracture lines are caused directly by impact of the projectile. Radial fractures may occur alone, but concentric heaving fractures are never seen without radial fractures.

Concentric Heaving Skull Fracture in Gunshot Wounds

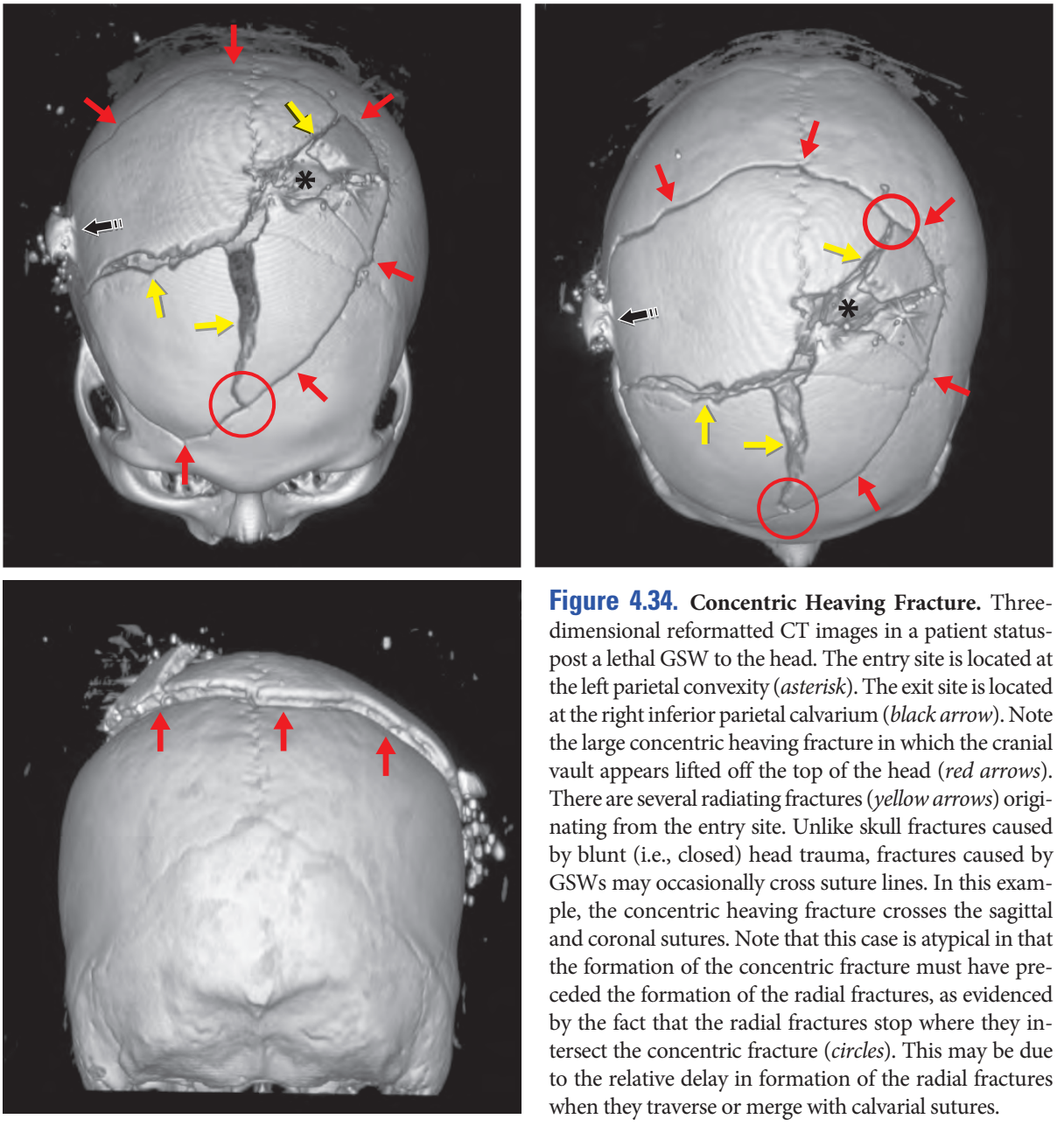


Figure 4.34. Concentric Heaving Fracture. Three-dimensional reformatted CT images in a patient status-post a lethal GSW to the head. The entry site is located at the left parietal convexity (*asterisk*). The exit site is located at the right inferior parietal calvarium (*black arrow*). Note the large concentric heaving fracture in which the cranial vault appears lifted off the top of the head (*red arrows*). There are several radiating fractures (*yellow arrows*) originating from the entry site. Unlike skull fractures caused by blunt (i.e., closed) head trauma, fractures caused by GSWs may occasionally cross suture lines. In this example, the concentric heaving fracture crosses the sagittal and coronal sutures. Note that this case is atypical in that the formation of the concentric fracture must have preceded the formation of the radial fractures, as evidenced by the fact that the radial fractures stop where they intersect the concentric fracture (*circles*). This may be due to the relative delay in formation of the radial fractures when they traverse or merge with calvarial sutures.

★**KEY POINT** In most cases, fractures do not cross fracture lines and do not cross sutures. The pattern of intersecting fracture lines is thereby used in forensics to assess the sequence and direction of fire in multiple GSWs to the head.

Patients with a thin calvarium are extremely sensitive to the sudden increase in ICP produced by a bullet entering the cranial cavity. In such cases, fractures may be seen remote from both the entry and exit sites. Secondary remote fractures can also occur in patients with a thicker skull if the weapon was fired at close range (secondary to gas discharged into the skull). In this setting, large pieces of the skull and brain are typically blown away, with maceration of the residual brain. In GSWs fired from a distance, gas entering the cranium plays no part in the production of fractures, and the fractures are caused by the pressure buildup and elevated ICP in the skull as a result of temporary cavitation. The transfer of cavitating energy can also be strong enough to result in remote fractures of the relatively thin orbital roof (see **Figs. 4.35** and **4.36**).

Orbital Roof Blow-Down Fracture in Gunshot Wounds

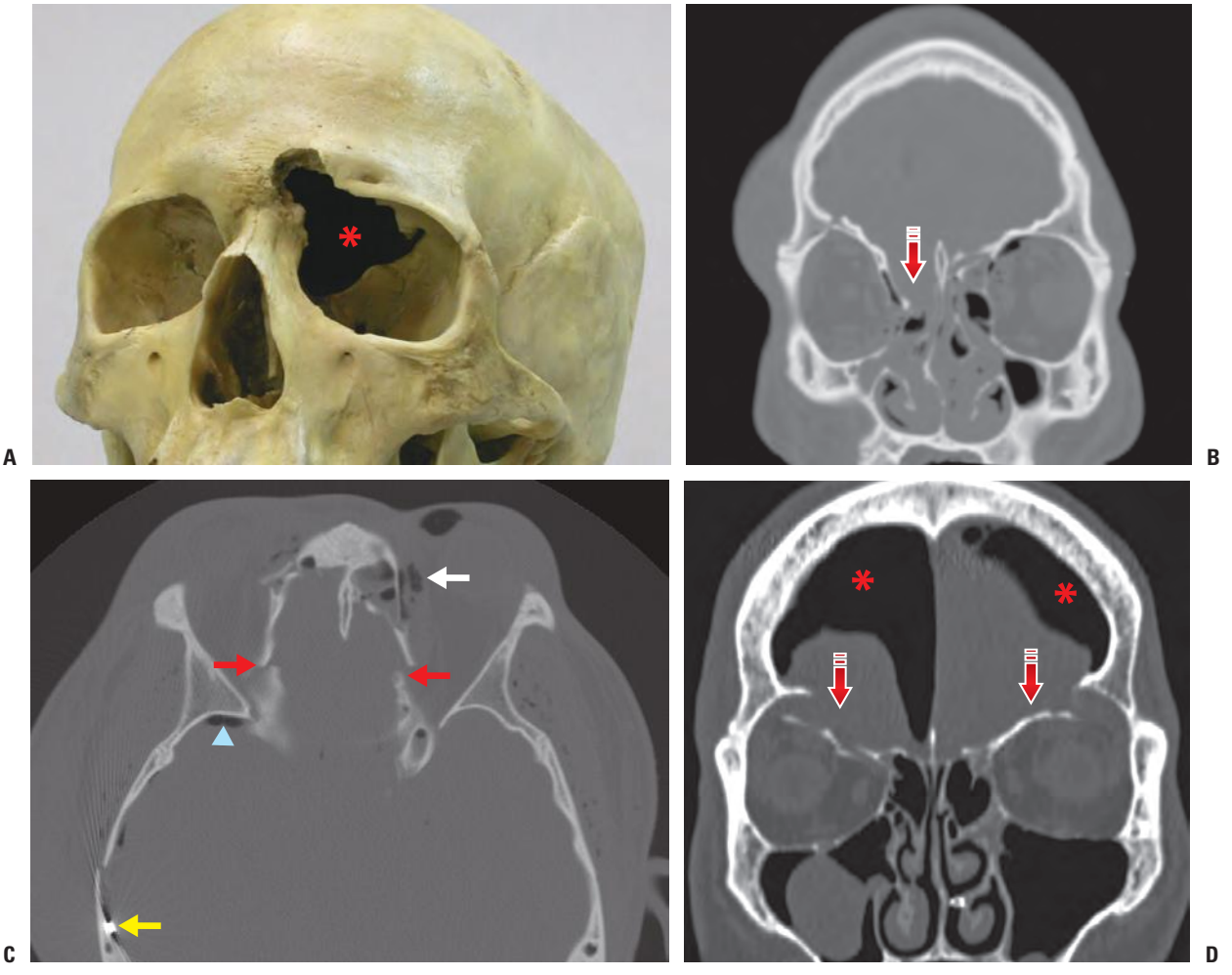


Figure 4.35. **"Blow-Down" Orbital Roof Fracture. A.** Skull of a young man status-post an intracranial GSW shows a large defect of the left orbital roof (*asterisk*). **B.** Coronal CT in a different patient demonstrates the typical *blow-down* fracture of the orbital roof into the ethmoid (*arrow*). **C.** Axial image from the same patient demonstrates bilateral medial orbital wall fractures extending from orbital roof fractures (*red arrows*); preseptal soft tissue air (*black arrow*); postseptal air, that is, orbital emphysema (*white arrow*); pneumocephalus (*blue arrow*); and a small residual bullet fragment adjacent to the fractured temporal squamosa (*yellow arrow*). **D.** Coronal CT from a different patient status-post an intracranial GSW showing bilateral blow-down orbital fractures and bilateral pneumocephalus (*asterisk*).

★**KEY POINT** The "blow-down" fracture is a unique injury that is attributed to a sudden and massive rise in intracranial pressure ICP. Consequently, it is seen almost exclusively with intracranial GSWs. The blow-down fracture is a calvarial fracture caused by elevated ICP at the site of least resistance (i.e., the thin orbital roof and ethmoid air cells). Disruption of the orbital roof can create an orbital communication with the intracranial cavity, leading to a CSF leak, encephalocele, intracranial infection, ophthalmoplegia, and the need for complex orbital and anterior cranial vault reconstruction.

Lethal Gunshot Wound

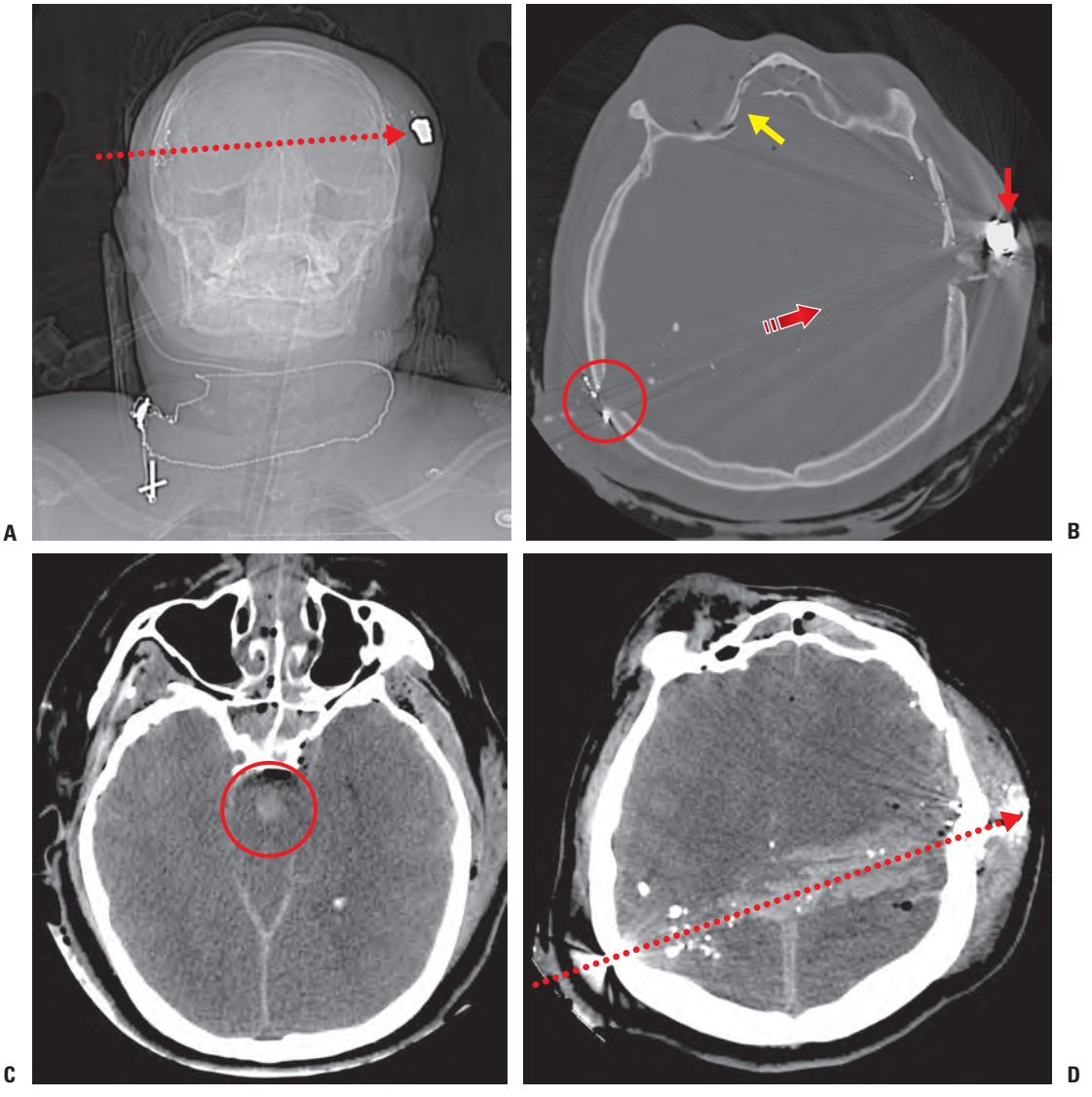
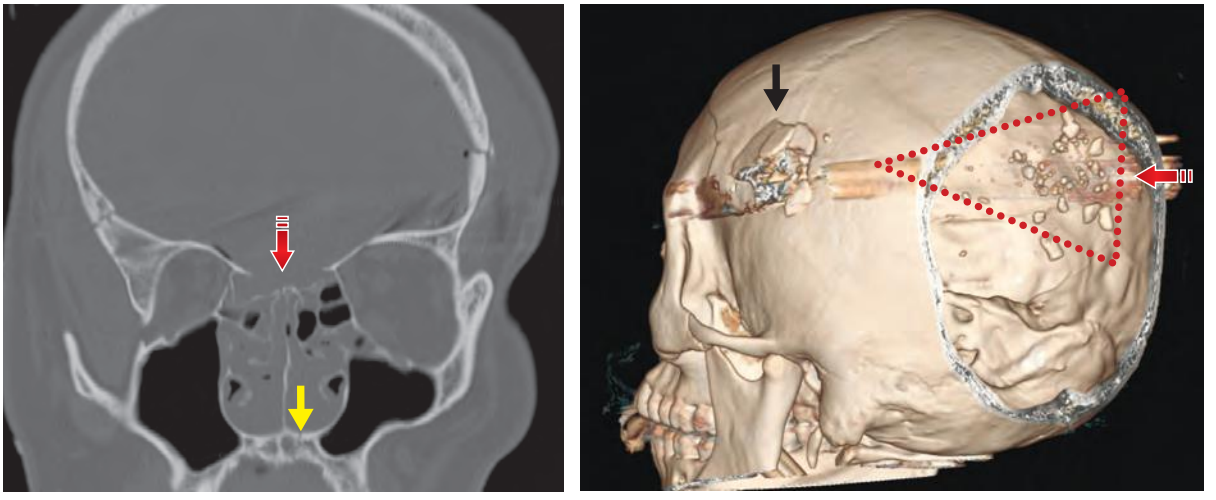


Figure 4.36. Lethal Gunshot Wound. A. AP scout view shows a right-to-left GSW with the major bullet fragment located within the left frontal scalp (*arrow*). **B.** Axial bone window CT shows the right-to-left trajectory (*large red arrow*) with characteristic beveling of the inner table of the skull at the entry site (*circle*) and a larger exit wound. The major bullet fragment is lodged in the left frontal scalp soft tissue (*small red arrow*). A blow-down fracture of the right orbito-ethmoid region is identified (*yellow arrow*). **C.** Noncontrast CT image at the level of the perimesencephalic cisterns shows an area of high density within the central pons, consistent with a Duret hemorrhage. Decreased gray-white matter differentiation is also present, consistent with diffuse cerebral edema. **D.** CT at a higher level shows the right-to-left trajectory with bone and bullet fragments scattered along the trajectory (*arrow*). There is sulcal effacement and early loss of gray–white differentiation. (*Continued*)



E F

Figure 4.36. (*Continued*) **E.** Coronal bone window demonstrates the blow-down fracture of the anterior ethmoid air cells (*red arrow*). Note also a fracture of the left palate caused by abrupt craniofacial distortion at the time of impact. **F.** Cutaway view from a 3D CT demonstrates numerous intracranial bone and bullet fragments that have a cone-shaped distribution (*triangle*) at the entry site (*red arrow*). The exit site within the left posterior frontal region (*black arrow*).

★**KEY POINT** This case illustrates several imaging findings typically found in intracranial GSWs: (1) characteristic beveling of the entry and exit sites, (2) early loss of gray–white matter differentiation in lethal injuries, (3) ballistic fractures remote from the entry and exit sites (especially orbital roof), and (4) the Duret hemorrhage secondary to the massive sudden rise in ICP.

The **keyhole fracture**, sometimes referred to as a *gutter fracture*, occurs when the projectile strikes the surface of the skull in a tangential manner.[46] It has a characteristic appearance that simultaneously exhibits features of both an entry and exit site (see **Fig. 4.37**). The keyhole fracture has a circular entrance defect and a triangular exit defect (hence, the analogy to a keyhole). External examination of the wound can be confusing, manifesting signs of both entrance- and exit-type trauma. Keyhole fractures induce the same shearing force of the volume-restricted cavitating energy, coupled with bone fragments serving as secondary missiles, as typical in penetrating and perforating GSW. This makes brain injuries that occur with keyhole fractures often devastating and in excess of what is erroneously predicted from visualization of the tangentially appearing entrance wound.[47]

Of interest, the autopsy report of President Abraham Lincoln is remarkably consistent with many of the skull findings described previously[48]:

> *There was a gunshot wound of the head around which the scalp was greatly thickened by hemorrhage into its tissue. The ball entered through the occipital bone about one inch to the left of the median line and just above the left lateral sinus, which it opened. The wound in the occipital bone was quite smooth, circular in shape, with beveled edges. . .the opening through the internal table being larger than that through the external table. The track of the ball was full of clotted blood and contained several little fragments of bone with small pieces of the ball near its external orifice. Both of the orbital plates of the frontal bone were fractured.*
>
> —Autopsy of Abraham Lincoln, 1865

Gunshot Wound Fracture Patterns: Keyhole Fracture

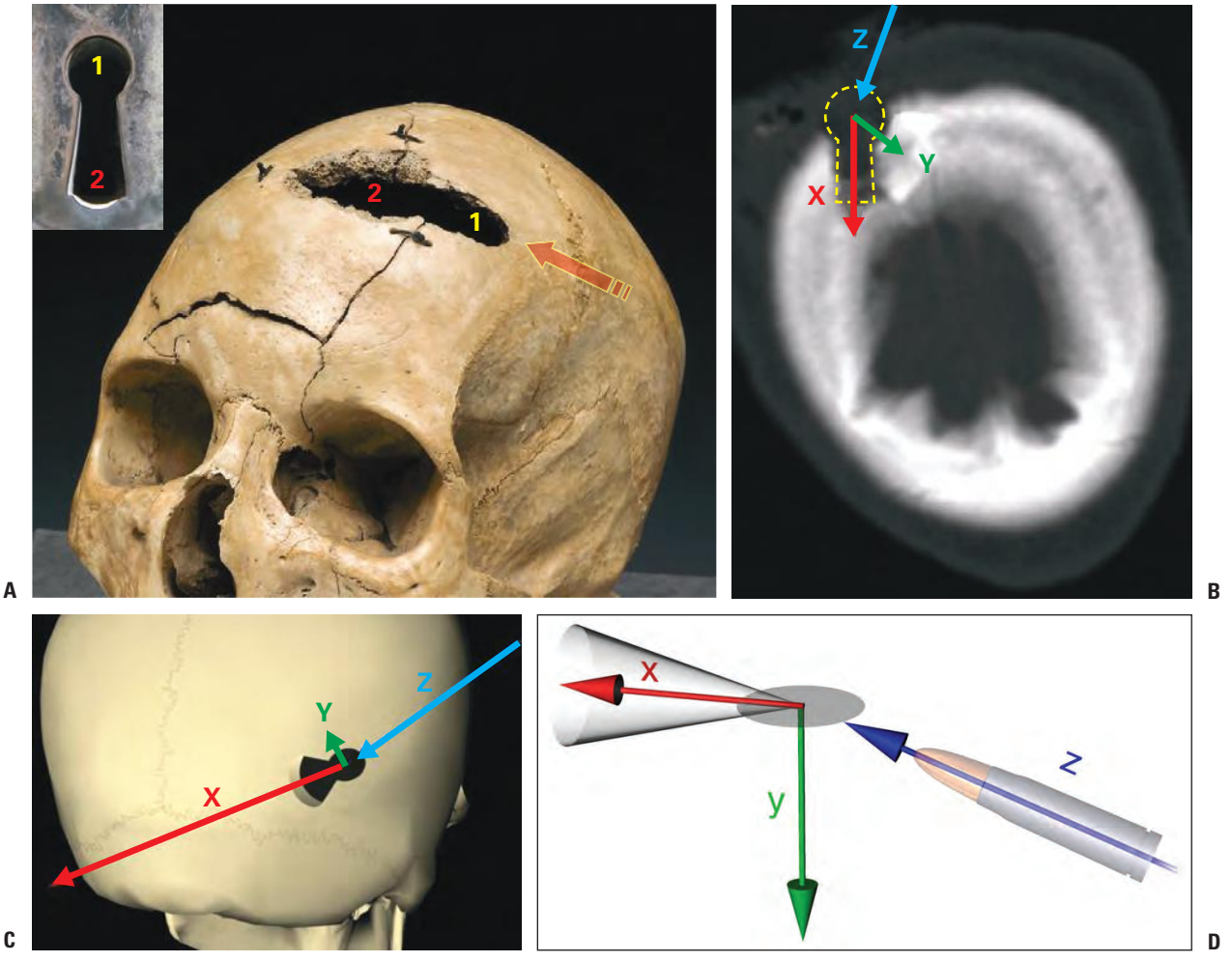


Figure 4.37. **Keyhole Fracture. A.** Photograph of a skull showing the characteristic combination of both entry and exit features at the site of impact. Note the sharp margin at the entry site (*1*) and irregular beveling of the outer table of the skull at the distal end of the wound (*2*). Both the size of the exit hole and the size of the sloped bevel are larger at the exit site than at the entry site. (Image courtesy of the National Library of Medicine.). Photograph of a typical door keyhole symbolizing the circular entrance defect and a triangular exit defect. **B.** CT example of a keyhole fracture with the trajectory forces outlined. **C.** Drawing of the keyhole mechanism in the skull. Note that the projectile does *not* enter the skull, but the force of the tangential impact causes significant intracranial injury. **D.** Mechanism of the keyhole fracture. Note that the projectile impacts the skull tangentially (*Z*), but the projectile itself does not enter the brain. The impact with the skull does, however, displace bone fragments perpendicularly into the brain (*Y*). As the projectile continues its course, it grazes the surface of the skull and causes beveling of the outer table of the skull (*X*). (*Continued*)

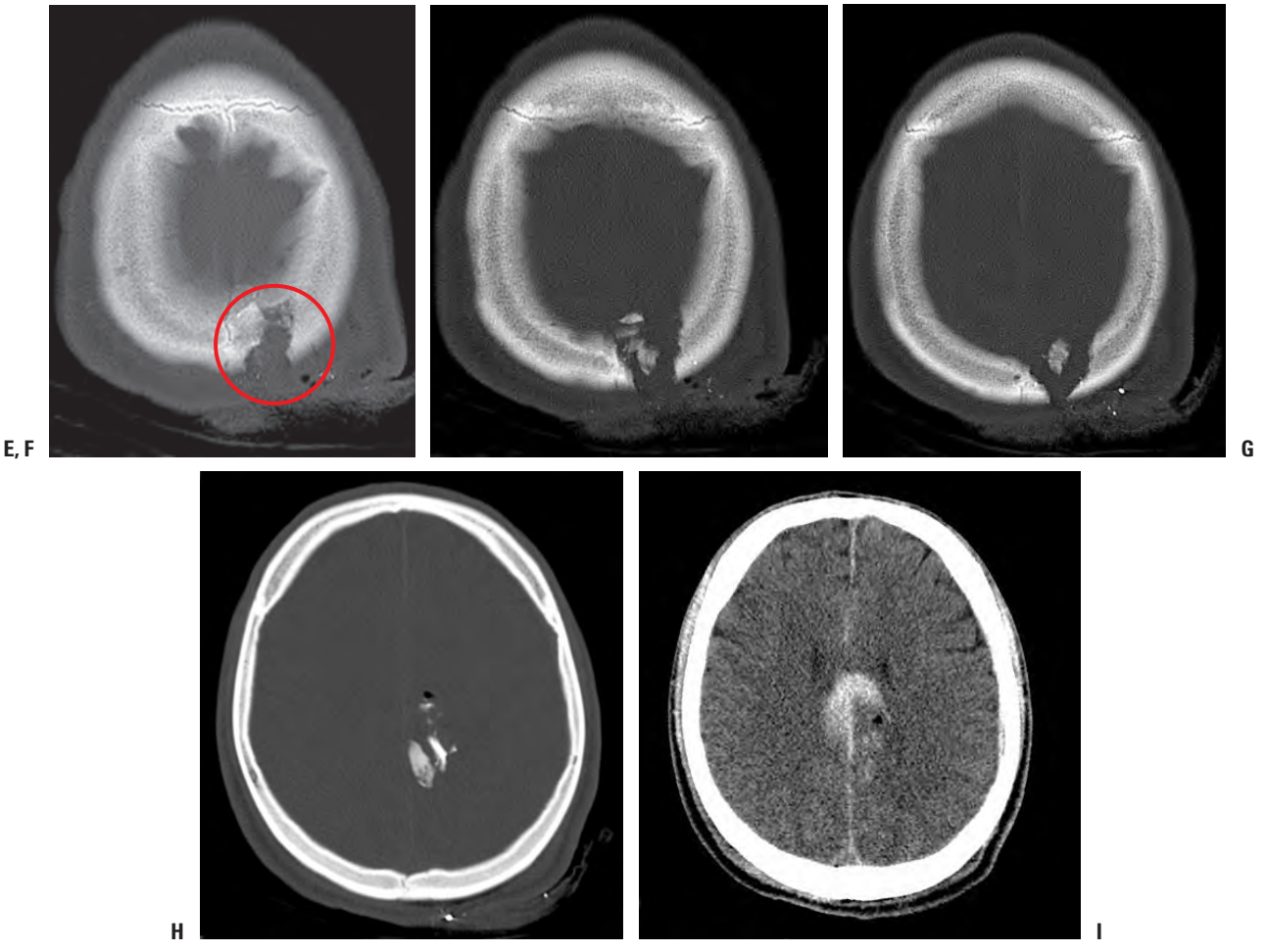


Figure 4.37. (*Continued*) **E–H.** Contiguous axial images viewed with bone windowing shows inward displacement of bone fragments. Note the upside-down keyhole fracture shape illustrated in **(E)** (*circle*). **I.** Brain windowing shows intracranial hemorrhage extending to the depth of the corpus callosum. (Adapted with permission from Jackson A, Searcey B, Smirniotopoulos J, Folio L. Keyhole fracture of the skull. *Mil Med* 2008, Dec;173[12].)

★**KEY POINT** The keyhole fracture, sometimes referred to as a gutter fracture, occurs when the projectile strikes the surface of the skull in a tangential manner. The shearing force of the volume-restricted cavitating energy, coupled with bone fragments serving as secondary missiles, makes brain injuries subsequent to keyhole fractures often devastating and in excess of what is erroneously predicted from visualization of the entrance wound.

Treatment and Prognosis. There has been an evolution of the treatment paradigms for penetrating injury over the past 50 years. Prior to the Vietnam War, there was aggressive debridement. This switched to conservative debridement in the Lebanese/Israel War. *The current approach is aggressive decompression as indicated for the brain injury itself, with a conservative approach to management of the entrance and exit wounds. Conservative wound management incorporates gentle irrigation, wound debridement and watertight closure of the dura, if possible, and scalp.*[49,50]

In many cases, brain swelling precludes repair of the dura, and thus a secure closure of the scalp is the best second-line defense against intracranial infection (see **Figs. 4.26** to **4.28**). Surgeons in the First Persian Gulf War (1990 to 1991) and Operation Desert Storm found that the initial goal in the treatment of shrapnel wounds of the brain should be to maximally preserve cerebral tissue. This entails either washout and debridement of scalp wounds when a surgical procedure is not necessary or limiting the wound debridement in situations when a craniectomy procedure is performed. This more conservative approach involves minimal manipulation of the injured and swollen brain and specifically avoids earlier practices of searching for shrapnel fragments that did not present themselves during gentle irrigation or hemostatic maneuvers.

Fears of intracranial abscess formation and development of chronic seizure disorders have been the main objections to the conservative approach. However, this concern is countered by the argument that a conservative debridement technique may result in reduced cortical tissue injury, less cortical volume loss, and therefore a diminished risk of post-traumatic seizures from the penetrating injury. Over 40% of patients with penetrating brain injury suffer from post-traumatic epilepsy. Furthermore, some believe that brain abscesses are less likely to form around a metallic shrapnel fragment, macerated brain, or other in-driven debris due to the sterilizing effect of the heat generated by the metal fragment.[51] In the absence of significant mass effect, small-entrance bullet wounds to the head are treated with local wound care and more extensive wounds with nonviable scalp, bone, or dura are debrided before closure. In the presence of mass effect, necrotic brain tissue is resected, but surgical debridement of the missile tract has not been shown to improve outcomes.[52]

Migration of a bullet fragment may occur days to years from the time of injury and is in general an indication for retrieval of a fragment, assuming that it is surgically accessible. At the time of the injury, exploration for bullet retrieval from within the brain is not recommended during initial wound management and surgery. However, bullets visible on the brain surface within a craniotomy or craniectomy exposure should be extricated. Consideration should be given to prophylactic removal of bullet fragments that come to rest in the CSF ventricles or cisterns due to their increased propensity to subsequently migrate. The timing of removal in these situations is individualized depending on the severity of the brain injury, the degree of brain swelling, and the likelihood that migration would lead to further brain injury or entail a more inaccessible or risky surgical retrieval of the bullet. Bullet fragments, as well as other foreign bodies, juxtaposed to intracranial vascular structures should be carefully analyzed for retrieval if surgically feasible.

The prognosis after penetrating injuries follows the rule of real estate: location, location, location. A small-caliber bullet or ice-pick injury to the brain stem is typically

fatal. Similarly, bihemispheric or multilobar injury and intraventricular hemorrhage are correlated with poor outcome.[53] Lateral perforating wounds typically have the worst outcome, particularly when they traverse the *zona fatalis* (i.e., approximately at the level of the thalamus).[54] This is the typical trajectory for a suicide GSW, which is essentially a high-energy contact injury that is associated with high mortality. As in civilian trauma, the mortality of combat-penetrating injuries with trajectories crossing the deep midline structures of the brain approximates 100%. However, patients with bilateral injuries limited to the more superficial aspects of the cerebral cortices can do remarkably well (**Figs. 4.28**, **4.29**, and **5.24**).[55] This may be because penetrating injuries to the cerebral cortices, even if bilateral, spare the basal vasculature and brain stem. By comparison, posterior fossa injuries generally have a poor prognosis, as the brain stem is likely to be injured.[56] Ventricular injuries have also been reported to have a high mortality rate.[57]

In general, the overall mortality of penetrating GSWs is approximately 85%. Admission GCS, pupillary status, and hemodynamic status seem to be the most significant clinical predictors of outcome.[8,58] Patients with a GCS score >8, normal pupil reaction, and single lobe of brain injury are most likely to benefit from early aggressive management. After surgical intervention, the mortality rate of craniocerebral GSWs is around 10%.[59] However, more effective onsite patient stabilization and management, faster transport to a tertiary trauma facility, and judicious use of aggressive surgical treatment may enable better GCS scores at presentation, earlier intervention, and improved outcomes. Finally, as alluded to earlier, the severity of a GSW is determined not by the amount of KE possessed by the bullet but rather by the amount of energy that is lost in the tissue. That is to say, it is the transfer of energy from the moving projectile to the target that brings about tissue damage. The main determinants of the amount of KE transferred by a bullet, and therefore, its wounding potential, are (1) shape of the bullet, (2) angle of yaw at the time of impact, (3) change in the presented area of the bullet in its passage through the brain, (4) construction of the bullet, and (5) biologic characteristics of the tissue through which the bullet passes.

GSW injuries to the brain bring about a number of pathophysiologic processes, including an elevation in ICP, brain swelling, and vascular injury with ischemic damage (see **Fig. 4.27**). Apnea and possible anoxic brain injury may occur from direct brain stem injury, or more commonly, from a pressure-wave shock injury to the brain stem. Traumatic vasospasm and/or vascular thrombosis caused by the shock wave may also contribute to anoxic injury. In either case, the instantaneous damage inflicted by the bullet is soon compounded, over the next few minutes to hours, by intracranial hypertension. Signs of raised ICP may be absent or subtle on an initial CT performed early after the injury. Sulci effacement, compression of basal cisterns, and loss of cortical gray–white matter discrimination evolve to advanced degrees, portending life-threatening brain herniation in a surprisingly short period of time after a GSW injury.

In summary, the resultant damage from a penetrating injury is caused by crushing, shredding, and stretching forces imparted directly from the projectile to the brain tissue, compounded with damage from secondary missiles and bone fragments. Together, these primary and secondary damaging forces culminate in a sudden increase in intracranial pressure, with

shock wave coup and contrecoup forces acting on the brain against the skull, dura, and other firm structures. By comparison, in the case of a knife injury, the brain injury may be quite local, without any additive temporary cavitation. In GSWs, however, the injury is far more complex, and the severity of the wound is directly related not simply to the mass and velocity of the fired bullet but to the amount of KE transferred from the projectile to the tissue. If the bullet enters the tissue but does not exit, all of its KE is applied to wound formation. If the bullet exits the tissue, the moving projectile retains some of its KE, and less of its KE is transferred to effect damage to the brain. The skull is a relatively innocent bystander in that it simply reacts to the focal impact of the bullet and the sudden increase in ICP. Depending on the thickness of the skull and the angle of impact, the skull can display a number of characteristic fracture patterns unique to penetrating GSW injuries.

TABLE 4.2 Imaging Checklist for Gunshot Injuries to the Head

✓ What is the location of the missile fragment(s)? Use the CT scout view.
✓ Is it a superficial, penetrating, or perforating injury?
✓ If perforating, what are the locations of the entry and exit sites?
✓ Is the projectile intact or fragmented?
✓ Are there retained fragments (bone, bullet, glass, other)?
✓ Is the injury unihemispheric or bihemispheric? Multilobar? Transventricular?
✓ Does the missile tract traverse the paranasal sinuses and/or mastoid air cells?
✓ Describe the trajectory of the bullet in three dimensions (i.e., is the path anterior or posterior, left or right, and superior or inferior?); the scout view can be helpful.
✓ Does the bullet path have a ricochet component?
✓ Are the basal cisterns preserved effaced? Is there midline shift?
✓ Is there hemorrhage remote from the primary injury?
✓ Is there a fracture remote from the primary entry and exit sites (e.g., orbital roof)?
✓ Describe the skull fracture(s): linear, comminuted, depressed, keyhole, or heaving?
✓ Does the fracture traverse the carotid canal or a dural venous sinus?
✓ Is CTA or catheter angiography indicated?
✓ On follow-up imaging, is there a change in position of bone and/or ballistic fragments?
✓ On follow-up imaging, is there new intracranial hemorrhage or enhancement to suggest interval development of a traumatic pseudoaneurysm?

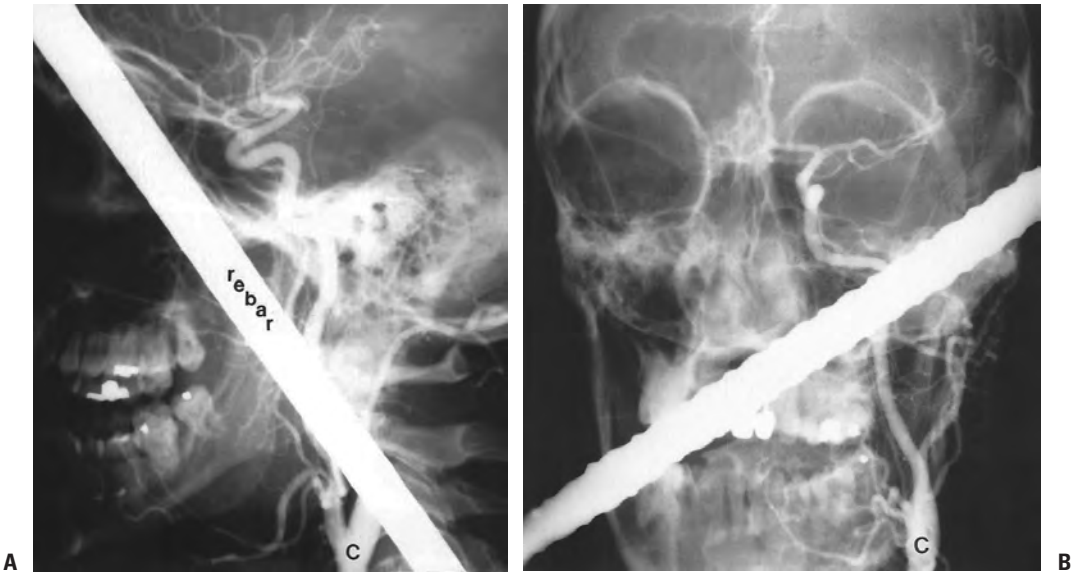
Nonballistic Penetrating Injury

Patients with nonballistic penetrating injuries often present to the emergency department (ED) with the penetrating weapon still in place (**Figs. 4.38** to **4.44**). Non-contrast CT, and occasionally plain radiographs, are initially obtained to estimate the depth and location of injury. CTA should be performed to exclude potential vascular injury, although metallic artifacts from a penetrating weapon may limit the usefulness of these studies (**Figs. 4.40** and **4.44**).[28,60] CTP may be helpful to identify ischemic brain tissue (**Fig. 4.46**). If CTA is suboptimal to exclude vascular injury, formal catheter angiography is performed prior to surgical removal of the weapon (**Figs. 4.39**, **4.41**, and **4.44**).[61] It is also helpful in the postoperative phase to exclude interval development of a traumatic pseudoaneurysm (**Fig. 4.40**; see also Chapter 5, Lessons 3 and 10). With handheld penetrating weapons (e.g., knife, hammer, pneumatic nail gun), the severity of the injury depends on the properties of the weapon, sharp versus dull and smooth versus irregular, as well as the physical characteristics of the skull at the point of impact.[62] In most cases, the dura and pia-arachnoid are torn, fragments of bone are propelled into the wound, and the weapon crushes brain tissue and disrupts intervening vascular structures along its path. The final injury often appears as a canalicular hemorrhagic wound canal (**Figs. 4.41**, **4.42**, **4.47**, and **5.24**). *Unlike GSWs, there is no temporary cavitation, and the final wound is due solely to permanent cavitation*. The degree of injury from handheld weapons depends on the depths to which the weapon penetrates. Brain injury resulting from blunt handheld weapons, such as a hammer, tend to be more superficial. Skull fractures and cortical contusions are common injuries from these weapons. However, if impact leads to fracture, causing thrombosis or laceration of an underlying major venous sinus, even a superficial injury can be fatal (**Fig. 4.45**). Similarly, a sharp handheld weapon can cause significant vascular injury depending on the depth to which it passes. Superficial sharp injury may similarly impair dural venous sinuses. Sharp, handheld weapons may, unlike their blunt counterparts, also penetrate deeply and injure major venous as well as arterial vessels (**Figs. 4.43** and **4.46**). Acutely, passage of a handheld weapon into the brain may cause perturbations and spasm in nearby and intervening arteries, and the wound may not bleed or may bleed only minimally. Bleeding into the wound track may ensue if transient spasm resolves early, before mechanisms of hemostasis have had time to seal disrupted sites in the vessel. On the other hand, prolonged vessel spasm may be of sufficient severity and duration to impair perfusion to downstream brain tissue, and infarction may result in the territory supplied by the injured arteries (see **Fig. 4.46**). Devastating nonballistic penetrating TBI may also result from nonhandheld weapons (**Fig. 4.48**). Penetrating injuries to the neck may lead to not only local vascular injury but they may also be the source of thromboembolic complications to the cerebral vasculature (**Fig. 4.50**). A pseudoaneurysm may develop from an injured vessel and yet not come to medical attention for several months or even years (**Fig. 4.49**).[63] Penetrating neck trauma is also typically imaged first with CTA with a low threshold for follow-up cerebral angiography in cases of obscuration of the vessels by artifact, uncertainty as to possible subtle abnormalities, and injuries requiring further definition and/or endovascular intervention. In the chronic phase, considerable mixed glial-mesodermal scarring and adhesions between brain and the dura can be identified, which contributes to the high rate of post-traumatic epilepsy in penetrating TBI.

Figure 4.38. Penetrating TBI. Photograph of a victim from the 2003 terrorist attack on the United Nations in Baghdad. During the explosion, framing from a window became a secondary projectile and impaled the victim's head. The patient underwent a staged procedure with proximal cervical carotid exposure and craniofacial exposure with projectile removal and skull base repair. (Courtesy of Rocco Armonda, MD, Col [ret], MC, USA.)

★**KEY POINT** As shown in **Figures 4.38** to **4.44**, patients with penetrating injuries often present to the ED with the foreign object in place, and angiography is usually performed before surgical removal of the object.

Nonballistic Penetrating TBI (Rebar Impalement)



A B

Figure 4.39. Penetrating TBI. A 30-year-old construction worker who fell three stories onto a metal concrete reinforcement bar framework (i.e., rebar). While a CTA was without obvious signs of arterial injury, the study was severely limited by streak artifact, which obscured optimal visualization of the vasculature. **A.** Lateral and **(B)** AP views from a left common carotid artery angiogram demonstrate a metal bar extending through the left face and right neck. Remarkably, no vascular injury was evident, even on the cerebral angiogram study. Common carotid artery **(C)**.

Nonballistic Penetrating TBI (Accidental Nail Gun Injury)

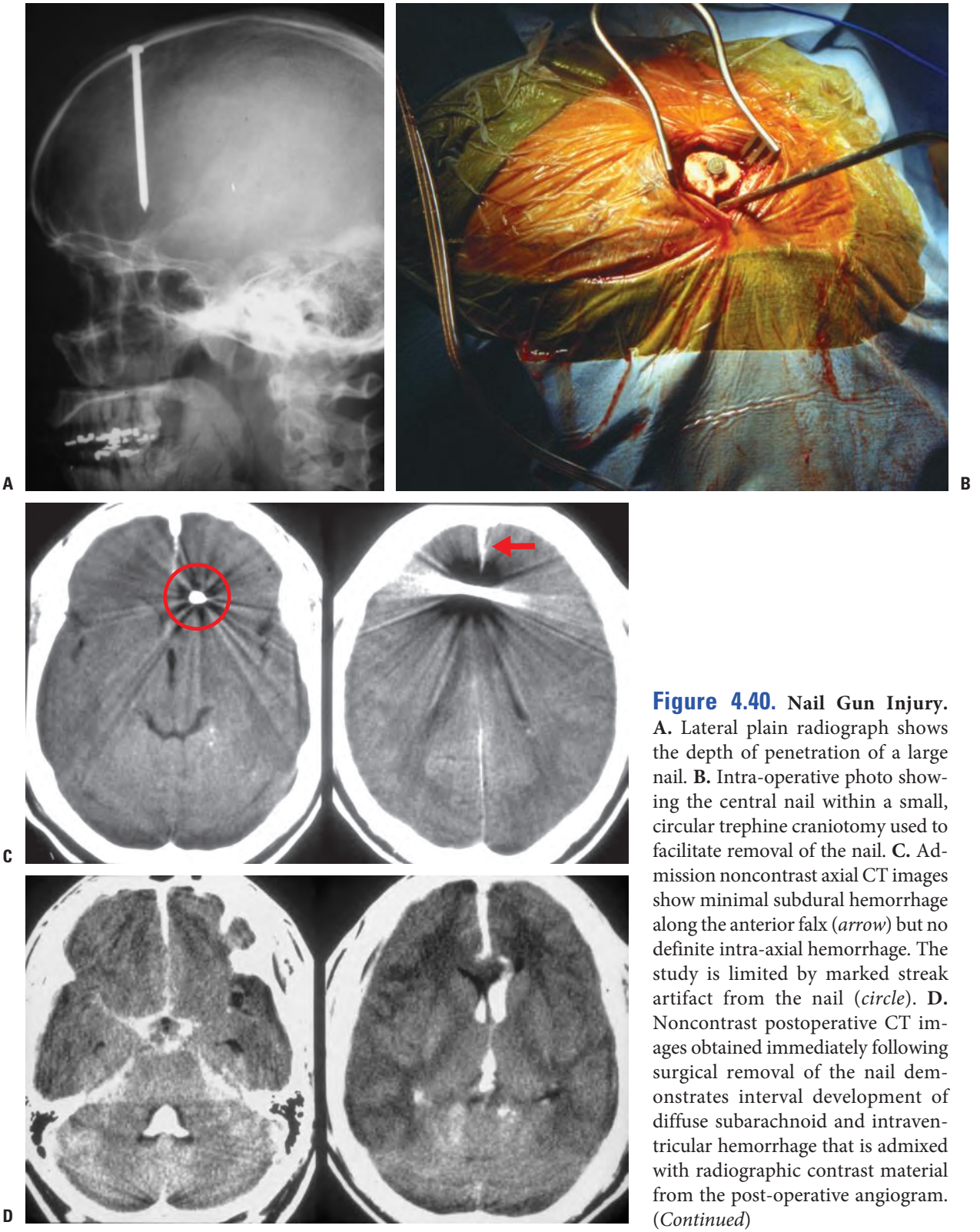
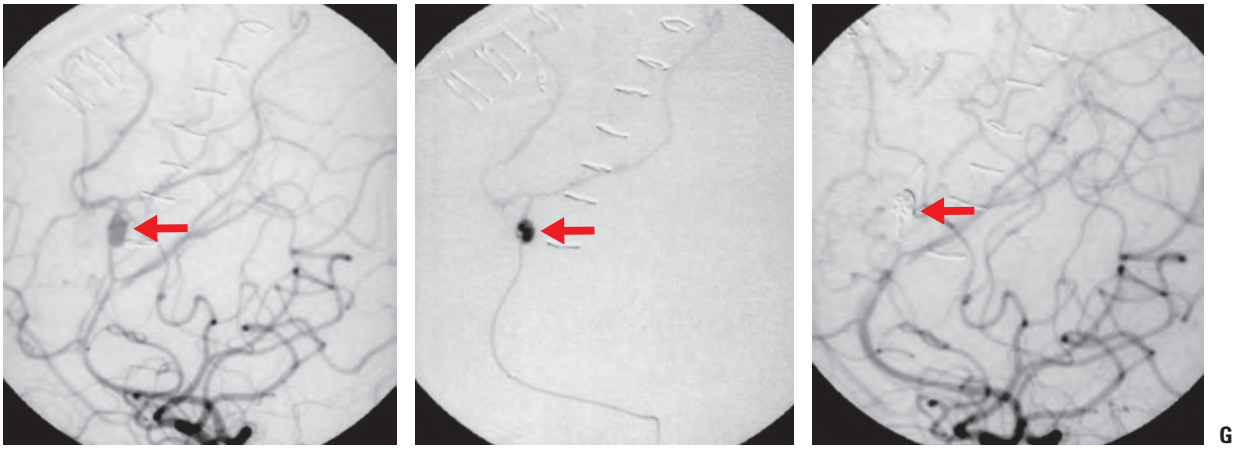


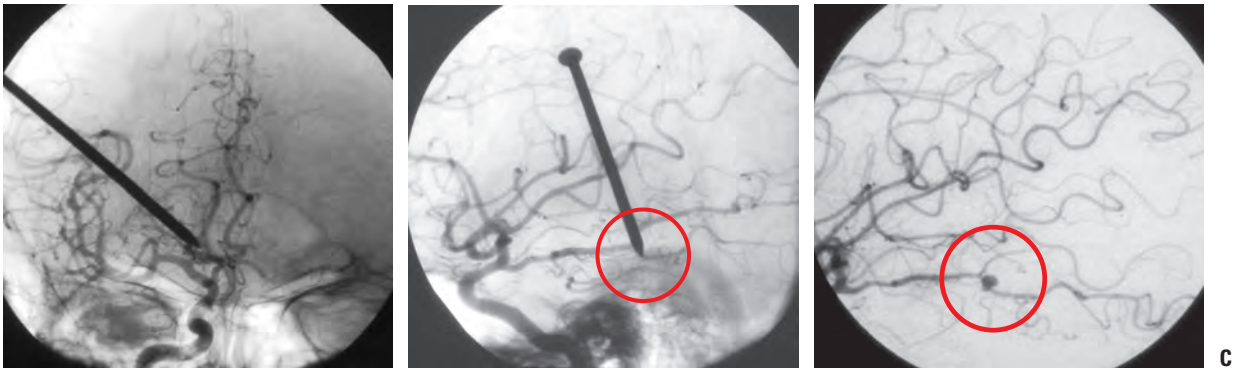
Figure 4.40. Nail Gun Injury. **A.** Lateral plain radiograph shows the depth of penetration of a large nail. **B.** Intra-operative photo showing the central nail within a small, circular trephine craniotomy used to facilitate removal of the nail. **C.** Admission noncontrast axial CT images show minimal subdural hemorrhage along the anterior falx (*arrow*) but no definite intra-axial hemorrhage. The study is limited by marked streak artifact from the nail (*circle*). **D.** Noncontrast postoperative CT images obtained immediately following surgical removal of the nail demonstrates interval development of diffuse subarachnoid and intraventricular hemorrhage that is admixed with radiographic contrast material from the post-operative angiogram. (*Continued*)



E, F G

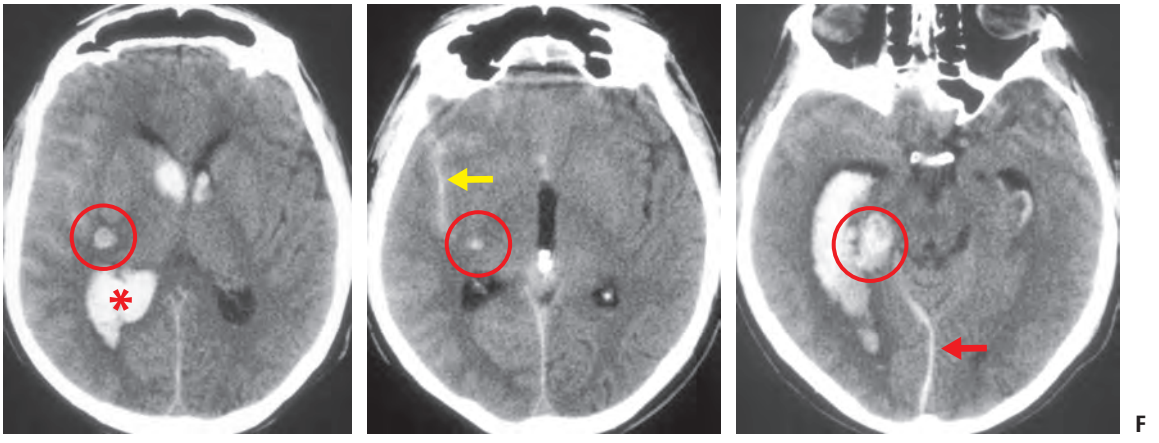
Figure 4.40. (*Continued*) **E.** Lateral view from the postoperative internal carotid artery catheter angiogram shows a pseudoaneurysm of the callosomarginal artery (*arrow*). **F.** Selective catheterization of the callosomarginal artery shows the tip of the microcatheter located within the pseudoaneurysm (*arrow*). Semisubtracted craniotomy skin staples are noted. **G.** Postembolization angiogram demonstrates coil occlusion of the aneurysm. (Courtesy of Chris Dowd, MD, UCSF.)

Nonballistic Penetrating TBI (Accidental Nail Gun Injury)



A, B C

Figure 4.41. **Nail Gun Injury. A.** Unsubtracted frontal and **(B)** lateral views from admission catheter angiography of the right internal carotid artery show a large intracranial nail coursing right to left, superior to inferior, and slightly anterior to posterior. Note the fetal origin of the posterior cerebral artery (PCA). The tip of the nail abuts the PCA, but no definite vascular abnormality is identified. **C.** Following removal of the nail, a postoperative angiogram, lateral subtracted view, shows a traumatic pseudoaneurysm of the PCA (*circle*) and mild vasospasm. (*Continued*)



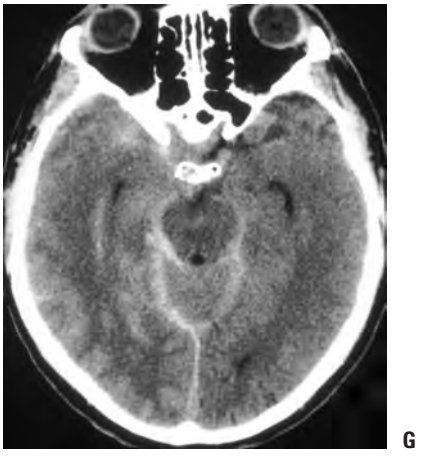


Figure 4.41. (*Continued*) **D–G.** Corresponding postoperative noncontrast CT images show the hemorrhagic track of the nail (*circles*). Note also intraventricular (*asterisk*), subarachnoid (*yellow arrow*), and falcine subdural (*red arrow*) hemorrhage. The hemorrhagic footprint of the nail track is largest at the level of the midbrain **(F)**, corresponding to the location of the PCA and pseudoaneurysm and suggesting that pseudoaneurysm rupture was the cause of the hemorrhages noted throughout. Tumefactive intraventicular hemorrhage is causing acute obstructive hydrocephalus.

★**KEY POINT** The previous three cases emphasize how preoperative and postoperative catheter angiography are indicated when a projectile is located near a major vessel.

Nonballistic Penetrating TBI (Lawn Dart Injury)

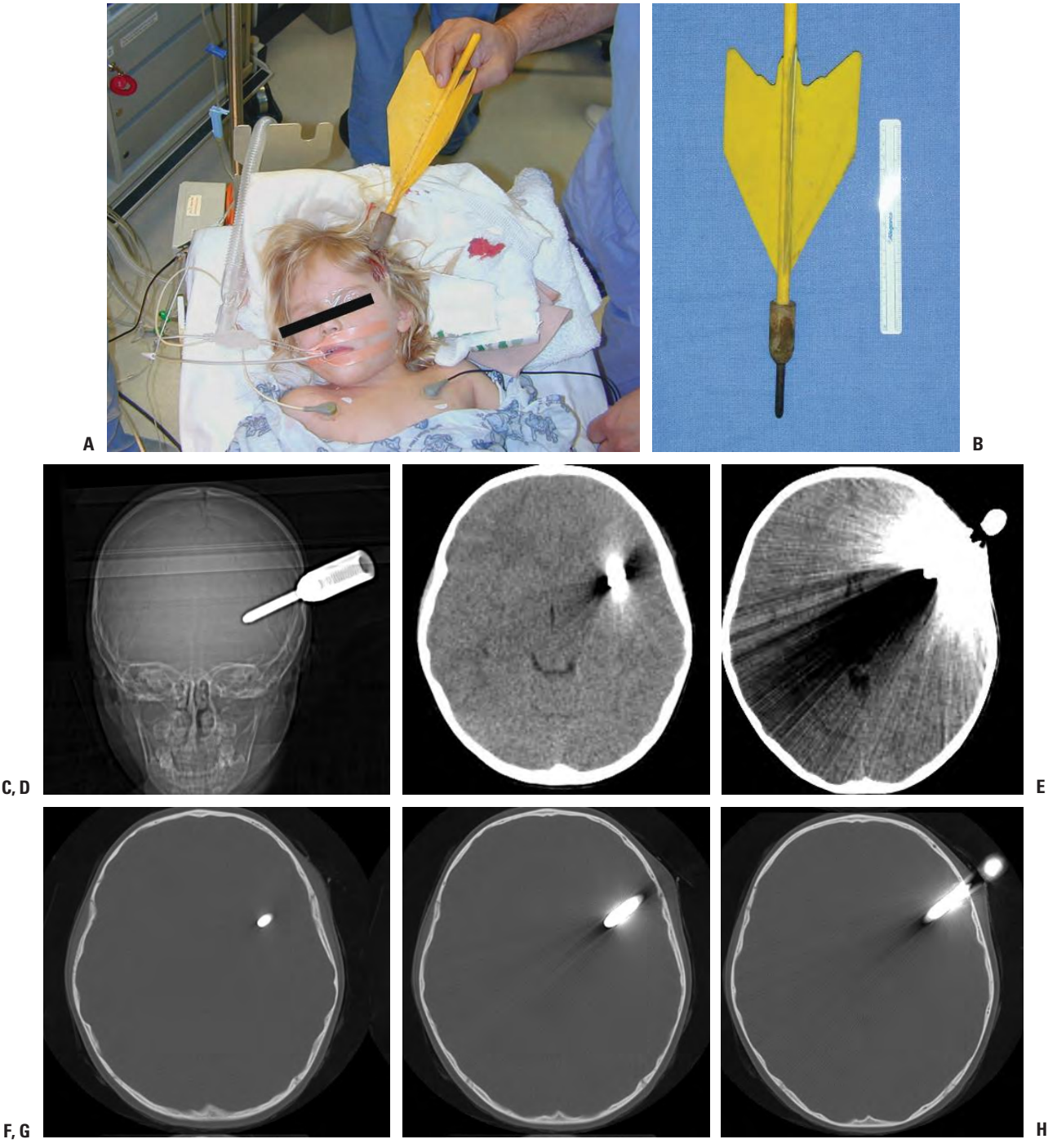
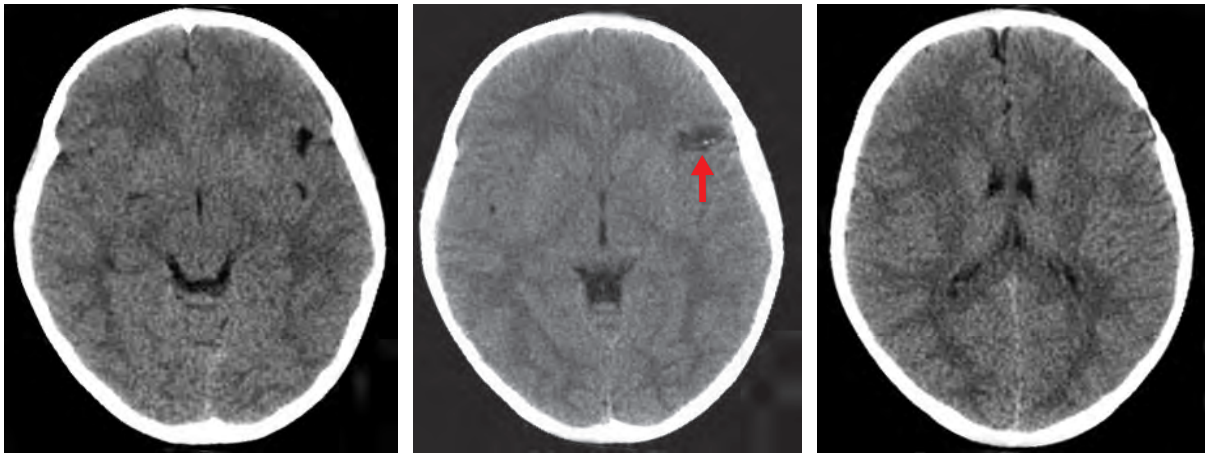


Figure 4.42. **Penetrating TBI. A.** Photograph of young girl with a penetrating injury to the left frontal region from an airborne lawn dart. **B.** Photograph of the dart after extraction. **C.** Admission CT AP scout view. **D, E.** Soft tissue CT windows show marked streak artifact precluding optimal visualization of the brain parenchyma. **F–H.** Bone windowing shows the depth of penetration with minimal disruption of the calvarium. (*Continued*)



I, J K

Figure 4.42. (*Continued*) **I–K.** Follow-up CT obtained 1 month later, the patient having had a seizure, reveals a small residual linear focus of encephalomalacia (*arrow*). (Courtesy of Howard Rowley, MD.)

★**KEY POINT** Owing to the absence of a cavitation component in handheld penetrating trauma, such lesions can result in relatively little injury to brain tissue.

Penetrating TBI (Non-Accidental Nail Gun Injury)

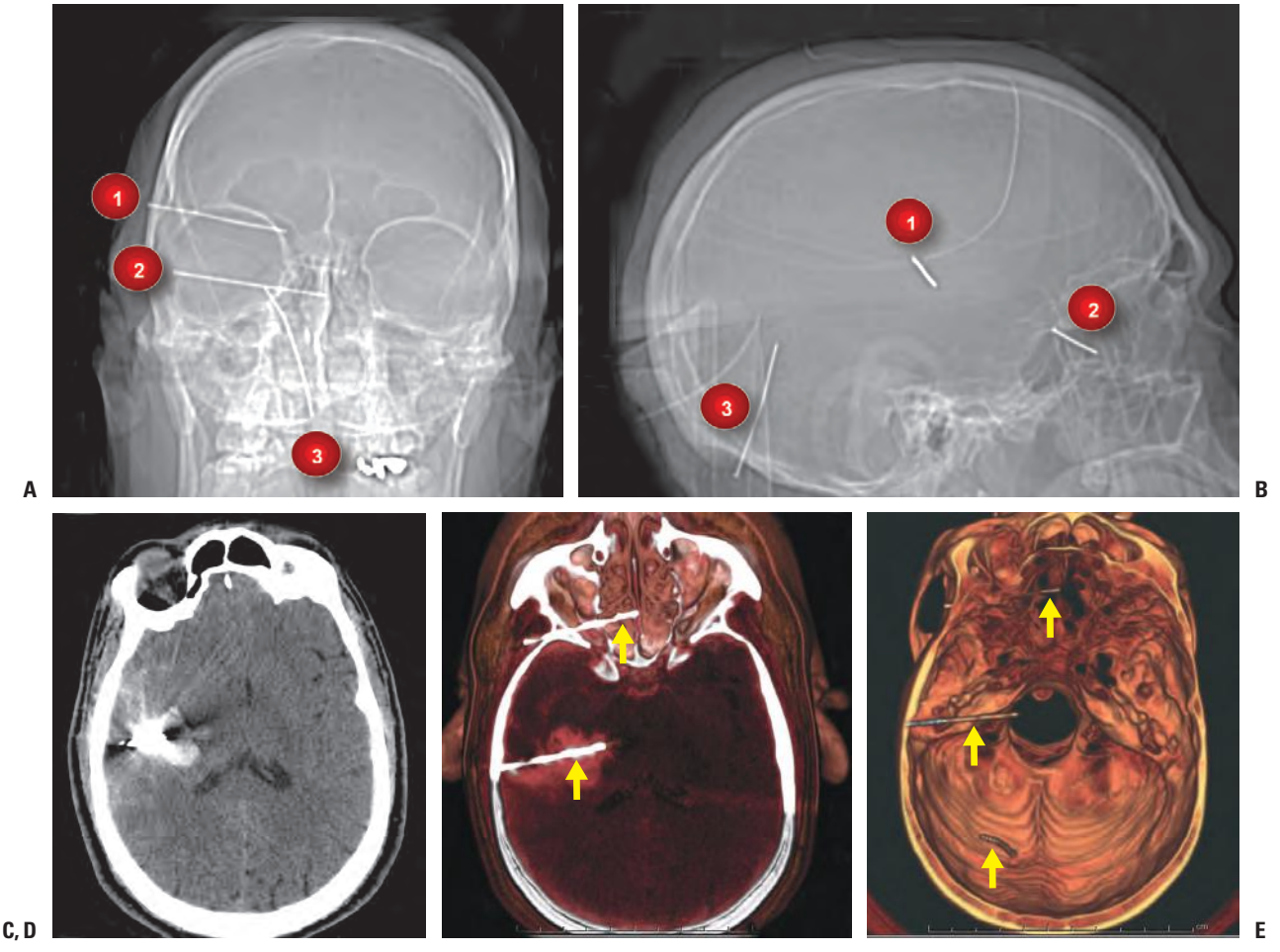


Figure 4.43. **Penetrating TBI. A,B.** AP and lateral CT scout images demonstrate three intracranial nails. **C.** Axial CT viewed at brain window shows intraparenchymal hemorrhage and SAH associated with a penetrating injury to the right temporal lobe. **D.** Thick slab 2D multiplanar reformation and **(E)** 3D, volume-rendered, shaded surface display images with virtual exclusion of the calvarial vault help visualize the trajectory of each nail (*arrows*). (Courtesy of Pete Hildenbrand, MD.)

★**KEY POINT** The zone of injury for nail gun injuries can be larger than expected for typical handheld, nonballistic penetrating weapons because of the pneumatic firing mechanism that causes concomitant cavitational injury.

Penetrating Craniocervical Injury (Chopstick)

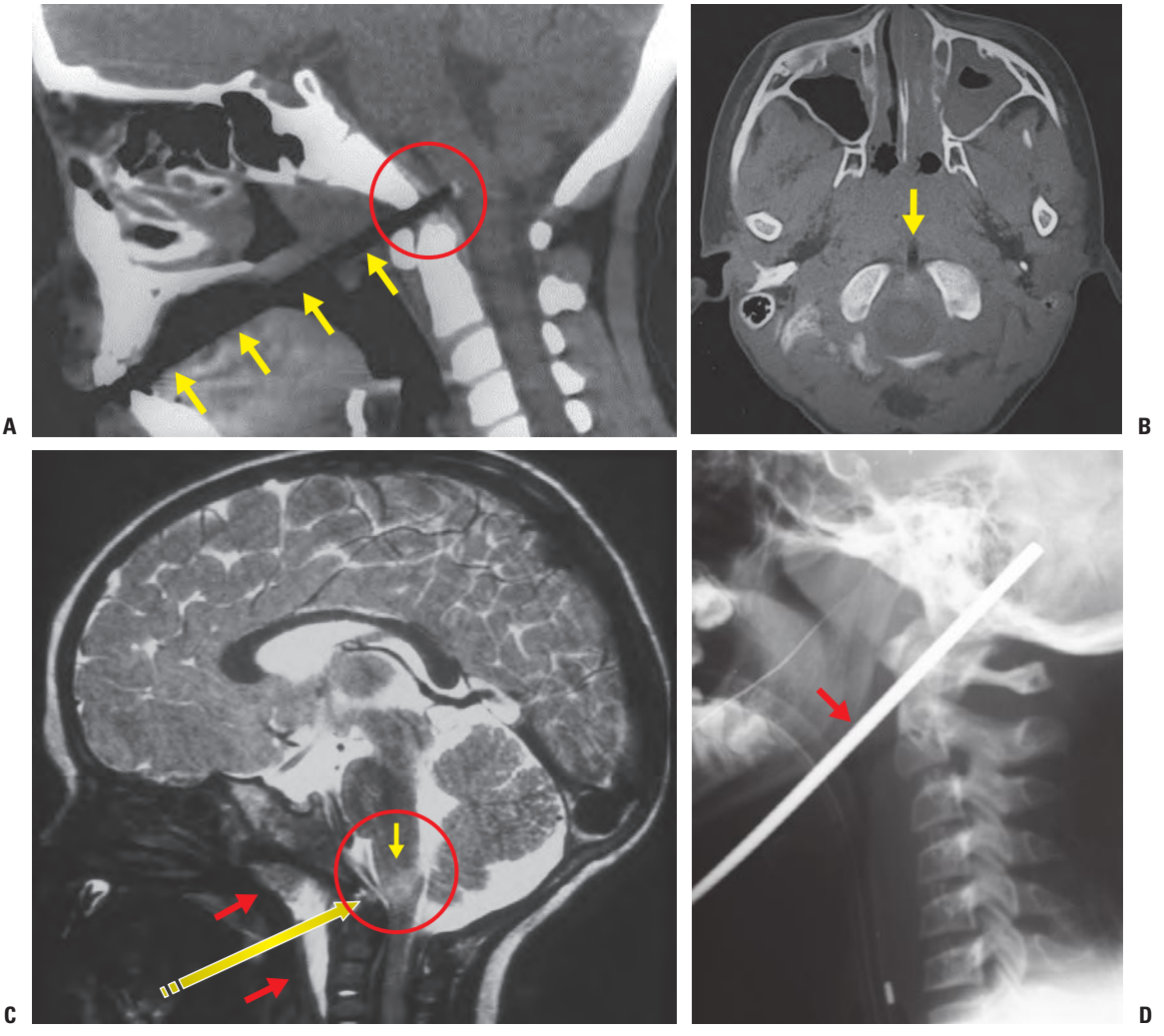


Figure 4.44. **Craniocervical Penetrating Injury (Three Different Young Patients). Patient 1** (Chopstick): **A.** Sagittal reformatted CT image in a young girl presenting to the ED with a chopstick stuck in her mouth. There is a linear radiolucent foreign body (*yellow arrows*) within the oropharynx, traversing the nasopharynx, and penetrating the craniocervical junction (*circle*). The tip of the chopstick abuts the ventral medulla oblongata. *Note that dry wood mimics air on CT* (hence its radiolucent density). **B.** Axial CT viewed with bone windowing shows a portion of the chopstick (*arrow*) at the level of the occipital condyles. **Patient 2** (Chopstick): **C.** Sagittal T2-weighted MRI in a patient who presented to the ED without the chopstick in situ. Note the focal T2 hyperintensity lesion within the medulla (*small yellow arrow*) and swelling of the nasopharyngeal soft tissues (*red arrows*). The hypothetical trajectory is outlined in the *large yellow arrow*. **Patient 3** (Chopstick-like metal rod): **D.** Lateral radiograph shows the foreign body entering the upper neck and traversing the craniocervical junction (*arrow*). (*Continued*)

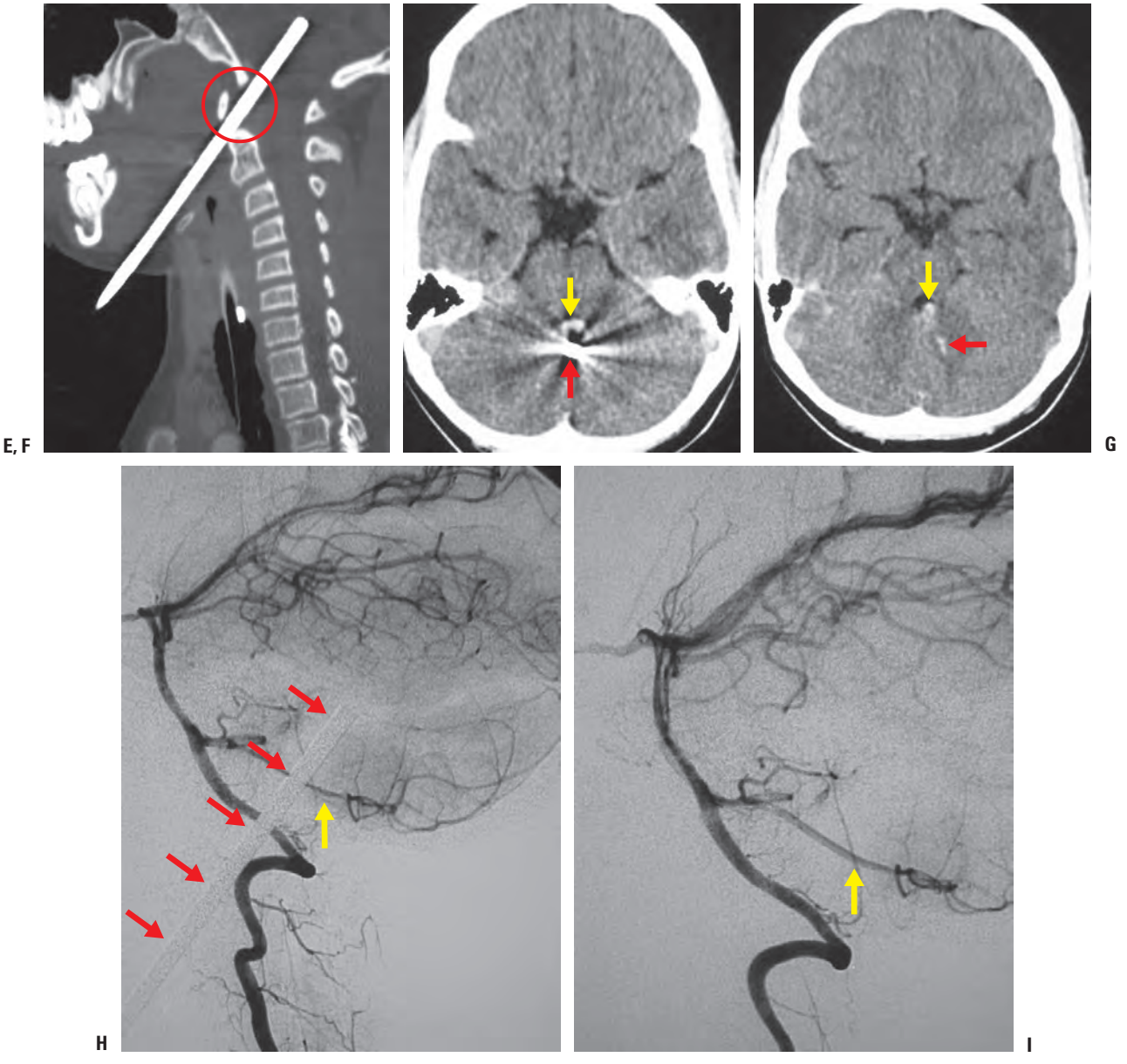


Figure 4.44. (*Continued*) **E.** Sagittal reformatted CT image demonstrates termination of the foreign body within the posterior fossa. Again, note how the rod enters the brain via the craniocervical junction (*circle*). **F.** Noncontrast preoperative axial CT shows extensive metallic artifact radiating from the tip of the foreign body (*red arrow*), which terminates just posterior to the fourth ventricle. A small amount of intraventricular hemorrhage is present (*yellow arrow*). **G.** Adjacent noncontrast axial CT image at the superior extent of the injury shows a tiny focus of intra-axial hemorrhage within the left corpus medullaris of the cerebellum (*red arrow*). A small amount on intraventricular hemorrhage is again seen (*yellow arrow*). **H.** Lateral catheter angiogram following injection of the left vertebral artery shows the rod "subtracted out" (*red arrows*), overlying the proximal V4 segment of the vertebral artery. Remarkably, there is no sign of disruptive injury (dissection, pseudoaneurysm, or transection) to the vasculature. Minimal vasospasm of the posterior inferior cerebellar artery (PICA) is present (*yellow arrow*). Note that the PICA originates from a dominant anterior inferior cerebellar artery (AICA) in this patient. **I.** Postoperative view following surgical removal of the foreign body shows improvement in the filling of the PICA (*arrow*). (*Continued*)

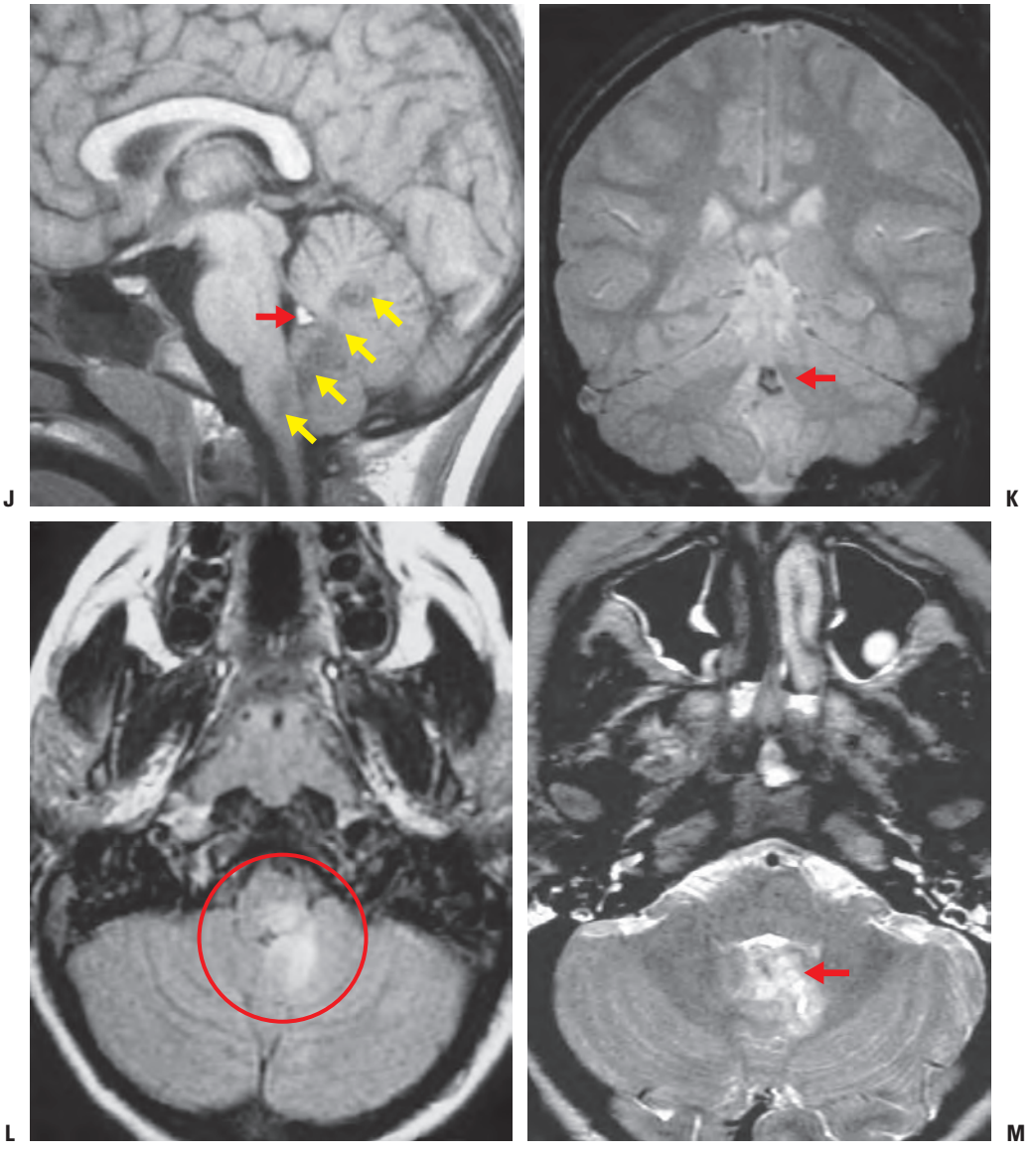


Figure 4.44. (*Continued*) **J.** Sagittal T1-weighted MRI performed the following day after injury shows a small focus of hemorrhage layering dependently (in a supine patient) in the fourth ventricle (*red arrow*). Note the tract-like area of T1-hypointensity within the medulla and cerebellum consistent with edema at the site of the extracted metal rod. **K.** Coronal gradient-recalled echo (GRE) image confirms the presence of intracellular methemoglobin within the fourth ventricle (*arrow*). **L.** Axial fluid-attenuated inversion recovery (FLAIR) image reveals abnormal T2 hyperintensity within the left posterior medulla and cerebellar tonsil (*circle*). **M.** Axial T2-weighted image at a higher level shows T2 hyperintensity within the left corpus medullaris, tonsil, and nodulus of the cerebellar vermis (*arrow*). The brain stem is normal at this level. The patient made a complete recovery. (Courtesy of Eduard Michel, MD, PhD.)

★**KEY POINT** The craniocervical junction is a common point of entry for penetrating objects. The three cases shown previously demonstrate how the foreign bodies pass between the tip of the clivus (i.e., basion) and the atlantoaxial joint.

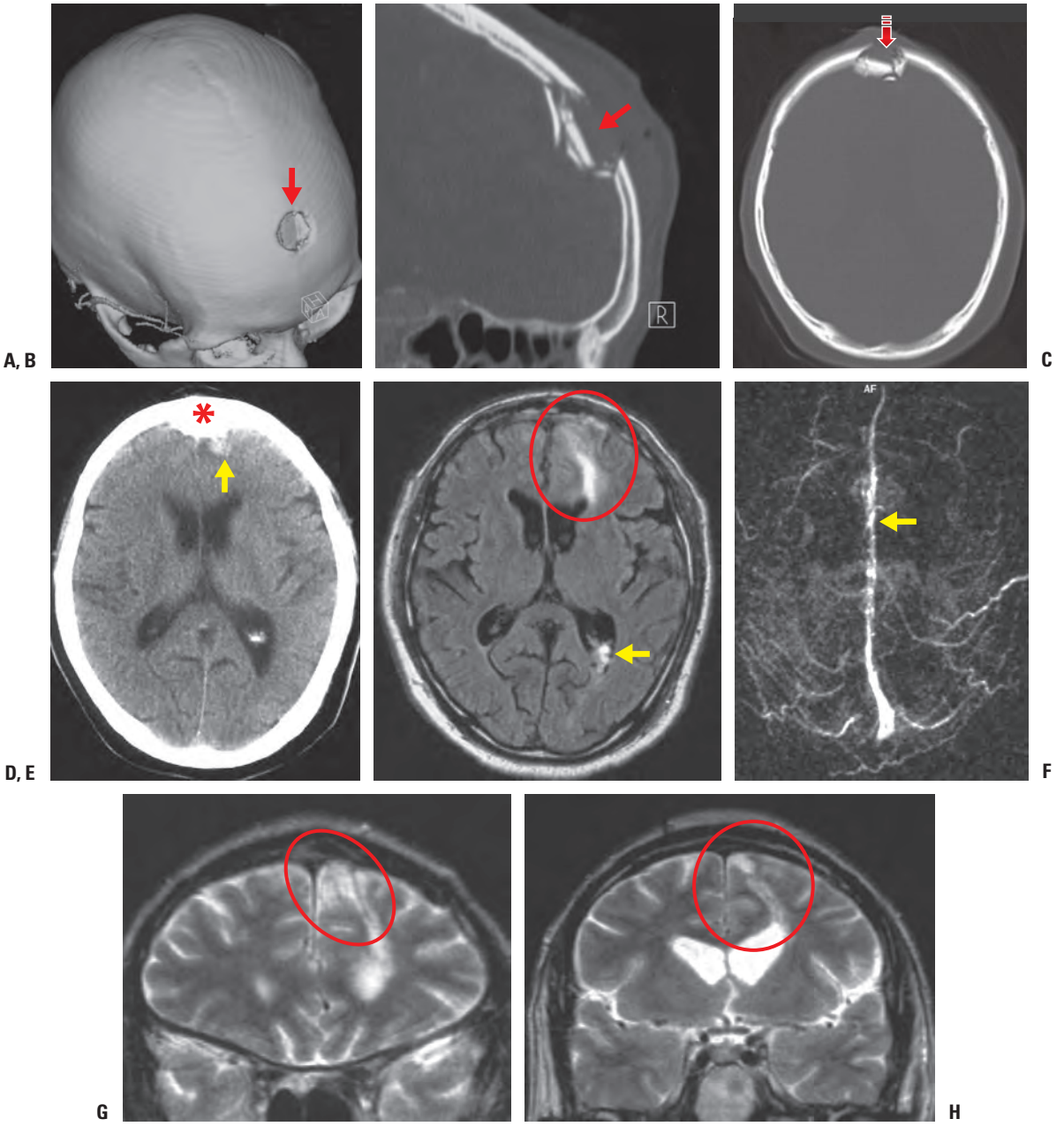


Figure 4.45. Nonballistic Penetrating TBI (Hammer to Head). A 57-year-old male assaulted with a hammer presented to the ED with a GCS = 15. **A.** Admission 3D CT performed 3 hours after the injury demonstrates a focal, well-defined, round defect in the midfrontal calvarium (*arrow*). The size and shape of the fracture corresponds exactly to the size and shape of the hammer. **B.** Admission parasagittal CT reformatted image shows a comminuted depressed fracture with preferential involvement of the inner table of the skull. **C.** Admission axial CT demonstrates the punched-out defect of the outer table of the skull (*arrow*). **D.** Admission axial CT viewed at brain window shows the depressed fracture (*asterisk*) and a small cortical contusion (*arrow*). **E.** Axial FLAIR MRI performed 4 hours after the injury reveals an area of T2 hyperintensity extending from the cortex to the frontal horn of the left lateral ventricle (*circle*); note the normal flow void in the superior sagittal sinus (SSS). A small amount of intraventricular hemorrhage is present (*arrow*). **F.** Preoperative MR venography confirms no evidence of occlusion of the SSS (*arrow*). **G,H.** Coronal T2 MRI reveals abnormal T2 hyperintensity extending from the SSS to the left frontal horn (*circle*). The flow void within the SSS appears normal. (*Continued*)

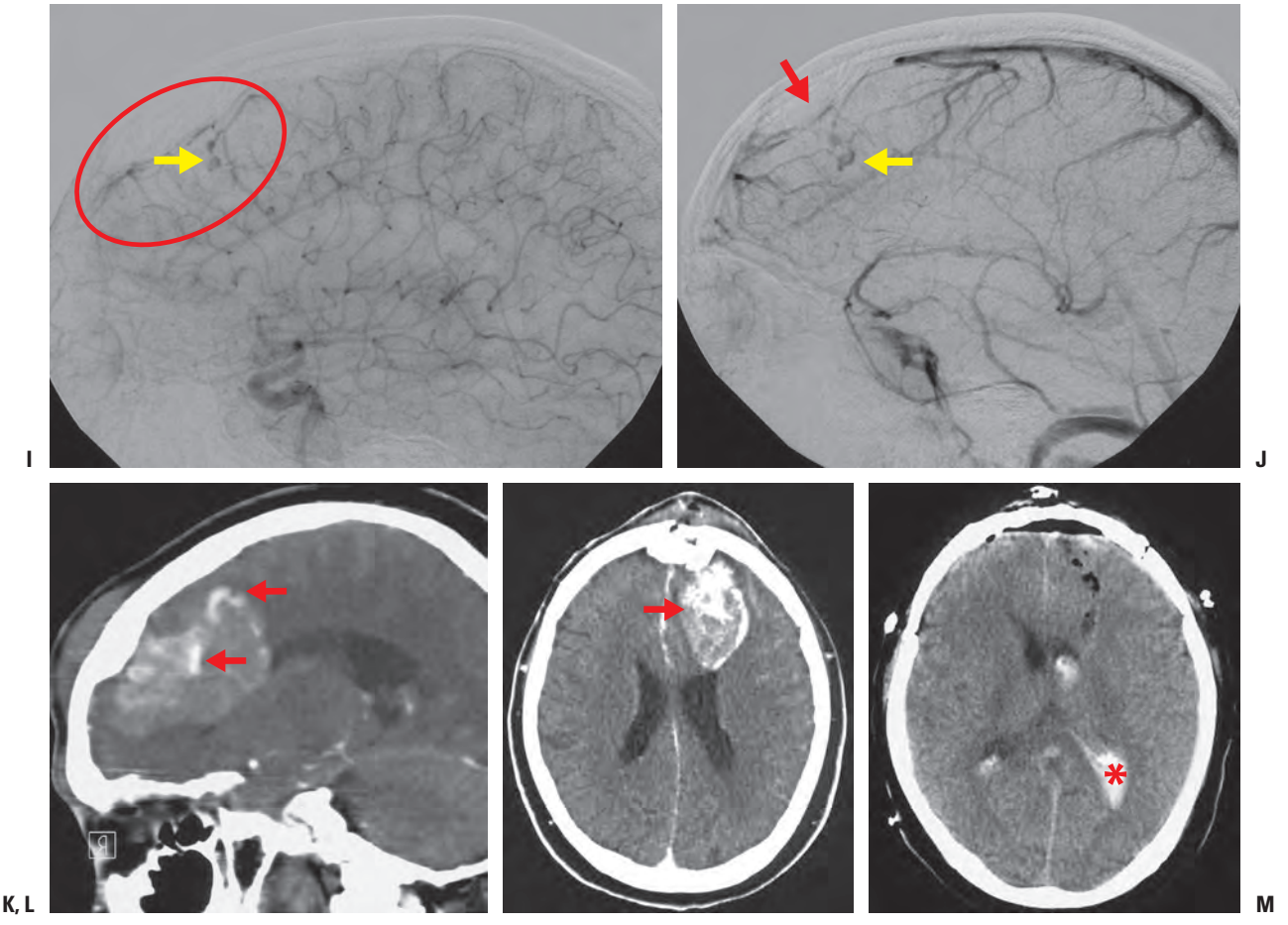


Figure 4.45. (*Continued*) **I.** Corresponding venous phase shows a patent, but compressed SSS (*red arrow*) and increased contrast extravasation (*yellow arrow*). **J.** Lateral view from a left internal carotid artery catheter angiogram performed 4 hours after injury shows abnormal contrast extravasation (*arrow*) at the site of injury (*circle*). **K.** Parasagittal and **(L)** axial images from a CT angiogram obtained immediately following the conventional catheter angiogram shows marked enlargement of the frontal hematoma with multifocal contrast extravasation (*arrows*). Compression of the anterior SSS likely contributes to the hemorrhage via venous hypertension. Note that findings at surgery confirmed compression of SSS, together with laceration of both the dura and a surface cortical draining vein, 5 mm lateral to the SSS. The injured veins were coagulated and recanalization of the SSS, enabled through elevation of the depressed fracture fragment, was confirmed via Doppler sonography prior to closure. **M.** Postoperative CT demonstrates interval evacuation of the intraparenchymal hematoma and presence of intraventricular hemorrhage (*asterisk*). (*Continued*)

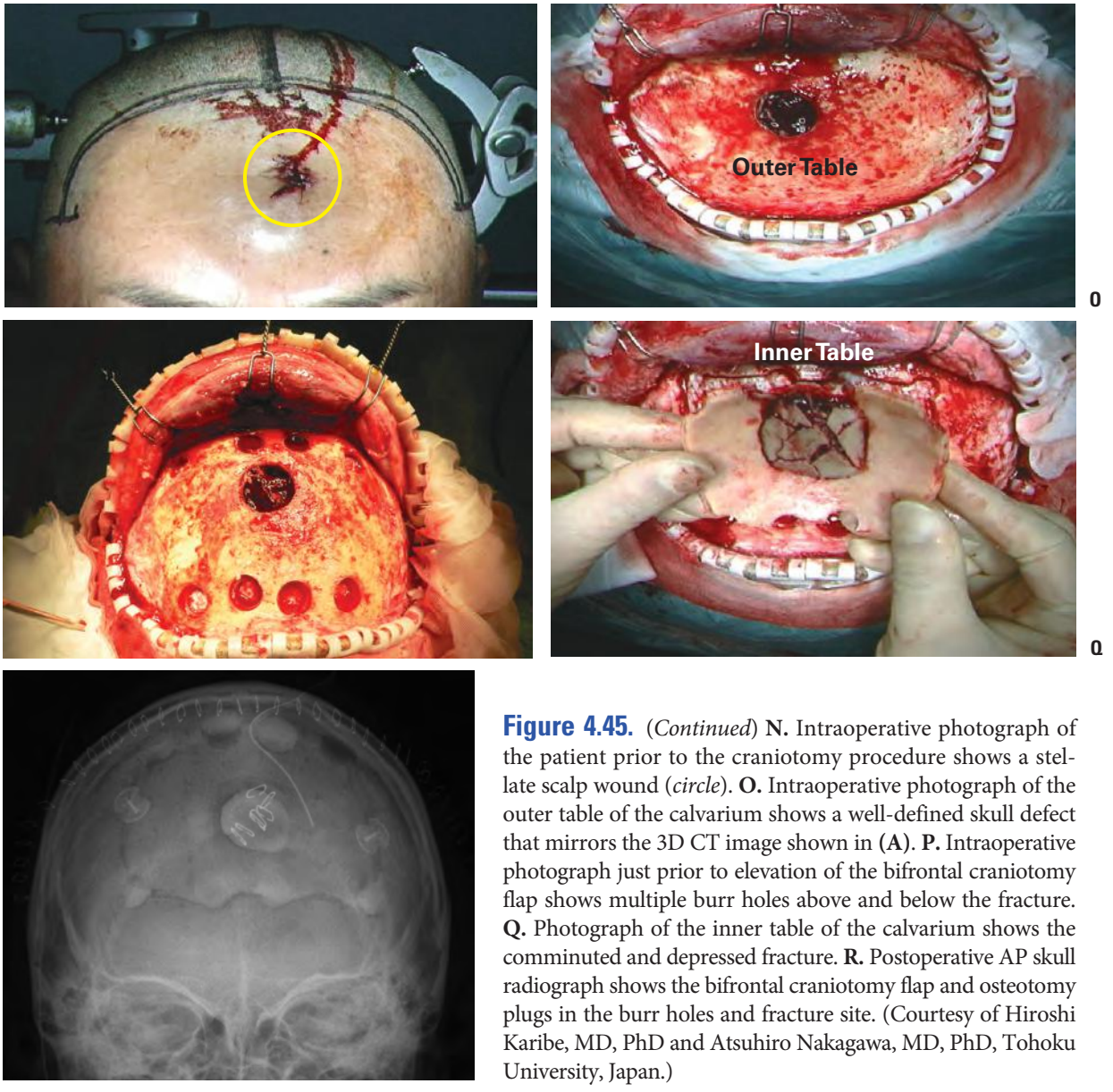


Figure 4.45. (*Continued*) **N.** Intraoperative photograph of the patient prior to the craniotomy procedure shows a stellate scalp wound (*circle*). **O.** Intraoperative photograph of the outer table of the calvarium shows a well-defined skull defect that mirrors the 3D CT image shown in **(A)**. **P.** Intraoperative photograph just prior to elevation of the bifrontal craniotomy flap shows multiple burr holes above and below the fracture. **Q.** Photograph of the inner table of the calvarium shows the comminuted and depressed fracture. **R.** Postoperative AP skull radiograph shows the bifrontal craniotomy flap and osteotomy plugs in the burr holes and fracture site. (Courtesy of Hiroshi Karibe, MD, PhD and Atsuhiro Nakagawa, MD, PhD, Tohoku University, Japan.)

★**KEY POINT** As in ballistic TBI, preferential involvement of the inner table relative to the outer table of the skull is seen in handheld penetrating trauma. This case also shows the role of CTA and cerebral angiography in predicting hematoma expansion through detection of active extravasation from vessels. In addition, a fracture that compresses or occludes a dural venous sinus can cause venous hypertension, thereby increasing the risk for intracranial hemorrhage.

Nonballistic Penetrating TBI (Phillips Screwdriver)

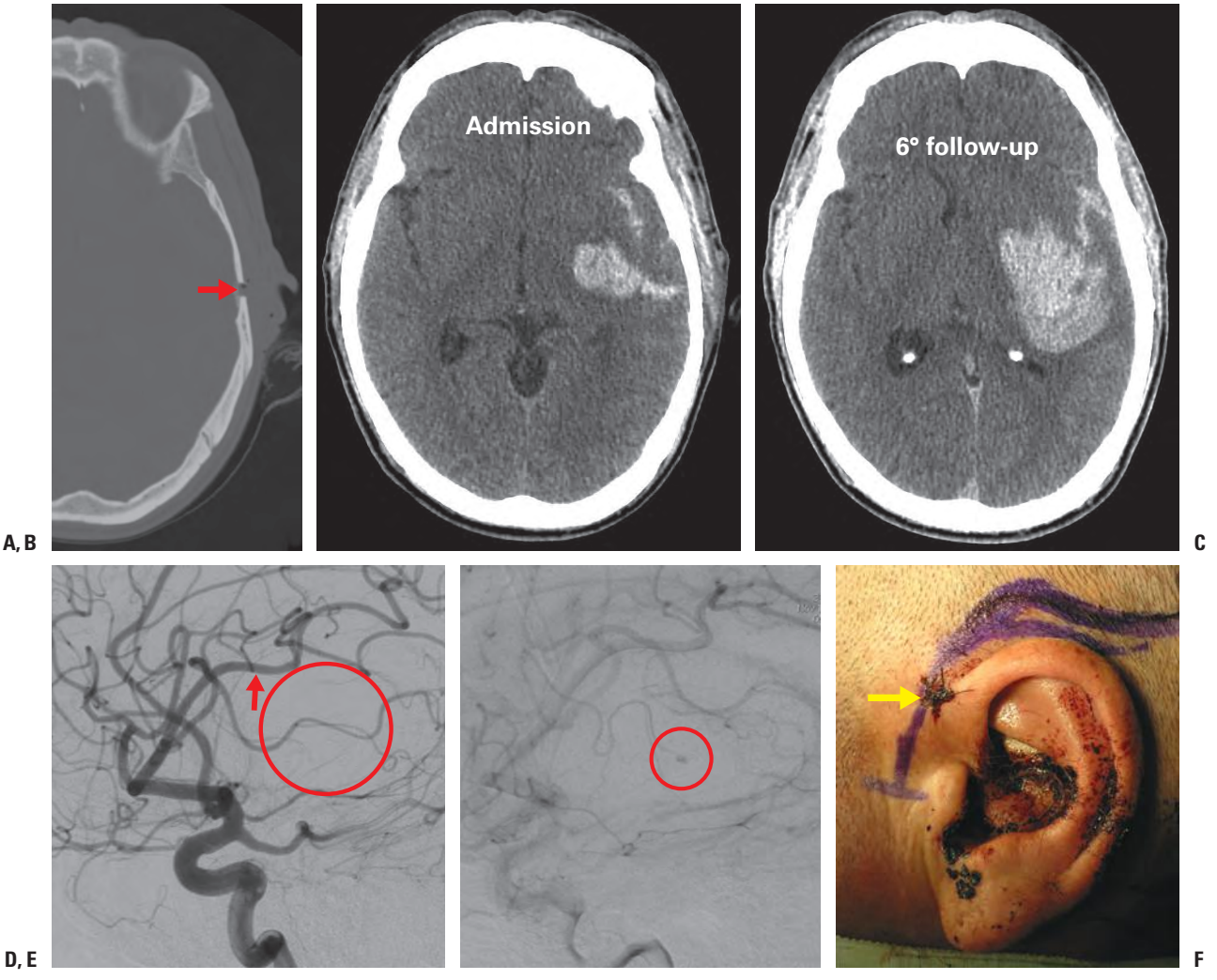


Figure 4.46. **Penetrating TBI (Screwdriver). A.** Admission CT bone window image shows a very focal defect in the skull at the entry site (*arrow*). **B.** A left temporal hemorrhage with a small amount of subarachnoid blood located at the anterior sylvian fissure is evident on the brain window image. **C.** Six hour follow-up CT scan performed after the patient developed worsening neurologic signs shows an increase in the intra-axial hemorrhage that necessitated neurosurgical evacuation. **D.** Subtracted lateral view from the arterial phase of the preoperative catheter internal carotid artery angiogram shows no definite vascular injury. There is mild superior displacement of the sylvian triangle (*arrow*) by an area of hypovascularity (*circle*), secondary to the left temporal hematoma. **E.** The venous phase of the angiogram shows a small focus of abnormal contrast extravasation (*circle*), corresponding to the wound track of the screwdriver. **F.** Preoperative photograph of the patient demonstrates a tiny cutaneous entrance wound anterior to the ear (*arrow*). Note the outline for the anticipated craniotomy drawn on the skin in purple. (Courtesy of Mathieu Laroche, MD, McGill University). (*Continued*)

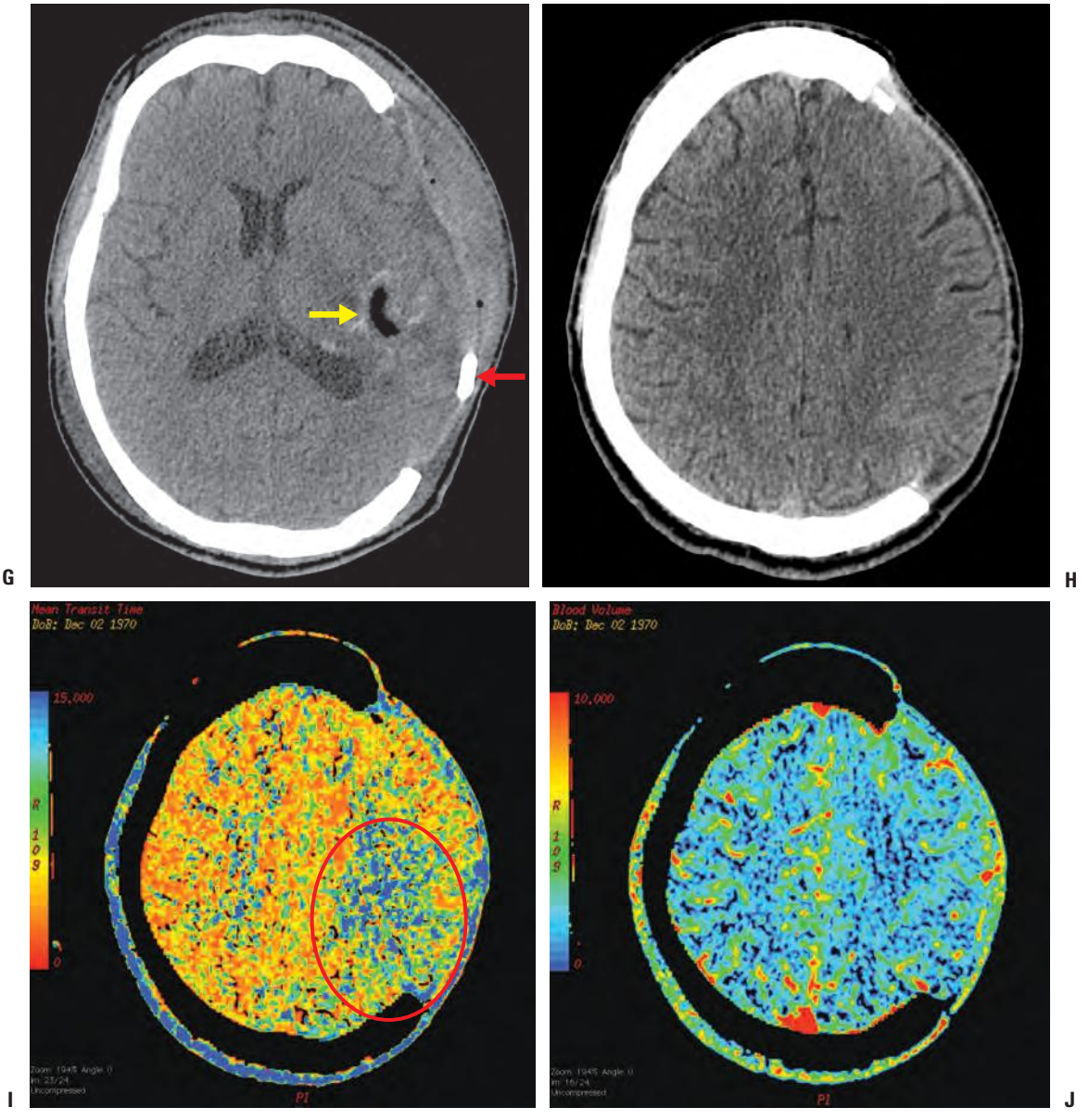


Figure 4.46. (*Continued*) **G.** Follow-up noncontrast CT scan 4 days later demonstrates an interval left holohemispheric decompressive hemicraniectomy and a Jackson-Pratt drainage catheter within the extra-axial/myocutaneous space (*red arrow*). Note the comma-shaped area of low attenuation in the operative bed that represents a piece of hemostatic Gelfoam (not air!) placed during surgery (*yellow arrow*). **H.** Two-month follow-up noncontrast CT image obtained above the level of injury shows no abnormality. **I.** Axial CTP image performed at the same time and at the same level shows abnormal prolongation of mean transit time (MTT) within the posterior left frontoparietal region (*circle*). **J.** Normal CTP cerebral blood volume (CBV) confirms that this area represents tissue at ischemic risk resulting from prior vascular injury. This finding can have important clinical management implications.

Nonballistic Penetrating TBI (Typical Knife Wound to the Head)

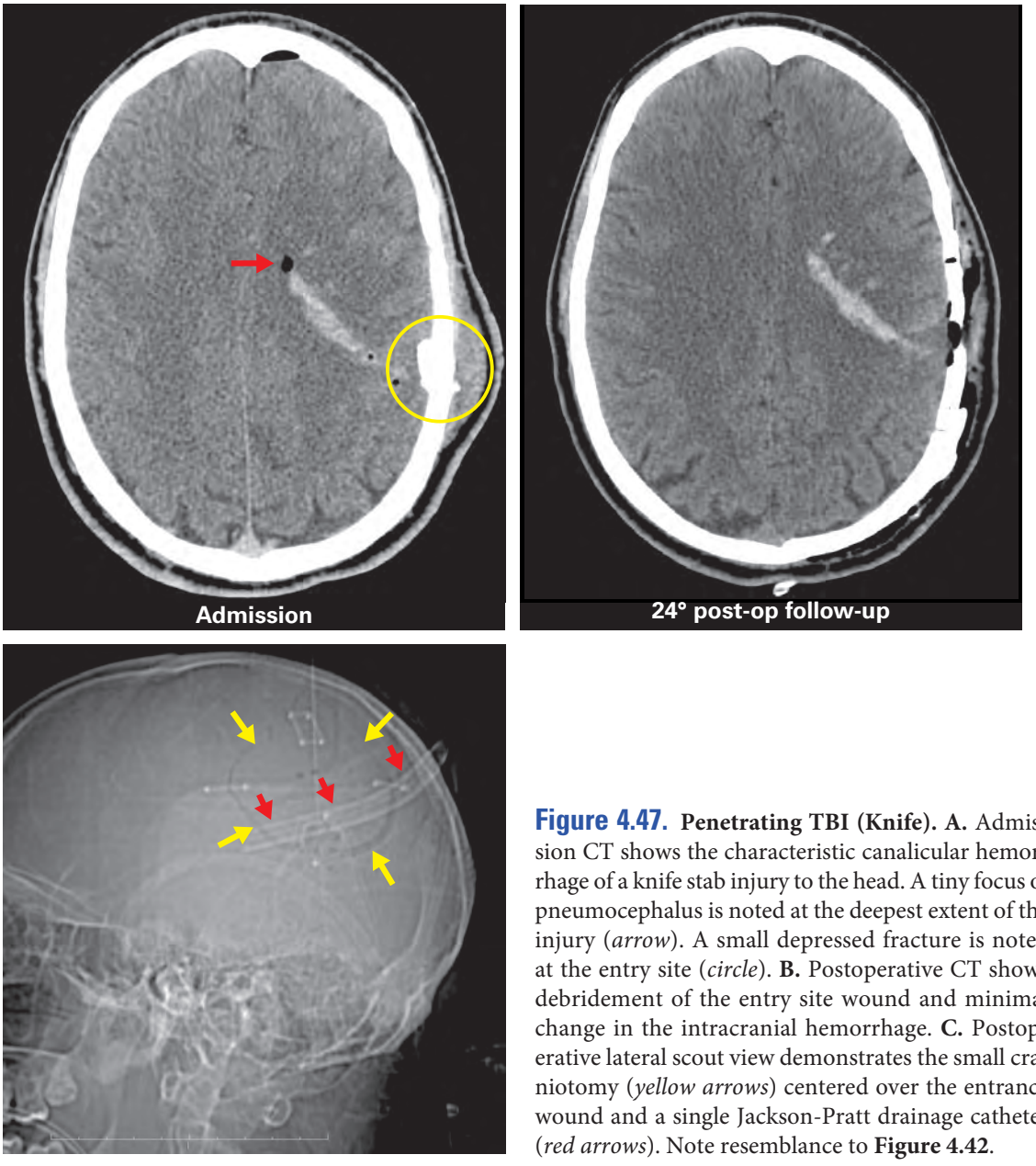


Figure 4.47. Penetrating TBI (Knife). A. Admission CT shows the characteristic canalicular hemorrhage of a knife stab injury to the head. A tiny focus of pneumocephalus is noted at the deepest extent of the injury (*arrow*). A small depressed fracture is noted at the entry site (*circle*). **B.** Postoperative CT shows debridement of the entry site wound and minimal change in the intracranial hemorrhage. **C.** Postoperative lateral scout view demonstrates the small craniotomy (*yellow arrows*) centered over the entrance wound and a single Jackson-Pratt drainage catheter (*red arrows*). Note resemblance to **Figure 4.42**.

★**KEY POINT** Due to the absence of temporary cavitation in handheld penetrating TBI, the injury typically appears as a canalicular hemorrhagic wound canal. Acutely, there may be minimal parenchymal bleeding because of arterial spasm.

Nonballistic Penetrating TBI (Surfboard to Head)

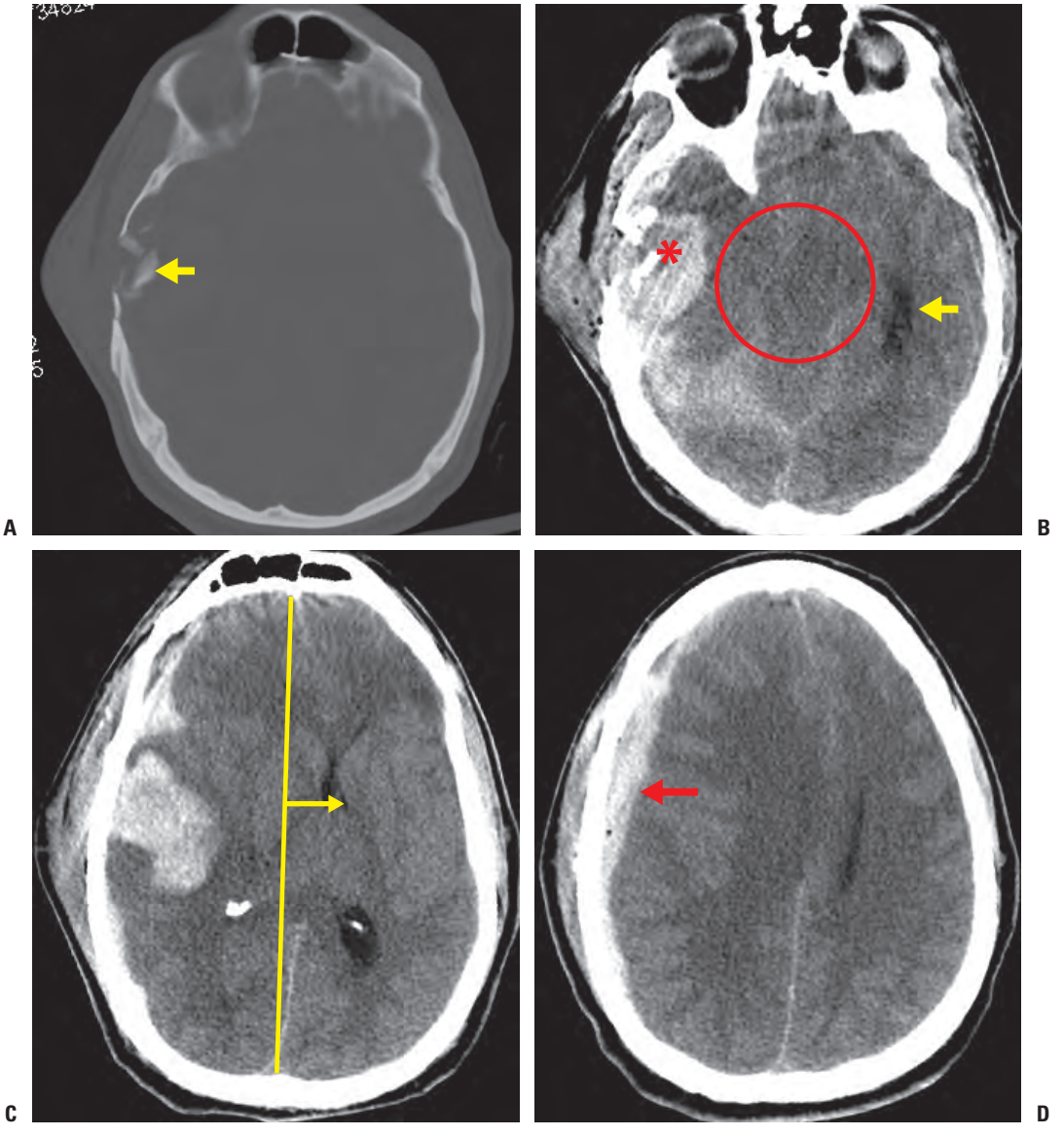


Figure 4.48. **Penetrating TBI with Post-Herniation Infarction and Duret Hemorrhage. A.** Admission CT shows a comminuted and depressed fracture of the right temporal squamosa (*arrow*). **B.** Admission noncontrast CT at the same level shows an underlying coup contusion (*asterisk*), subdural hemorrhage layering along the tentorium, and ominous effacement of the perimesencephalic cisterns (*circle*). Note trapping of the left temporal horn (*arrow*) due to aqueductal compression. **C.** Admission CT at the level of the basal ganglia shows significant midline shift (*arrow*) and subfalcine herniation. Together with the severe effacement of the basal cisterns seen in **(B)**, this imaging appearance is indicative of acute downward cerebral herniation. **D.** Admission CT at the level of the corona radiata shows a right frontal subdural hematoma (*arrow*) and effacement of the right lateral ventricle. (*Continued*)

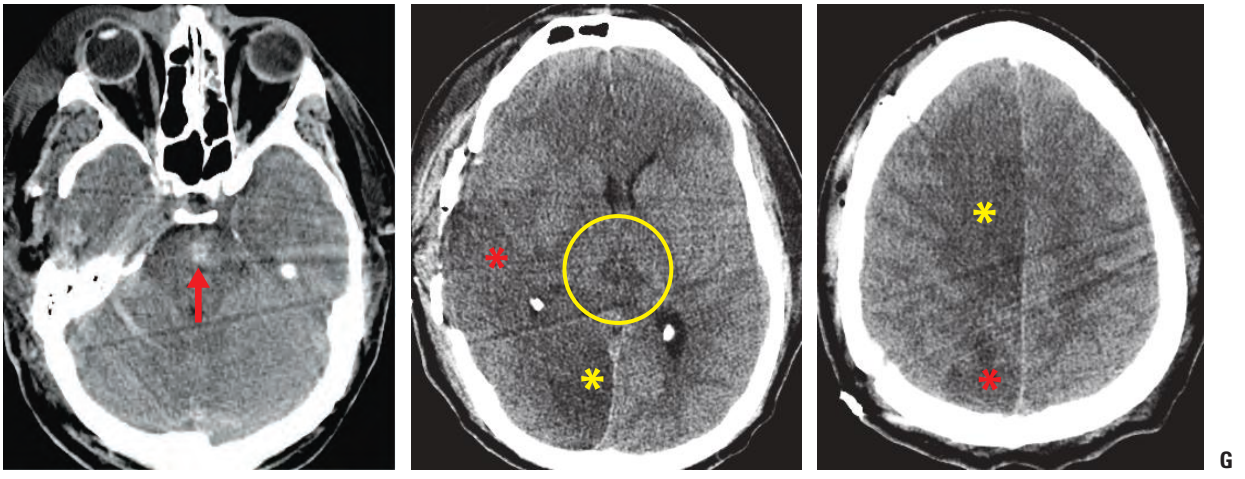


Figure 4.48. (*Continued*) **E.** Immediate postoperative CT shows a focus of high attenuation within the central pons (*arrow*) consistent with a Duret hemorrhage caused by the acute downward cerebral herniation evident in **(B)** and **(C)**. **F.** Postoperative CT obtained at the same level shown in **(C)** demonstrates interval post-herniation ischemic infarction of the right occipital lobe (*yellow asterisk*) and thalami (*circle*) due to PCA compression. Abnormal low attenuation is also noted within the territory of the right temporal lobe (*red asterisk*), which is due to one or more of the following mechanisms: vascular injury from the initial trauma, perihematoma vasogenic edema, and/or compressive ischemia from preoperative mass effect. **G.** Postoperative CT at the level of the centrum semiovale reveals additional post-herniation ischemic infarction in the territory of the right anterior cerebral artery (*yellow asterisk*), due to prior subfalcine herniation. Also, the right PCA infarct is noted to extend superiorly to this level (*red asterisk*).

★**KEY POINT** Even though the mechanism of injury in this penetrating TBI case is due solely to permanent cavitation (i.e., without superimposed temporary cavitation), the rapid development of mass effect from the intracranial hemorrhage in this young patient caused post-herniation ischemic infarction and a Duret hemorrhage.

Remote Nonballistic Penetrating TBI (Traumatic Pseudoaneurysm)

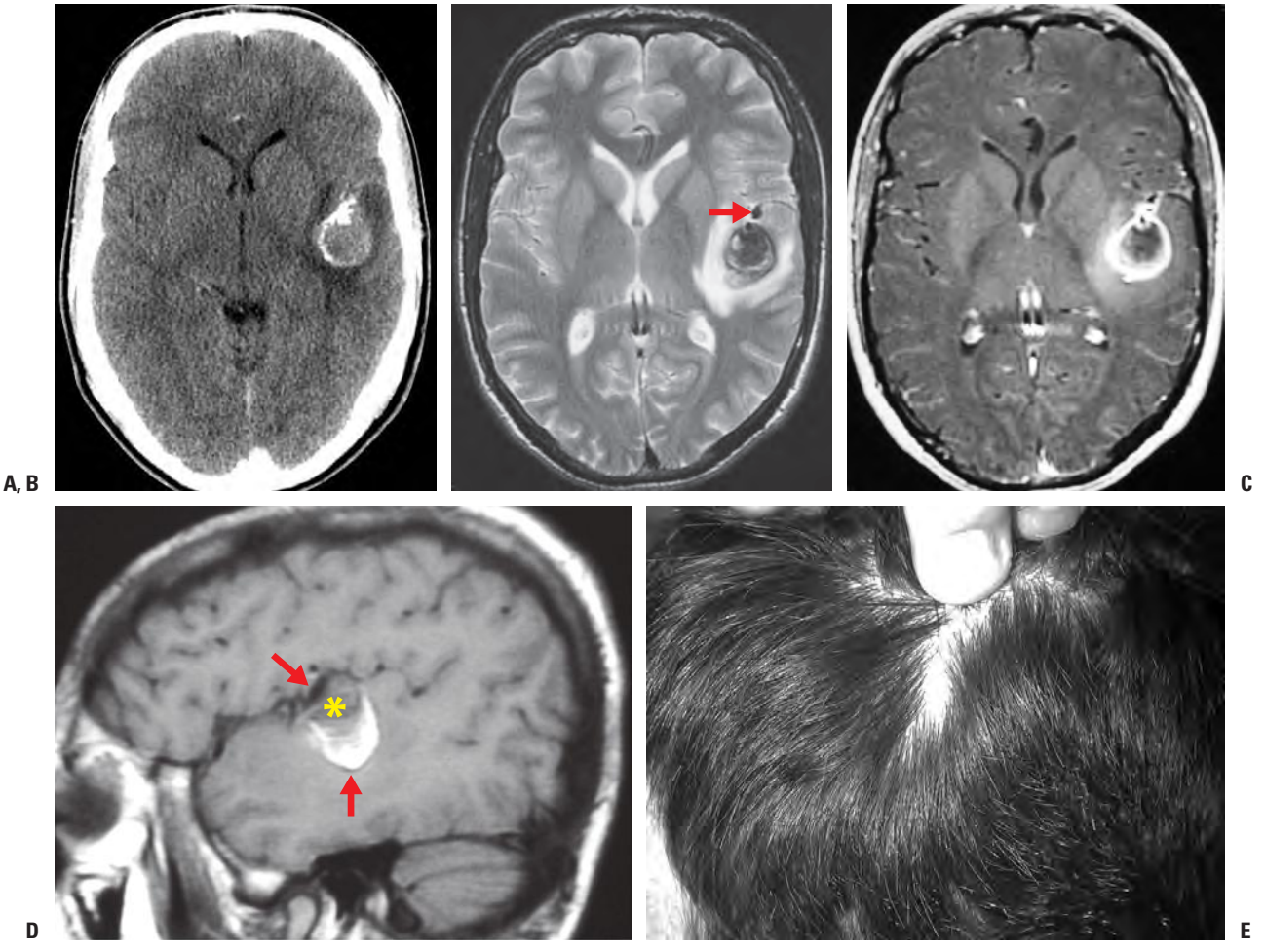
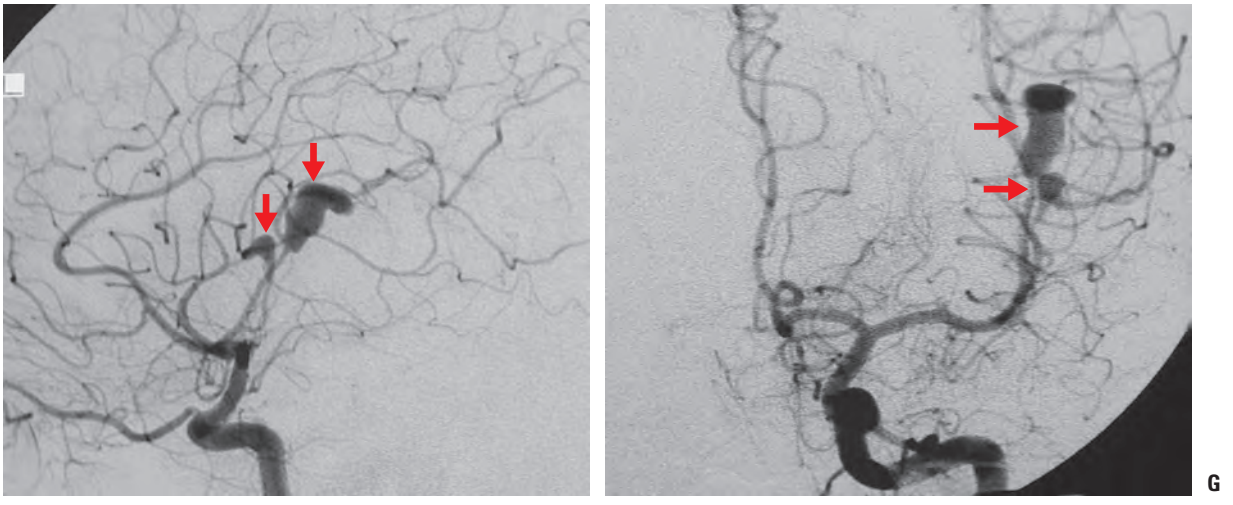


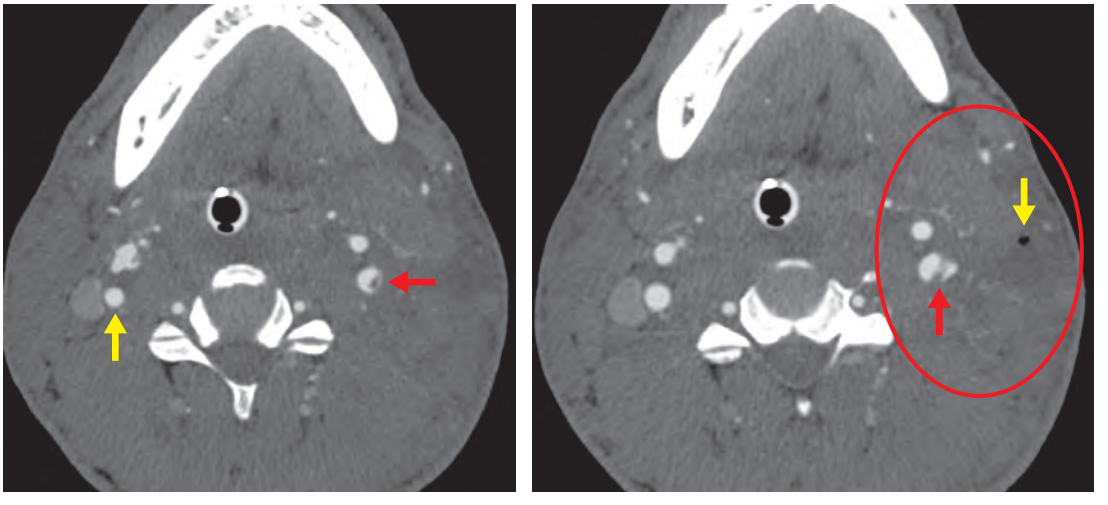
Figure 4.49. **Traumatic Pseudoaneurysm.** This 22-year-old presented with a seizure 14 years after having hit his head against the sharp corner of a table. **A.** Noncontrast axial CT shows a rounded, hyperdense, left temporal mass with surrounding vasogenic edema. Note partial peripheral rim calcification along the medial margin of the mass, consistent with a long-standing lesion. **B.** Axial T2-weighted MRI reveals a predominately hypointense lesion with surrounding hyperintense vasogenic edema. Note the abnormal flow void adjacent to the anterior aspect of the lesion (*arrow*), representing one of the pseudoaneurysms identified on the angiogram. **C.** Contrast-enhanced axial T1-weighted MRI shows peripheral enhancement of the lesion. **D.** Noncontrast parasagittal T1-weighted image shows a crescent of peripheral hyperintensity within the hematoma, consistent with subacute methemoglobin (*arrows*). The more superior component of the hematoma is isointense, consistent with acute deoxyhemoglobin (*asterisk*). Together, the imaging suggests a longstanding pseudoaneurysm that recently bled. The imaging appearance is also consistent with myxomatous aneurysms, but this was ruled out clinically. **E.** Photograph of the patient's scalp showing the focal alopecic scar overlying the region of the intracranial lesion. (*Continued*)



F G

Figure 4.49. (*Continued*) **F.** Lateral and **(G)** frontal views from a conventional catheter angiogram following injection of the left internal carotid artery show two adjacent pseudoaneurysms (*arrows*) arising from opercular branches of the left middle cerebral artery. Both lesions are irregular, without a definite neck, and contrast stagnation was noted on the late arterial and venous phases of the angiogram. (Courtesy of Jose Cohen, MD.)

Stab Wound to Neck (Embolic Infarction)



A B

Figure 4.50. **Stab Wound Injury to the Neck with Embolic Infarction. A, B.** Contiguous axial CTA images show an intraluminal filling defect within the proximal left internal carotid artery consistent with a luminal thrombus (*red arrows*). Compare with the normal right ICA (*yellow arrow*). Nonenhancing soft tissue swelling consistent with myofascial hemorrhage is also evident (*circled region*). A punctate focus of air (*blue arrow*) within the swollen soft tissues indicates the location of the wound tract. (*Continued*)

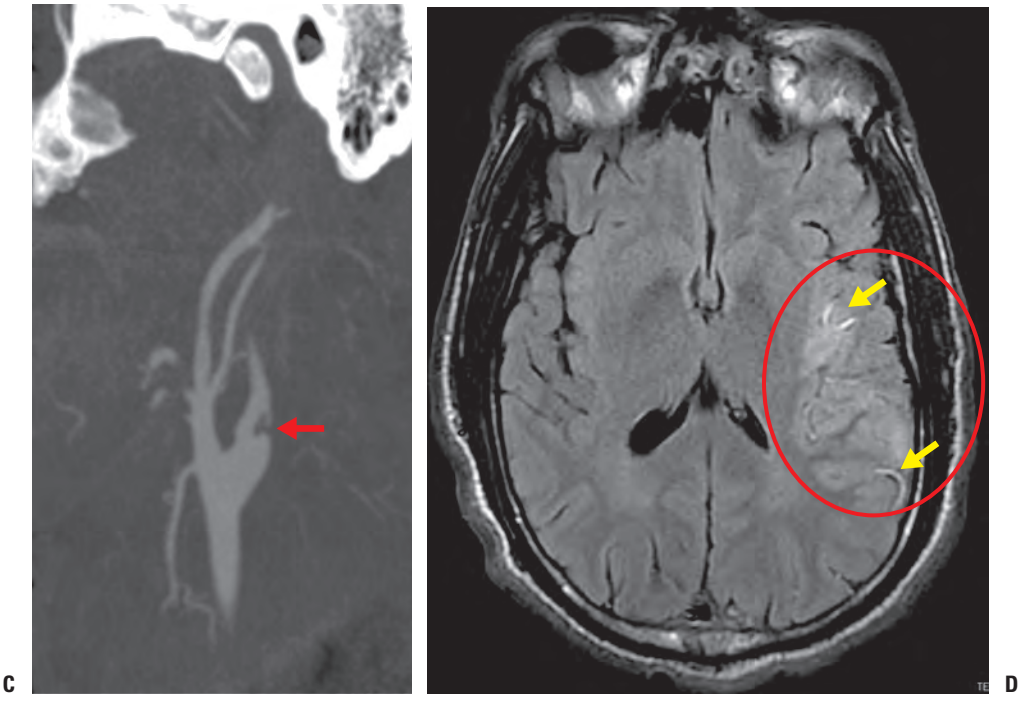


Figure 4.50. (*Continued*) **C.** Oblique parasagittal reformatted CTA image of the left cervical carotid artery demonstrates a pedunculated filling defect, consistent with an intraluminal thrombus (*arrow*). The thrombus appears to be attached to the posterolateral wall of the ICA. **D.** Axial FLAIR image of the brain shows mild swelling, as evidenced by subtle sulcal effacement of the sulci and hyperintensity of the left temporal cortex, consistent with an acute left middle cerebral artery infarction (*circled region*). Note abnormal hyperintensity within several MCA tertiary vessels (*arrows*), consistent with either slow flow, retrograde collateralization, or, less likely, thrombus.

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CHAPTER 5

How Does Combat TBI Differ from Civilian TBI? 12 Lessons

LESSON 1: MILITARY PATIENTS ARE MORE HOMOGENEOUS THAN CIVILIAN PATIENTS

Combat patients are typically young, extremely healthy, motivated men. Their youth and excellent pre-injury health, together with their drive to recover, so that they can return to help their fellow soldiers, lead to a more favorable prognosis for combat victims. One of the interesting explanations for improved outcomes for military patients is the upregulation of trophic factors, such as brain-derived neurotrophic factor (BDNF), that occurs in physically fit individuals who suffer traumatic brain injury (TBI).[1] One may then contrast the combat victim with the endangered population in the civilian milieu during wartime—the injured include women, children, and seniors—who may suffer from other medical comorbidities and low physiologic reserve. In addition, civilian trauma patients do not have the benefit of protective body armor during the injury. Therefore, data from the large, relatively homogeneous military population with the potential for long-term follow-up may not be directly applicable to the mass casualty experience of future terrorist attacks and natural disasters at home. However, some data in preparation for these civilian scenarios may be collected from over 25% of U.S. troops in the National Guard and Reserve who are older than active-duty soldiers and have the expected age-related medical comorbidities. In addition, privately hired contractors in the war zone are another heterogeneous group, typical of a civilian trauma population, that can be used to study injuries in terrorist and disaster situations. Finally, the number of women in the military has doubled in the past decade. Women now comprise about 13% of Americans serving in uniform, which translates to more than 255,000 women having served in Iraq and Afghanistan. Under current U.S. Army rules, women are not officially assigned to units whose primary mission is direct

TABLE 5.1 Differences between Combat and Civilian Traumatic Brain Injury (TBI)

Austerity versus Aristocracy
1. Military combatants are a more homogenous subset of patients than civilian patients.
2. Patient triage and transport are different.
3. The imaging approach is different, with low thresholds for CTA or cerebral angiography to rule out vascular injury and limitations on magnetic resonance imaging (MRI) due to retained metallic foreign bodies.
4. Blast-related trauma is the most common mechanism of injury. As a result, *penetrating* injuries are more common.
5. Polytrauma (*extreme radiology*) is more common.
6. Life-threatening hemorrhage is more common.
7. Hyperthermia and burns are common in combat injury but rare in civilian injury.
8. Assessment of the full extent of traumatic brain injury (TBI) in the acute setting is difficult.
9. Facial injuries are more common and complex.
10. Stroke and cerebrovascular injuries are more common.
11. Secondary TBI is a frequent complicating feature.
12. Post-traumatic stress disorder (PTSD) is more common.

CTA, computed tomography angiography.

combat on the ground but can be assigned to other roles in combat zones.

LESSON 2: PATIENT TRIAGE AND TRANSPORT ARE DIFFERENT

Urban civilian trauma care typically involves emergency surgery with definitive/reconstructive surgery *at a single site and usually at a single point in time*. In contrast, because of the "remote" location of battlefield injury, delayed evacuation from a hostile, often hot and dirty environment is the rule. To deal with complex medical evacuation (MEDEVAC), five levels (also known as *roles*, *echelons*, and *stages*) of patient care have been implemented. Precise protocols for all aspects of care and operations are in place at each of the five levels (**Fig. 5.1**).[2,3] The actual practice of medicine on the battlefield is similar to trauma systems around the United States. What is unique, however, is the fact that this care can be rapidly deployed and sustained in the most austere and remote locations on the earth and can be conducted under extremely chaotic and hazardous conditions. Studies of TBI in austere environments have shown rates of death that are two to three times as high as those in environments where advanced care is available.[4]

In brief, **Level 1** care is performed by the field combat medic, who establishes emergency stabilization of the soldier at the moment of injury. This level is also termed the *Battalion Aid Station*. There is no surgery or patient holding capacity at this level, and the casualty is triaged to either return to duty or evacuated to the next level. **Level 2** involves the Forward Surgical Team (FST), which is a 100% mobile (usually six Humvees with trailers) surgical team that remains in close proximity to the front-line infantry and provides emergency damage control surgery (DCS). It typically consists of 20 personnel and two operating tables. The FST carries a small ultrasound machine and sometimes a portable X-ray machine. The FST has a 1.5-hour set-up and an 8-hour evacuation time (**Fig. 5.2**). The mainstay of initial surgical care provided at this level are completion amputations, delayed primary closures, and local wound care for extremity injuries when revascularization is not a viable option. During this early phase, debridement and skeletal fixation are essential components in preparation for later major reconstructive procedures. Once stabilized by the trauma team at the Level 2 facility, the patient is transferred for computed tomography (CT) imaging and neurosurgical care at the next level. MEDEVAC travel time can take 15 to 45 minutes. In the setting of a severe TBI, speed is critical to minimize the time between injury and access to a CT scan and neurosurgical treatment.

Level 3 in the continuum of care is the combat surgical hospital (CSH), located in the larger cities (Balad, Baghdad, and Kabul) (**Fig. 5.3**). It is the highest level of medical care available within the combat zone, and it is the first location where both CT and neurosurgery are represented. The CSH continues the *full court press* approach to patient care and performs minimal yet optimal surgical intervention. It consists of about 250 beds and 550 personnel. Infectious disease, neurology, neurosurgery, ophthalmology, head and neck surgery, pathology, and renal subspecialty services are available. These facilities are similar to a moderate-sized community hospital in the United States with emphasis on trauma management. Digital X-ray, CT, fluoroscopy, ultrasound, and interventional radiology are usually present at the CSH. Casualties who are not able to return to their units within 1 week are aeromedically evacuated via a veritable *flying ICU*, a C-17 cargo plane transformed into a critical care air transport (C-CAT) plane (**Fig. 5.5A–C**), to **Level 4** of care: the European Command hospital in Landstuhl, Germany. Landstuhl Regional Medical Center (LRMC) is the largest North Atlantic Treaty Organization (NATO)–controlled air base in Europe. It is the first stop for *all* American casualties leaving the conflict in Iraq and Afghanistan. LRMC is electronically linked to the battlefield so that images and medical records of the wounded can be accessed prior to arrival (**Fig. 5.4**). On arrival to LRMC, physicians continue to treat and triage the wounded. Because of the daily influx of wounded troops, patient turnover is by design (and necessity) remarkably quick, with an average hospital stay of 3 days.

Once stabilized at LRMC, the injured soldier is again transferred via C-CAT to the Continental United States (CONUS in military parlance) for definitive care. This represents **Level 5** of care. Remarkably, the C-CAT intensive care unit (ICU) care team has had zero mortality in transporting casualties from the battlefield to LRMC and from LRMC to the United States. The vast majority of troops are air evacuated to Washington, DC Severely burned soldiers are sent to Brooke Army Medical Center (BAMC). Advances in computer technology have enabled weekly video-teleconference, a highly effective communication system. During this approximately 2-hour video-teleconference,

treating physicians from Iraq, Afghanistan, LRMC, Walter Reed National Military Medical Center, and BAMC communicate online (see **Fig. 5.5F**). The multisite physicians discuss interval surgery, medical treatment, patient complications, and outcome. It is a remarkable opportunity for the treating physicians at the different levels of care to maintain continuity of care and obtain patient follow-up.

The carefully choreographed continuum of care developed by the military is, indeed, a medical marvel. It has resulted in an unprecedented survival of injured soldiers from even the most severe of penetrating injuries. It has decreased morbidity and provided important insights into the natural history of combat wounds. However, increased survival by its nature sets up a prolonged injury course with risks of secondary injury, secondary insults, and complications to the wounded brain. Precarious physiologic issues come into play when sick patients are taken into flight for 8 to 16 hours. Unlike civilian trauma, casualties from war are managed by numerous different physicians at the varying levels of medical care, in and out of the combat zone. From the time of injury to their return to the United States, injured soldiers pass through as many as five treatment facilities, spending only a few days at each facility. Nevertheless, this well-oiled military medical machine provides optimal care of the injured brain with minimal risk of secondary damage and complications.

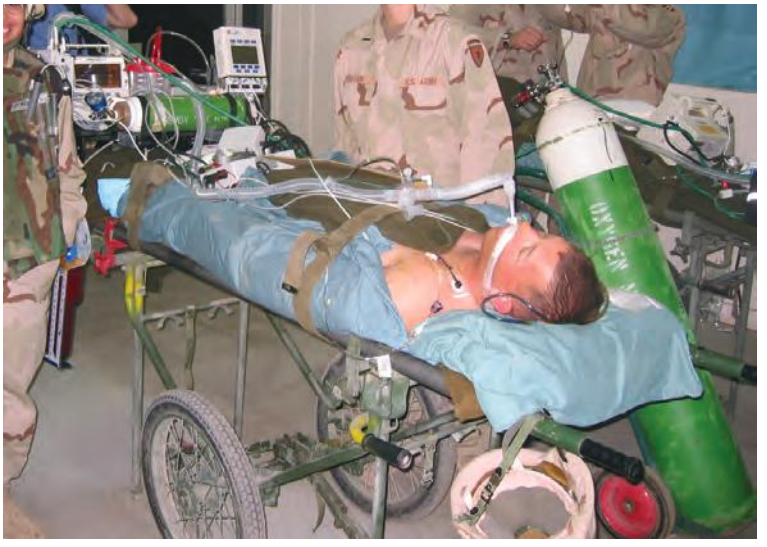
Figure 5.1. Combat Casualty Transport. MEDEVAC of casualties follows a carefully choreographed transport system. See previous section's explanatory text.

Level 2: Forward Surgical Team (FST)

A

B

Figure 5.2. **Forward Surgical Team. A.** Level 2 consists of a 100% mobile surgical team that remains in close proximity to the frontline infantry and provides emergency "damage control" surgery. It typically consists of 20 personnel and two operating tables. It has a 1.5-hour set-up and an 8-hour evacuation time. **B.** Portable radiography is the only imaging technology available to the FST. (*Continued*)



C

D

Figure 5.2. (*Continued*) **C.** Photograph of a wounded soldier on a wheeled NATO *litter*. The litter is a lightweight canvas stretcher that serves as the main mode of patient transport from one level to the next. It is used to evacuate the wounded from the time of injury on the battlefield until they arrive stateside. **D.** Photograph of a litter positioned on a hospital gurney at Level 4. As a civilian, I never realized how crucial this seemingly primitive device is to patient transport.

★**KEY POINT** Transport of the injured soldier occurs more often than transport of the civilian trauma patient due to the multiple levels of military medical care. The transport of trauma patients is associated with an increased risk of secondary brain injury.

Level 3: Combat Support Hospital (CSH)

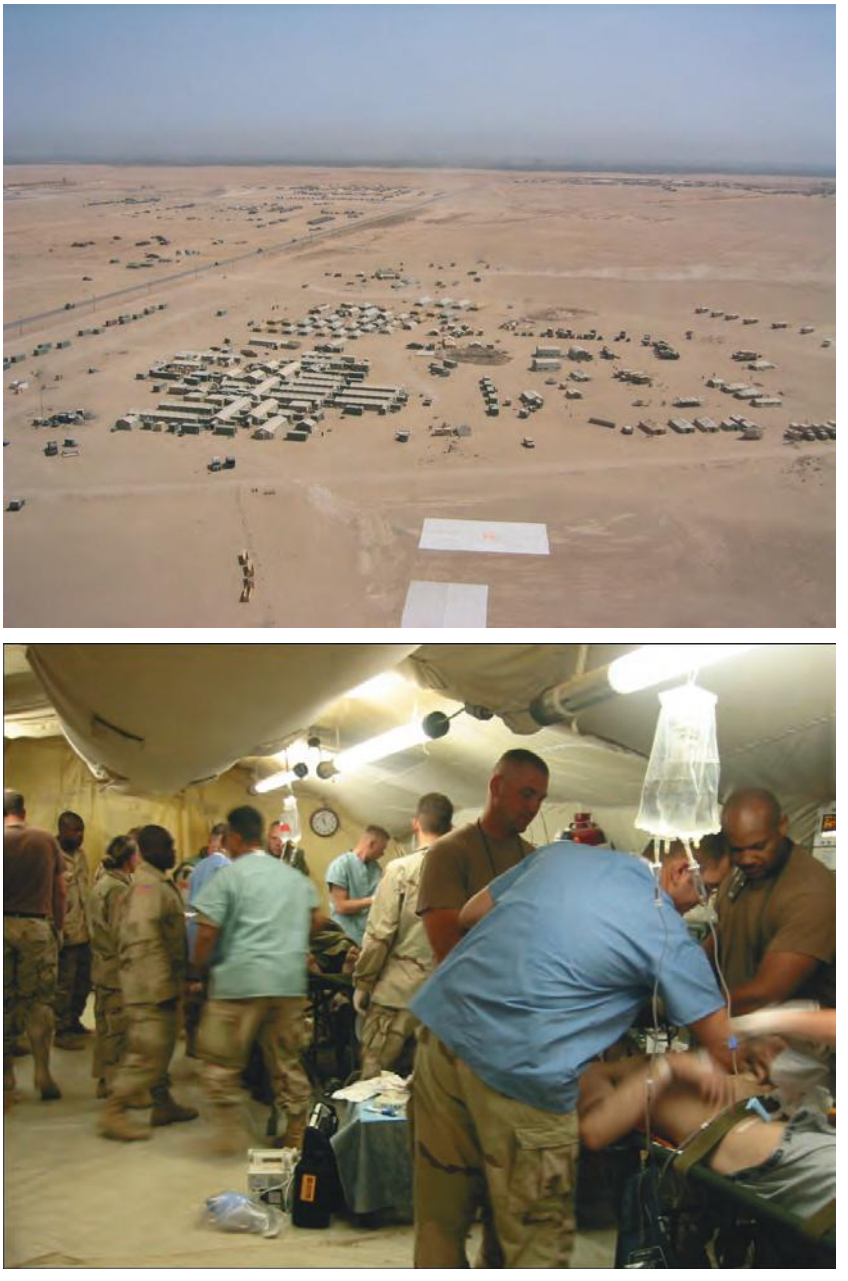


Figure 5.3. Level 3: **Combat Support Hospital. A.** As can be appreciated from this photograph, heat and dust associated with the Baghdad CSH is environmentally unfriendly to advanced neuroimaging. Nevertheless, digital X-ray, CT, fluoroscopy, interventional radiology, and ultrasound are usually present at the CSH. Magnetic resonance imaging (MRI) became available in Afghanistan in 2011. The CSH consists of about 250 beds and 550 personnel. These facilities are similar to a moderate-sized community hospital in the United States with emphasis on trauma management. Casualties who are not able to return to their units within 1 week are aeromedically evacuated to Level 4 care. **B.** Receipt and recovery area. (*Continued*)

C

D

Figure 5.3. (*Continued*) **C.** The physician wearing the orange cap is the mass casualty "MASCAL" chief who directs the controlled chaos and decides who goes to radiology or the operating room and in what order. Note the omnipresent transport litter (*arrows*). **D.** Combat casualty within the CT scanner. (*Continued*)

E

F

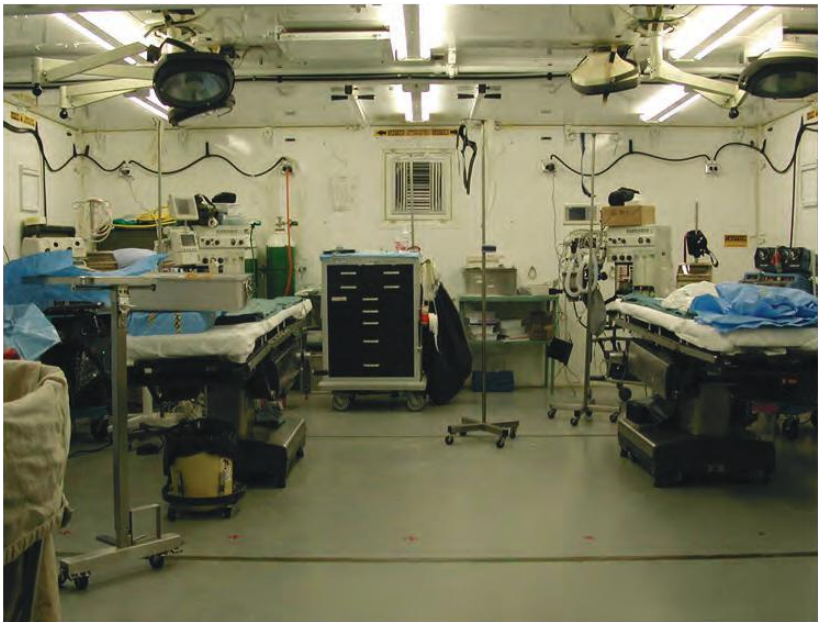


Figure 5.3. (*Continued*) **E, F.** Inside the CSH operating rooms. The CSH operating rooms provide minimal yet optimal surgical intervention. (*Continued*)

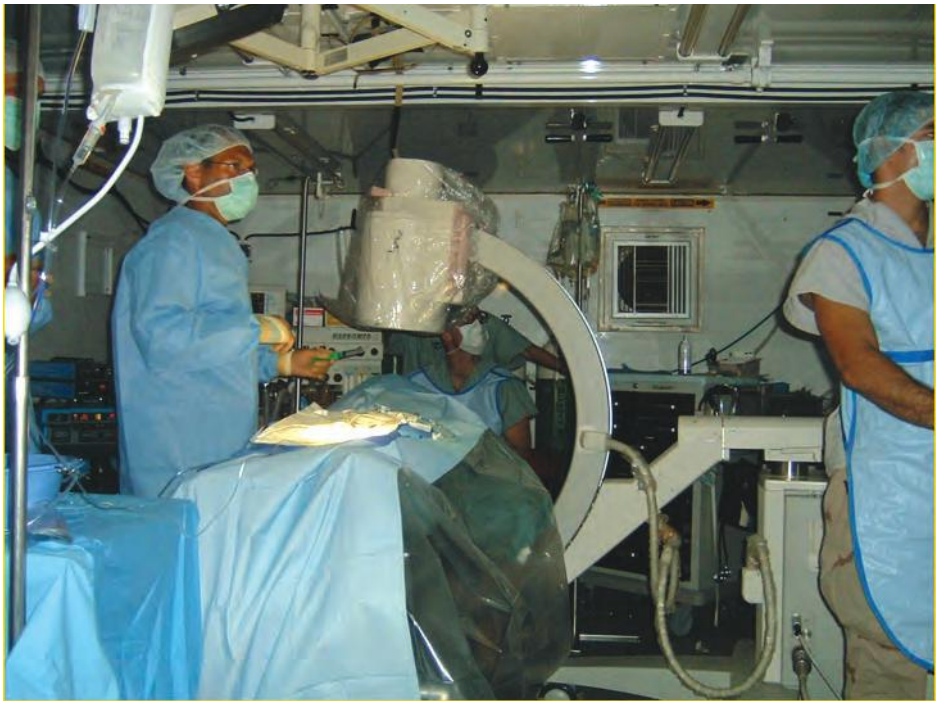
G 

Figure 5.3. (*Continued*) **G.** Dr. Rocco Armonda performs cerebral catheter angiography with C-arm assistance.

★**KEY POINT** The CSH continues the full court press approach to patient care. The CSH is characterized by a multidisciplinary team with several subspecialists treating the same patient simultaneously under the direction of one trauma surgeon. It is the highest level of medical care available within the combat zone.

Combat Casualty Communication

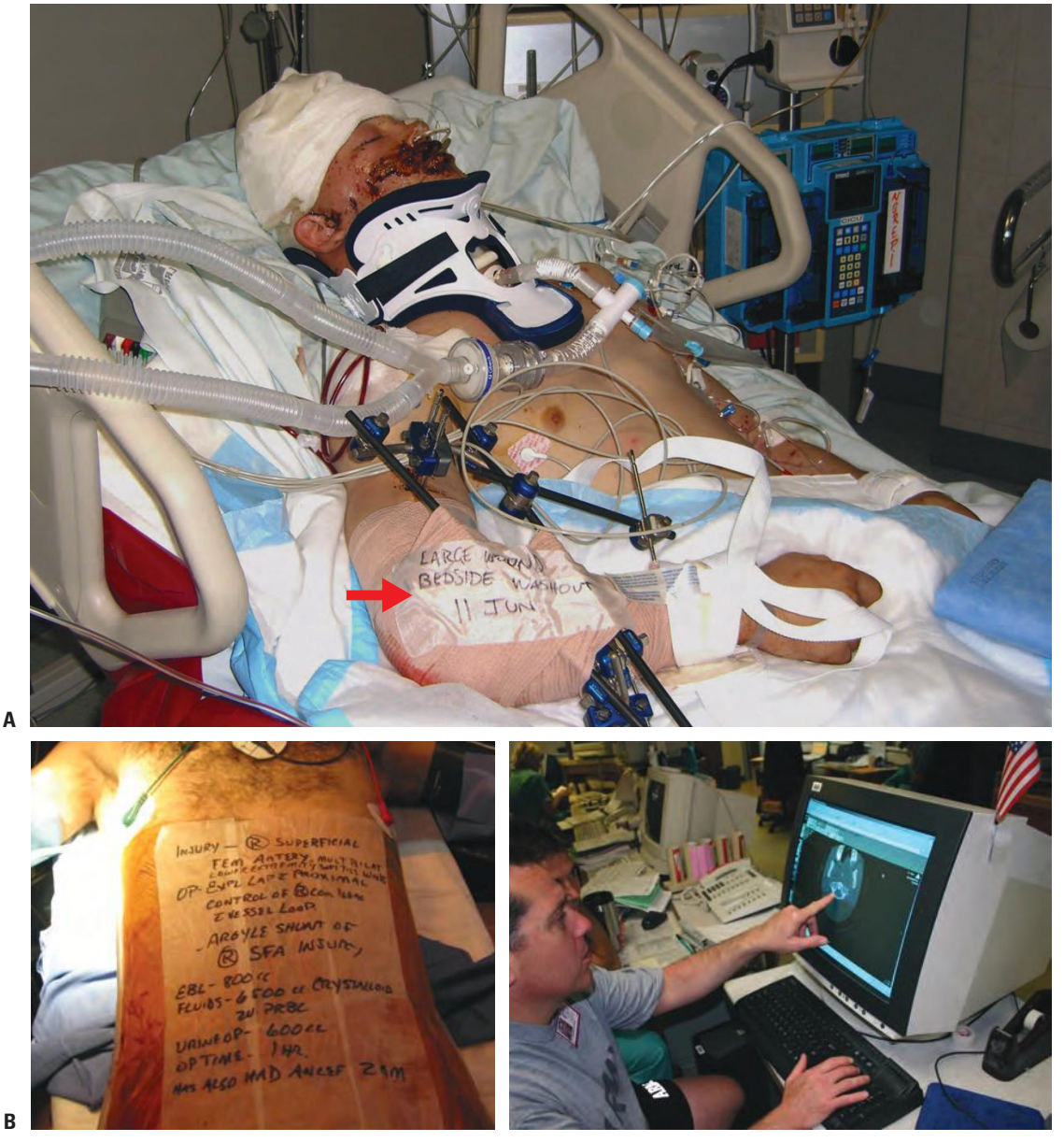


Figure 5.4. **Interfacility Combat Casualty Communication. A.** Photograph of an LRMC polytrauma patient showing external fixation of a right humeral fracture, facial burns, cervical spine trauma, tracheostomy, and TBI. Note the information handwritten directly on his extremity dressing (*arrow*). **B.** Photograph of a soldier's torso immediately following FST surgery and prior to transport to the CSH. Note the handwritten information on the anterior abdominal wall bandage that briefly describes the injury and treatment. **C.** Teleradiology from the battlefield. Images of combat casualties are electronically transferred in advance of the patient's arrival to LRMC, Germany.

★**KEY POINT** Owing to frequent rapid transport from one location to another, casualties often have details of their recent medical care handwritten directly on them. Patients are sent by MEDEVAC from the FST to the CSH and then flown from the CSH to LRMC and later from LRMC to the United States with the help of flight surgeons on the C-CAT team.

Transport from Level 3 to Level 4

Figure 5.5. Level 4: **Landstuhl Regional Medical Center.** Soldiers who are unable to return to their units within 1 week of injury are air evacuated via C-CAT to Level 4 of care in Germany. **A.** Inside the C-CAT showing triple-stacked litters carrying wounded soldiers. **B.** C-CAT flight surgeon patch. **C.** Critically injured soldier on transport litter (*red arrow*) in C-CAT. Note the extensive life support equipment (*arrow*) that accompanies severely wounded soldiers during their flight. This particular soldier suffered polytrauma and was severely burned, hence the protective "packaging" of the patient. Only a very small portion of his face is exposed (*yellow arrow*). *(Continued)*

D

E

F

Figure 5.5. *(Continued)* **D.** Transport bus from Ramstein Air Force Base delivering the wounded soldiers to LRMC emergency entrance. **E.** Transporting the soldier to LRMC ICU. Note extensive portable monitoring equipment. **F.** A 2008 screenshot of a weekly video-teleconference that occurred between LRMC, Walter Reed Army Medical Center (WRAMC), Wilford Hall Medical Center (WHMC), Brooke Army Medical Center (BAMC), and Bethesda. This unique conference allows military physicians at multiple sites to discuss the continuum of care from the time of the soldier's injury on the battlefield to his or her final location in the United States.

LESSON 3: THE IMAGING APPROACH IS DIFFERENT IN WAR AND TERRORISM

The intensity of war is high, and the operating environment can be extremely difficult, from both a combat operations perspective and an imaging perspective. Medical imaging in the combat zone is different from civilian trauma in several ways (**Table 5.2**). The primary purpose of acute imaging in a war zone is to determine whether immediate damage control surgery is necessary. The equipment must be rugged, reliable, and preferably portable. There is limited power in the war zone and equipment service and support can be problematic. Yet, based on an explosive mechanism of injury, with risk of severe injuries of every body part, a liberal approach to imaging is often warranted. Strong consideration is given to whole-body CT scanning for any individual with multiple shrapnel wounds.[5] Several recent studies of whole-body CT/computed tomography angiography (CTA) in civilian trauma suggest that this comprehensive imaging protocol could play a role on the battlefield as part of a broad screening protocol to promptly identify all tissue and vascular injuries.[6,7] Because of the complex polytraumatic nature of the injuries with concomitant difficulties in patient transport, the battlefield may be the perfect place for a portable CT scanner such as the CereTom; that is, bring the imaging to the patient and not the reverse (**Fig. 5.6**). There are several imaging options for evaluating neurotrauma suffered in war and terrorism (**Table 5.3**).

TABLE 5.2 Differences between Combat and Civilian Imaging

1. *Total-body imaging* is routine on the battlefield because polytrauma is common in combat injury.
2. *Plain radiographs are obtained more often* because of the large number of extremity injuries and because of *the higher incidence of foreign bodies* in combat injuries that are well visualized on plain films. Note: Plain films are also often indicated in civilian penetrating trauma.
3. The *evolving nature* of combat lesions warrants *more frequent follow-up* imaging.
4. The combination of total-body scans with multiple follow-up imaging studies *increases the radiation* exposure to this young patient population.
5. Polytrauma and the severity of the injuries make patient *transport to imaging more hazardous*.
6. *MRI is rarely performed in the combat zone* and must be performed judiciously because of the frequent contamination of the wounds by ferromagnetic foreign bodies.
7. *Catheter angiography is performed more frequently* largely because there is an overall increase in neurovascular injury in combat, relative to civilian, TBI. In addition, less invasive CTA studies may be inadequate as imaging artifacts from fragments and debris can obscure subtle vascular anatomy.
8. Electrical power sources and equipment service and support are less available in the austere combat environment.

CTA, computed tomography angiography; MRI, magnetic resonance imaging; TBI, traumatic brain injury.

Mobile CT Imaging

Figure 5.6. Portable CT Imaging. A. Note the size of the CereTom portable CT scanner relative to a 6-foot man. **B.** Photograph showing the mobile CT located within the back of an ambulance (*arrow*).

★**KEY POINT** Complex polytrauma leading to tenuously stable patients and difficult transport logistics can make field management of the combat victim challenging. Level 2 surgical forward stations next to the battlefield may be the perfect place for a portable CT scanner such as the CereTom.

Plain radiographs are obtained more often in combat. Combat and terrorist attacks frequently generate severe and diffuse injuries, with metallic foreign bodies inflicting damage in numerous parts of the body simultaneously (**Figs. 4.4, 5.7, 5.8, 5.19,** and **5.36**). In this setting, the Lodox/Statscan can enable rapid identification of the extent of injury and target specific sites for more detailed additional CT imaging (**Fig. 5.8E**).[8,9] The Statscan is a novel low-dose digital radiography machine that can perform a whole-body exam in approximately 15 seconds. The maximum direct dose of radiation to the patient is on the order of 1 mGy, that is, equivalent to one adult chest radiograph.[10] Plain radiographs are particularly useful for (1) assessing the trajectory of the penetrating fragments, (2) identifying the presence and location of residual fragments from a bullet that has exited, and (3) assessment of the type of ammunition used.

Plain radiographs are particularly useful to rule out the possibility of retained bullets, such as devastator bullets, which carry risk of detonation during manipulation at surgery or autopsy. Imaging of the wound track can also distinguish full versus partial jacketed ammunition. In a partial jacketed bullet, the less dense copper jacket can be distinguished from the lead core, and small fragments of metal are often noted along the wound track. In contrast, the absence of a metallic trail is often indicative of an full metal jacketed bullet. All metallic foreign bodies cause significant beam-hardening streak artifact on CT. Unlike MRI, however, their presence poses no hazards to patient imaging.

Typical IED Blast Injuries to the Extremities

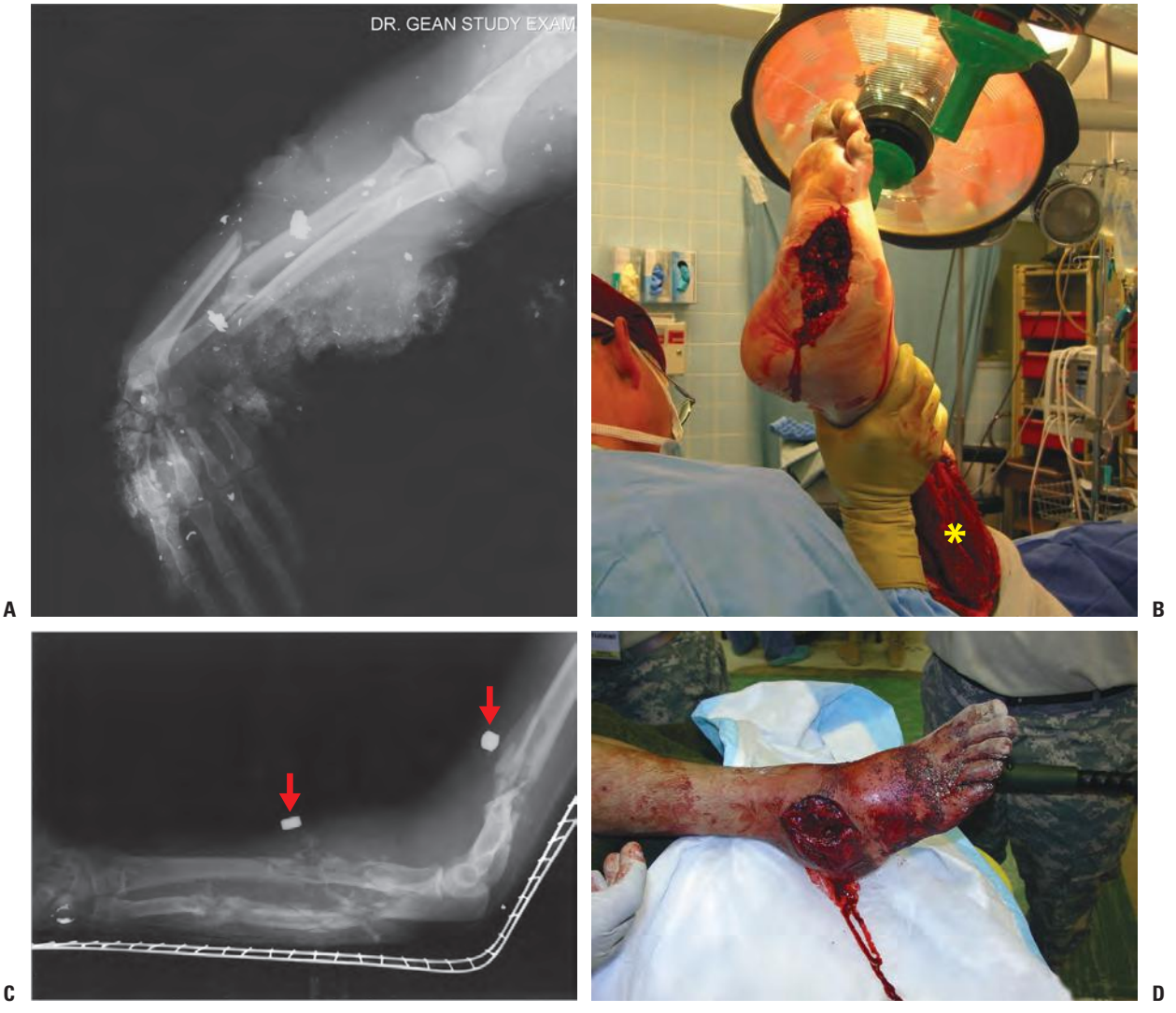


Figure 5.7. Typical IED Blast Injuries to the Extremities. A. Mangled forearm and hand with multiple radiopaque foreign bodies. Note that there is no urgency to removing retained fragments. At surgery, if retained foreign bodies are easily accessible in the wound, then they are routinely removed. However, if they are remote from the surgical site, they can be removed electively. **B.** Intraoperative view of an "exploded" foot; note also the lower extremity fasciotomy (*asterisk*). **C.** Markedly comminuted forearm and humerus fractures with several metal nuts lodged in the soft tissues (*arrows*). **D.** Photograph of an open and contaminated ankle fracture.

★**KEY POINT** In contrast to civilian trauma, combat trauma is associated with more extremity mutilation and amputations. The fractures have, quite literally, an exploded appearance and they usually occur in the setting of an open and contaminated wound.

TABLE 5.3 TBI Imaging Options in War and Terrorism

Combat and Terrorism Traumatic Brain Injury: Imaging Options
Skull Film
Rarely recommended (except for foreign bodies in penetrating trauma)
Computed Tomography
Recommended in the acute setting
Preferred exam for skull fractures
CT cisternography for suspected CSF leak
Computed tomography angiography (CTA) for vascular injury
Computed tomography perfusion (CTP) for dysautoregulation, permeability/blood–brain barrier injury, and penumbra assessment
Magnetic Resonance Imaging
Recommended in the acute setting when neurologic findings are unexplained by CT
Recommended for subacute and chronic TBI
T1-weighted with fat suppression for vascular dissection
Magnetic resonance angiography (MRA) for suspected vascular injury
FLAIR imaging for cortical contusions, SAH, and TAI
Diffusion-weighted imaging (DWI) for post-traumatic infarction, TAI, and fat emboli
Diffusion tensor imaging (DTI) for TAI and connectivity assessment
T2* gradient-recalled echo (GRE) and susceptibility-weighted imaging (SWI) for blood
High-field (3–7T) imaging for temporal lobe injury, TAI, and for advanced MRI techniques
MR Spectroscopy, Magnetization Transfer Imaging, Functional MRI
May be helpful in TAI and predicting long-term prognosis; role in BINT unknown
SPECT, PET, and Magnetic Source Imaging
Usefulness still limited; role in BINT unknown
Catheter Angiography
Recommended to confirm and/or treat a vascular abnormality suspected on CTA

BINT, blast-induced neurotrauma; CSF, cerebrospinal fluid; CT, computed tomography; FLAIR, fluid-attenuated inversion recovery; MR, magnetic resonance; PET, positron emission tomography; SAH, subarachnoid hemorrhage; SPECT, single photon emission computed tomography; TAI, traumatic axonal injury; TBI, traumatic brain injury.

Typical IED Blast Trauma

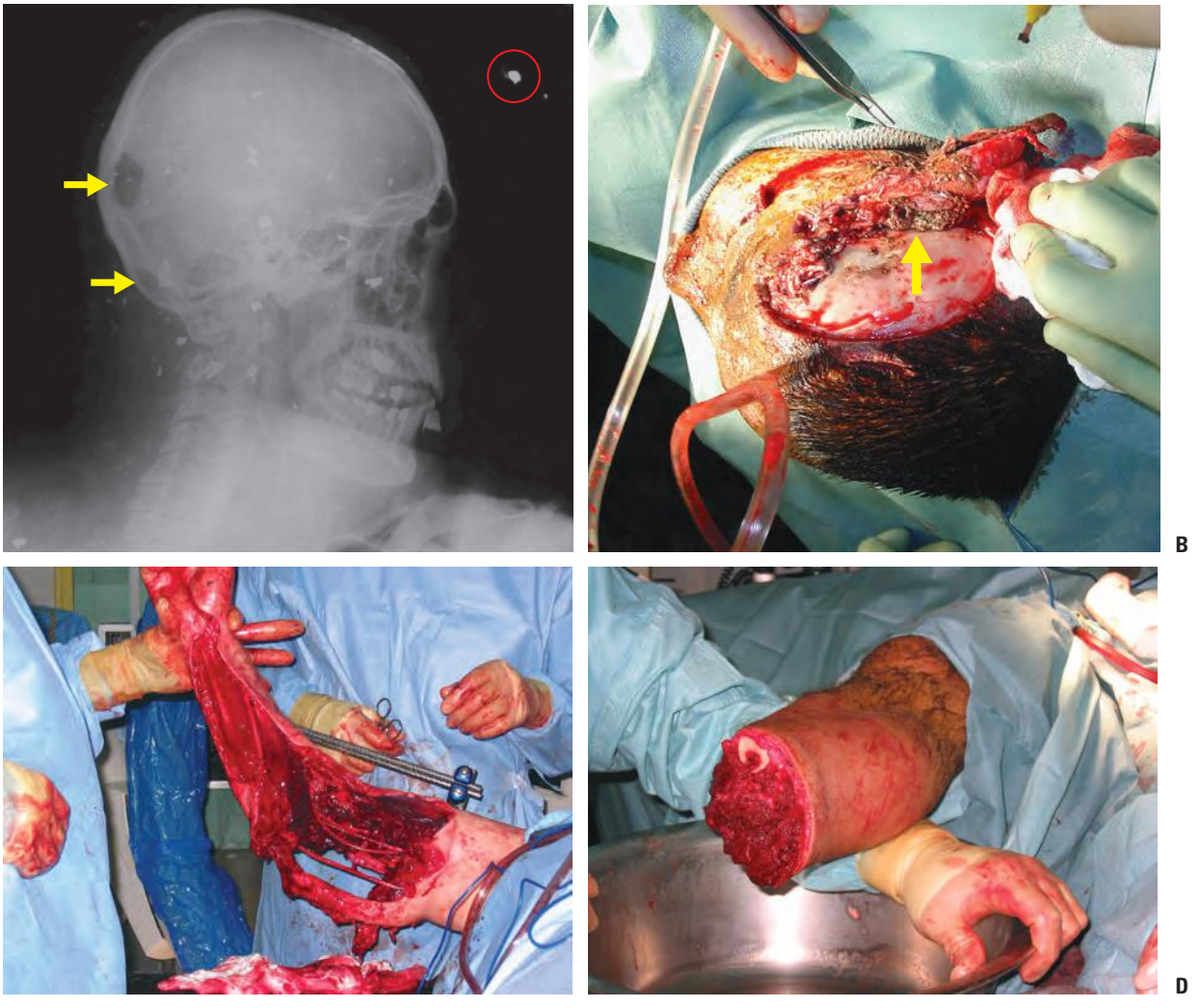


Figure 5.8. Comminution, Contamination, Mutilation, Penetration, and Amputation: A Frequent Theme in Combat and Terrorist Attacks. A. Oblique plain radiograph demonstrates multiple skull defects (*arrows*) and retained radiopaque foreign bodies, one of which is outside the body but present on the transport litter (*circle*). **B.** Intraoperative photograph showing cutaneous burns of the face, tissue maceration, and mud in the scalp flap (*arrow*). Blast wounds are universally contaminated wounds. They are frequently soiled with dirt, clothing, enteric contents, and other debris (see also **Fig. 5.7**). **C.** Intraoperative photograph showing the typical mangled and mutilated wound to the right upper extremity following an IED blast injury. The patient necessitated emergency damage-control *guillotine* amputation **(D)**. *(Continued)*

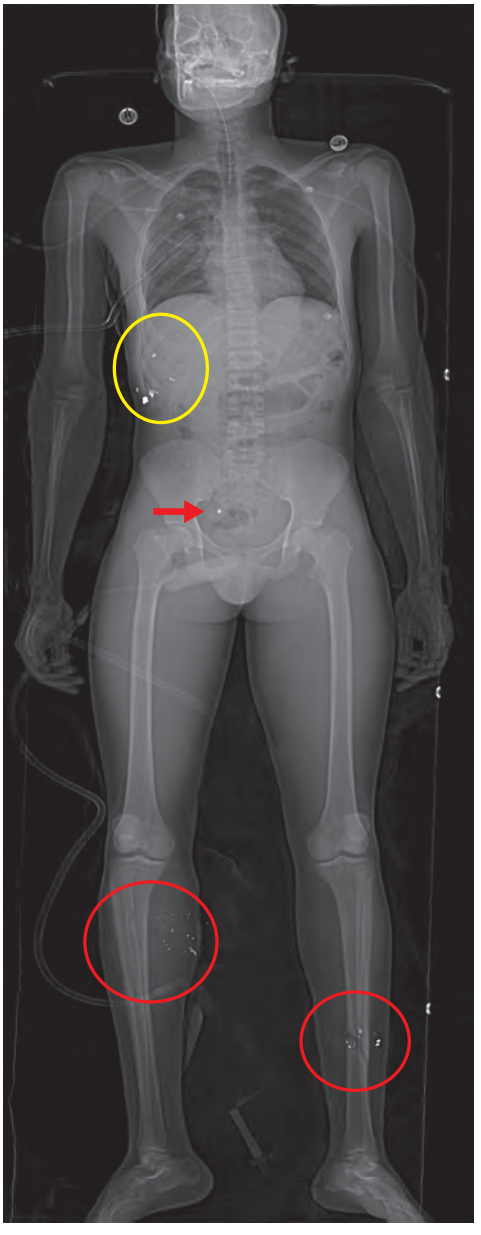


Figure 5.8. (*Continued*) **E.** Full body radiography using Statscan (Lodox) in a different patient who is status-post multiple gunshot wounds (GSWs). The Lodox scan reveals comminuted fractures of the left tibia and fibula (*red circles*). Multiple retained ballistic fragments are present in both lower extremity wounds as well as in the right chest (*yellow circle*). An additional small metallic fragment is identified within the pelvis, remote from the entry sites (*arrow*). Multiple injuries and foreign bodies can be rapidly and simultaneously identified with this low-dose digital technology.

MRI is often contraindicated in war, terrorism, and natural disasters. In contrast to plain radiography and CT imaging, the presence of metal should be considered a relative contraindication to MRI. In military and civil ballistic trauma, bullets and projectiles from secondary blast injury may contain almost any kind of metal, the common ones being steel, iron, lead, copper, aluminum, and various alloys. The permissibility of an MRI in the setting of a foreign metallic body is not absolute and depends on the following criteria: type and location of the projectile, proximity and fragility of vital tissue, time elapsed since the initial injury, the potential value of information from the MRI study to patient management, and whether this information would be accessible through other imaging modalities.[11–13] Rotation and movement of a projectile that contains ferromagnetic materials during the MRI scanning can lead to devastating consequences. Ferromagnetic metals have unpaired electrons and exhibit a strong attraction to a magnetic field. The atoms of ferromagnetic metals have a permanent magnetic moment and retain their magnetic properties even after the external magnetic field is removed. The primary ferromagnetic metal of concern in MRI scanning is iron, and steel is forged from iron. Nickel is also ferromagnetic. Nonferromagnetic metals do not possess a permanent magnetic moment and are safe to image with MRI. Copper, lead, tin, titanium, brass, and aluminum are examples of non-ferromagnetic metals. When the metallic nature of a foreign material is unknown, one should evaluate the risk versus benefit of whether or not to perform the MRI based on the assumption that the material could contain steel.

In addition to foreign projectiles, medical implants and devices that have metallic content can also be dislodged or become dysfunctional during MRI scanning.[14] Contraindications to MRI currently include cochlear

implants, ocular prostheses, magnetically activated dental implants, and tissue expanders. Swan-Ganz catheters, cerebral ventricular shunt tube connectors, ferromagnetic intravascular coils/filters/stents that have been placed within several weeks before imaging, and all cerebral aneurysm clips of unknown composition should not be enlarged with MRI. All biomedical implants and devices of uncertain ferromagnetism should be first evaluated with ex vivo MRI scanning techniques. A helpful website to assist in the decision to take an MRI of patients with biomedical implants and devices is *www.MRIsafety.com*. Imaging nonferromagnetict objects with MRI should still be approached with caution because technical specifications might not list small amounts of ferromagnetic contaminants.

Image distortion will often occur when imaging metal with MRI, depending on the amount and magnetic susceptibility of the metal, the shape of the object, and the pulse sequence. Gradient-recalled echo (GRE) sequences, and especially susceptibility-weighted sequences, yield larger artifacts because of the local field distortion. Nonferromagnetic foreign bodies cause minimal image degradation on MRI. Aluminum, although it is nonmagnetic, may cause some distortion, whereas stainless steel causes marked image degradation. The slightly diamagnetic nature of copper can result in a perimeter artifact, seen as an area of signal distortion and misregistration. Lead, which is more diamagnetic, rarely has enough phase distortion on spin-echo sequences to interfere with image interpretation. However, nickel can be mildly ferromagnetic and is a frequent trace element in lead bullets. Thus knowledge that the retained fragment was from a lead bullet does not completely exclude the possibility that it is contaminated by nickel impurities, which impart risk of rotation and movement of that fragment during MRI. Lead shot causes minimal image distortion on spin-echo sequences despite the severe image degradation that it causes on CT scans. Therefore, the associated contusional injury along the tract of the wound is better appreciated on MRI than on CT images when non-ferromagnetic munitions are used.

MRI only became available in the war zone in October of 2011. There are currently three mobile 1.5T MRI machines in Afghanistan, and the majority of the images are reviewed remotely via military teleradiology networks. The main purpose of MRI in the combat zone is for research and gathering data on U.S./NATO troops. It is also used for imaging host nationals and military contractors. The role of MRI has been carefully expanded to include medical conditions where the results could change mission requirements and/or medical management of individual soldiers (**Fig. 5.9**). In addition to TBI, musculoskeletal trauma, tumors, infectious/inflammatory diseases and congenital disorders are among the afflictions diagnosed by mobile MRI. However, even in uninjured soldiers, MRI must be performed judiciously because prior events in the war zone may have led to wounds that were contaminated with ferromagnetic foreign bodies that were retained after the wounds healed. It some cases, it may be necessary to surgically extract a foreign body in order to perform important MRI studies, such as a spine MRI for suspected cord injury.

Role of MRI in Combat (and Concussion)

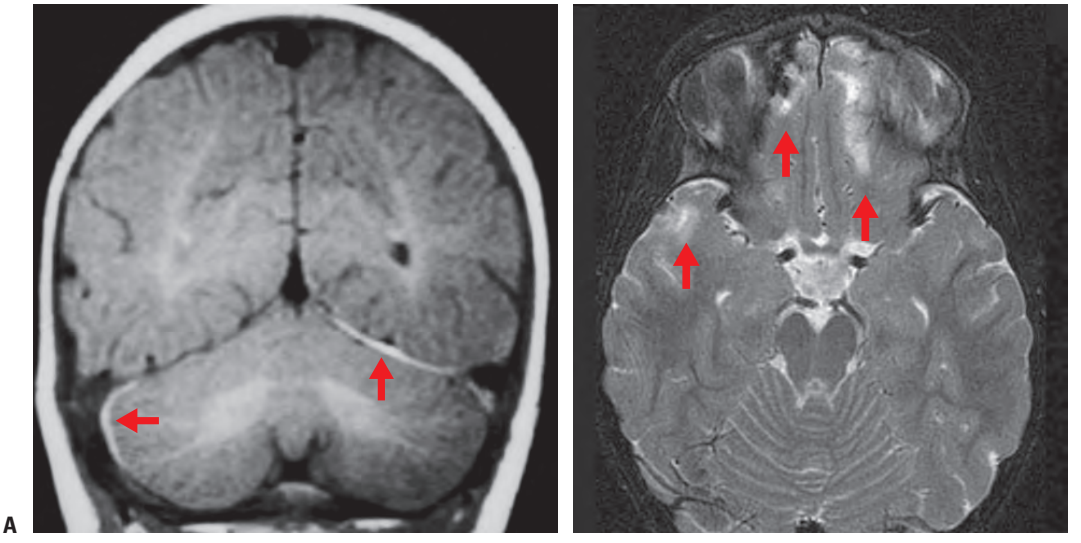


Figure 5.9. **Role of MRI in Combat (and Concussion). A.** T1-weighted coronal MRI demonstrates a thin layer of T1 hyperintensity along the left tentorium as well as lateral to the right cerebellar hemisphere, consistent with subacute subdural hemorrhage (*arrows*).This subdural hematoma (SDH) was not evident on the CT imaging study. **B.** T2-weighted axial MRI reveals abnormal hyperintensity with the right anterior temporal cortex and orbitofrontal lobes (*arrows*). The CT exam did not demonstrate these abnormal findings. Although both of these patients were civilians, similar MRI findings in soldiers would warrant immediate removal from combat.

★**KEY POINT** MRI first became available in the war zone in October of 2011. This case is an example of how patients with a normal CT may benefit from MRI to identify abnormalities that may change mission requirements and/or medical management.

CT-angiography and conventional catheter angiography are performed more often in the setting of war and terrorism. This is due to the unusually high incidence of neurovascular injury with IED explosions in comparison to civilian trauma (see Lesson 10). Although catheter angiography remains the gold standard, multislice CTA has replaced catheter angiography as the initial diagnostic study for penetrating head and neck injuries. CTA has acceptable sensitivity and specificity to detect vascular injuries in these patients and offers advantages over angiography in terms of ease and timing, decreased cost, noninvasive nature, and the low requirement for endovascular intervention in these patients.[15] In the absence of retained metal foreign bodies, an MRI study of the brain is helpful to better identify TAI and the extent of TBI and to assess for the presence or risk of ischemic complications from cervical vascular injury (**Figs. 3.20** and **5.9**). Ischemic risk to brain tissue may not be detected by routine CT brain imaging, although CT-perfusion (CTP) studies may be helpful when MRI is contraindicated.

MRI and CT can be complementary for evaluating injuries from penetrating wood or other nonmagnetic objects (**Fig. 5.10**). In addition, when the soldier returns to the United States, there are various functional imaging techniques that may provide evidence of metabolic dysfunction that cannot be detected by macroscopic anatomic imaging modalities. It is hoped that the results of newer imaging techniques such as diffusion tensor imaging (DTI), functional magnetic resonance imaging (fMRI), single photon emission computed tomography (SPECT), fluorodeoxyglucose positron emission tomography (FDG-PET) scanning, magnetoencephalography (MEG), and magnetic source imaging (MSI) in TBI patients will be validated and permit prediction of patient outcome. These advanced imaging technologies are reviewed in the following sections with reference to blast TBI and post-traumatic stress disorder (PTSD).

Wooden Foreign Body (Tree Branch) Mimicking a Pseudoaneurysm on MRI

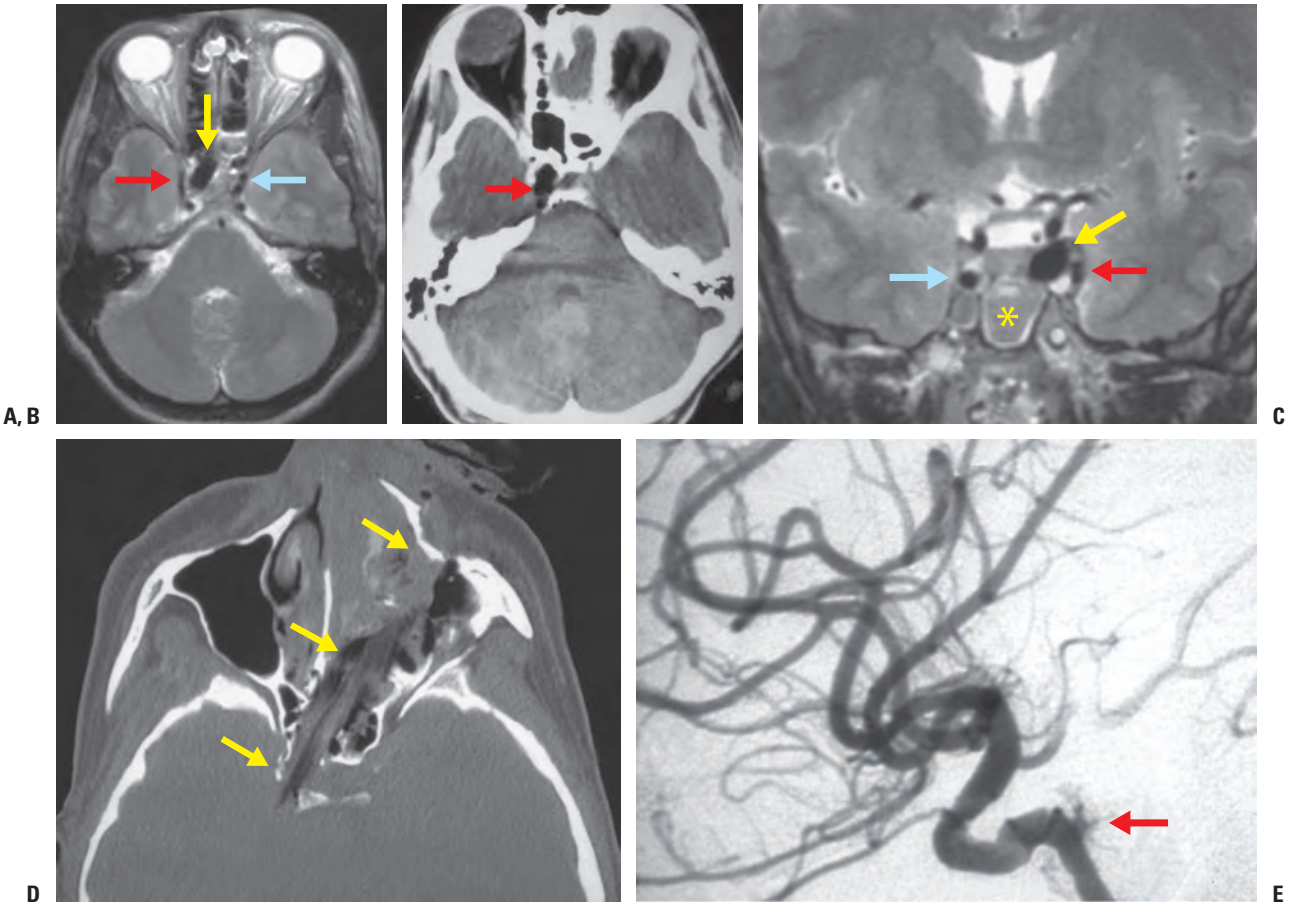


Figure 5.10. **Orbitocranial Wooden Foreign Body. A.** Axial and (**C**) coronal T2-weighted MRI show an abnormal area of hypointensity (*yellow arrow*) within the right cavernous sinus that suggests either air or a vascular flow void. The sphenoid sinus is opacified (yellow asterisk) and the adjacent cavernous internal carotid artery (ICA) (*red arrow*) directly abuts the lesion, making it impossible to exclude a pseudoaneurysm on MRI. Compare with the normal left cavernous ICA flow void (*blue arrow*). **B.** Axial CT demonstrates ovoid *air density* in the right cavernous sinus (*arrow*). **D.** Axial CT bone window better delineates the wooden foreign body traversing the inferior left orbit and terminating in the right cavernous sinus (*arrows*). **E.** Lateral catheter angiogram reveals irregularity of the cavernous and precavernous right ICA (*arrow*) but no definite pseudoaneurysm. (Courtesy of Bertil Leidner, MD, Karolinska University Hospital, Stockholm, Sweden.)

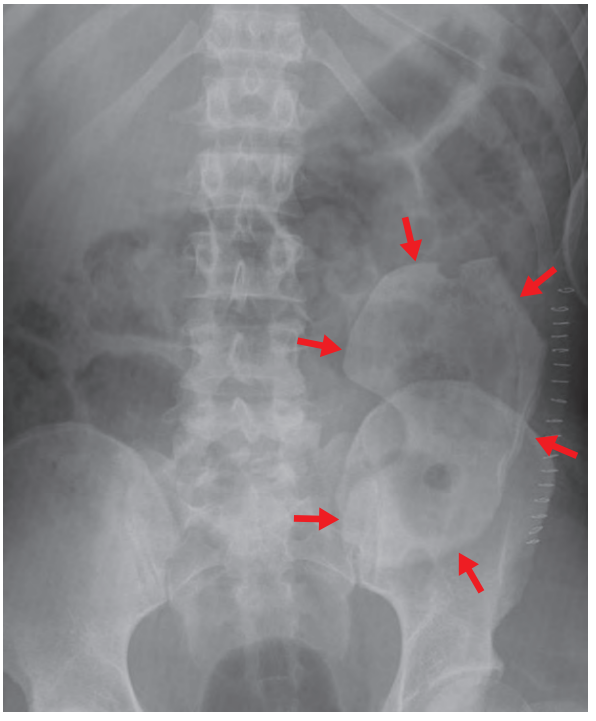
★**KEY POINT** The density of wood on CT is a function of its water content; that is, dry wood has air density and wet wood has soft tissue density on CT.

LESSON 4: BLAST-RELATED TRAUMA IS THE MOST COMMON MECHANISM OF INJURY IN WAR AND TERRORIST ATTACKS

Although rare in civilian society, explosions are a daily occurrence on the battlefield and in war-torn urban areas. An explosive mechanism accounts for about 80% of injuries incurred in Operation Iraqi Freedom (OIF)/Operation Enduring Freedom (OEF) combat, and this is the highest proportion of explosive injuries seen in any large-scale conflict to date.[16–18] Nearly one-third of all combat forces have been exposed to a blast force, and nearly 60% of all blasts have resulted in TBI.[19] The vast majority of these blast injuries are caused by IED explosions, designed to cause massive bodily destruction by propelling large numbers of fragments toward the intended victims. This is in contrast to blunt trauma, which predominates in civilian injury. Indeed, TBI may be the signature injury of the wars in Iraq and Afghanistan, and the IED is the signature weapon. In 2005 alone, for example, the U.S. military reported 10,953 IED attacks, at an average of 30 per day.[20] The use of explosive devices, particularly roadside IEDs, initiates complex, multi-mechanistic forces on the body from the explosion itself and also by vehicle translocation causing rollover, elevation, and upending or colliding with other objects, resulting in occupant blunt trauma. The remaining 20% of hostile injuries are caused by bullets from small arms. These tend to be of higher kinetic energy (e.g., AK47s) than those encountered in civilian trauma practice. The approximately 25% of non–combat-related injuries in the deployed forces are known as nonbattle injuries and include vehicle crashes, falls, and sports injuries. However, many of these also differ from civilian trauma. Military personnel are equipped with personal protective equipment, such as helmets, goggles, and body armor consisting of a Kevlar jacket. These alter the way in which energy is coupled to the body. Body armor substantially protects certain areas from fragment injury. In addition, vehicles may be heavily protected (e.g., the A1M1 tank or mine-resistant ambush protected [MRAP] vehicle or relatively unarmored (e.g., the originally deployed high mobility multipurpose wheeled vehicle [HMMWV], which was later up-armored). It's worth emphasizing that explosive trauma resulting from terrorist attacks is not mitigated by the protective equipment afforded in combat.

Blast trauma typically results in complex, contaminated penetrating injuries. Furthermore, thermal injury is a common occurrence in blast trauma. This has introduced a unique group of burn casualties, separate from the typical fire and smoke inhalation victims, into trauma medicine. As discussed in Chapter 3, combat neurotrauma usually results from a variable combination of the four mechanisms of blast injury, and is therefore called "*blast-plus*" TBI, as opposed to blast-induced neurotrauma (BINT).[21] (**Figs. 5.11** to **5.23**) The frequent cerebral swelling that accompanies blast trauma has resulted in an unprecedented number of decompressive craniectomies (DCs) in this conflict in comparison to prior wars.[22–24] In austere environments and special situations (like combat injuries), the removed bone flap from the craniectomy may be placed into the abdominal wall for preservation, pending replacement after resolution of the acute brain swelling and aeromedical transport to a U.S. military medical center. However, this trend is changing due to the unique infection potential in other continents and improved prosthetic technology. In addition, perioperative infections at the time of cranioplasty surgery to replace the skull bone have raised concerns about replacement of the patient's own bone as compared to a prosthetic skull implant. Prosthetic implants also circumvent long-term complications of bone resorption, which are commonly seen following autologous cranioplasties.

Decompressive Hemicraniectomy Performed in a Combat Field Hospital



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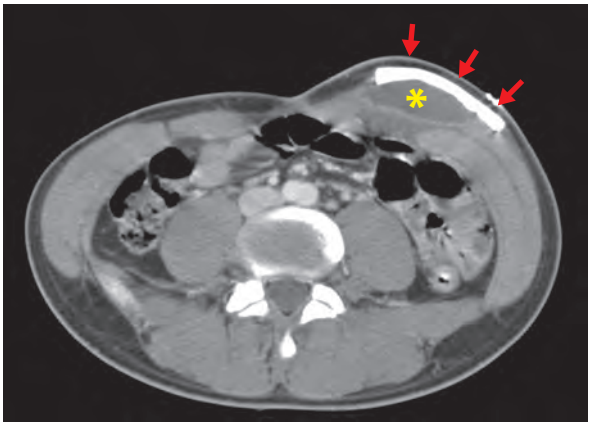

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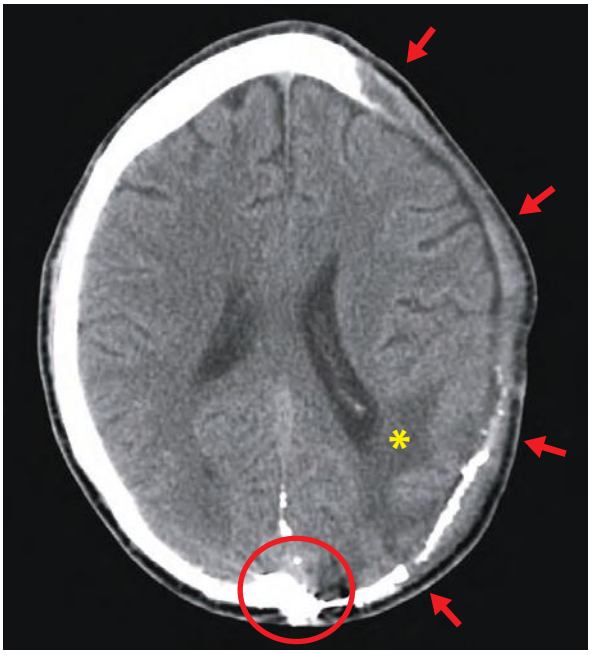


C

Figure 5.11. Decompressive Craniectomy Skull Flap "Banked" in the Abdomen. A. Plain film of the abdomen demonstrates the skull flap in the left lower quadrant (*arrows*). Surgical staples are seen. **B.** Contrast-enhanced axial CT of the abdomen shows the craniectomy flap (*arrows*) with an underlying homogeneous fluid collection (*asterisk*) in the left anterior abdominal wall. The differential diagnosis of the fluid collection includes liquified hematoma, seroma, abscess, or proteinaceous fluid collection. Because the cultures were negative for bacteria, liquified hematoma was the working diagnosis. **C.** Axial CT of the brain demonstrates the large craniectomy site (*arrows*). Encephalomalacia is noted in the left parieto-occipital lobe (*asterisk*). Beam hardening artifact is noted from residual metallic shrapnel (*circle*). Following recovery from the craniectomy surgery, the patient ambulated with a hockey helmet for protection of the brain while awaiting his cranioplasty. After surgical removal of skull flap from the abdomen, it was decided to use a prosthetic cranioplasty implant. Developing literature on *Acinetobacter* species infection in autologous bone flaps is one of several factors weighing into the decision of whether to use a synthetic cranioplasty implant instead of the patient's native bone flap. (Adapted with permission from Folio L, Craig S, Singleton, B, et al. Emergency decompressive craniotomy with banked skull flap in subcutaneous pocket of abdominal wall for preservation. Military Medicine Radiology Corner, May 2006).

"Blast-Plus" Brain Injury

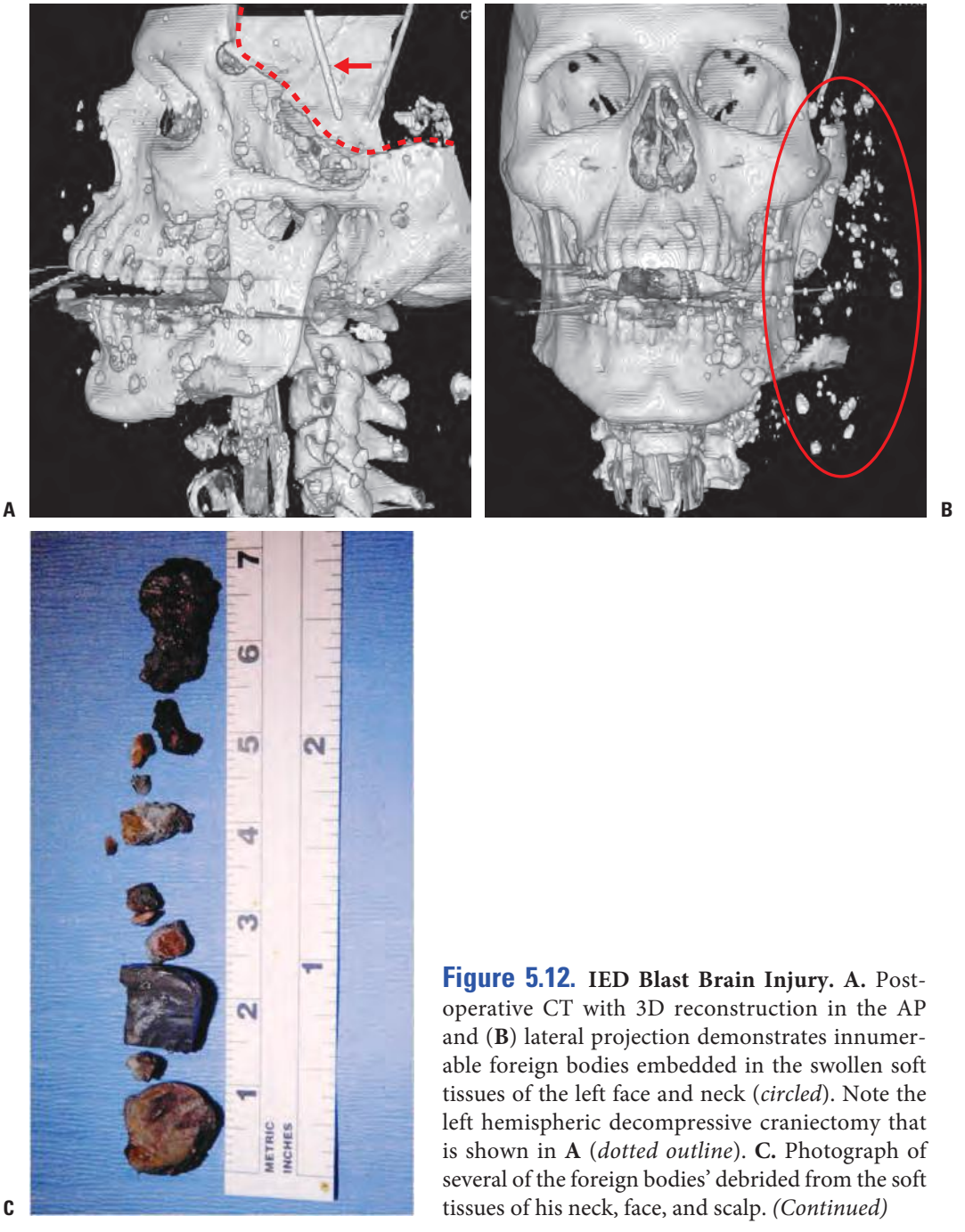
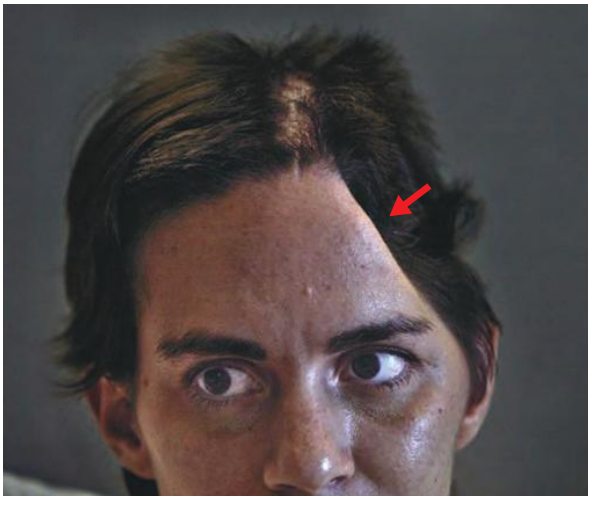


Figure 5.12. IED Blast Brain Injury. A. Postoperative CT with 3D reconstruction in the AP and (**B**) lateral projection demonstrates innumerable foreign bodies embedded in the swollen soft tissues of the left face and neck (*circled*). Note the left hemispheric decompressive craniectomy that is shown in **A** (*dotted outline*). **C.** Photograph of several of the foreign bodies' debrided from the soft tissues of his neck, face, and scalp. *(Continued)*



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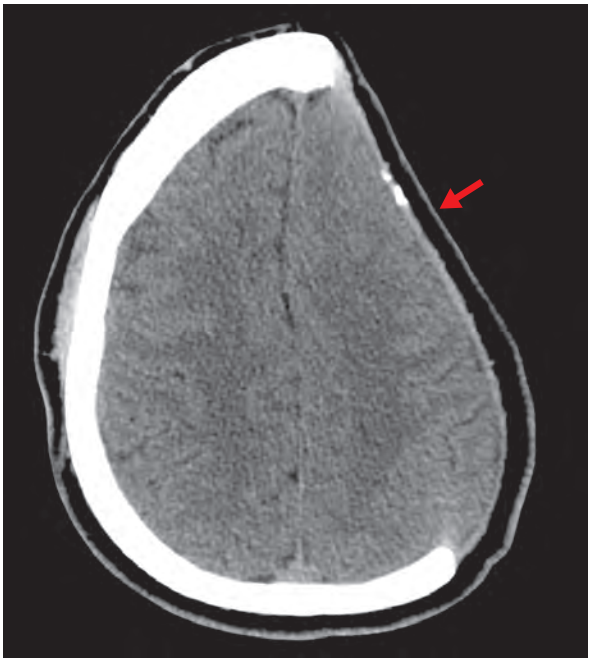


F

Figure 5.12. *(Continued)* **D.** Photograph of another victim of an IED explosion that was obtained 2 months after a decompressive craniectomy illustrates the characteristic "sunken scalp" deformity of left hemicranium (*arrow*). **E.** Photograph of the patient 3 months following successful cranioplasty. **F.** Axial CT obtained 2 months after the decompressive craniectomy shows the typical concavity of the left hemicranium (*arrow*); compare with (**D**).(Courtesy of Guy Rosenthal, MD, Hadassah Hospital, Israel.)

"Blast-Plus" Brain Injury

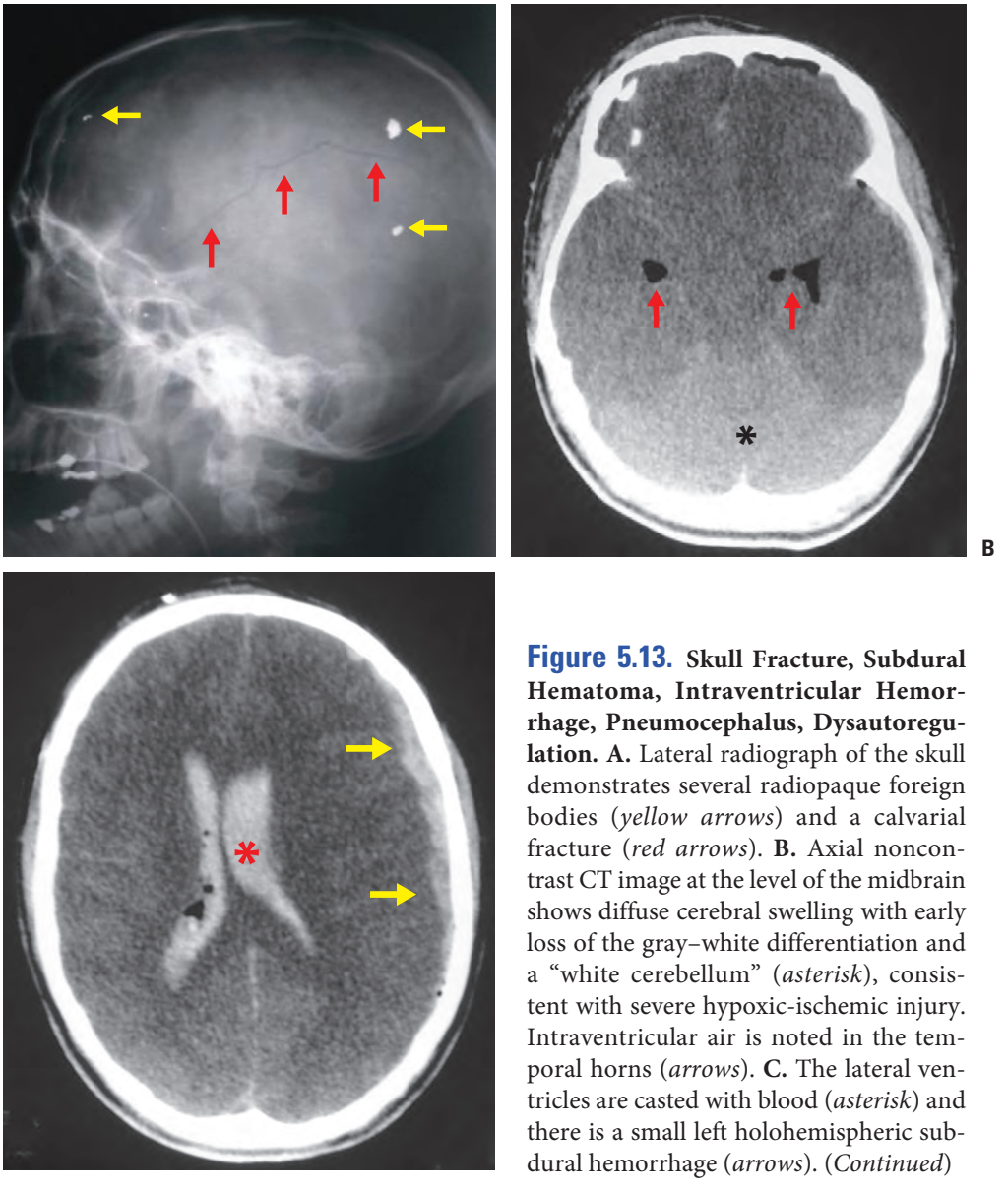
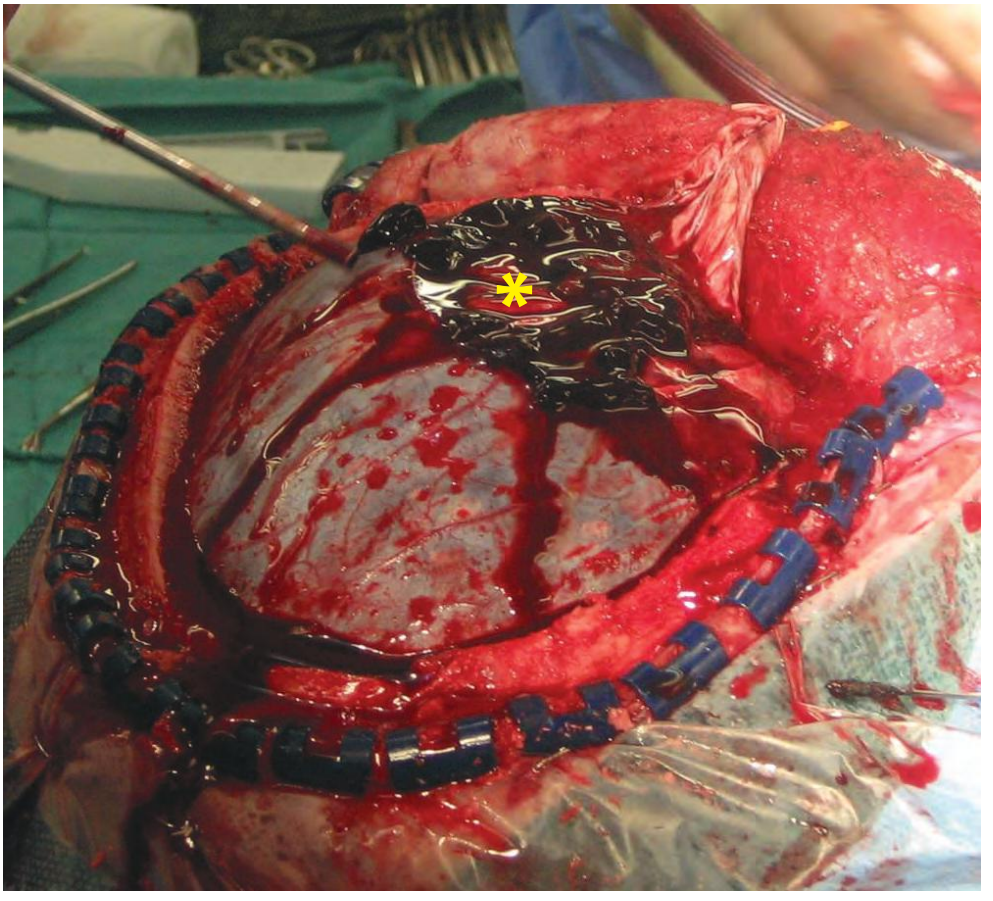


Figure 5.13. Skull Fracture, Subdural Hematoma, Intraventricular Hemorrhage, Pneumocephalus, Dysautoregulation. A. Lateral radiograph of the skull demonstrates several radiopaque foreign bodies (*yellow arrows*) and a calvarial fracture (*red arrows*). **B.** Axial noncontrast CT image at the level of the midbrain shows diffuse cerebral swelling with early loss of the gray–white differentiation and a "white cerebellum" (*asterisk*), consistent with severe hypoxic-ischemic injury. Intraventricular air is noted in the temporal horns (*arrows*). **C.** The lateral ventricles are casted with blood (*asterisk*) and there is a small left holohemispheric subdural hemorrhage (*arrows*). (*Continued*)



D

Figure 5.13. (*Continued*) **D.** Intraoperative photograph shows the craniotomy with the myocutaneous scalp flap reflected anteriorly (upper right area in the photograph). Note the epidural blood being suctioned from above the dura (*asterisk*), consistent with epidural hemorrhage. The underlying subdural hemorrhage is seen as a bluish discoloration beneath the dura. (Courtesy of Rocco Armonda, MD, Col (ret), MC, USA).

"Blast-Plus" Brain Injury

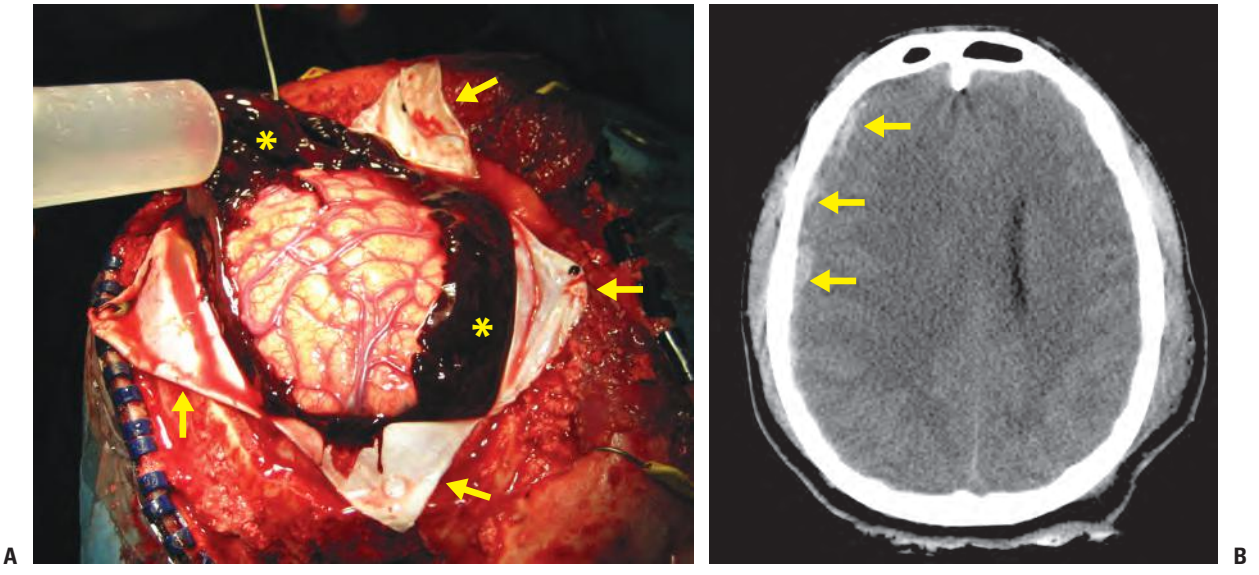


Figure 5.14. **Acute SDH following a Roadside IED Explosion. A.** Intraoperative photograph shows subdural hemorrhage (*asterisks*) beneath the opened dural leaflets (*arrows*). (Courtesy of Rocco A. Armonda, MD, Col (ret), MC, USA). **B.** Admission CT shows a heterogeneous right frontal subdural hematoma (*arrows*) causing effacement of the right lateral ventricle. The heterogeneity of the subdural hematoma (SDH) is consistent with active hemorrhage. Note how the SDH is typically contrecoup to the site of impact, denoted by left parietal scalp swelling.

"Blast-Plus" Brain Injury

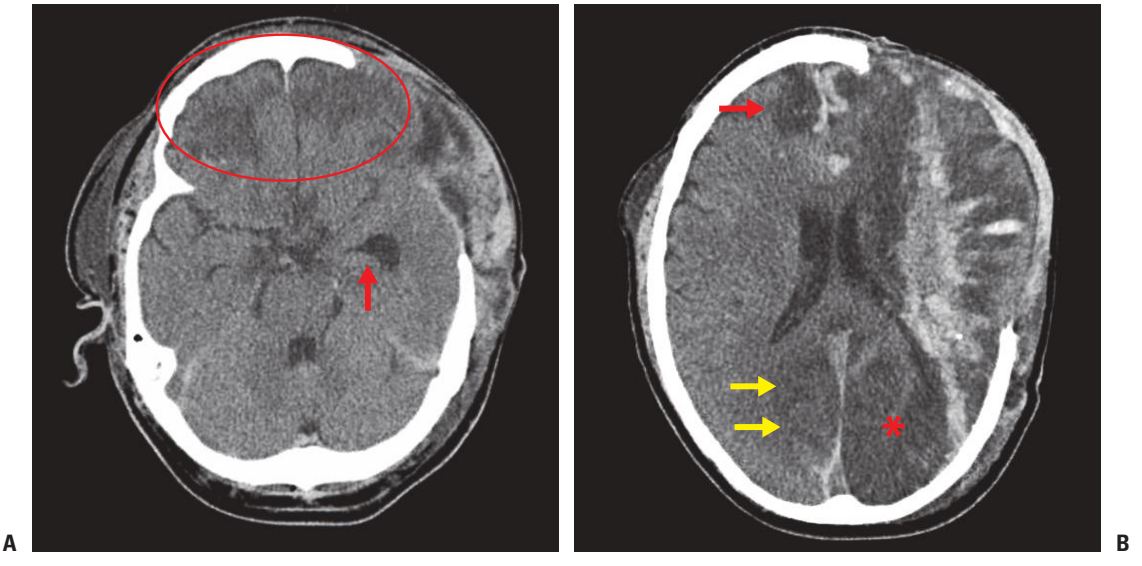


Figure 5.15. **IED Brain Injury. A.** Noncontrast axial CT image on postop day 3 demonstrates a small left frontotemporal decompressive hemicraniectomy, bifrontal edema (*circle*), and a "trapped" left temporal horn (*arrow*). **B.** Note extensive left hemispheric hemorrhagic injury and external herniation. Post-herniation infarction is seen in the territory of both posterior cerebral arteries (*yellow arrows*), left (*asterisk*) greater than right (*arrows*), and the right anterior cerebral artery (*red arrow*).

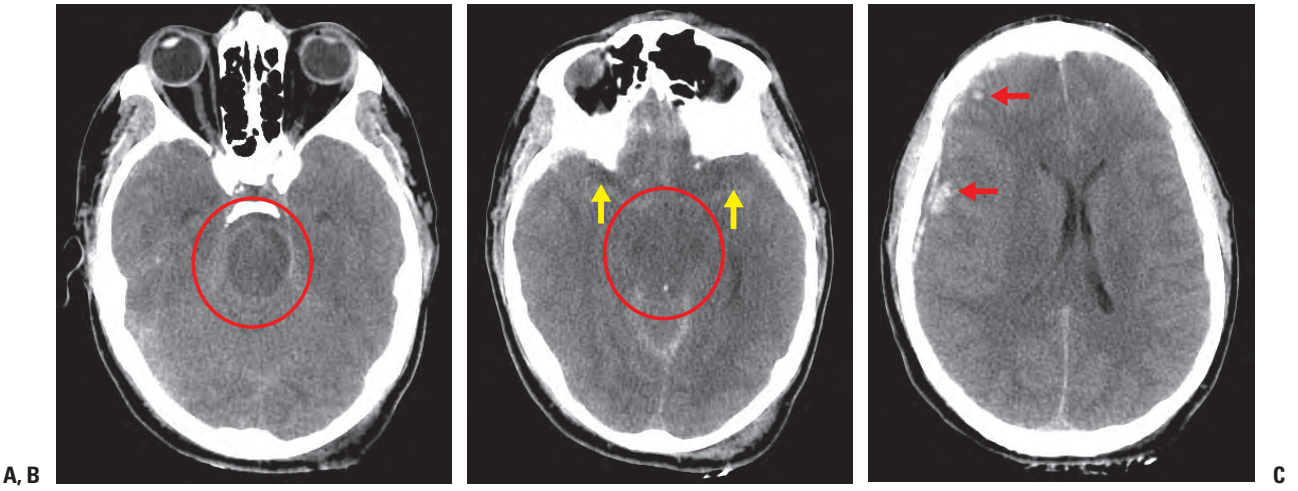


Figure 5.16. Cerebral Hyperemia, Cortical Contusions, and Subarachnoid Hemorrhage. A, B. Admission noncontrast CT images demonstrate complete effacement of the cerebral sulci and perimesencephalic cisterns (*circle*). Ill-defined high attenuation is seen in region of the M1 segment of the middle cerebral arteries, consistent with subarachnoid hemorrhage (*yellow arrows*). The coup site is identified by the left occipital subgaleal scalp hematoma (*red arrow*). **C.** Small right frontal contrecoup cortical contusions and a tiny SDH are also noted (*red arrows*). Note that while there is diffuse sulcal effacement, the gray–white matter differentiation is preserved at the level of the lateral ventricle.

★**KEY POINT** Diffuse cerebral hyperemic swelling (i.e., cerebral hyperemia) is diagnosed on CT imaging when there is evidence of diffuse mass effect with preservation of the gray–white matter differentiation. It is called cerebral edema when there is mass effect with loss of the gray–white matter differentiation (note that this refers to cytotoxic edema, not vasogenic edema). Cerebral hyperemia is much more common in younger patients than in older patients, and this may partially explain why it is particularly problematic in combat TBI.

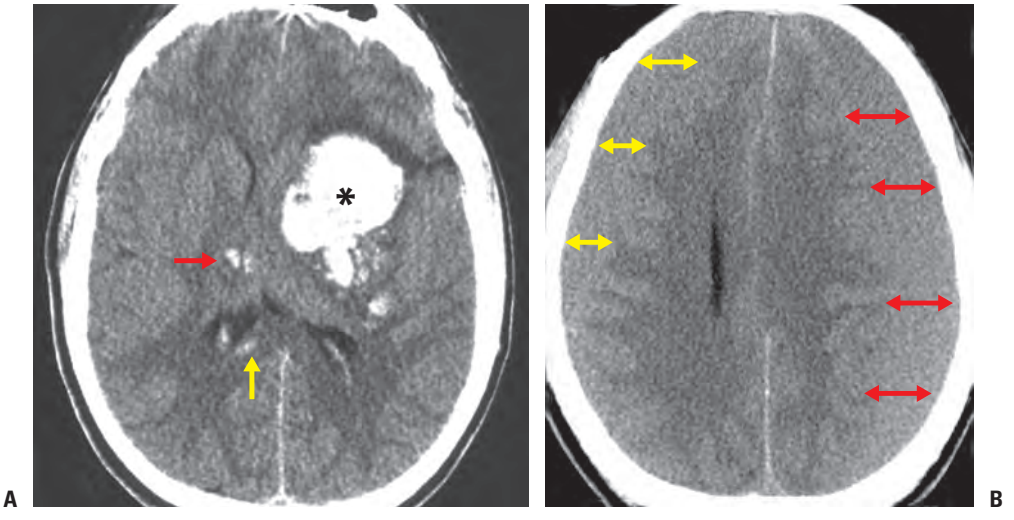


Figure 5.17. "Blast-Plus" Brain Injury. A. Traumatic axonal injury (TAI). Noncontrast axial CT image demonstrates acute hemorrhage in the left basal ganglia (*asterisk*), right thalamus (*red arrow*), and splenium of the corpus callosum (*yellow arrow*), consistent with TAI. **B. Isodense SDH.** This 28-year-old patient presented with persistent headaches several weeks after injury. Axial CT demonstrates complete effacement of the left lateral ventricle and abnormal separation of the cortical mantle from the calvarium (*red arrows*), consistent with an isodense subdural collection. A smaller isodense SDH is also present on the right (*yellow arrows*).

"Blast-Plus" Brain Injury (Traumatic Axonal Injury)

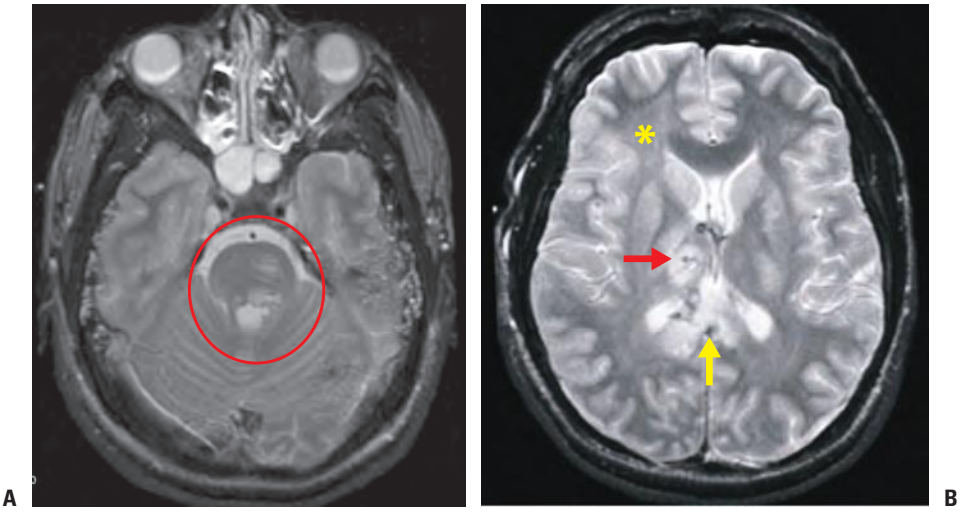


Figure 5.18. **Traumatic Axonal Injury. A.** Axial T2-weighted MRI demonstrates hyperintense lesions within the left pons and superior cerebellar peduncle (*circle*). **B.** Axial T2-weighted image at the level of the basal ganglia shows a hemorrhagic lesion within the right thalamus (*red arrow*), abnormal right frontal white matter T2-hyperintensity (*asterisk*), and an enlarged and hyperintense splenium of the corpus callosum (*yellow arrow*). The combination of lesions located in the lobar white matter, corpus callosum, and brain stem is consistent with Grade 3 TAI.

★**KEY POINT** The imaging appearance of TAI and extra-axial hemorrhagic collections from combat trauma appears similar to that found in civilian TBI. It is unknown whether the primary blast force exacerbates 3° blast (i.e., blunt) trauma.

"Blast-Plus" Brain Injury (Bus Explosion/Suicide Bomb)

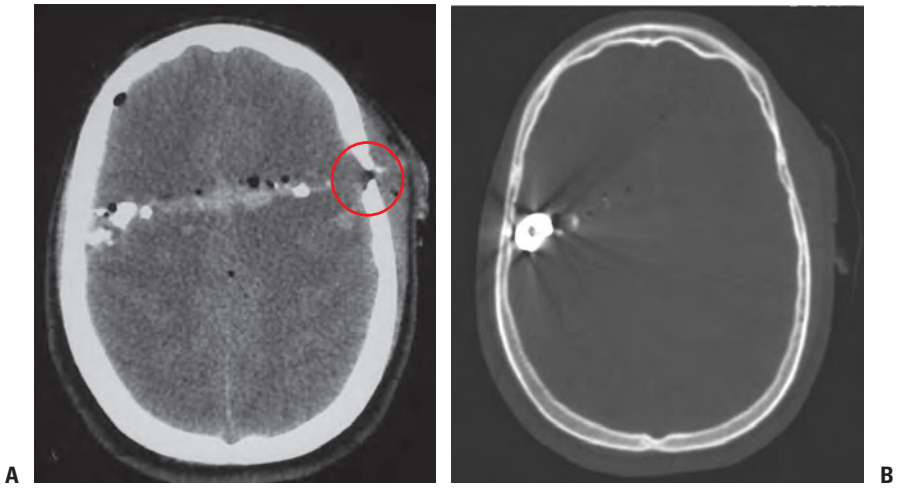


Figure 5.19. **Multiple Intracranial and Extracranial Foreign Bodies. A.** Admission CT shows a bifrontal penetrating injury (i.e., the projectile did not exit). Note beveling of the inner table of the calvarium, consistent with the entry site (*circle*). **B.** Bone windowing at a slightly lower level better demonstrates the nature of the foreign body. (*Continued*)

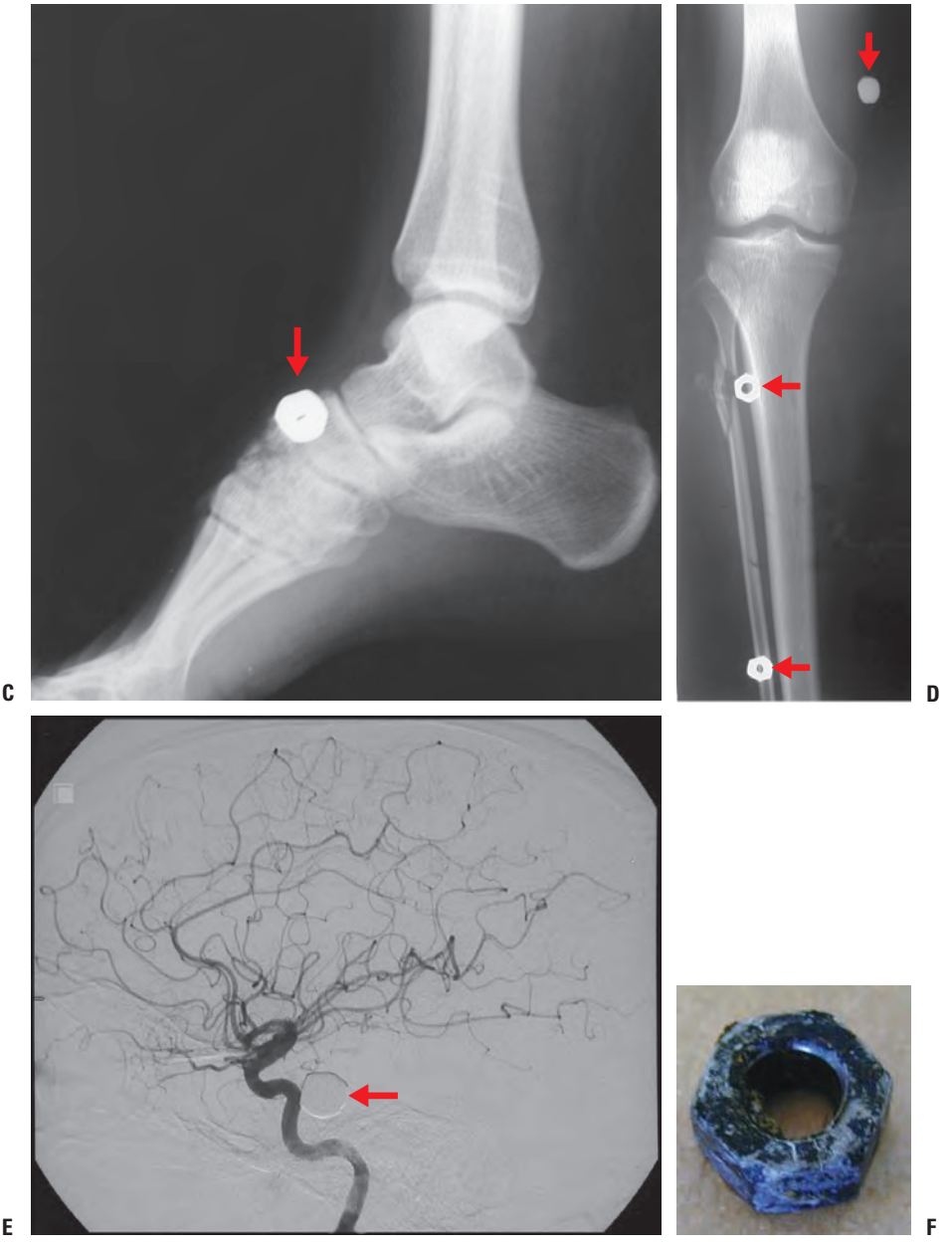


Figure 5.19. (*Continued*) **C, D.** Ankle and knee radiographs demonstrate additional foreign bodies (*arrows*). **E.** Lateral subtracted view from an ICA catheter angiogram shows the foreign body (*arrow*) and normal cerebrovascular anatomy without sign of injury. **F.** Photograph of one of the surgically removed rusty hexagonal nuts. Remarkably, the patient survived with a Glasgow Outcome Scale (GOS) = 3.

Another imaging finding that is commonly found is blast-related *vasospasm* (**Figs. 5.20, 5.66,** and **5.67**). As stated in Lesson 10, it is one of many causes for the increased incidence of stroke in young soldiers.

Cerebral Vasospasm (IED Blast)

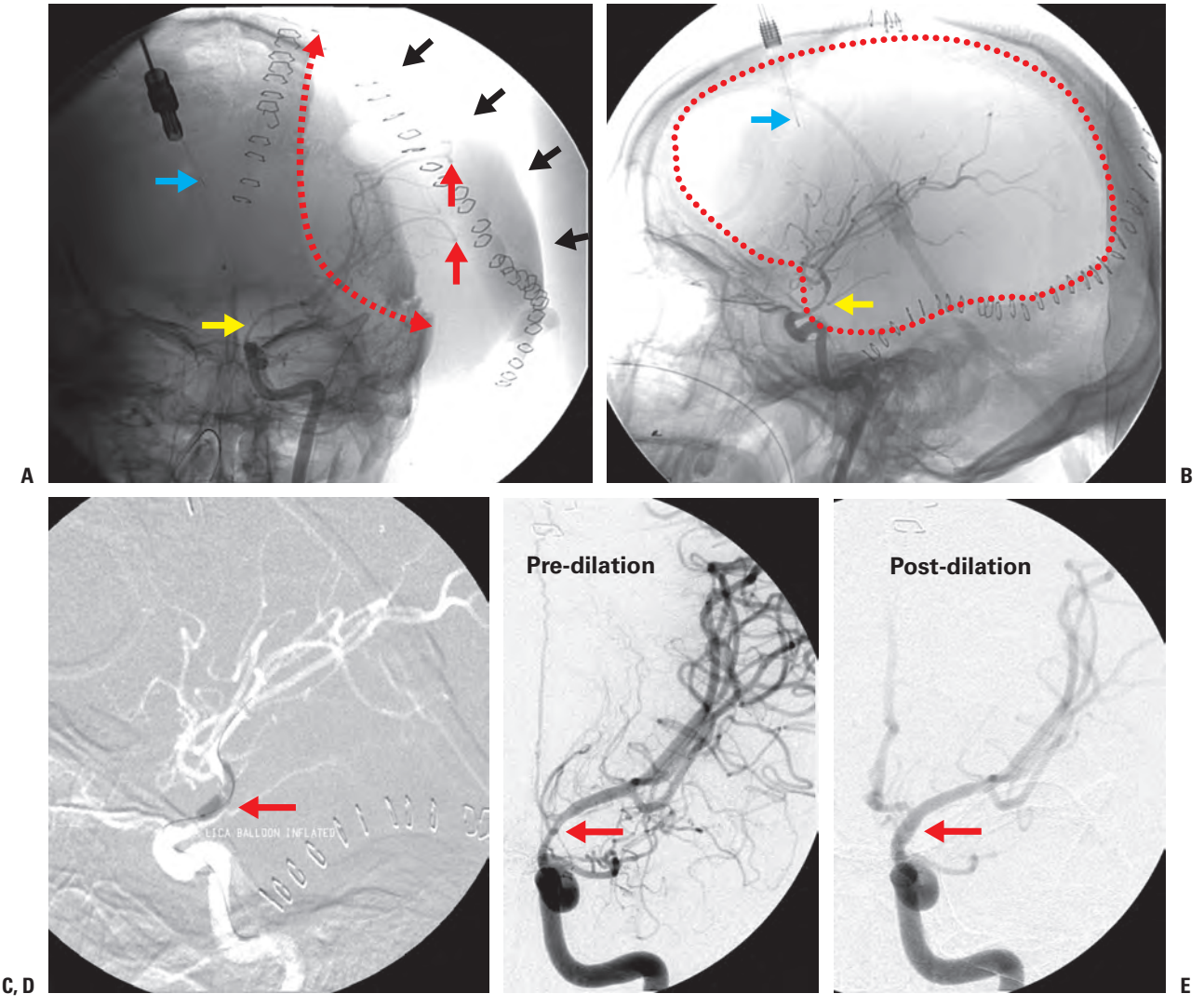


Figure 5.20. Cerebral Vasospasm and Decompressive Hemicraniectomy. This soldier was injured by a large blast inside a booby-trapped house. **A.** AP and (**B**) lateral views from a left ICA angiogram demonstrate severe supraclinoid ICA vasospasm (*yellow arrows*). The patient is status-post a large left decompressive hemicraniectomy (*dotted line*). A brain tissue oxygen monitor is positioned within the right frontal lobe (*blue arrow*). Note marked external herniation of the brain through the craniectomy defect with middle cerebral artery branches located beyond the margin of the skull (*red arrows*) and the scalp surface located far outside the expected skull margin (*black arrows*). **C.** Balloon (*arrow*) dilation of the segmental ICA vasospasm. **D.** Pre- and (**E**) post-balloon dilation showing interval increase in caliber of the left supraclinoid ICA (*arrow*). (Courtesy of Rocco A. Armonda, MD, Col (ret), MC, USA).

★**KEY POINT** The supraclinoid ICA, where it is attached at the distal dural ring, has the highest incidence of delayed vasospasm.

"Blast-Plus" Brain Injury (Subacute Traumatic Axonal Injury)

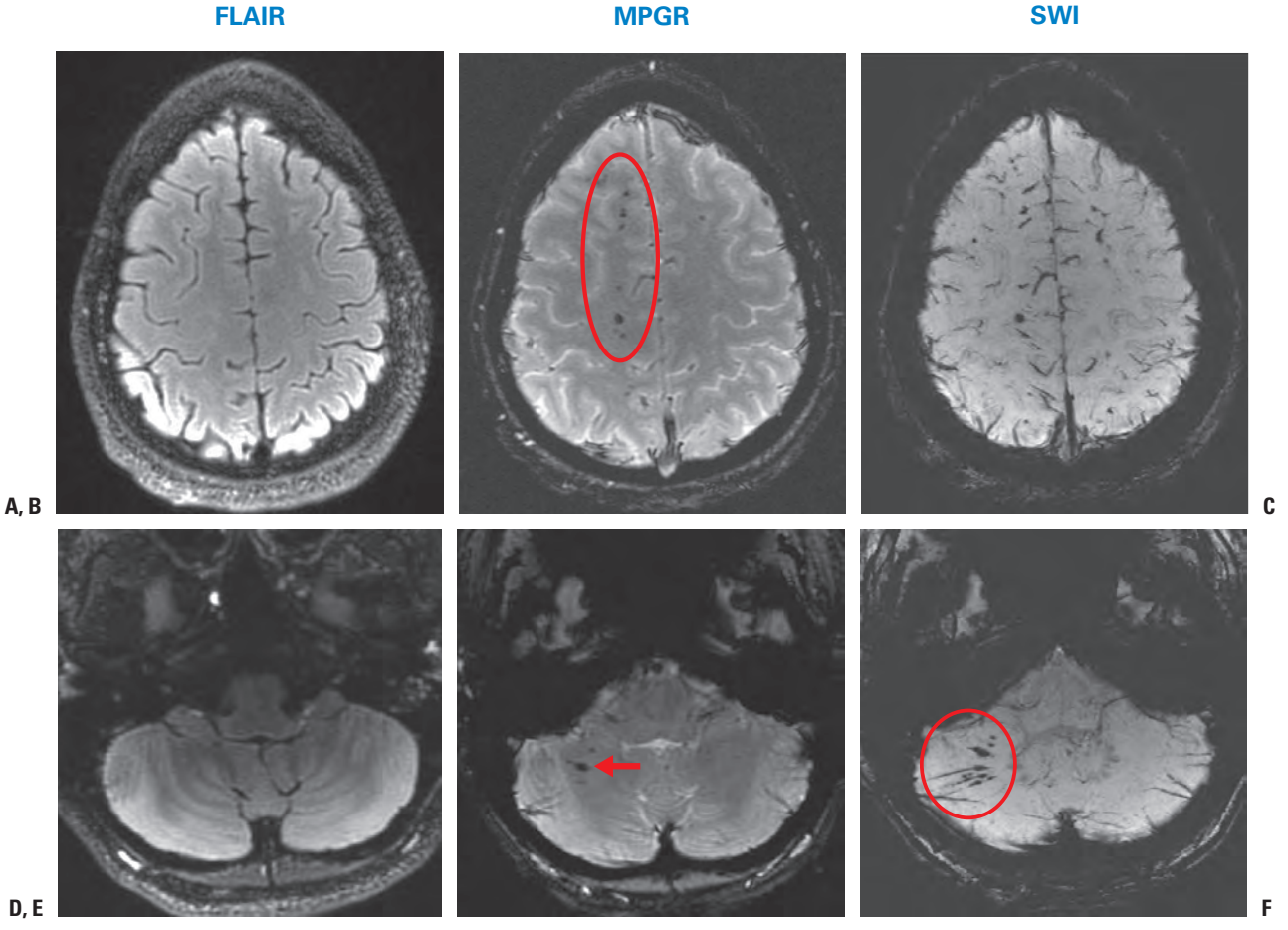


Figure 5.21. **Blast TBI.** This soldier presented with persistent balance problems, short-term memory loss, and diplopia 6 weeks after suffering mild TBI from an IED explosion. He had no recollection of the explosion event and suffered mild retrograde amnesia. **A.** Axial FLAIR image shows no abnormality. **B.** Corresponding GRE image demonstrates multiple right frontal subcortical microhemorrhages (*circle*). **C.** Corresponding SWI sequence is somewhat limited due to the signal loss from normal venous structures, which thereby decreases the conspicuity of the lesions seen in (B). **D.** Axial FLAIR image through the cerebellum is normal. **E.** Corresponding GRE image shows several petechial microhemorrhages within the right cerebellar hemispheric white matter. **F.** SWI imaging increases the conspicuity of these lesions (*circle*). (Courtesy of Gerard Riedy, MD, PhD, Walter Reed National Medical Center).

★**KEY POINT** In the absence of an occipital skull fracture, the cerebellum is rarely injured in civilian blunt TBI.

"Blast-Plus" Brain Injury (Interval Cerebral Atrophy)

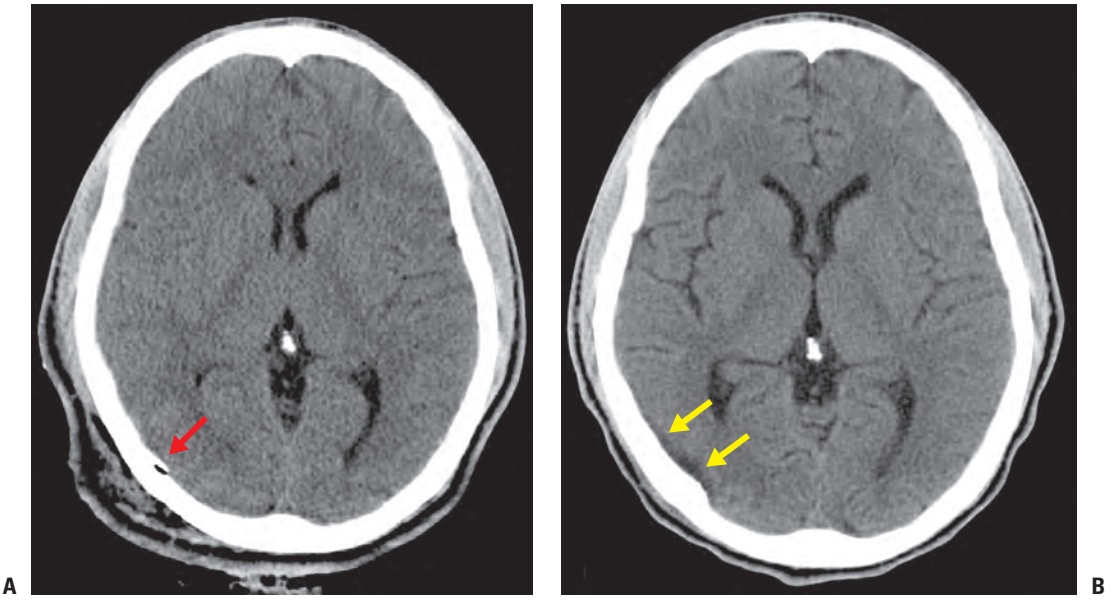


Figure 5.22. Interval Development of Cerebral Atrophy. This 30-year-old male is status-post an IED explosion. **A.** Noncontrast axial CT on admission shows a minimally depressed skull fracture beneath a right occipital scalp laceration but no focal intracranial abnormality. **B.** Two-month follow-up CT shows an interval increase in size of the cerebral sulci, cisterns, and ventricles, consistent with diffuse cerebral atrophy. A small focus of encephalomalacia of the cortical surface is identified at the site of the prior fracture (*arrows*).

★**KEY POINT** TBI is a well-known cause of global cerebral volume loss. It is unknown whether the primary blast force exacerbates the atrophic process.

"Blast-Plus" Brain Injury (Chronic)

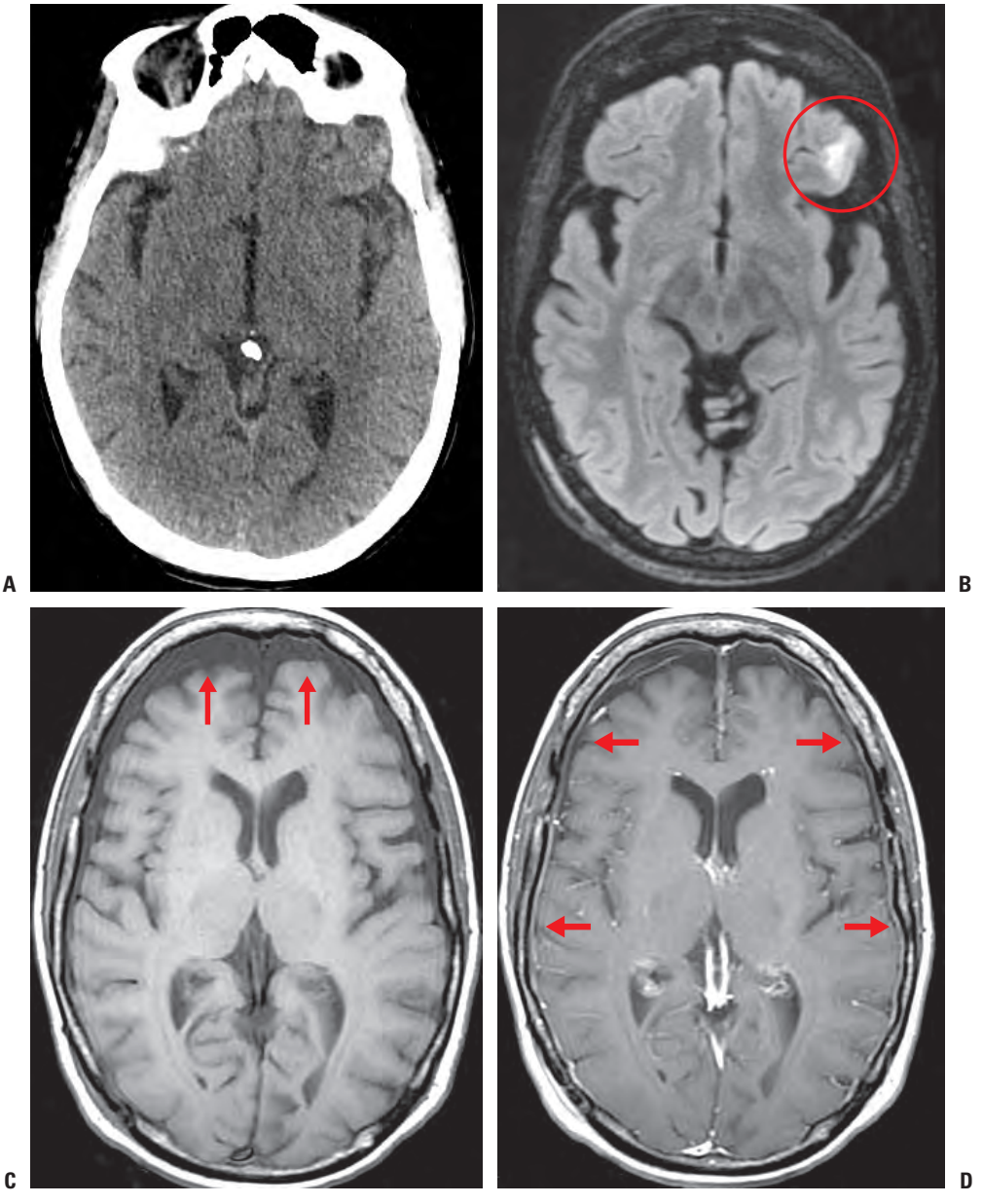
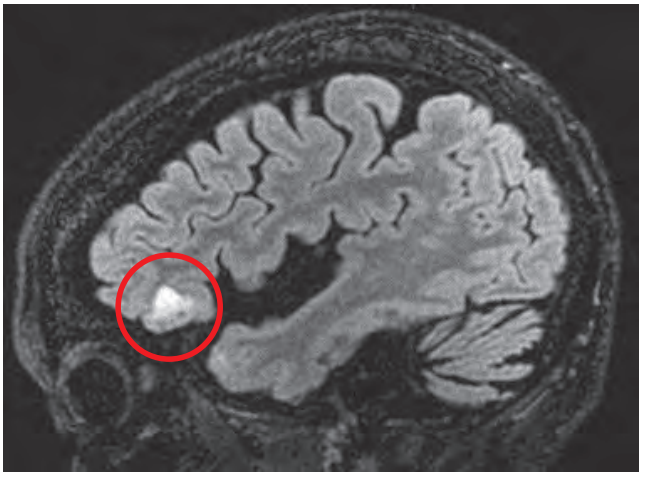


Figure 5.23. Remote Cortical Contusion, Atrophy, Chronic Subdural Hematoma. This 29-year-old male presented with a seizure 2 years status-post IED explosion. He remembered having hit his head at the time of the injury but denied loss of consciousness (LOC). **A.** Noncontrast axial CT shows abnormal global volume loss for a patient this age but no focal abnormality. **B.** Axial FLAIR MRI performed 1 week later and obtained at the same level reveals a focal hyperintense lesion within the left orbitofrontal lobe (*circle*). Even in retrospect, it is impossible to see this lesion on the corresponding CT. **C.** Noncontrast axial T1-weighted MRI at a higher level shows mild prominence of the bifrontal extra-axial space (*arrows*). **D.** Contrast-enhanced T1-weighted MRI at the same level reveals subtle diffuse thin dural enhancement (*arrows*), consistent with dural thickening from a prior subdural hemorrhage. (*Continued*)



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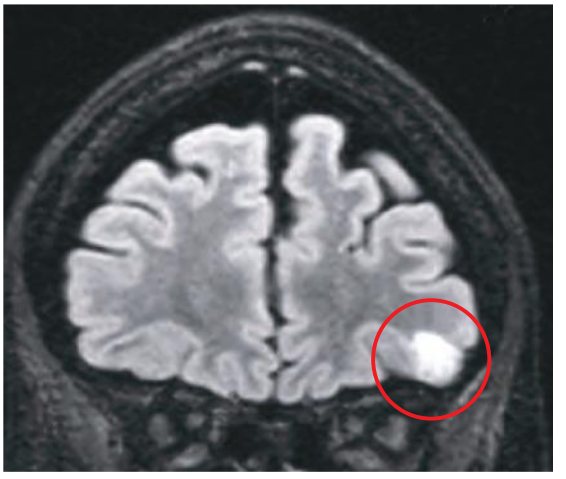

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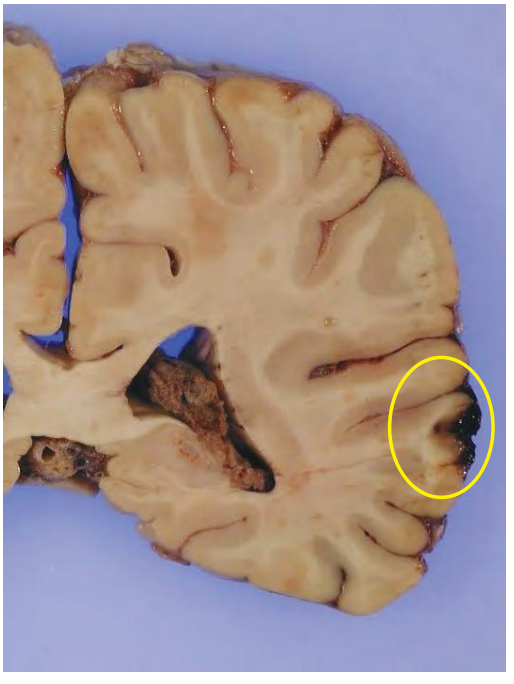


G

Figure 5.23. (*Continued*) **E.** Parasagittal and **(F)** coronal FLAIR MRI shows typical wedge-shaped cortical hyperintensity characteristic of a remote cerebral contusion (*circle*) that could be the cause of the patient's seizure. **G.** Coronal autopsy specimen in a different patient with a similar lesion shows a subtle hemorrhage involving the surface of the left middle temporal gyrus (*circle*), consistent with a small cortical contusion.

★**KEY POINT** This case illustrates how the typical superficial location of small cerebral contusions can make it difficult to identify them on CT. It also illustrates how contrast-enhanced MRI can help detect recent and remote subdural hemorrhage.

Advanced Neuroimaging Techniques in Blast TBI

In contrast to the obvious findings present on CT, MRI, and angiography shown in the previous examples, the imaging manifestations of blast brain injury are frequently more subtle and controversial. This is an area of active research that has accelerated the use of advanced imaging modalities in TBI. DTI, fMRI, MEG, MSI, SPECT, and FDG-PET have all been used to evaluate these patients, but in this author's opinion, as of 2013, these advanced neuroimaging techniques are not quite ready for the routine clinical setting.

Diffusion Tensor Imaging

In an unrestricted environment, diffusion is isotropic; the random motion of water molecules is equally likely to take place along any direction in space. DTI capitalizes on the fact that water diffusion in the brain is anisotropic. Less hindered by cell membranes, myelin, and other cytoarchitectural barriers, water molecules diffuse along greater distances when oriented parallel to intact white matter fibers than they do when oriented perpendicular to them. In contrast to conventional diffusion-weighted imaging (DWI), in which the diffusion of water molecules is measured in 3 orthogonal directions in order to estimate only the average rate of diffusion, DTI uses measurements in at least 6 but typically 25 to 30 directions in order to characterize the orientational anisotropy of water movement. Because water diffusion follows axonal tracts, DTI is used to deduce axonal orientation and to create images of white matter tracts in the brain.[25] Because diffusion occurs on a submillimeter spatial scale on the order of fiber tracts, DTI parameters such as fractional anisotropy (FA), mean diffusivity (MD), axial diffusivity (AD), and radial diffusivity (RD) characterize microstructural changes in the white matter. Although the biophysical basis of these parameters is complex,[26] FA reflects both axonal density and myelin content, and MD quantifies the average rate of diffusion in all directions. AD and RD are derived parameters that conceptually represent the ease which water diffuses along or across the axons, respectively.

DTI has provided important insights into TBI over the past decade, particularly concussions. It is the study of choice when evaluating the integrity and directionality of white matter fiber tracts, and it has been extensively applied as a research tool in the study of TAI.[27–32] When axons are injured, the cytoskeletal network impairs axoplasmic transport, causing changes in diffusivity that can be visualized and quantified on DTI.[33] Recent studies suggest that the extent of damage to white matter structures on DTI may correlate with the extent of cognitive impairment and functional outcome following TBI.[29,30] The genu of the corpus callosum appears particularly vulnerable, and changes in the corpus callosum are related to the clinical severity of the head trauma.[34] Several DTI studies have revealed an abnormal decrease in white matter FA when the corresponding, conventional MRI studies were normal.[27–29,35–37] DTI has been shown to be more sensitive than conventional 3T MRI sequences in detecting TAI. Furthermore, DTI parameters change over time; follow-up DTI examination in most patients shows partial or complete correction/resolution of many areas with diminished FA within 30 days of injury.[33] This normalization of quantitative FA measurements is not interpreted as definite evidence for regeneration of neurons; rather, it suggests that a cellular repair mechanism may have corrected the cytoskeletal dysfunction or misalignment, abating a permanent disconnection of the

injured axons. In chronic TAI, FA is frequently reduced. Recently, researchers have made significant progress in mapping the brain's inordinate number of connections, in what has been termed the *connectome*. The high interconnectivity of the human brain might minimize the effects of local damage, such that the multiple hubs function as an integrated unit able to compensate for focal impairment.[38] On the other hand, a small-scale injury could have major functional repercussions because one disrupts not just that local portion of the brain, but a large interconnected network.

While DTI is providing remarkable insights into TBI, many believe that there is an irrational exuberance for it in the literature, especially for 3D color tractography (**Figs. 5.24, 5.32,** and **5.33**).[39] Abnormalities within the tractogram, although visually very appealing, are not specific for TBI. Indeed, a normal Virchow–Robin space can misleadingly appear to prune a tract, as easily as a TAI lesion can. Additionally, small differences in the techniques and parameters used can lead to wide variability in the white matter tracts generated with DTI tractography. For example, tractography images are very prone to thresholding, and one needs to know the connectivity threshold that was used before the data can be interpreted and trusted. Thus, images can be made to look a certain way simply by varying the parameters that are used for data acquisition and imaging processing. Moreover, the sensitivity of fiber-tracking algorithms to many physical and computational variables is still poorly understood in normal brain and even less so in injured tissue.

Multimodality Imaging (including DTI) in TBI

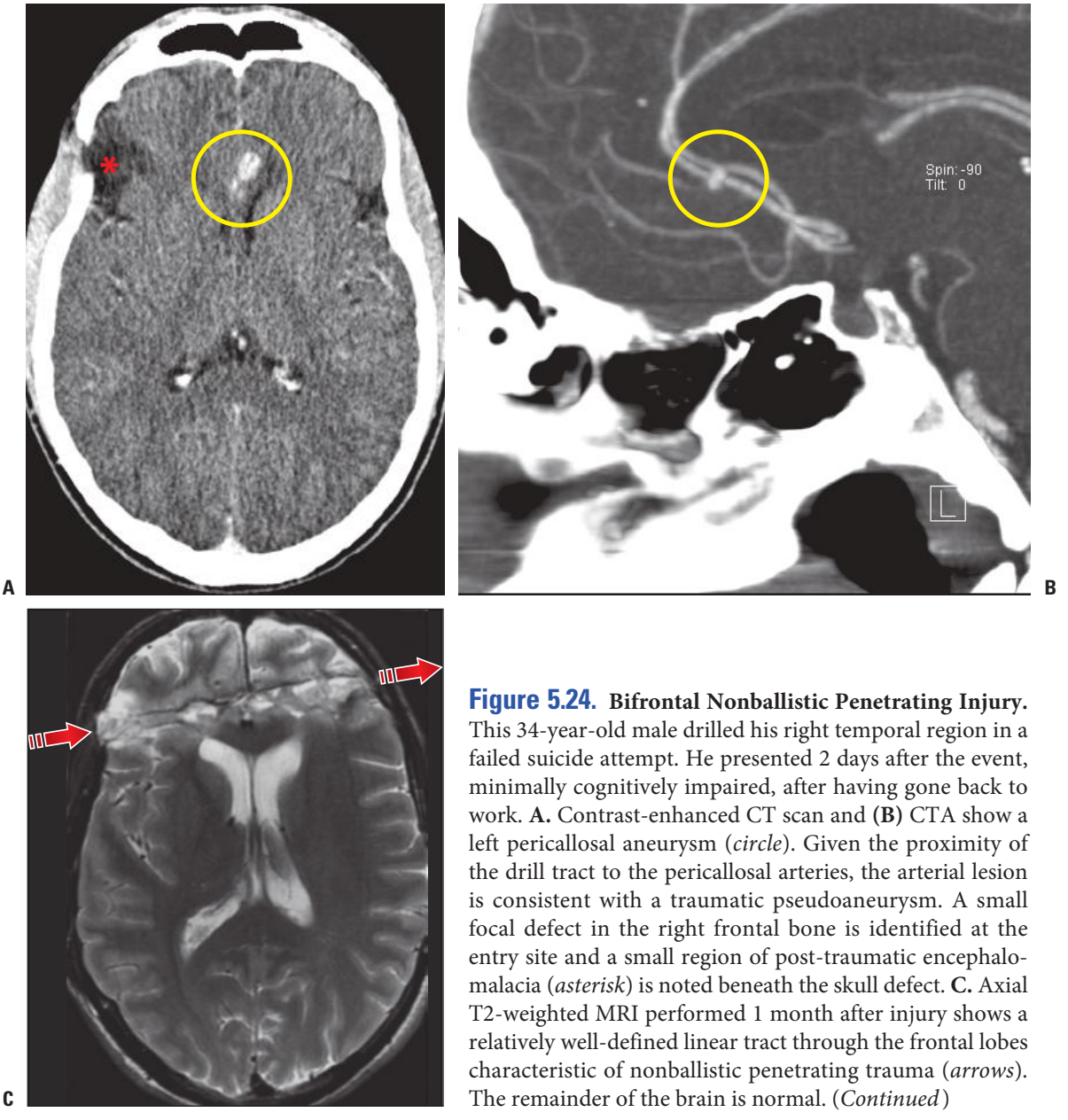


Figure 5.24. Bifrontal Nonballistic Penetrating Injury. This 34-year-old male drilled his right temporal region in a failed suicide attempt. He presented 2 days after the event, minimally cognitively impaired, after having gone back to work. **A.** Contrast-enhanced CT scan and **(B)** CTA show a left pericallosal aneurysm (*circle*). Given the proximity of the drill tract to the pericallosal arteries, the arterial lesion is consistent with a traumatic pseudoaneurysm. A small focal defect in the right frontal bone is identified at the entry site and a small region of post-traumatic encephalomalacia (*asterisk*) is noted beneath the skull defect. **C.** Axial T2-weighted MRI performed 1 month after injury shows a relatively well-defined linear tract through the frontal lobes characteristic of nonballistic penetrating trauma (*arrows*). The remainder of the brain is normal. (*Continued*)

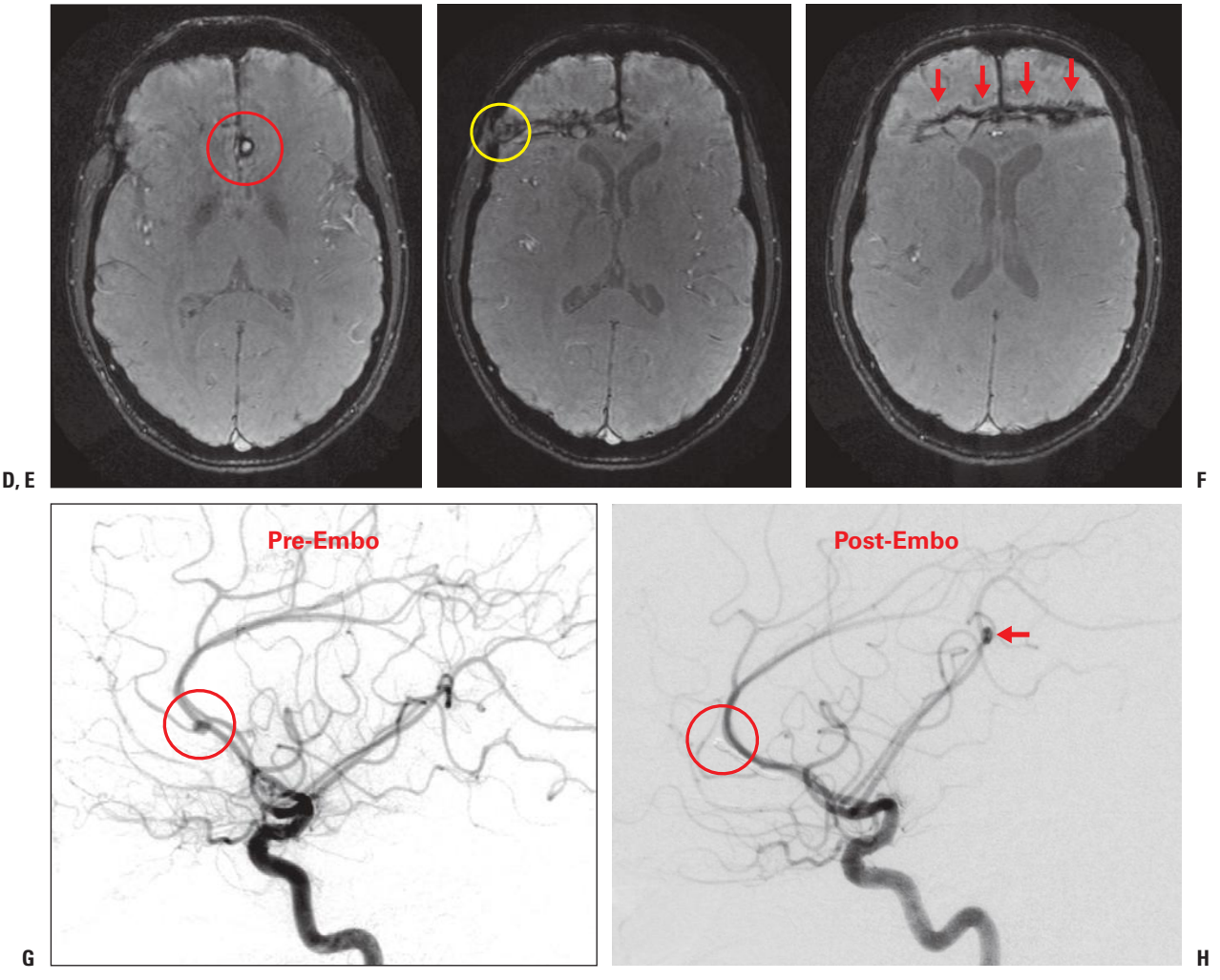
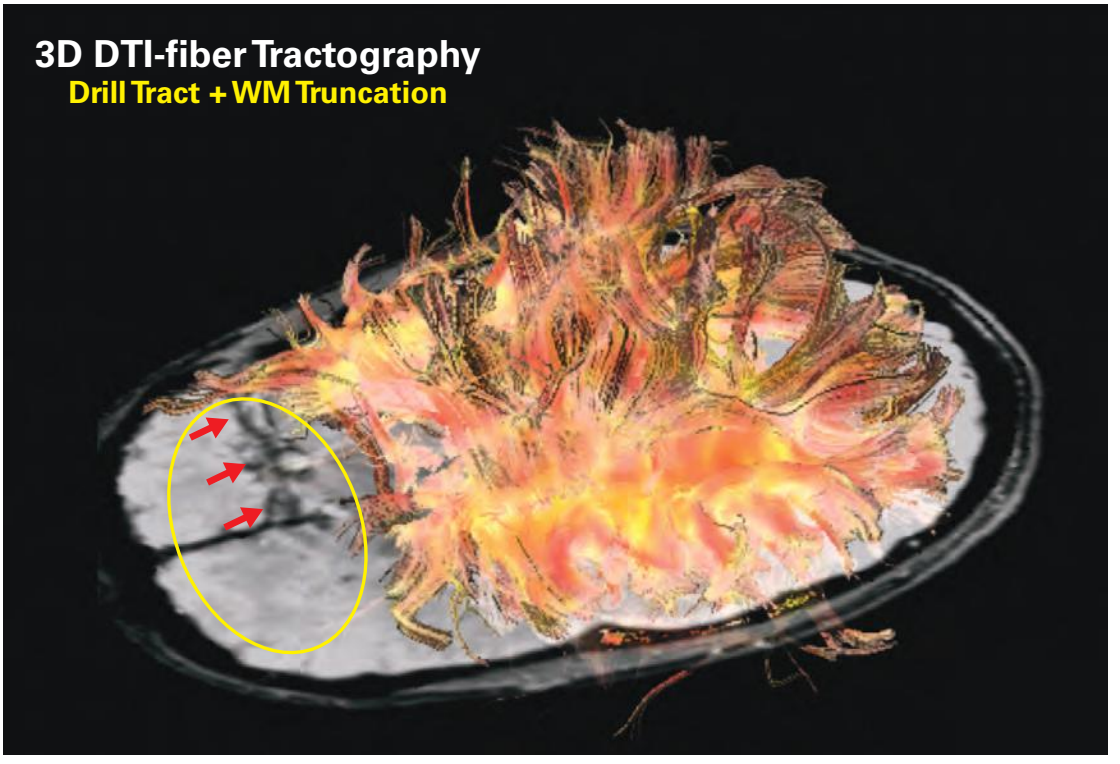


Figure 5.24. (*Continued*) **D–F.** High-resolution T2*-weighted images show a linear zone of abnormal signal intensity, consistent with edema and blood products (*arrows*). The aneurysm (*red circle*) is seen as central high signal with a surrounding rim of low-signal intensity. The entry site of the drill bit is seen as a small focal defect in the right frontal bone (*yellow circle*). **G.** Lateral view from an ICA catheter digital subtraction angiogram shows the pericallosal aneurysm (*circle*). **H.** Corresponding view from a postembolization angiogram demonstrates successful occlusion of the pseudoaneurysm (*circle*). The small contrast accumulation noted at the apex of the Sylvian triangle is a normal vascular loop and not a pseudoaneurysm (*arrow*). (*Continued*)



I

Figure 5.24. (*Continued*) **I.** 3D color MRI DTI-fiber tractography shows normal white matter tracks in orange, yellow, and black. Note the abrupt termination of frontal lobe white matter fiber tracts (*circle*) along the drill tract (*arrows*). (Courtesy of Marion Smits, MD, PhD, Rotterdam, NL.)

Numerous factors must be accounted for to reduce false positives and false negatives when interpreting DTI studies, with or without tractography. For example, FA and apparent diffusion coefficient (ADC) are affected by imaging variables such as field strength and resolution. Subject variability such as age, gender, and the presence or absence of preexisting disease can also be important. For example, patient age affects the interpretation of FA because low FA may be expected during early development and late senescence. In addition, there is an inherent 5% to 10% variation in most parameters between different scanners. Therefore, unless one does all of one's studies on the same scanner, with age, gender, and so on matched in control data (also from the same scanner), it is difficult to state with confidence how a measured diffusion parameter, such as ADC, FA, AD, RD, or fiber count, compares to other injured subjects or to normal controls.

DTI suffers from severe artifacts in the presence of magnetic field inhomogeneities. In addition, the significant T2* decay that can occur during long echo trains makes high-resolution acquisitions challenging. With the recent advance of parallel imaging technology, less spatial distortion and higher signal-to-noise ratios (SNR) can be achieved in a time-efficient manner with single-shot echo-planar imaging (EPI). More case-controlled studies with age-matched comparisons between patients and corresponding controls are necessary to assess the utility of these techniques to DTI imaging. Finally, the difficulties of postprocessing and the need for statistical analyses to evaluate the effects

on FA maps of spatial distortion correction induced by gradient nonlinearity, misregistration, image processing, and variable protocol parameters (e.g., b value, number of diffusion-encoding gradient directions, number of excitations, software data analysis package, etc.) are currently keeping DTI primarily in the research arena.

Diffusion kurtosis imaging (**DKI**) is a new extension of conventional diffusion imaging. With current DTI techniques, only a very small subset of the full diffusion map is sampled. Diffusion data acquired is analyzed as a single compartment, using Gaussian distribution curves. This Gaussian assumption, however, may not accurately apply to fiber tracks/crossings in biologic tissue, which may exhibit restricted, non-Gaussian diffusion.[40] DKI techniques measure water diffusion in non-Gaussian tissue and thus it is more accurate than DTI. Kurtosis is a dimensionless statistical measure of the departure of a distribution from a Gaussian curve. DKI is thought to be an imaging marker of tissue structure complexity (i.e., cellular compartments and barriers) with considerable promise for increasing our understanding TBI in the future.

Functional Magnetic Resonance Imaging

Functional MRI (**fMRI**) is an imaging technique that relies on the relationship between physiologic function, energy consumption, and blood flow to depict brain activity. It indirectly observes the activity of the brain via detection of alterations in the ratio of cerebral blood deoxyhemoglobin to oxyhemoglobin in response to particular tasks.[41,42] Blood oxygen level dependent (BOLD) signal is the MRI technique used to detect deoxyhemoglobin to oxyhemoglobin ratios to create functional imaging maps. fMRI relies on the principle that changes in the BOLD signal are caused by changes in CBF, which in turn are thought to be caused by changes in neuronal activity. As neuronal activity increases, blood flow overcompensates such that the local blood oxygenation, in the form of oxyhemoglobin, actually increases and the deoxyhemoglobin concentration decreases. Deoxyhemoglobin is paramagnetic, whereas oxyhemoglobin is diamagnetic. Therefore, a decrease in the concentration of deoxyhemoglobin is reflected by an increase in magnetic resonance (MR) signal intensity on GRE images. This principle has been validated in animal studies, but the fundamental mechanisms underlying the coupling between neuronal activity and blood flow changes have not been elucidated. In TBI, abnormalities in fMRI signals could be caused by either abnormalities in neuronal activity (as is usually assumed), or by abnormalities in the coupling of neuronal activity to the regulation of cerebral blood flow (which is rarely assessed). In either case, it is important to emphasize that neither fMRI nor DTI directly images brain anatomy or brain activity. Instead, each is the result of many layers of analysis and interpretation, far removed from the raw data.

fMRI can be performed at either 1.5T or 3T, but higher field strength is preferred. A high-resolution, 3D-spoiled gradient recalled (SPGR) acquisition in the steady state, T1-weighted, whole brain study is initially obtained. Then, for fMRI data collection, gradient-recalled echo (GRE) EPI is performed, preferably with parallel imaging. Although resting state fMRI can be performed, most fMRI studies employ measuring the magnetic evoked response to a stimulated task. The magnetic evoked responses to the stimulated tasks (e.g., working memory) are subsequently coregistered with the high-resolution MRI. BOLD fMRI studies performed in patients with mild TBI who had normal routine MR structural imaging have shown differences in brain

activation during various tasks as compared with healthy control subjects (**Fig. 5.25**).[43–46] In most studies, compared with controls, patients with TBI show increased activity, suggesting adaptive mechanisms in this group. However, reduced brain activity in concussed individuals has also been noted. To date, TBI fMRI research has centered primarily on the assessment of deficits after mild TBI, and these studies are advancing our understanding of the brain's ability to reorganize after injury.[47,48]

During most MRI procedures, there is no need for interaction between the physician and the patient. This is contrasted by fMRI, where the physician must assess the neurologic status of the patient and select an appropriate task and protocol for the study based on the patient's neurologic deficits and cognitive limitations. Further, the paradigm must be delivered to and successfully performed by the patient while in the MRI machine. A major challenge facing task-related fMRI studies is that the changes in BOLD signal are strongly dependent on how well the task is performed. Thus, interpretation of fMRI changes for TBI patients may be confounded by a brain-injured patient's inability to fully cooperate with a task. Cognitive impairments, such as concentration difficulties, working memory deficits, susceptibility to fatigue, and depression arising as a result of the TBI may lead to poor task performance during fMRI studies. These may be the same functional deficits that the fMRI is attempting to study. Thus, distinguishing whether patient cooperation or neuronal tissue injury is the cause of BOLD signal changes during an fMRI study may be difficult. Careful selection of the stimulated task in fMRI for TBI patients is also critically important. Unfortunately, residency and fellowship training and annual scientific society meetings generally do not provide educational sessions on how to optimally devise and perform fMRI tasks. Resting-state fMRI is a new technique that does not depend on patient interaction. Preliminary studies have shown disrupted default-mode network connectivity in patients with mild TBI.[49,50]

Functional Magnetic Resonance Imaging (fMRI) in Repetitive Head Injury

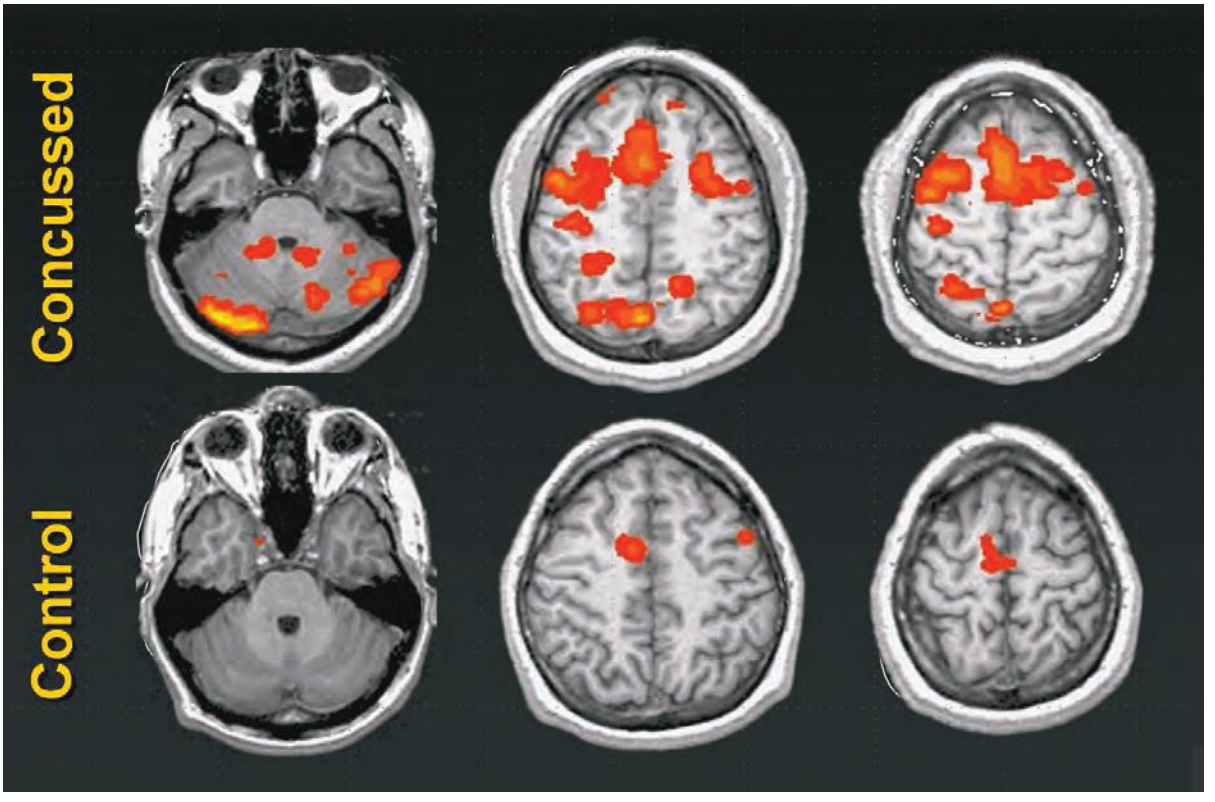


Figure 5.25. **fMRI in Concussion.** This figure demonstrates within subject differences for two football players, one who experienced a concussion **(top)** and one who did not **(bottom)**. Both players were imaged prior to the start of the football season. The concussed player was imaged again within a week following his concussion. The control player was imaged again at the end of the season. Colored areas show cortical and subcortical regions where the second imaging session resulted in significantly greater activation than the first imaging session. Note significantly greater activation in the concussed player as compared with the nonconcussed player. (Courtesy of Kelly J. Jantzen.)

Many of the limitations described earlier for DTI also exist for fMRI. Both are powerful tools, but there is a need for improved standardization of data acquisition and analysis protocols. A major limitation of fMRI is unintentional bias, which is inherent to the data processing. As one processes the data, input from the analyst is required to optimize the data into interpretable images, and the resultant images can change or vary depending on that input. For example, the signal that fMRI detects is noisy, and researchers must statistically process the data in order to make the resulting data interpretable. One of the most common approaches is known as spatial smoothing, which involves averaging the activity of each brain region with that of its neighbors. Recent studies of spatial smoothing have suggested that traditional methods of fMRI analysis may be inaccurate.[51] Another

criticism of fMRI in TBI patients is the lack of a baseline (i.e., pretrauma) study. Therefore, like DTI, fMRI has been used in number of research studies of TBI patients but is not yet part of routine neurotrauma clinical care.

Magnetoencephalography

Magnetoencephalography (MEG) is a noninvasive functional imaging technique with high temporal resolution (<1 millisecond) and spatial localization accuracy (2 to 3 millimeters) at the cortical level. MEG provides a selective reflection of activity in dendrites oriented parallel to the skull surface. MEG is predicated on the fact that electrical currents flowing within dendrites give rise to a surrounding magnetic field that can be measured by superconducting quantum interfering devices (SQUIDs). Unlike normal brain tissue, which generates alpha waves with frequencies above 8 Hz, brain-injured tissue generates abnormal low-frequency delta waves (1 to 4 Hz), and these can be directly measured and localized using MEG. MEG can detect abnormal, focal slowing when other imaging modalities are normal. Indeed, MEG appears to be more sensitive than conventional MRI and SPECT in its ability to detect abnormalities in TBI patients. Abnormal MEG delta waves have been observed in subjects without an obvious DTI abnormality, indicating that MEG may also be more sensitive than DTI in diagnosing mild TBI.[52] Unfortunately, there are currently only about 20 whole-head MEG systems in the United States and fewer than 150 systems installed worldwide. The limited availability of MEG systems, not only for TBI research but also for potential clinical care, will likely be a significant impediment to advancement of MEG applications for TBI for, at least, the near future. **Magnetic source imaging (MSI)** utilizes MEG to localize weak magnetic signal generated by neuronal electrical activity and then integrates the electrophysiologic data into anatomic data obtained with conventional MRI (**Figs 5.26**, **5.30,** and **5.44**). Simplistically, MSI = MEG + MRI. In two recent studies, MSI showed abnormal low-frequency magnetic activity in mild TBI patients with postconcussive syndromes.[53,54] These abnormal slow waves are thought to originate from cortical gray matter areas that have undergone deafferentation from white matter axonal injury. Application of MSI in the evaluation of TBI has been lacking due to the limited availability of MEG and mainly due to cost. Additional research is necessary before MSI is adopted in the clinical setting.

Magnetoencephalography (MEG) and Magnetic Source Imaging (MSI) in TBI

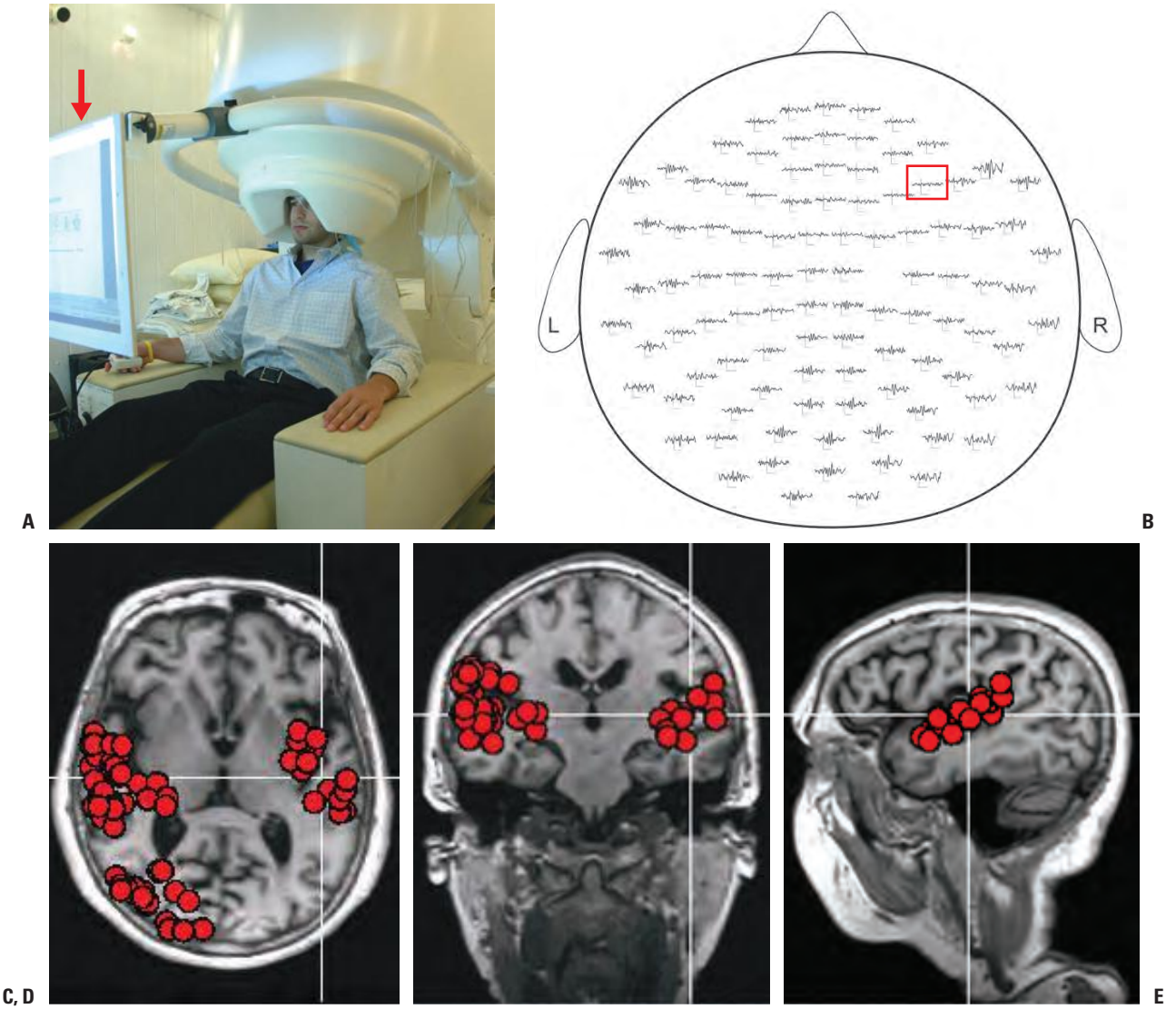
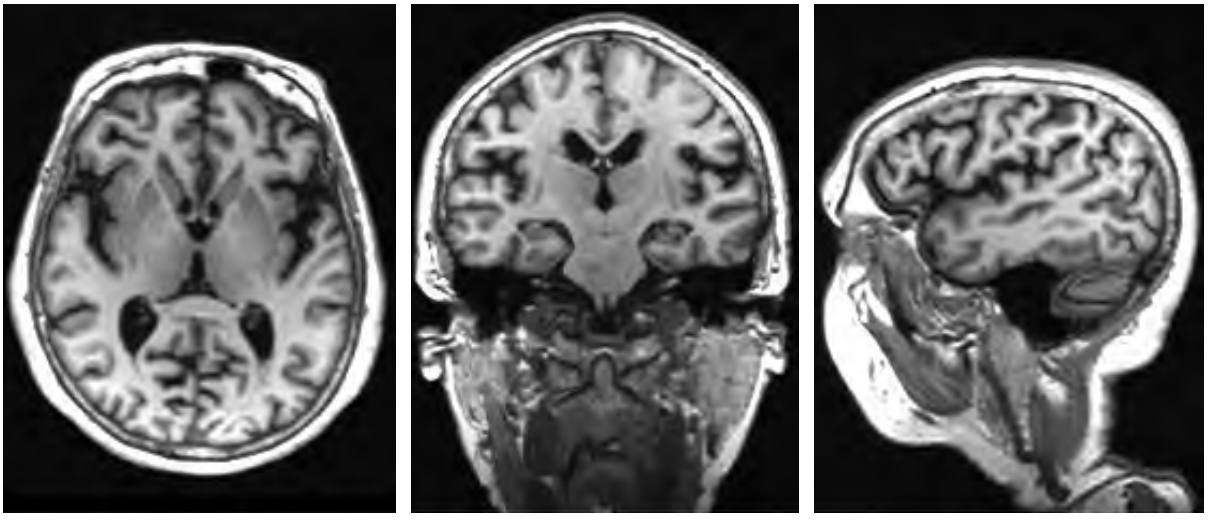


Figure 5.26. Magnetoencephalography (MEG) and Magnetic Source Imaging (MSI) in TBI. A. Photograph of a patient undergoing an MEG scan. Note the interactive screen directly in front of the patient (*arrow*) and the resemblance of the MEG scanner to a giant hairdryer. (Courtesy of the National Institutes of Health, Department of Health and Human Services.) **B.** Illustration of the neuronal electrical activity acquired from the MEG scanner, which will subsequently be used to formulate the MSI. Note the resemblance of the data to an electroencephalogram (EEG). The red box outlines a sample brain wave acquired from the right frontal lobe. **C.** MSI images in the axial **(C)**, coronal **(D)**, and sagittal **(E)** planes in a 57-year-old patient who suffered mild TBI from a fall. The red dots represent abnormal slow waves identified in the MEG data. (*Continued*)



F, G H

Figure 5.26. (*Continued*) **F–H.** These images are the same T1-weighted MRI images without the superimposed MEG data. Other than mild sulcal and ventricular enlargement for a patient this age (i.e., global volume loss), the images are normal. This patient had normal CT, DTI, and conventional MRI examinations.

Magnetic Resonance Spectroscopy

Magnetic resonance spectroscopy (**MRS**) noninvasively measures the relative amounts of metabolites in brain tissue, which are quantified from a small selected volume of tissue (voxel). Metabolites are most readily measured with proton (^{1}H) MRS, although carbon-13 and phosphorus-31 MRS can also be performed. The most common metabolites measured by proton (^{1}H) MRS include *N-acetylaspartate* (NAA), *creatine* (Cr), *choline* (Cho), *glutamate* (Glu), *lactate* (Lac), and *myoinositol* (mI). The measurement of brain neurochemistry in head trauma is based on the principle that primary and secondary TBI insults result in alterations in cellular metabolism, thereby leading to changes in concentrations of various brain metabolites. Metabolite concentrations are generally measured as areas under the resonance signal for that metabolite. They are expressed as ratios, relative to similar measurements for Cr or Cho that are used as controls. The metabolites displayed from right to left are NAA, Cr, Cho, and mI (**Figs. 5.27** and **5.45**). A line drawn to connect the peaks of the metabolites is called *Hunter's angle*. In normal patients, Hunter's angle has a 45-degree slope. This aids in the evaluation of normal versus abnormal MRS, although it is not specific for a pathologic diagnosis.

MRS is another tool that can identify abnormalities in the setting of a normal conventional MRI exam.[55–58] A reduction in NAA, a marker of axonal and neuronal functional status, typically occurs in areas of injured brain after TBI. Changes in NAA over time following injury have been shown to reflect the dynamic nature of recovery after TBI. NAA remains low in patients with poor recovery and returns to normal in patients with good outcomes.[59–62] Decreased NAA can result from neuronal loss or from neuronal mitochondrial dysfunction. Neuronal mitochondrial dysfunction can be reversible, and NAA signal may recover after the resolution of energy failure induced by the traumatic event. A reduction in the NAA/Cr ratio may be seen in contused brain within 24 hours after TBI.[63] Perturbations in Glu, a key neurotransmitter, are also seen following TBI. Cho is a precursor

for cell membrane synthesis and is considered a surrogate indicator of membrane structural integrity. It is typically increased in TBI, and this is thought to be due to myelin injury and accumulation of membrane myelin degradation products after the white matter shearing injury. A reduction of NAA and an elevation of Cho correlate with the severity of injury as measured by the Glasgow Coma Scale (GCS) and duration of post-traumatic amnesia.[64] Cr, a composite signal consisting predominately of Cr and phosphocreatine, is considered to be a measure of cellular density and is especially high in glial cells (e.g., post-traumatic gliosis). Cr is also a marker of cell energy metabolism and mitochondrial function. Increased Cr levels may be part of a repair mechanism associated with increased mitochondrial function in areas of injury. Lactate, a by-product of anaerobic glycolysis, may be diffusely elevated in TBI and has been correlated with a poor clinical outcome. mI is typically elevated and is also associated with a poor neurologic outcome. The reasons for its elevation remain unclear but may be due to astrogliosis or to a disturbance in osmotic function.

Magnetic Resonance Spectroscopy (MRS) in TBI

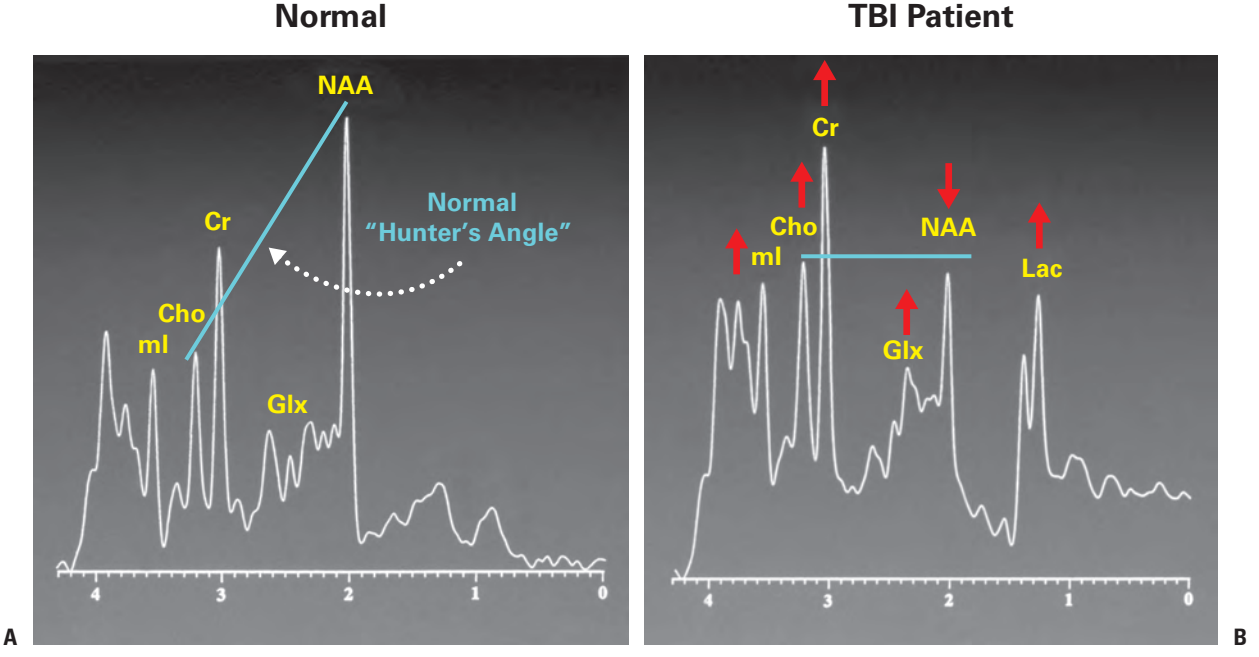


Figure 5.27. Magnetic Resonance Spectroscopy in TBI. A. Normal MR spectrum. The metabolites displayed from right to left are N-acetylaspartate (NAA), creatine (Cr), choline (Cho), and myo-inositol (mI). A line drawn to connect the peaks of the metabolites is called *Hunter's angle* (*blue line*). Note how Hunter's angle has a 45-degree slope. This aids in the evaluation of normal versus abnormal MRS, although it is not specific for a pathologic diagnosis. **B.** Abnormal MRS in a TBI patient. Note the abnormal decrease in NAA and the abnormal increase in lactate (Lac), glutamate (Glx), Cho, and mI. In addition, Hunter's angle has lost its normal 45-degree slope (*blue line*).

Despite its proven role in neurooncology and metabolic disorders, MRS is not routinely performed in TBI and is usually reserved for experimental studies. Data interpretation is complicated by variability from factors including the severity of the initial injury, the time between injury and the MRS scan, the types and location of spectral acquisitions, and the outcome measures used to monitor recovery. In addition, results may have been influenced by technical factors involved in the acquisition and processing of MRS data. One cannot account for voxels with spectra that are so distorted that the identity of the metabolites cannot be determined. In addition, large amounts of blood products, as is frequently the case with TBI, can lead to voxels with no measurable metabolites. This later limitation may cause an underestimation of the overall effect of hemorrhagic damage. Further, because metabolite ratios are used instead of quantitative concentrations, changes in the concentration of a control metabolite (Cho or Cr) by the TBI injury process, can lead to errors in interpretation. This may explain why the Cho/Cr ratio is slightly lower in some brain regions in patients with poor outcomes, even though Cho concentrations are elevated in TBI and are proportionately higher in severe, as compared to mild injury. Finally, until MRS examinations are reimbursable by Medicare, the clinical use of this technique will continue to be limited.

Higher Field Strength (3T to 7T) Imaging

High-field MRI is becoming more commonplace in today's medical arena. The relatively new 3T machines lack some of the advantages that have evolved for 1.5T imaging, including larger bores and wider fields of view, but they are evolving quickly. Currently, claustrophobia and body size can limit the number of patients that can benefit from a 3T machine. In addition, because 3T images are more prone to susceptibility artifacts, surface contusions may be missed. At 7T, the imaging quality of the posterior fossa is very poor. Nevertheless, the increased SNR inherent in a 3T machine is roughly double than at 1.5T, and this higher SNR can be used to reduce image acquisition time, improve resolution, or achieve a combination of the two. High-end applications such as functional studies based on BOLD contrast, MRS, and DTI clearly benefit from 3T. Further, new-phased array coil systems combined with parallel imaging technique offer the promise of even faster scanning, which is particularly helpful in the world of trauma imaging.

Ultra–high-field strength systems are defined as having a static magnetic field strength (B_0) of 7T or higher. Currently, this includes 7, 8, and 9.4T whole-body human scanners, with FDA approval of field strengths up to 8T. The advantages of 7T MRI include the expected gains in SNR, improved spectral resolution in MRS, shorter scan time, and submillimeter visualization of microvasculature with phase-sensitive and susceptibility-weighted imaging techniques that have facilitated the detection of traumatic microbleeds. The downsides of higher field MRI include (1) safety issues related to the more powerful magnet, (2) the difficulty of performing spin-echo sequences because of the issue of specific absorption rates and geometric distortion, and (3) contrast resolution in conventional T1-weighted and T2-weighted images is inherently reduced due to shorter relaxation times. The success of ultra–high-field MRI in more mainstream imaging applications will hinge on the safety and effectiveness of the modality as well as its ability to cost-effectively address an unmet critical need. It has the potential to noninvasively integrate functional information with superior morphology and drive them together toward molecular imaging. Indeed, many physicians

believe that 3T is now considered the state-of-the-art field strength of choice over 1.5T for evaluation of most neurologic diseases and that a similar transition to 7T will continue to enhance our understanding of TBI, assist in defining MRI markers that are the most relevant to patient prognosis, and lay the groundwork for future trials of more effective treatments.

Single Photon Emission Computed Tomography

Single photon emission computed tomography (**SPECT**) is a nuclear medicine study that uses gamma-emitting isotopes such as xenon-133 (^{133}Xe) and technetium-99-m-hexamethylpropylamine-oxime (^{99m}Tc-HMPAO) to image cerebral perfusion. Brain SPECT imaging is performed with a gamma camera 2 hours following the intravenous injection of ^{99m}Tc-HMPAO. The normal adult brain shows a bilateral symmetric tracer distribution, with higher activities in temporal, parietal, and occipital cortices; the basal nuclei; thalami; and the cingulate gyrus. The discovery of significant changes in CBF in patients with TBI makes SPECT a promising tool to evaluate cerebral perfusion in patients with TBI.[65–67] Frontal and temporal lobe hypoperfusion is commonly seen in head injury, presumably due to the gliding effect of the brain over the contours of the underlying skull (**Figs. 5.28, 5.44,** and **5.45**). Cerebral hypoperfusion in TBI has generally been attributed to the cerebral edema that surrounds the damaged brain, limiting cerebral blood flow. However, it may also result from vasospasm, direct vascular injury, and/or perfusion changes due to diaschisis (or disturbances) in the coupling between neuronal activity and blood flow.[68] SPECT can show areas of perfusion abnormality following head trauma while corresponding conventional CT and MRI imaging studies are normal. Abnormal SPECT perfusion deficits have been shown to correlate more accurately than structural CT and MRI with the acute clinical status of the patient.[69,70] SPECT may also provide a better long-term prognostic predictor in comparison to CT or conventional MRI.[71] For example, multiple CBF abnormalities; larger alterations in CBF; and defects that involve the basal ganglia, temporal and parietal lobes, and brain stem on SPECT imaging have been associated with worse prognosis following TBI. Areas of acute areas of hypoperfusion in the acute stage have been associated with subsequent findings of brain atrophy at 6 months following injury. This suggests the possibility that secondary ischemic damage in the acute phases of injury may have detrimental long-term consequences.[66] ^{99m}Tc-diethylenetriaminepentaacetic (DTPA) brain scintiangiography can also be a useful tool to confirm brain death. When standard apnea tests are contraindicated, DTPA brain scintiangiography can be a useful adjunct to clinical criteria for brain death declaration because of its sensitivity, medicolegal acceptance, and ability to be performed at the bedside.[72] Despite the importance of these findings, SPECT imaging has some limitations. Due to its inherent low spatial resolution, SPECT is less sensitive than MRI in detecting small lesions. In addition, there is no standard protocol followed by all investigators for SPECT acquisition, processing, and interpretation of the data. There is also lack of quantitation of the regional cerebral perfusion using the current radiopharmaceuticals approved by the FDA. Therefore, SPECT imaging may be complementary to, but not a replacement for, MRI in the evaluation of TBI.

Single Photon Emission Computed Tomography (SPECT) in TBI

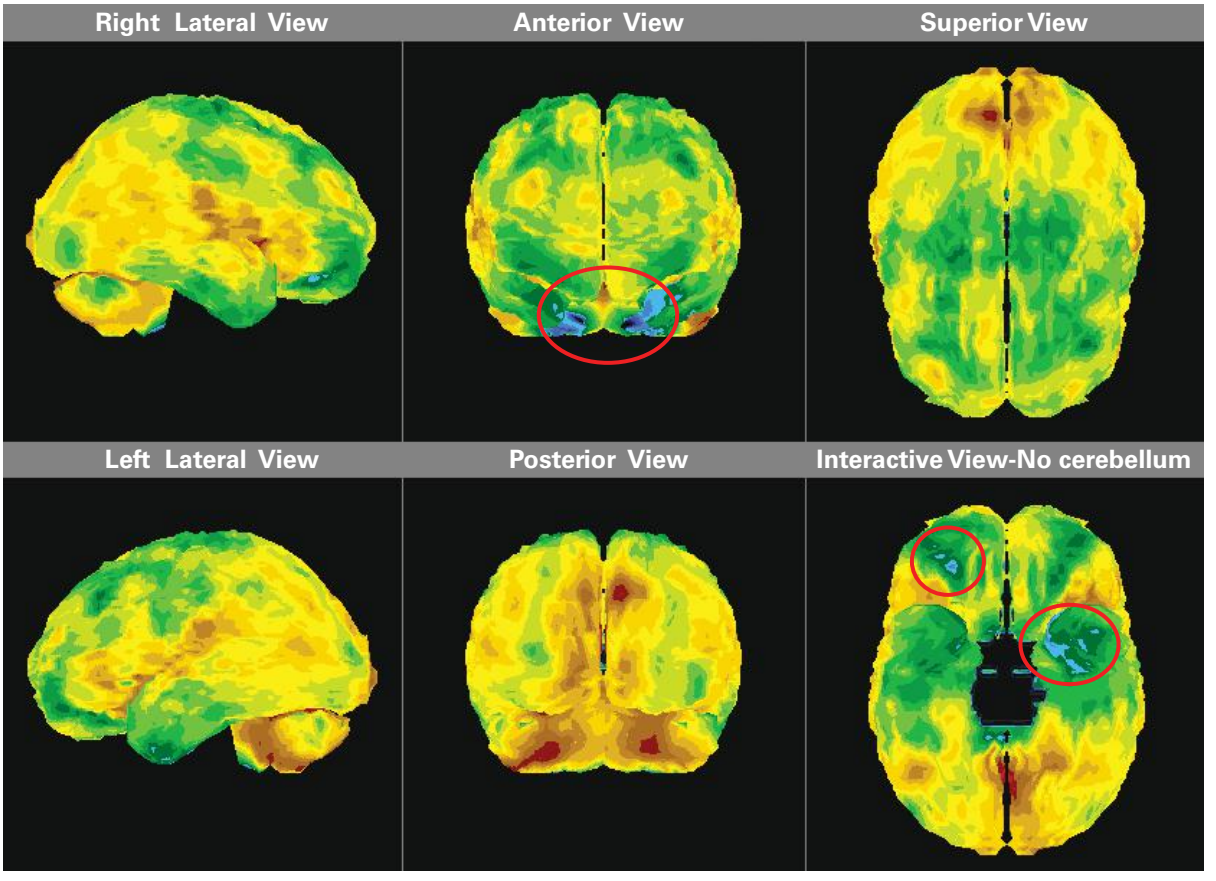


Figure 5.28. 3D Talairach Cortical Perfusion SPECT in TBI. Note the bilateral, relatively symmetric, decrease in cerebral perfusion to the frontotemporal lobes (*green areas*). Conventional MRI (not shown) demonstrated very subtle post-traumatic encephalomalacia involving the anteroinferior left temporal lobe and the right orbitofrontal cortex. These areas are shown in blue on the SPECT study (*circles*).

Positron Emission Tomography

Positron emission tomography (**PET**) imaging uses spatial biodistribution of positron-emitting isotopes, commonly oxygen-15 (^{15}O), to measure cerebral perfusion and oxygen metabolism, and 2-[fluorine 18] fuoro 2-2-fluoro-2-deoxy-D-glucose (^{18}F-FDG), to measure cerebral glucose metabolism. Acutely injured brain cells show increased glucose metabolism (hyperglycolysis) owing to intracellular ionic perturbation, a fall in oxidative metabolism, and uncoupling of cerebral blood flow.[73] Therefore, FDG-PET permits us to directly image foci of brain dysfunction. By comparison, SPECT imaging measures cerebral perfusion, from which function is inferred. Following the initial hyperglycolysis state, injured brain cells show a prolonged period of regional hypometabolism, lasting up to 1 month. Based on the principle that regional glucose metabolism reflects the neuronal activity of the region, focal hypometabolism on PET indicates an area of neuronal dysfunction (**Fig. 5.29**). To account for the metabolic reduction, two main mechanisms have been proposed: (1) local neuronal loss

and (2) decreased neuronal activity due to disturbances in metabolic function of injured, but viable, neurons. PET technology enables functional brain imaging with significantly higher resolution than that achieved through perfusion SPECT analyses. Oxygen-15 positron emission tomography (^{15}O) can define potential ischemic areas after brain injury, which is associated with poor outcome.[74–76] Early human studies of PET imaging for TBI had limited success in demonstrating consistent results regarding regional glucose metabolism. Due to the heterogeneous nature of TBI, studies found both hypermetabolism and hypometabolism in the same regions across different TBI patients.[77] The metabolic abnormalities were also found to extend far beyond the lesions, and this, surprisingly, was especially noted for SDH and epidural hematoma (EDH).[78] Cortical contusions, intracerebral hematomas, and encephalomalacia all tend to show more regional metabolic abnormalities confined to the specific lesions. A more recent study has shown hypermetabolism within the cerebellar vermis of the injured brain.[79] Some studies have suggested that PET has a greater sensitivity than MRI for the detection of abnormalities in patients with mild and moderate TBI who have persistent cognitive or behavioral complaints.[68] Unfortunately, although PET provides information on cerebral metabolism, which can be invaluable to the study of TBI patients, it is relatively expensive to perform. Further, because an onsite cyclotron is needed to generate the radioactivity used, the availability of PET imaging is limited. The role of other radiolabeled neuroreceptor ligands to evaluate head injury by PET imaging is theoretically promising but remains to be determined.

Positron Emission Tomography (PET) Imaging in TBI

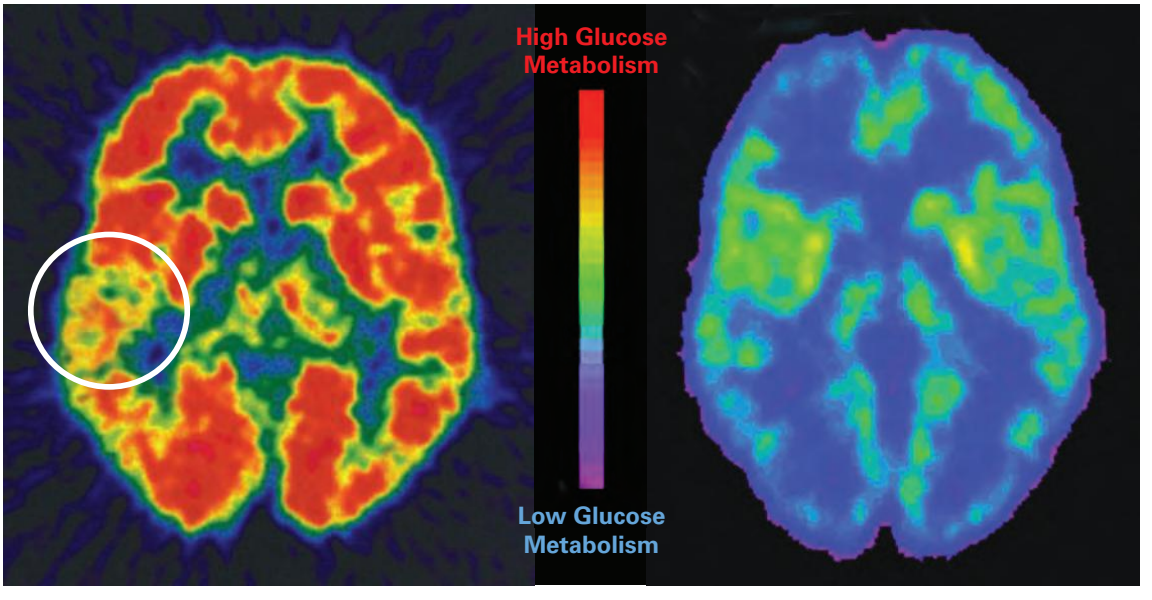


Figure 5.29. **Positron Emission Tomography Imaging in TBI. A.** Axial image in a 23-year-old male several months after a fall shows abnormal glucose hypometabolism within the right temporal lobe manifested by a focal asymmetric decrease in red coloration (*circle*) in comparison to the left temporal lobe. The corresponding CT and MRI studies were unremarkable. **B.** Axial image in a comatose 21-year-old several days following severe TBI shows a diffuse decrease in cerebral metabolism; note the complete absence of red coloration throughout the cerebral cortex.

In summary, DTI, fMRI, MEG, MSI, MRS, SPECT, and PET scanning have already provided important insights in the TBI research arena, and they hold tremendous promise for routine clinical application in the future. I truly believe that it's only a matter of time until many, if not all, of these technologies will be used to assist in the diagnosis of blast brain injury. However, there are just too many current unknowns and/or obstacles to allow them to be reliably used in today's routine clinical practice. Innumerable variables factor into the making of the images, and we simply do not know (yet) how to interpret the findings. We need to first establish an acceptable normal database that is controlled for demographic variables. Given the frequently heterogeneous nature of blast TBI, careful consideration must be made to control for clinical factors such as the injury characteristics, patient age, comorbid conditions, and the time since injury during the interpretation of imaging studies. Technical factors also need to be defined, including the development of protocols that are sensitive to TBI lesions and deficits in blast victims.[80] Therefore, further research with comprehensive analysis needs to be done before there is widespread adoption of these new techniques into our routine clinical imaging armamentarium. Fortunately, several investigators and centers are currently in the process of doing just that.

The majority of explosions result in "blast-plus" TBI, and only a handful of pure primary blast TBI (i.e., BINT) cases have been published (Table 5.5).

The current imaging manifestations of isolated BINT include the following:

- Decreased glucose metabolism within the cerebellum and brain stem has been identified on FDG-PET scans in soldiers suffering persistent postconcussive symptoms following repetitive blast exposure.[81] A recent mouse model of blast injury also demonstrated multifocal axonal injuries in these regions. Mild TBI caused by a non-blast mechanism is also associated with glucose metabolic depression. The duration of the hypometabolism reflects a period of increased cerebral vulnerability in which a second injury can dramatically worsen outcome.[82] Another recent PET study showed hypometabolism in the right superior parietal region in TBI patients who had suffered blast injury, as compared to blunt TBI veterans.[83] The results of this pilot study suggested that pure blast force mild TBI may have greater postconcussive sequelae, including deficits in attentional control and regional brain metabolism, compared to blunt mild TBI. The magnitude and duration of the hypometabolic phase after TBI varies with the severity of the injury and the age of the patient.[73,84] The mechanism underlying glucose hypometabolism also changes over time. Mechanisms active during the acute phase of injury may subside, and different mechanisms may become apparent as secondary cascades evolve and contribute to continued depression of glucose metabolism.
- Various TBI-relevant fMRI tasks have investigated the neuronal circuits affected by blast-related TBI.[85,86] These tasks evaluate the most commonly reported functional sequelae of blast TBI, such as problems with working memory, attention, and changes in emotional response. In addition to task-based fMRI methods, *resting state* connectivity analysis may provide information relevant to diffuse axonal injury due to blast-induced and non–blast-induced mild TBI.[87,88]
- One DTI study of mild to moderate blast TBI veterans did *not* show evidence of white matter damage despite the presence

of residual clinical symptoms in these patients.[89] In contrast, two other studies did identify abnormal DTI findings in U.S. military personnel troops with blast-linked trauma and concussion. Moore and colleagues[90] found a *more diffuse pattern of white matter damage* by DTI imaging, as compared to brain trauma victims with concussions caused by direct impact or acceleration. This diffuse pattern of white matter damage was identified as decreased FA *and* ADC. They also found signs of inflammation several months after the blast trauma, when most symptoms of concussion had typically disappeared. This finding suggested that there are prolonged subacute or chronic inflammatory effects following blast injury. These delayed effects may be related to diffuse synaptic loss and/or axonal impairments. DTI evidence of TAI has also been observed in soldiers with blast-related mild TBI, many of whom had normal conventional MRI studies.[91] Abnormalities were detected up to a year after injury, suggesting evolution not resolution of axonal injury. Of note, all of the patients in this study suffered "blast-plus" TBI, meaning that they were exposed to a primary blast force *plus* blunt force impact. Preferential injury to the cerebellum was noted, an area rarely affected in civilian mild TBI. DTI studies have also attempted to identify areas of metabolic dysfunction following blast TBI as measured by PET.[92] Decreased FA on DTI imaging has been shown to be significantly correlated with reduced glucose metabolism in regions of TAI. Damage to axons and/or decreases in synaptic connectivity can directly affect cerebral activity levels, cerebral glucose metabolism, and behavioral outcome.[93] A DTI study in combat veterans has further found a significant association between blast exposure, PTSD, and DTI abnormalities (manifested as decreased FA).[94] They speculated that brain changes detected on DTI may be due to neurochemical alterations induced by chronic stress and/or by subclinical brain injury from blast exposure.

- Abnormal MEG in blast TBI patients has been reported by Huang and colleagues[52,95] (**Fig. 5.30**). MEG slow waves, generated from the lateral prefrontal cortex (PFC), orbitofrontal cortex, anterior cingulate, and temporal lobe, were detected in blast-injured victims. These abnormal MEG slow waves have been attributed to cortical deafferentation due to TAI and decreased axonal fiber FA. DTI imaging in these patients showed that the AP-oriented diffusion in the left superior longitudinal fasciculus was thinner in the blast TBI patient as compared to a normal control. The routine MRI was normal. The authors concluded that MEG low-frequency source imaging may be useful to distinguish blast, from nonblast, TBI.

Magnetoencephalography (MEG) and Diffusion Tensor Imaging (DTI) of "Blast-Plus" Brain Injury

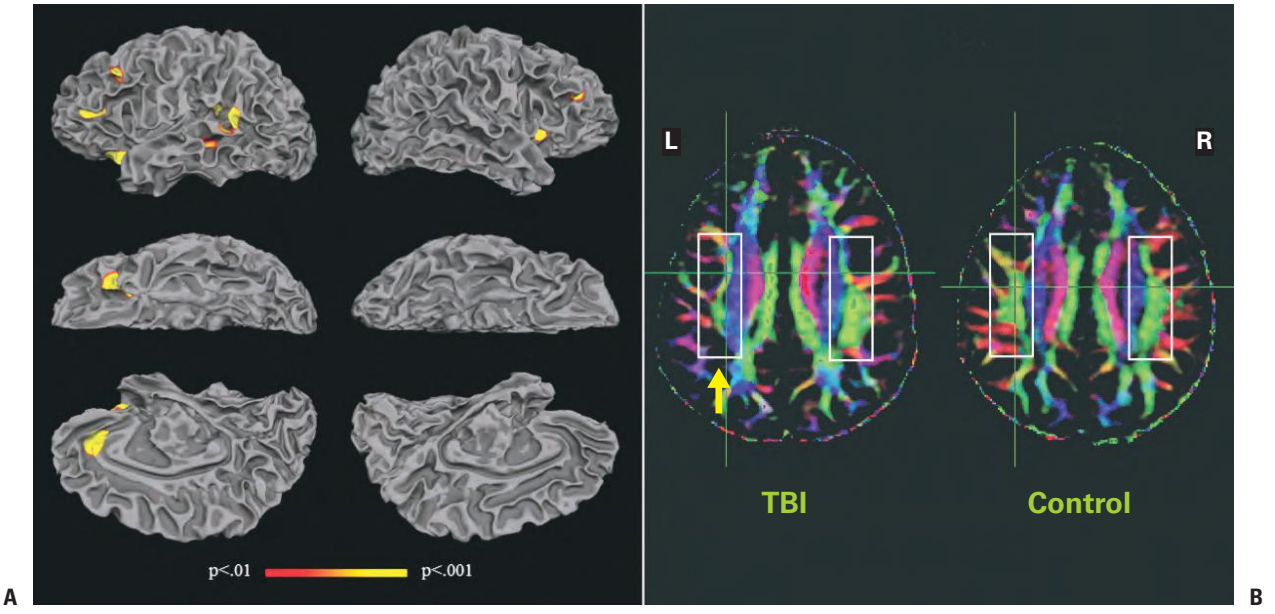


Figure 5.30. **Diffusion Tensor Imaging and Magnetoencephalography in Blast TBI.** This 27-year-old soldier experienced a brief LOC after his Humvee was hit by an IED. His conventional MRI was normal. **A.** 3D volumetric MRI with MEG registration performed 1 year later shows abnormal slow waves (delta waves) within the left lateral prefrontal cortex, orbitofrontal cortex, anterior cingulate, and left temporal lobe (*yellow areas*). **B.** DTI shows thinner AP-oriented diffusion in the left superior longitudinal fasciculus (compare the amount of green in the *white boxes* of the blast TBI patient (*arrow*) versus the normal control. (Courtesy of Roland Lee, MD, PhD.)

■ The first case report of BINT documented an extra-axial hemorrhage on CT following an industrial blast explosion (**Fig. 5.31**A).[96] The explosion did not cause any direct contact or impact to the patient. Two nonreported cases of combat BINT, both of which showed traumatic SAH, are also shown in Figure 5.31B,C.

Blast-Induced Neurotrauma (BINT)

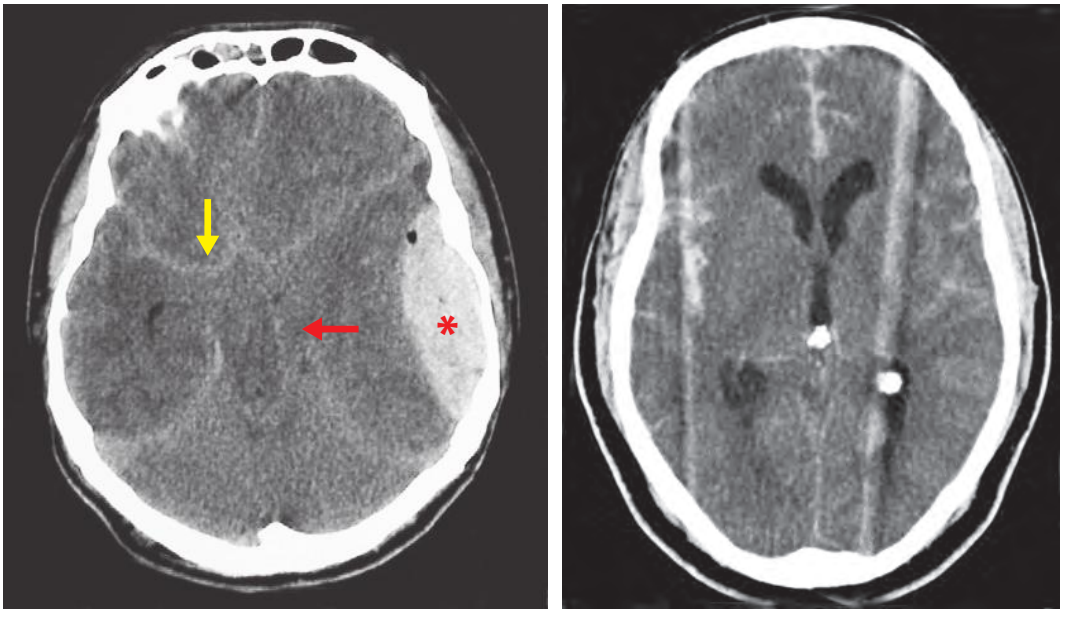
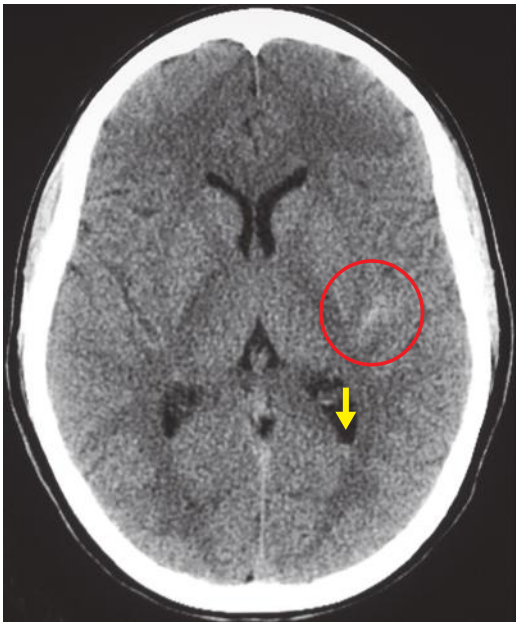


Figure 5.31. **Blast-Induced Neurotrauma. A.** This 36-year-old male is status-post a blast injury from a steam boil explosion. He was admitted to the emergency department (ED) with a GCS = 15. Other than a left tympanic membrane rupture, there were no blunt or penetrating wounds on physical examination. His GCS quickly declined to 8, and he underwent emergent CT imaging. Noncontrast axial CT images demonstrate a left temporal EDH (*asterisk*), complete effacement of the perimesencephalic cisterns with brain stem compression (*red arrow*), and high attenuation within the subarachnoid space, consistent with either true and/or pseudo–SAH (*yellow arrow*). A nondisplaced left temporal bone fracture was also identified. The patient underwent emergency neurosurgical evacuation of the EDH but died 3 weeks later. (Reproduced with permission from Serkan Yilmaz, Kocaeli University, Turkey.) **B.** This 21-year-old soldier died immediately following primary blast injury. His noncontrast CT is of limited quality due to technical factors and motion artifact. Nevertheless, it shows diffuse SAH and possible early loss of gray–white matter differentiation. (Courtesy of Roni Rooks, MD, LTC, MC.) (*Continued*)



C

Figure 5.31. (*Continued*) **C.** This 26-year-old soldier suffered hearing loss and headaches immediately following an IED explosion. He did not show evidence of secondary, tertiary, or quaternary blast trauma. Noncontrast axial CT shows subtle asymmetric high attenuation within the left sylvian fissure, consistent with acute SAH (*circle*). In addition, a small CSF–blood fluid level is identified within the left occipital horn, consistent with intraventricular hemorrhage (IVH) (*arrow*). No intra-axial abnormality is identified, and there is no evidence of scalp injury to suggest focal head impact or penetrating foreign bodies.

★**KEY POINT** Isolated primary blast brain injury, also known as blast-induced neurotrauma (BINT), appears uncommon. The majority of blast injuries to the brain involves variable combinations of primary, secondary, tertiary, and quaternary injuries, and is thus termed "*blast-plus" TBI*.

- Injury to the arcuate fasciculus identified on DTI tractography was the second case report of BINT.[97] (**Fig. 5.32**). In this case, a 23-year-old soldier was exposed to two separate blasts occurring 2 months apart. He was approximately 5 ft from the explosion in both cases, and he was wearing a helmet and goggles. After the second blast, he reported headaches, intermittent tinnitus, and conduction aphasia. Initial CT scanning was negative, and conventional MRI obtained nearly 3 years later was also normal. DTI tractography performed was positive for thinning of the left arcuate fasciculus fiber tracts. PET scanning showed subtle asymmetry in the same region. A follow-up DTI obtained 4 months later after intensive speech and language therapy demonstrated "slight sprouting around the locations that appeared to have been sheared." Although currently speculative, the change over time in white matter tracts observed using sequential DTI may reflect an improvement in connectivity.

Diffusion Tensor Imaging (DTI) in Blast-Induced Neurotrauma (BINT)

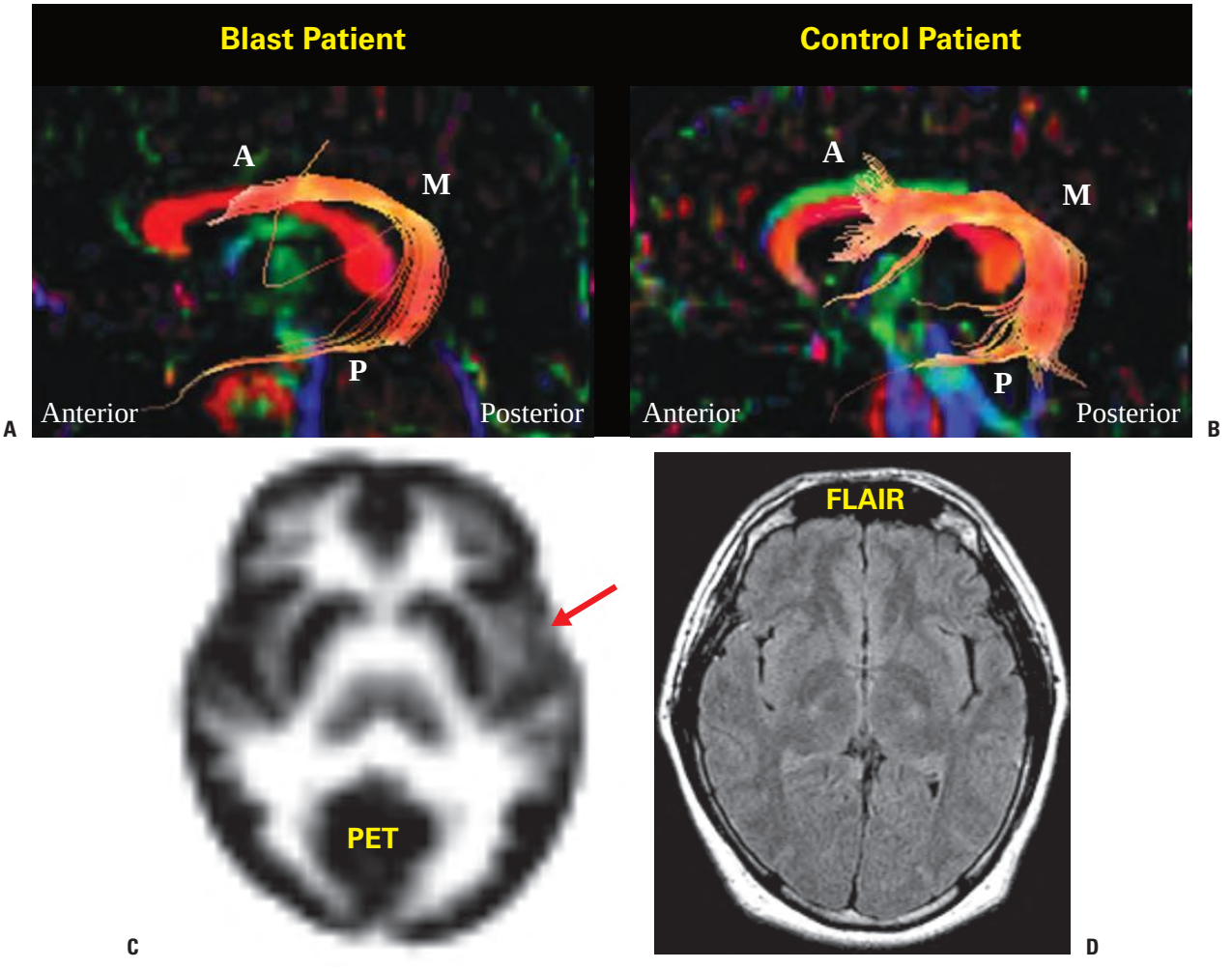


Figure 5.32. Arcuate Fasciculus Damage Seen on Diffusion Tensor Imaging in a Blast-Exposed Soldier. A 4T MR DTI in the sagittal projection demonstrates the left arcuate fasciculus (in orange) of a patient with BINT **(A)** versus a normal age-matched control patient **(B)**. Note that the patient's fiber tract is thinner than that of the control and how the control patient has extensive branching at both ends of the fasciculus. This blast-exposed patient presented with conduction aphasia (damage to the arcuate fasciculus is known to be associated with conduction aphasia). **C.** PET imaging shows subtle asymmetry in the temporal lobe with decreased activity in the region of the anterior termination of the left arcuate fasciculus (*arrow*). **D.** Corresponding FLAIR MR image is unremarkable. A, anterior end of the AF terminating at Broca's area; M, middle of the tract; P, posterior end of the AF terminating at Wernicke's area. (Courtesy of J. Wesson Ashford, MD, PhD; Yu Zhang, MD, and Les Folio, DO, MPH, Col [ret], USAF.)

★**KEY POINT** DTI has provided important insights into TBI over the past decade, and it has been extensively applied as a research tool in the study of traumatic axonal injury. However, numerous factors must be accounted for to reduce false positives and false negatives when interpreting DTI studies, with or without tractography (see above text).

■ The third-case report of BINT involved a focal cerebellar lesion with an ipsilateral internal auditory canal hemorrhage (**Fig. 5.33**).[98] The soldier was approximately 125 m from an explosion that occurred to her left. It was associated with a brief loss of situational awareness and vomiting. Immediately following the large blast, the patient experienced severe headache, ringing in the left ear and, aching ribs, but her neurologic examination upon receiving medical attention was normal. Transmission of the blast shock wave to the intracranial cavity was postulated to have occurred along the auditory canal, the path of least impedance. This is consistent with her symptoms of left-sided ear ringing. Her onset of vomiting in the acute phase is suggestive of possible cerebral swelling, a finding that may be more apparent in severe BINT. Imaging studies demonstrated abnormalities in the left cerebellum. It is possible that the cerebellar damage may have been induced by segmental arterial vasospasm or even an unidentified vertebral dissection secondary to blast wave propagation. As emphasized in Figure 5.21, injury to the cerebellum is very uncommon in civilian blunt TBI. Cerebellar injury from blunt TBI is usually limited to severe TBI or to injury associated with occipital skull fractures. Personally, during my 30 years of experience imaging TBI, I have not seen one case of cerebellar injury in a patient with mild TBI unless there was an adjacent skull fracture.

Primary Blast Brain Injury (Blast-Induced Neurotrauma [BINT])

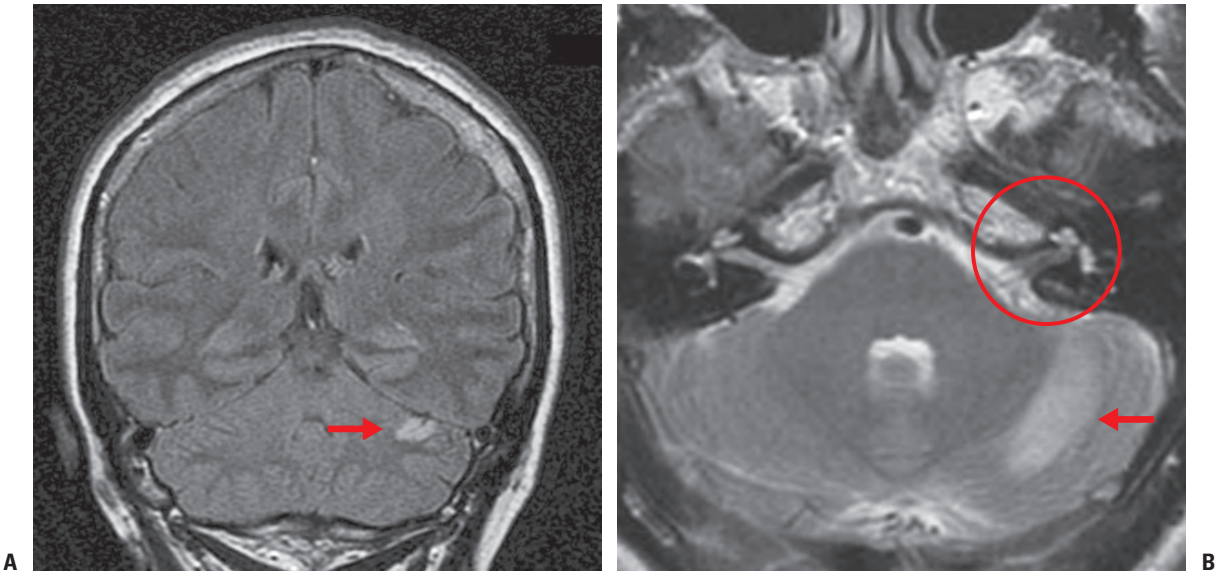
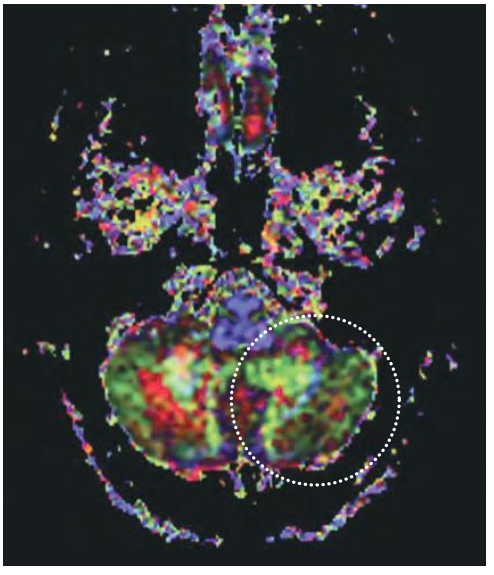


Figure 5.33. Primary Blast TBI, Also Known as Blast-Induced Neurotrauma (BINT). A. Coronal FLAIR image illustrates a small focal area of hyperintensity in the left superior cerebellar cortex, which may represent a small ischemic infarct (*arrow*). **B.** Axial T2 image at the level of the cerebellopontine angle cistern shows abnormal hypointensity within the internal auditory canal (*circle*) and a hyperintense area within the cerebellum (*arrow*) corresponding to the FLAIR abnormality in **(A)** The hypointensity in the auditory canal is thought to represent hemorrhage. (*Continued*)



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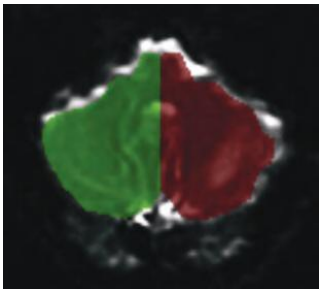

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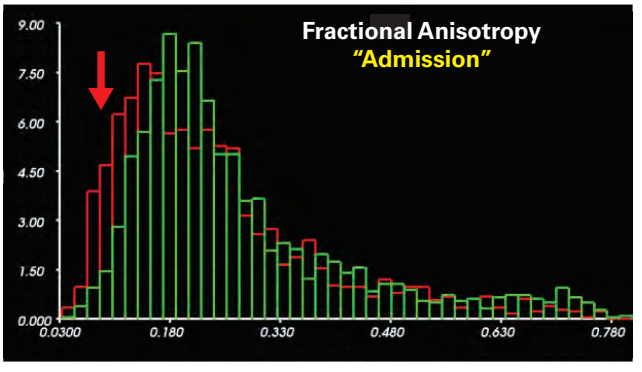

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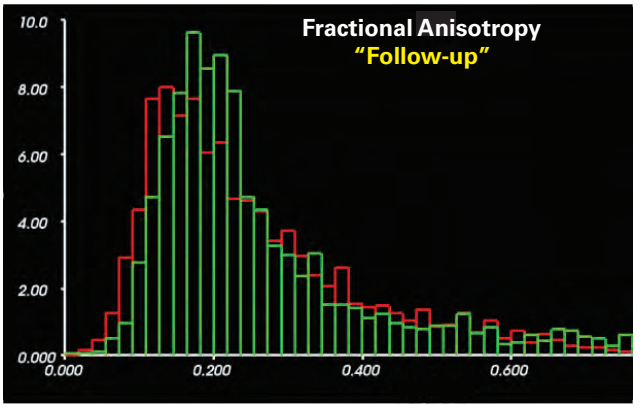

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Figure 5.33. (*Continued*) **C.** Axial diffusion tensor imaging (b = 0, 1000 [s/mm2], in 25 independent directions) of the cerebellum with right to left fascicles depicted in *red*, superior to inferior fascicles in *blue*, and anterior to posterior fascicles in *green*. The DTI imaging in this patient shows a decrease in the right-to-left fascicles (*red*) of the left cerebellar hemisphere (*circle*). **D.** 3D DTI color tractography shows the right and left crossing fibers of the cerebellum with asymmetric loss and thinning of fiber tracts in the left cerebellar hemisphere (*arrow*). **E.** Region of interest (ROI), used to compare the fractional anisotropy (FA) for the right (*green*) and left (*red*) cerebellar hemispheres in figures **(F)** and **(G)**. **F.** The initial FA histogram at the time of injury shows the right cerebellar FA (*green*) and left cerebellar FA (*red*) with a leftward FA shift (*arrow*). **G.** The 4-month follow-up study demonstrates more coherent overlap of the right and left FA histograms suggesting a decrease in tissue microheterogeneity for the right and left cerebellar ROIs. The soldier's symptoms had improved during the interim. (Courtesy of Gerard Riedy, MD, PhD, Walter Reed National Medical Center, Bethesda, MD.)

- A recent 3T MRI study following training sessions for breachers (individuals who use explosives in the military and law enforcement community) showed evidence of prominent perivascular spaces in 23% of participants and increased BOLD activation post-training but no evidence of fMRI, DTI, SWI, or FLAIR signal abnormality.[99]

- The largest study to date, in which DTI was used to examine veterans of the Iraq and Afghanistan wars, demonstrated a significantly higher number of white matter lesions in veterans who sustained mild TBI than those without TBI (**Fig. 5.34**).[100] In addition, veterans who had blast-related TBI showed more lesions than those with non–blast-related TBI. The difference in the number of lesions was not influenced by age, time since trauma, a history of mild TBI unrelated to deployment, or coexisting psychopathology. The number of lesions was correlated with the severity of TBI and with performance in executive functioning tasks.

"Blast-Plus" Brain Injury (Diffusion Tensor Imaging)

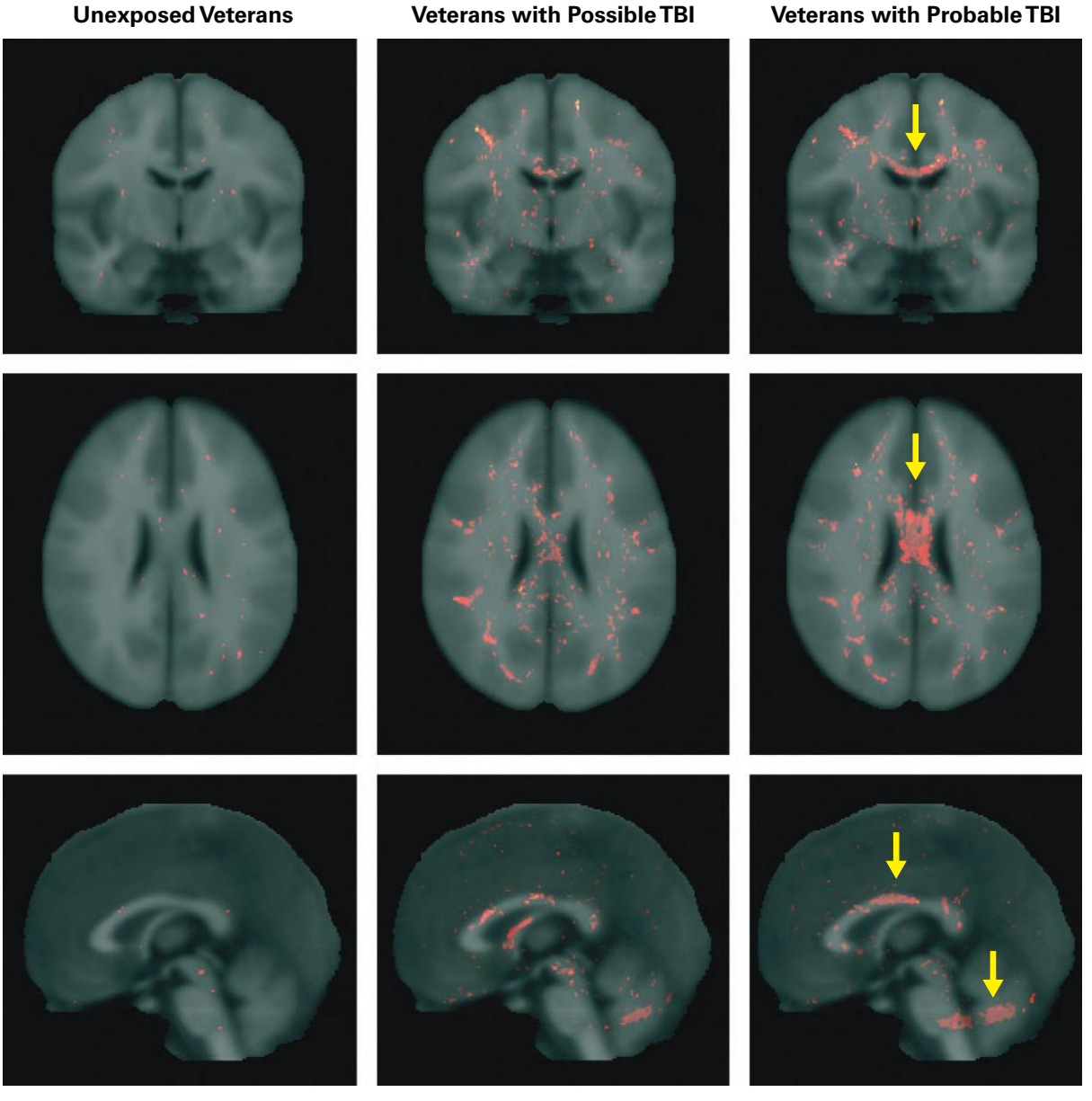


Figure 5.34. "Blast-Plus" TBI. DTI composite illustrations of white matter regions with abnormally low fractional anisotropy (FA) in veterans with *no* exposure to TBI, *possible* TBI, and *probable* TBI. Z-score images were given a threshold of −3.0. All patients had normal conventional MR exams. Overlap of lesions across subjects is represented in *red*. Note prominent involvement of the corpus callosum and cerebellum (*arrows*). (Adapted with permission from Ricardo Jorge, MD.)

Chronic traumatic encephalopathy (CTE) is a recently identified, tau protein–linked, neurodegenerative disease triggered by repetitive head trauma, as occurs in sports and in combat.[101] A very interesting recent study using the PET chemical marker known as FDDNP 2-(1-{6-[(2-[^{18}F]Fluoroethyl)(methyl) amino]-2-naphthyl}ethylidene)malononitrile showed abnormal binding of FDDNP to deposits of amyloid-β plaques and neurofibrillary tau tangles in the brains of five retired professional football players.[102] High concentrations of tau protein were identified in areas known to be affected by CTE, such as the amygdala and subcortical regions of the brain. Global volume loss was noted in all patients. Cerebral atrophy is well known; nonspecific sequelae of TBI and whether it is more or less common in blast trauma remains unknown (**Figs. 5.22** and **5.23**). The researchers in this small, preliminary, but promising study also found a direct correlation between the number of concussions a player had experienced and the amount of FDDNP binding. Currently, CTE is linked to dementia and depression, but it can only be diagnosed postmortem. It is hoped that early detection of tau proteins using this new imaging biomarker may advance our understanding of brain injury pathophysiology. The capability of imaging modalities to enable screening and detection of CTE may help guide strategies for early intervention and treatment. As mentioned later in Lesson 12, emotional issues, including PTSD and suicidal ideations, are a significant problem in veterans. Larger studies tracking patients over time will need to be done to determine how prevalent the disease is in the general population and whether certain individuals are more predisposed to developing the disease.

To date, PET imaging studies examining the potential role of tau protein deposits in head injury have been limited to civilian TBI. However, this technique holds exceptional promise for advancing our understanding of blast TBI. A recent landmark study by Goldstein and colleagues[103] demonstrated evidence of CTE in postmortem brains from veterans exposed to blast and/or concussive injury. These investigators also developed an experimental animal model that recapitulates CTE-linked neuropathology in mice 2 weeks after exposure to a single blast. In this model, blast-exposed mice demonstrated phosphorylated tauopathy, myelinated axonopathy, microvasculopathy, chronic neuroinflammation, and neurodegeneration in the absence of macroscopic tissue damage or hemorrhage. Blast exposure induced persistent hippocampal-dependent learning and memory deficits that persisted for at least 1 month. Behavioral and cognitive abnormalities correlated with impaired axonal conduction and defective activity-dependent long-term potentiation of synaptic transmission. Intracerebral pressure recordings demonstrated that shock waves traversed the mouse brain with minimal change in the intracranial pressure (ICP). By contrast, kinematic analysis revealed that blast-induced head oscillation occurred at accelerations sufficient to injure the brain. Further, head immobilization during blast exposure prevented blast-induced learning and memory deficits. Therefore, this well-designed, translational study of Goldstein and colleagues[103] provides persuasive evidence that injurious head acceleration from the primary blast shock wave may be a key mechanism leading to blast-related TBI and CTE. In summary, their results reveal common pathogenic determinants leading to CTE in blast-exposed military veterans and provide mechanistic evidence linking blast exposure to persistent clinical impairments. The results of these studies have obvious, direct relevance to head-injured athletes. It is hoped that future PET imaging research with FDDNP will allow

TABLE 5.4 Key Points in Blast-Related TBI

■ Blast injury is the most common cause of TBI in war and terrorist attacks.
■ Explosive blast TBI covers a wide spectrum of injury.
■ Blast trauma is divided into four mechanisms of injury: *primary* (resulting from the overpressure shock wave), *secondary* (resulting from penetrating injuries from foreign objects propelled by the explosion), *tertiary* (resulting from the patient being thrown or crushed), and *quaternary* (resulting from thermal trauma).
■ **Blast-induced neurotrauma (BINT)** is the name given to brain injury that results solely from the primary overpressure shock wave. **"Blast-plus" TBI** is the name given to brain injury resulting from a combination of BINT *plus* secondary, tertiary, and/or quaternary injury mechanisms.
■ The vast majority of blast TBI is "*blast-plus*" TBI.
■ It is clear from laboratory studies that the primary shock wave can induce brain injury in animals; the degree to with the primary shock wave causes damage in humans remains uncertain.
■ The *coupling* and transmission of pressure waves from body blast injury to the brain is, in part, the cause of BINT.
■ Multiple physical and genetic factors determine the extent of BINT.
■ Brain and soft tissue edema are common in blast trauma.
■ Vasospasm is common in blast-induced brain injury.
■ Little is known about the imaging manifestations of BINT.
■ There is a high association of mild TBI and PTSD with blast-induced brain injury.
■ Virtually nothing is known about the long-term consequences of blast-induced TBI.

PTSD, post-traumatic stress disorder.

early identification, advance our pathoanatomic understanding of combat brain injury, and enable prevention of the disease.

LESSON 5: POLYTRAUMA IS MORE COMMON

Those who receive the full force of the highly lethal weaponry used in modern-day combat and terrorist attacks suffer a variety of injuries not encountered in civilian practice. Severe TBI resulting from a blast injury is almost always combined with complex multiorgan trauma. Secondary, tertiary, and quaternary injury mechanisms give rise to blunt and penetrating injuries, as well as burns that include the complex polytrauma seen in blast injury victims. As such, the imaging of these patients is often termed "*Extreme Radiology*". Given the complexity of the injury mechanisms, it is difficult to separate and discern the extent to which the *primary* blast force plays a role in

causing injury. The majority of blast injuries are caused by IEDs, which are designed to cause massive bodily destruction. Large numbers of fragments are propelled toward the intended victims. In addition to these complex multi-mechanistic forces of injury, roadside IEDs also cause vehicle translocation resulting in occupant blunt injury/trauma from rollover, elevation, and upending or collision. Multiple systemic injuries are the rule, and they complicate both the identification and the treatment of TBI (**Figs. 5.35** and **5.36**). A typical admission history might be something like:

> *21-year-old M s/p IED blast with intracranial contusions, bilateral lower extremity above knee stumps, large segments of devitalized/necrotic tissue with dirt and shrapnel, pulmonary edema, open abdomen, 25% burns, left upper extremity fasciotomy/escharotomy, and hypovolemic shock.*

Combat, Terrorism, and Natural Disaster Polytrauma

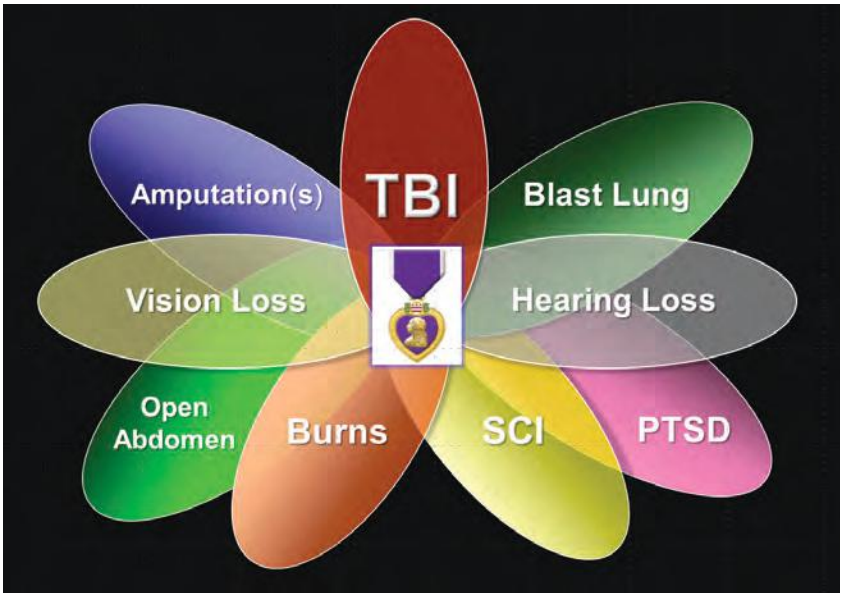


Figure 5.35. Polytrauma. Note the complex overlap of multisystem injuries that can occur in combat. At the center of the diagram is the venerable Purple Heart, awarded to those wounded or killed in action against an enemy of the United States. PTSD, post-traumatic stress disorder; SCI, spinal cord injury; TBI, traumatic brain injury.

Combat, Terrorism, and Natural Disaster Polytrauma

Figure 5.36. **Blast Polytrauma. A.** Intraoperative photograph illustrates bilateral lower extremity amputations, external fixation of a right femoral fracture, open abdomen due to blast-induced intestinal edema, and diffuse cutaneous burns. The soldier also suffered blast lung injury and TBI (not shown). He survived his horrific injuries. **B.** Venn diagram emphasizing the importance of acidosis, coagulopathy, and hypothermia in determining patient prognosis following severe trauma. The *arrow* points to the intersection of these three factors, known as the *triad of death*. INR, international normalized ratio.

Too Many "-ations"

Contamination, mutilation, amputation, penetration, perforation, aspiration, herniation, resuscitation, coagulation, laceration, dysautoregulation, inflammation, complication, and frustration (e.g., PTSD) are some of the unfortunate themes in war and terrorist attacks. Because the predominant mechanism of injury is an IED blast injury, the tissue literally explodes. It is shredded and crushed, rather than cleanly cut. Unlike AK47 or small arms wounds, all IED blast injuries are contaminated. In the extracranial soft tissues, including the scalp, embedded foreign bodies and soft tissue defects necessitate multiple wound washouts, serial debriding, and a heightened vigilance for sepsis.[104] Superimposed burn injuries (discussed in Lesson 7) also contribute to the heightened risk for infection. The soldier's body armor helps protect the torso, leaving the face and extremities exposed. This renders the soldier highly vulnerable to single, double, and triple amputations. Nearly 4% of all wounded warriors have suffered one or more amputations. Of casualties who died, nearly 25% suffered an amputation. Note that these amputation rates are much higher than those in any prior conflict in American history.[105] Of course, civilian victims of terrorists attacks are not protected by body armor, and these injuries can be even more severe.

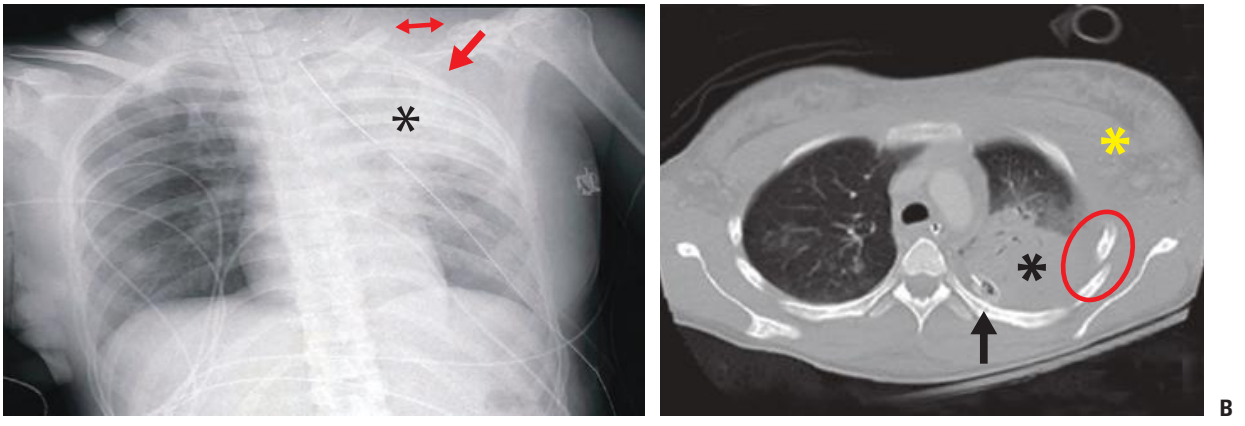
A soldier's lungs may be injured in combat via several mechanisms, all of which interfere with normal oxygenation to the brain. Pulmonary compromise can be caused by blast lung injury, pulmonary contusion/laceration, pulmonary emboli, aspiration, acute respiratory distress syndrome (ARDS), neurogenic pulmonary edema, and blast-induced air emboli (**Figs. 3.9, 5.37,** and **5.38**). As compared to civilian trauma, there are many unique features to combat pulmonary injuries. Interestingly, primary blast-induced pulmonary trauma is relatively uncommon in this war, in comparison to prior conflicts and civilian blast trauma. The classic chest radiographic findings of blast lung include bilateral lung opacities, a narrow mediastinum, an absence of pleural effusions, and a normal heart size. These findings are all considered consistent with increased permeability edema (i.e., capillary leak) rather than hydrostatic edema (fluid overload). Note that transfusion reactions can have a similar appearance. Blast overpressure injury can also cause direct lung injury in the form of pulmonary hemorrhage and contusion. Migratory ground glass opacities on the chest radiograph are less consistent with pulmonary contusion and more compatible with fluid shift in the lungs. The lungs can also be secondarily affected by a blast-induced injury to the heart. Cardiac contusions, arrhythmias, coronary artery obstructions by air emboli, or coronary vasospasm can occur following a blast overpressure wave and can lead to cardiac dysfunction with secondary manifestations on lung function.

ARDS occurs in combat trauma, usually in conjunction with other severe injuries such as traumatic amputation and hemorrhagic shock.[106] In combat trauma, ARDS is often caused by fat emboli, massive transfusions, or toxic inhalation. Both pulmonary emboli and fat emboli are more common in combat than in civilian trauma patients. Fat emboli are particularly common in patients with long bone fractures.[23,107] Air emboli are thought to arise from tears between the alveoli and the intralobular venules.[108] Fat emboli lead to complications of ARDS, whereas air emboli can be fatal.

Acute traumatic lung injury caused by neurogenic pulmonary edema is usually attributed to the catecholamine surge that accompanies a sudden extreme increase in ICP or a hypothalamic injury.[109] Neurogenic pulmonary edema is an anomaly, as it cannot be categorized into

either of the two major types of pulmonary edema. Features of both high-pressure and increased-permeability abnormalities appear to be involved, and the mechanisms responsible for these abnormalities are quite complex. The high-pressure insult results from one or more mechanisms, including systemic hypertension, pulmonary venoconstriction, negative and positive inotropic action, and intrinsic myocardial dysfunction, that arise from the catecholamine surge. Increased permeability leading to neurogenic pulmonary edema may be mediated by alpha and beta effects of the catecholamine surge on the pulmonary vasculature. This adrenergic activity increases the pulmonary capillary pressure, and under resultant increased pressure gradient, fluid is induced to move from the pulmonary microvasculature into the pulmonary alveoli.[110] In addition, catecholamines may stimulate an inflammatory response that may also contribute to leakiness of the pulmonary capillaries.

Blast Pulmonary Injury



A B

Figure 5.37. Blast Lung (Pulmonary Contusion). A. AP chest radiography demonstrates a left clavicle fracture (*double-headed arrow*), upper left rib fractures (*arrow*), an upper lobe pulmonary contusion *(asterisk)*, and a left thoracostomy tube. **B.** Axial contrast-enhanced CT of the chest viewed at bone windowing shows the left lung contusion (*black asterisk*), rib fracture (*circle*), anterior chest wall soft tissue swelling (*yellow asterisk*), and thoracostomy tube (*arrow*). An enteric tube is noted in the esophagus.

Pulmonary Laceration (Helicopter Crash)

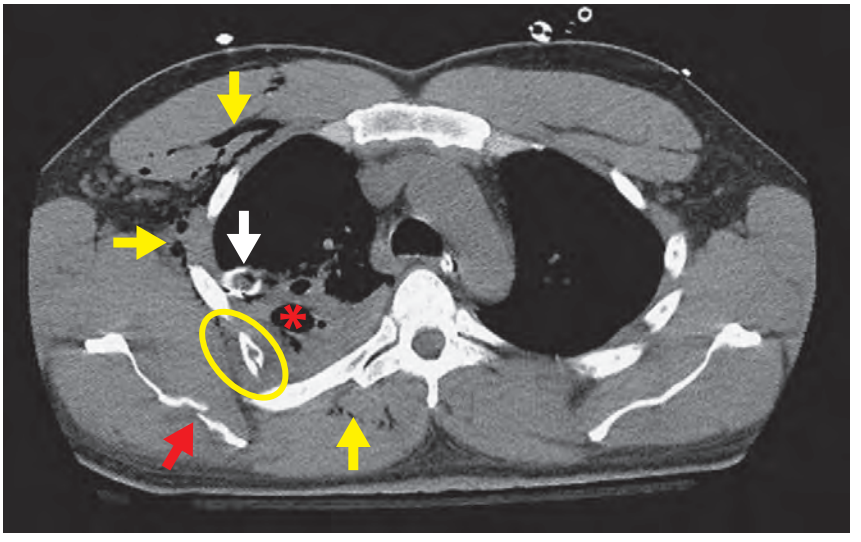


Figure 5.38. Pulmonary Laceration. This 21-year-old patient is status-post a helicopter crash. Noncontrast axial CT shows a comminuted fracture of the right scapula (*red arrow*) and right 4th rib (*yellow circle*). Note the underlying cystic lucency (*asterisk*) within an area of pulmonary consolidation, consistent with a pulmonary laceration and adjacent hemothorax. Air tracks into the right pectoralis and paraspinal muscles (*yellow arrows*). An enteric tube is present in the esophagus and a right thoracostomy tube is noted (*white arrow*).

The abdomen, like the chest, is protected by body armor, but this does not preclude abdominal injury. The bowel is rarely injured from the isolated blast wave itself. However, direct injury to the bowel may occur by shrapnel produced by the explosive blast.[111] Bowel injury is less common in this conflict in comparison to prior conflicts. In contrast, as discussed earlier, blast-induced TBI has been found to be much more common in this war. This disconnect is an enigma. One explanation is that modern body armor protect the lungs and intestines, but not the brain, from blast injury. Another possibility is that other physical forces may play a more prominent role in blast injury to the bowel versus blast injury to the brain.[112] That is, explosives produce effects other than just pressure waves, including light, acoustic, thermal, and electromagnetic forces, as well as toxic fumes. The relative contribution of these forces to blast TBI is uncertain, and these forces may exert effects differently on bowel and brain. Bowel contusions, perforations, mesenteric tears, and renal failure secondary to myoglobinuria can also be caused by crush injuries.

Abdominal injury also acts to amplify damage to the injured brain. For example, liver damage can initiate a systemic inflammatory response syndrome (SIRS) leading to the release of proinflammatory cytokines into the circulation that may trigger neuroinflammation.[113] A recent study has demonstrated that systemic inflammation outside the central nervous system (CNS) can activate innate immune mediators within the CNS and thereby augment axonal injury. The extent of both the SIRS and neuroinflammatory responses are dependent

on the severity of the liver injury.[114] In addition, elevated *intra-abdominal* pressure can be transmitted to the abdominal vena cava and cephalad to the cranial venous system. This elevation of pressure in the cranial draining veins exacerbates ICP in patients with TBI. Recognition of this relationship between abdominal pressure and ICP is important to consider in the treatment of raised ICP in patients with abdominal injury. The option of a decompressive laparotomy may be useful in patients with intracranial hypertension refractory to optimal medical therapy.[115]

Metabolic acidosis (lactic acidosis because of mitochondrial mayhem), a massive release of catecholamines, hypo- and hyperthermia, hyperglycemia, and cytokine release all contribute to the complexity of treating the wounded brain. Post-traumatic seizures may arise from or contribute to the metabolic disturbances. Post-traumatic seizures are more common in combat, in part due to the high incidence of penetrating TBI and because post-traumatic epilepsy is more common in men.[116] In the United States, about 6% of epilepsy is attributed to TBI. Mild TBI increases the subsequent risk of epilepsy by about twofold. Moderate TBI increases the risk of epilepsy by about threefold, whereas severe TBI is associated with about a 17-fold increase in risk, which translates into more than 15% of patients with severe TBI developing epilepsy.[117] For some subgroups, such as patients with a skull fracture or an SDH, more than 20% develop post-traumatic epilepsy.[118] Seizures affect as many as 50% of soldiers suffering penetrating head injury.[119] Early post-traumatic seizures exacerbate the structural damage to an already injured brain and may contribute to the development of a chronic epileptogenic focus.[120] Seizures may also induce blood pressure changes, hypoxic events, and elevate ICP. These events may lead to further damage, especially if they occur in the first few days after injury.[121,122]

Not to be overlooked, combat injury may also encompass spinal trauma and injury to the peripheral nervous system (**Figs. 5.39** to **5.43**). Multiple spinal fractures are often encountered. Maintaining spine immobilization in the midst of combat and terrorist activity can be especially challenging. Injuries to the peripheral nervous system are far more varied and common than in civilian trauma. Plantar fasciitis, tarsal tunnel syndrome (boot trauma), entrapment neuropathy (from constantly holding a weapon), penetrating peripheral nerve trauma, and demyelination from a blast injury are all encountered on the battlefield.

Blast Polytrauma

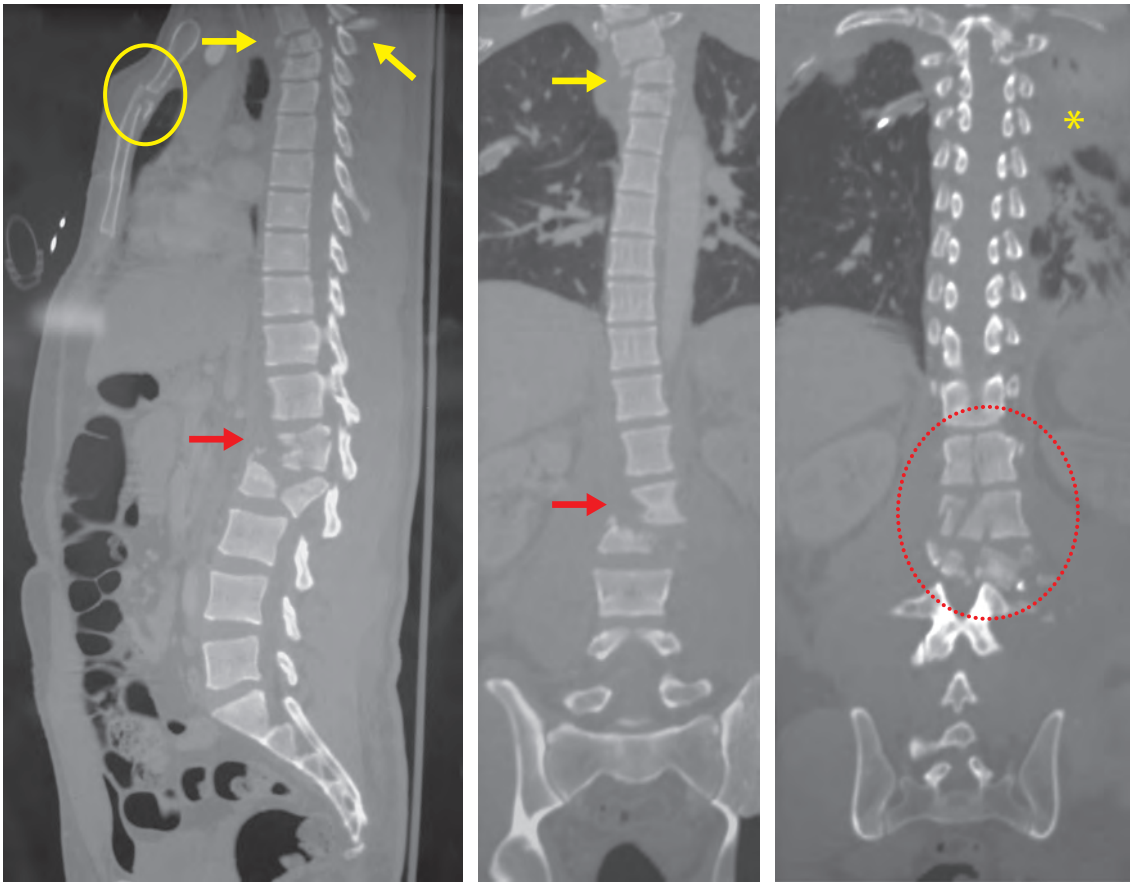


Figure 5.39. Blast Polytrauma. This 20-year-old soldier is status-post an IED blast while riding in a Humvee. **A.** Sagittal and **(B and C)** coronal reformatted CT images demonstrate an upper thoracic (*yellow arrows*) and thoracolumbar (*red arrows* and *circle*) burst fracture-dislocations. A fracture of the sternum (*yellow circle*) and blast lung injury (*asterisk*) are also noted.

★**KEY POINT** Roadside IED explosions often cause blunt trauma as crew members are thrown around inside the vehicle after the blast. Closed fractures of the upper and lower extremities and spine are common. In addition, when projectiles hit the vehicle, they create shock waves that travel through the vehicle as compression waves. These compression waves have sufficient force to break metal on the inside of the vehicle, and the resulting spall is dangerous to the crew and equipment. Spine injury stabilization in such polytrauma cases make patient management and transport even more difficult.

Spinal Trauma (Humvee IED Blast Injury)

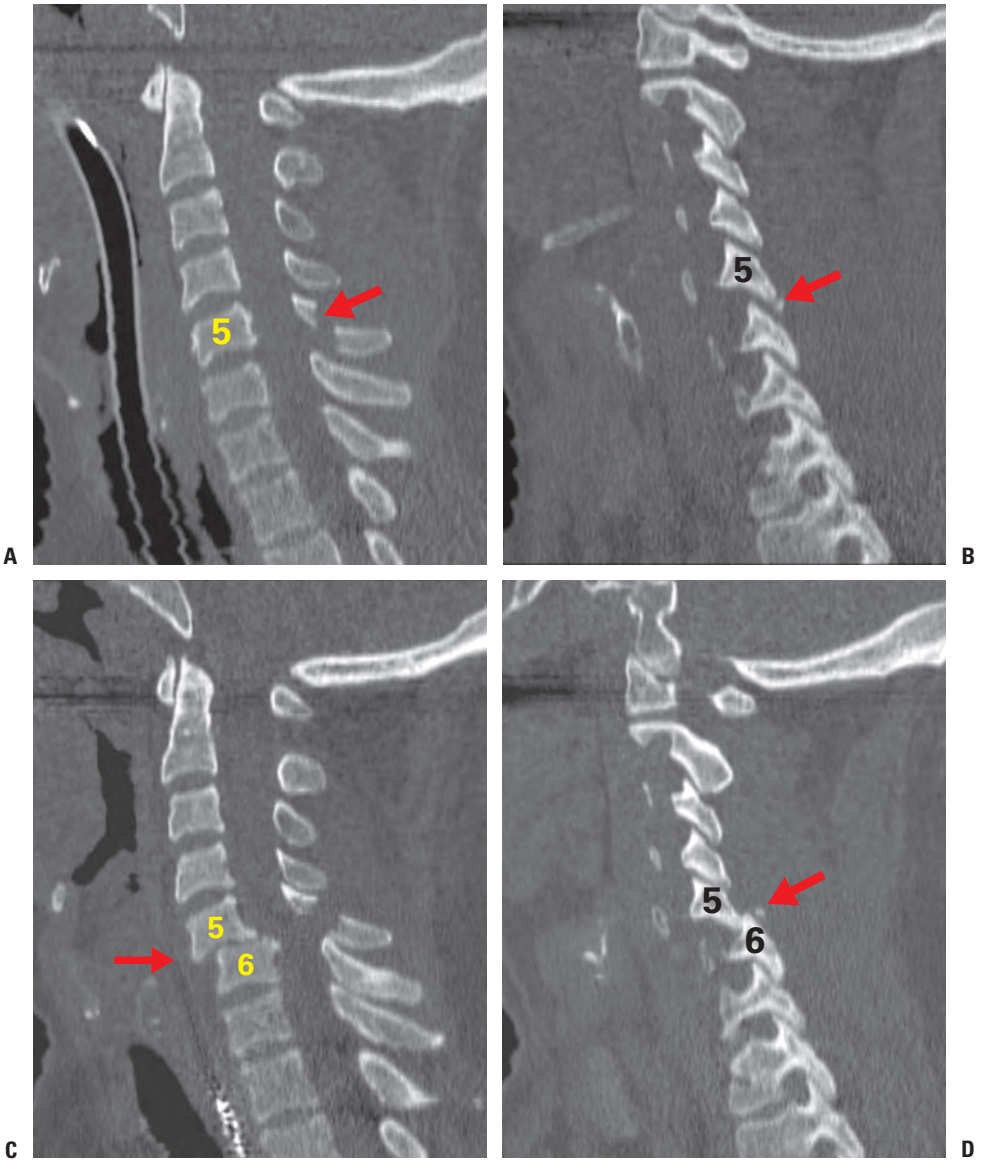
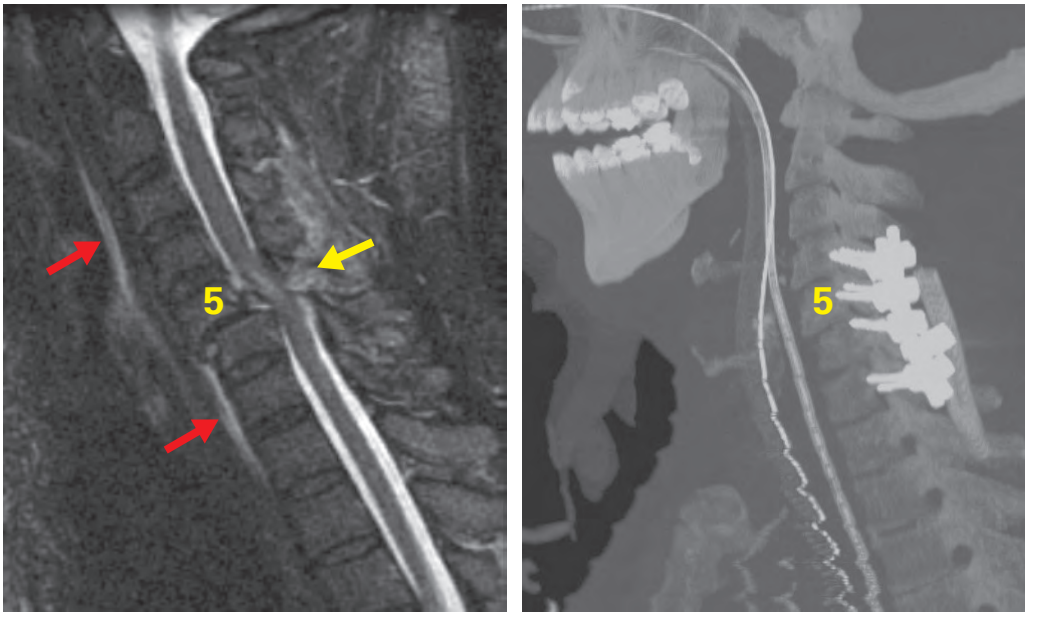


Figure 5.40. Cervical Intramedullary Contusion with Interval Development of Bilateral Jumped Facets. **A.** Midsagittal reformatted CT shows normal alignment and a fracture of the C5 spinous process (*arrow*). The patient is intubated and presented with quadriplegia. It is difficult to assess the prevertebral soft tissues because of intubation. **B.** Parasagittal reformatted CT shows a small fracture of the C5 inferior articular facet (*arrow*) with a slightly widened facet joint. **C.** Despite careful c-spine precautions, the 15-hour follow-up CT following transport to LRMC (in a C-collar) shows interval anterior subluxation of C5 on C6 (*arrow*). **D.** The corresponding parasagittal image reveals a C5 jumped facet (*arrow*). (*Continued*)



E F

Figure 5.40. (*Continued*) **E.** Midsagittal short TI inversion recovery (STIR) MR demonstrates abnormal intramedullary T2 hyperintensity consistent with a cord contusion (*yellow arrow*). Note also C5–6 disc retropulsion, mild prevertebral soft tissue swelling (*red arrows*), and subtle posterior soft tissue T2 hyperintensity (*yellow arrow*). **F.** Postoperative sagittal reformatted CT image after operative stabilization.

★**KEY POINT** MRI became available in the war zone in 2011 (i.e., 8 years after the beginning of the conflict). It has been particularly helpful in diagnosing mission-changing injuries such as neurologic decompression sickness, musculoskeletal injuries, SCI and TBI. It must be performed judiciously because of frequent contamination of wounds with ferromagnetic foreign bodies.

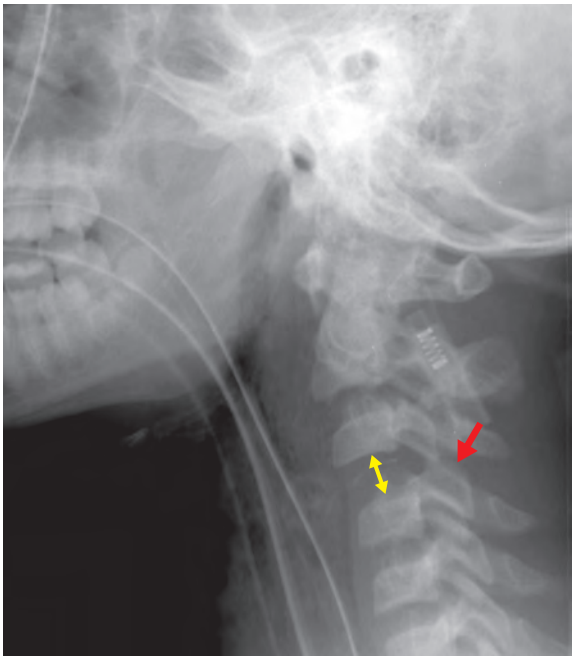
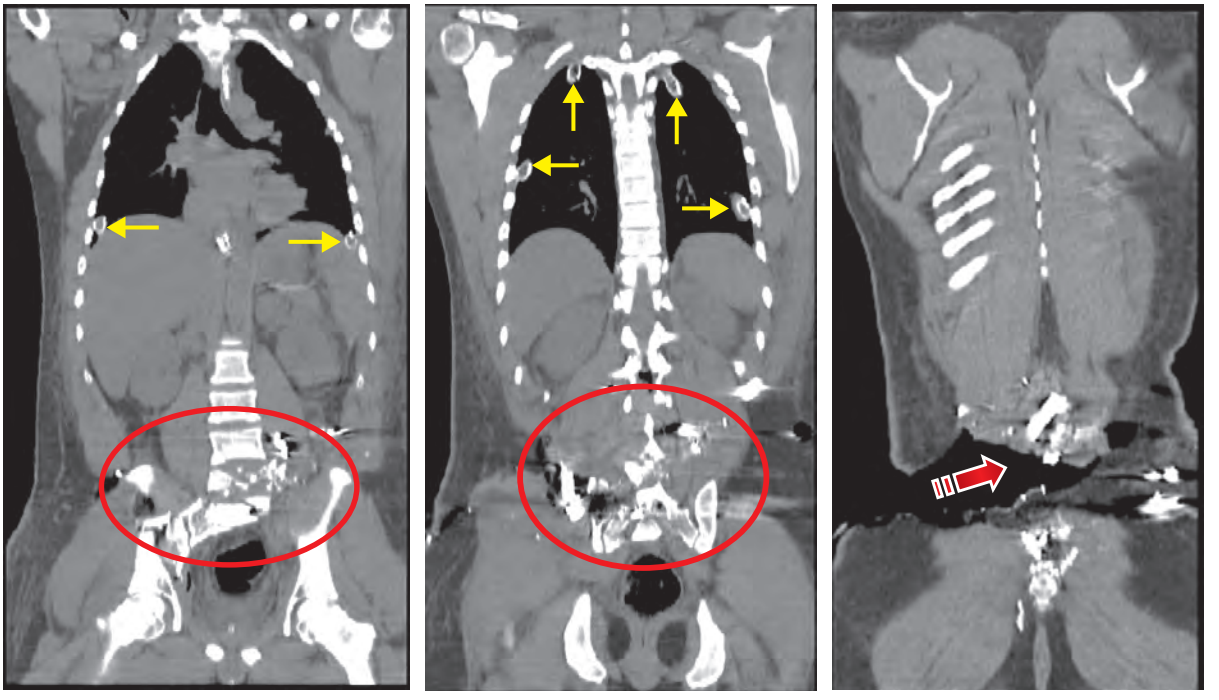


Figure 5.41. Cervical Distraction Injury (IED Blast Trauma). Lateral plain radiograph of the cervical spine in a 17-year-old soldier status-post IED explosion and Humvee rollover. The patient is intubated. Note abnormal increased disc space between the C3 and C4 vertebral bodies (*yellow arrow*), consistent with an acute distraction injury of the cervical spine (*arrow*). There is also uncoupling of the lateral masses at this level; note the "empty" superior articular surface of C4 (*red arrow*).

Spinal Trauma (IED Blast Injury)



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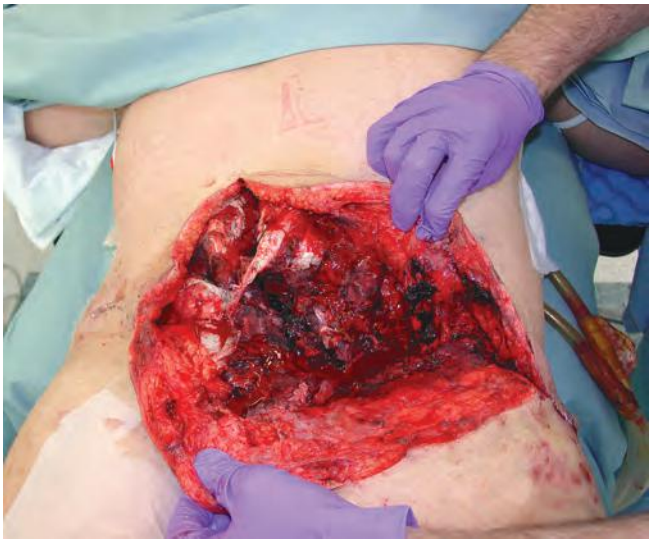
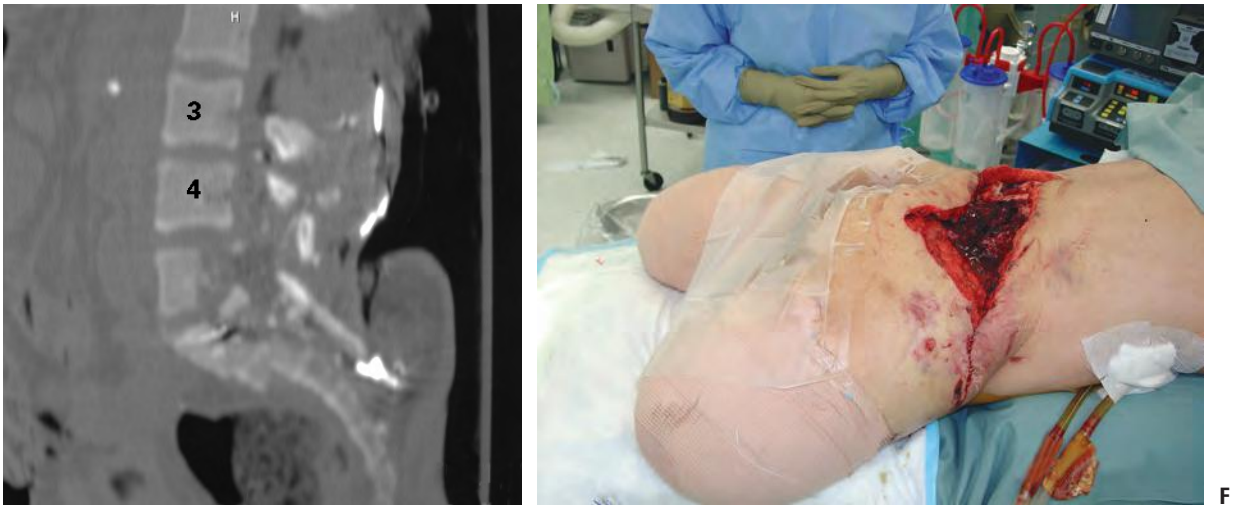


D

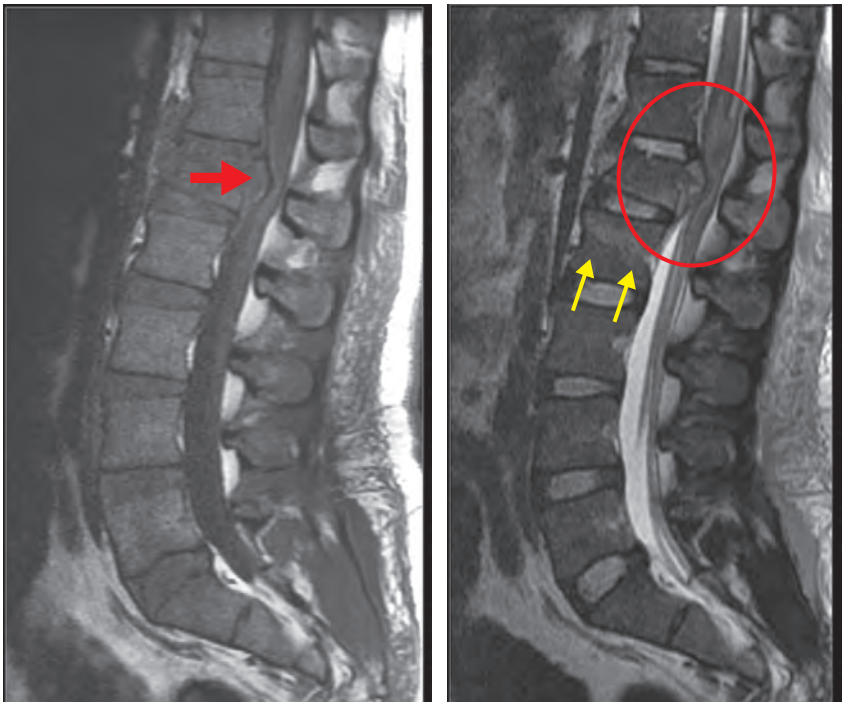
Figure 5.42. Spine Trauma (IED Blast). This 23-year-old soldier is status-post an IED explosion with total dissociation of the spine and pelvis. The thecal sac was tied off downrange at the L3 level. **A–C.** Admission coronal reformatted CT images of the chest/abdomen/pelvis study demonstrate a comminuted fracture-dislocation of the lumbosacral junction and pelvis (*circle*) with a large soft tissue defect (*red arrows*). Bilateral thoracostomy tubes are also noted (*yellow arrows*). **D.** Intraoperative photograph of the patient's lower back demonstrates extensive lumbosacral osseous and soft tissue injury. (*Continued*)



E F

Figure 5.42. (*Continued*) **E.** Sagittal reformatted CT shows comminuted fractures of the lumbosacral junction and several radiopaque foreign bodies lodged within the posterior paraspinal soft tissue. **F.** Intraoperative photograph reveals the soft tissue defect and bilateral above-the-knee amputations.

Spine Trauma (IED Blast Injury)



A B

Figure 5.43. **Spine Trauma (IED Blast).** This 19-year-old soldier is status-post an IED blast injury and Humvee rollover presented with back pain, lower extremity weakness, and bowel/bladder incontinence. **A.** Sagittal T1-weighted MRI performed following emergency transport to LRMC demonstrates an acute burst fracture of L1 with loss vertebral body height and retropulsion of the posterior vertebral body (*arrow*). **B.** Sagittal T2-weighted MRI reveals the L1 fracture and subtle abnormal hyperintensity of the conus (*circle*).

Imaging Approach Summary

The most important issue in acute blast trauma is the rapid detection of a potentially treatable lesion. Because of the speed, safety, availability, and power with which CT can evaluate the polytrauma patient, it is currently the initial imaging modality of choice. Concomitant CTA is often performed acutely for cases of suspected vascular injury. CT is particularly helpful in penetrating trauma because it easily detects the location of ballistic fragments (which contraindicate MRI) and small bone fragments within the brain. Although CT is relatively insensitive to TAI, brain stem injury, and very small extra-axial collections, it effectively and noninvasively identifies potentially treatable space-occupying intracranial lesions, midline shift, hydrocephalus, and impending or established areas of ischemia and/or brain herniation. However, CT has definite limitations for diagnosing TBI and SCI. Thus, although CT is generally preferred in acutely injured patients, if a discrepancy is found between a patient's clinical status and the CT findings, a subsequent MRI scan should be performed as it might identify a CT invisible injury and help in predicting neurologic recovery. In these cases, MRI should be performed within the first few weeks of injury because this appears to be the most sensitive time period for identifying traumatic lesions. If one waits longer than 2 to 3 weeks to image the patient, the edema associated with axonal disruption is likely to resolve and smaller injuries will be more difficult to detect.

The use of MRI in acutely traumatized, multiorgan-injured patients currently entails numerous technical challenges. Once these are overcome, the use of MRI in an acute setting will increase and become more routine. Further, because of its lack of ionizing radiation, MRI offers a particular advantage in the younger population, in whom CT imaging poses a small risk of iatrogenic carcinogenesis, especially when sequential imaging studies need to be performed, as is often the case in TBI patients.

MRI is preferable to CT in assessing subacute and chronic head injury. At present, the most basic MRI protocol for imaging TBI patients should include a GRE (or SWI) sequence to detect subtle white matter shearing hemorrhages, especially when using lower field-strength magnets. FLAIR images are recommended as the T2-weighted sequence of choice for confirming the GRE abnormality and for detecting cortical contusions and nonhemorrhagic TAI. Depending on the location of the lesion noted on the axial image, a supplementary sagittal or coronal sequence could help localize the lesion. Additional coronal and sagittal images are especially useful in evaluating the temporal lobes, subfrontal region, and corpus callosum. However, if one has access to 3D Cube FLAIR imaging, the supplementary planes can be obtained without additional time. DWI is obtained in virtually all patients and may also be obtained using new 3D techniques. The remaining MRI techniques described previously can be obtained at the discretion of the clinician and neuroradiologist. It should be remembered that imaging protocols should be used only as rough guidelines and that the imaging parameters should be tailored to each patient. Conventional MRI offers superior anatomic resolution, and, in the future, functional and hemodynamic MRI will likely become routine supplements to the imaging of neurotrauma patients.

Advanced neuroimaging techniques such as DTI/DKI, fMRI, MRS, MEG/MSI, PET, SPECT, and ultra–high-field MRI are all being investigated in patients with TBI with the hope of more accurately capturing the true burden of disease and improving our understanding TBI pathophysiology.[54,123,124] These advanced

imaging techniques show tremendous promise to probe functional and prognostic questions and to better delineate the heterogeneous and dynamic nature of TBI. They are currently, however, largely confined to the research arena. Combinations of advanced MRI techniques (e.g., DTI + MEG or MRS + DTI) may prove the best way to detect subtle neuronal and/or axonal injury that cannot be detected by conventional MRI or CT techniques (**Figs. 5.44** and **5.45**).

Unfortunately, most of the data available at present are from small case series or case reports, and there are numerous logistic hurdles to overcome before these techniques are "ready for prime time" in routine clinical practice. In addition, the financial burden of combining different neuroimaging techniques in today's cost-conscious, evidence-based, health care reform climate is a real issue. Unlike the prior decades, today's trend is toward simplicity, ease of use, increased efficiency, and lower cost. Therefore, while there are numerous promising imaging biomarkers for TBI currently available, they require further study to fully determine their clinical utility in day-to-day practice. Future imaging directions include (1) routine ultrafast whole-body trauma imaging with acquisition speeds that limits motion artifacts and thus improve image quality, (2) advanced iterative reconstruction algorithms that allow a reduction in radiation dose while still maintaining high-quality images, and (3) development of computer-aided detection devices/software allowing for rapid interpretation of critical findings.

Multimodality Imaging in TBI

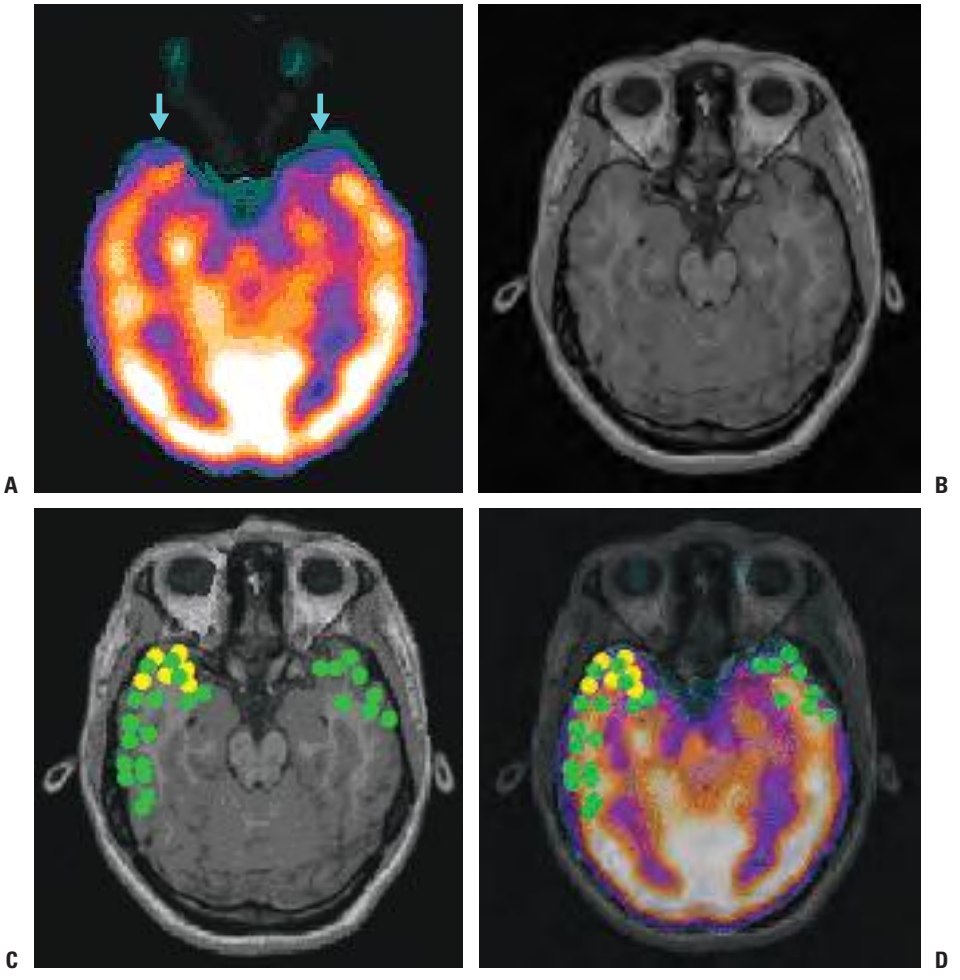


Figure 5.44. Multimodality Imaging in Head Trauma (SPECT + MRI + MEG). This 23-year-old female presented with persistent postconcussive symptoms 2 years following mild head trauma. **A.** Integrated single photon emission computed tomography (SPECT) reveals bitemporal hypoperfusion (*blue arrows*). **B.** T1-weighted MRI is normal, as were the remaining conventional MRI sequences (not shown). **C.** Magnetoencephalography (MEG) reveals right and left temporal focal slow waves (*green dots*) and interictal spikes (*yellow dots*). Interictal spikes are a diagnostic sign of epilepsy and they appear to play a fundamental role in epileptogenesis following brain injury. In this patient, MEG also showed rare left temporal epileptiform spikes even though the participant had never had a seizure. **D.** Fused image. (Courtesy of Jeff Lewine, PhD.)

★**KEY POINT** Conventional MRI offers superior anatomic resolution, and, in the future, functional and hemodynamic MRI will likely become routine supplements to the imaging of neurotrauma patients. It may turn out that *combinations* of advanced MRI techniques (e.g., DTI, MEG, MRS, SPECT) will prove the best way to detect subtle neuronal and/or axonal injury that cannot be detected by conventional MRI or CT techniques.

Multimodality Imaging in TBI

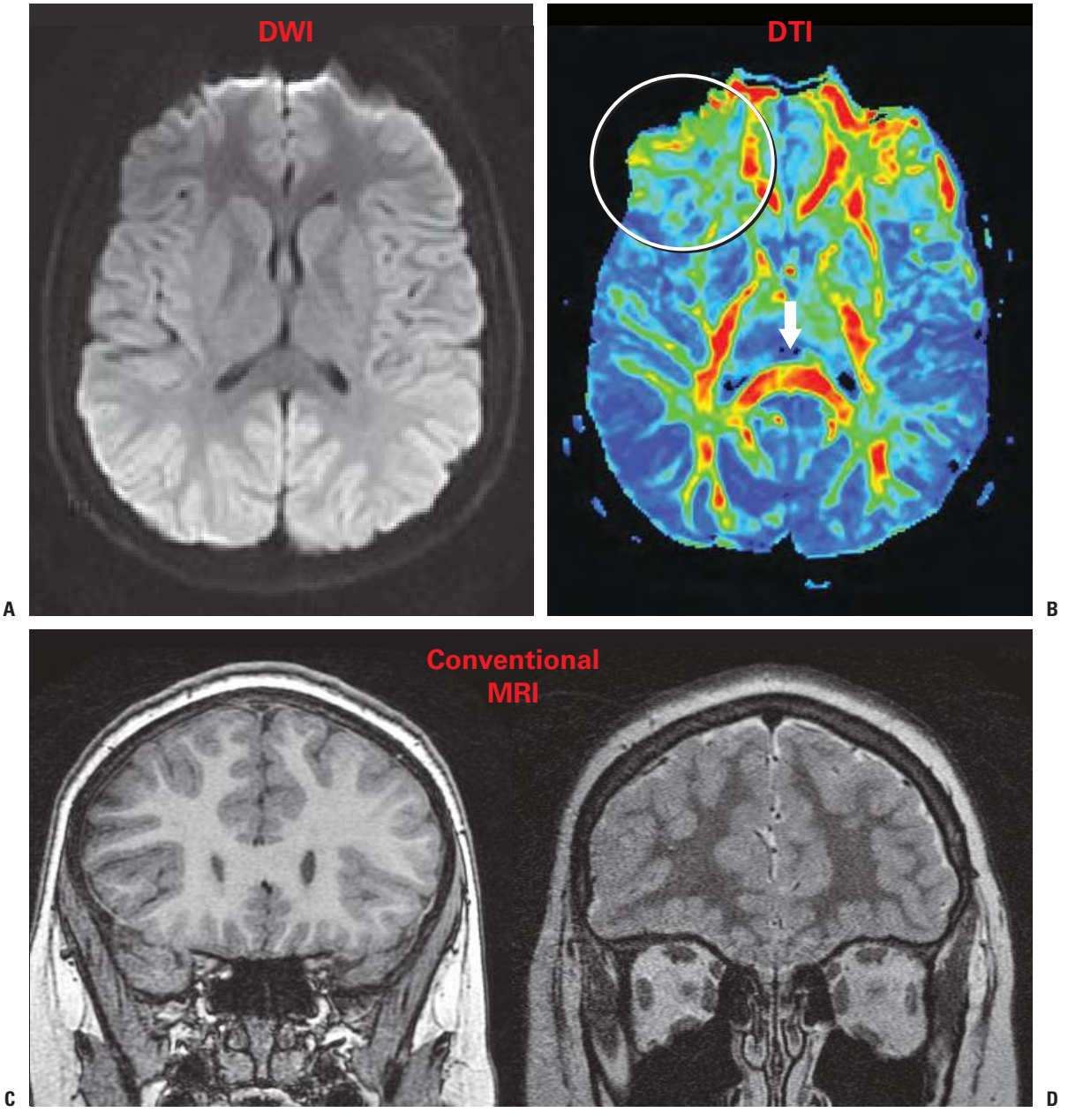
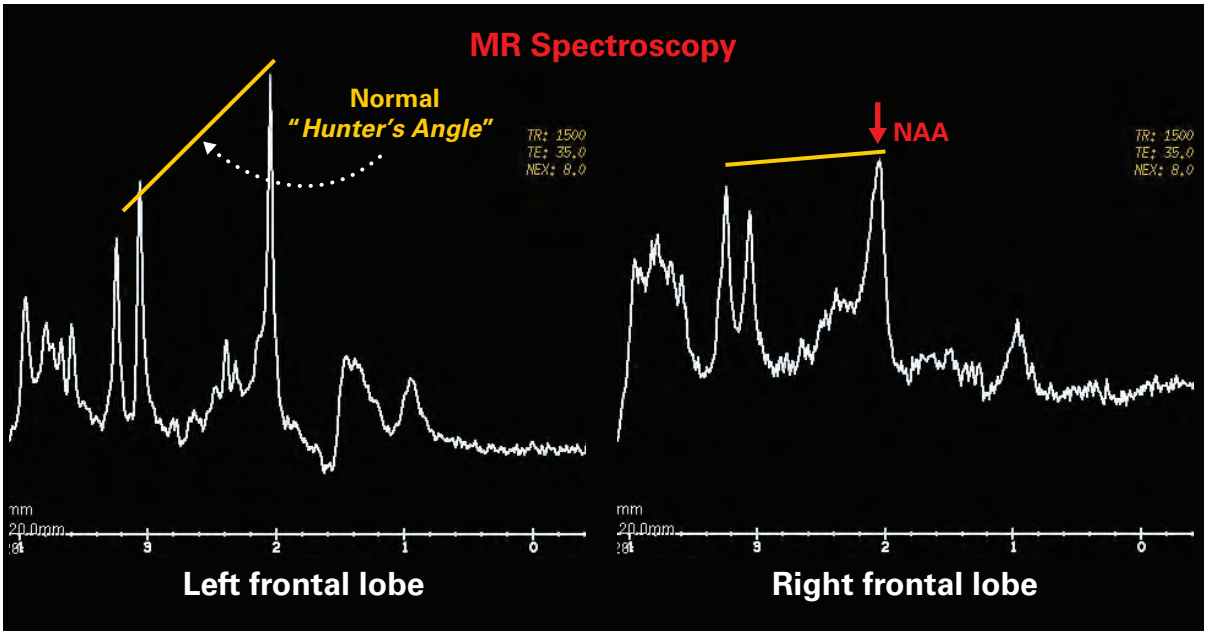


Figure 5.45. Multimodality Imaging in Head Trauma (DTI + MRI + MRS). This 18-year-old male presented with persistent postconcussive symptoms 1 year following mild head trauma. **A.** Axial diffusion -weighted image (DWI) is normal. **B.** Axial diffusion tensor imaging (DTI) shows decreased fractional anisotropy in the right orbitofrontal region (*circle*) and the splenium of the corpus callosum (*arrow*). **C.** Conventional and (**D**) T2-weighted coronal MR images are normal. (*Continued*)

★**KEY POINT** Conventional MRI offers superior anatomic resolution, and, in the future, functional and hemodynamic MRI will likely become routine supplements to the imaging of neurotrauma patients. It may turn out that *combinations* of advanced MRI techniques (e.g., DTI, MEG, MRS, SPECT) will prove the best way to detect subtle neuronal and/or axonal injury that cannot be detected by conventional MRI or CT techniques.



E F

Figure 5.45. (*Continued*) **E.** Single voxel magnetic resonance spectroscopy (MRS) of the left orbitofrontal region is normal. Note the normal 45-degree slope of Hunter's angle (*yellow line*). **F.** Right frontal lobe MRS shows flattening of Hunter's angle (*yellow line*) and a decrease in NAA (*arrow*).

LESSON 6: LIFE-THREATENING HEMORRHAGE IS MORE COMMON

Hemorrhage is the leading cause of preventable death on the battlefield. It is more common in combat than in civilian trauma because of the increased incidence of polytrauma and the increased time to treatment. The role that the primary blast overpressure wave plays in combat hemorrhage is unknown. Despite the higher rates of hemorrhage in combat as compared to civilian injuries, the introduction of DCS has made it possible to salvage severely injured soldiers who previously would have exsanguinated due to uncontrollable bleeding.

Nearly 25% of patients with combat-related injuries have physiologic derangements that do not permit a time-consuming vascular reconstruction, and aggressive DCS is essential for limb salvage.[125] The central tenet of DCS is to prevent patients from dying from the *bloody vicious cycle*. The bloody vicious cycle arises when bleeding causes patients to develop a triad of conditions that together further exacerbate the bleeding and feed back to further worsen the triad of conditions, further exacerbate the bleeding, and ultimately culminate into a spiraling, unsalvageable situation. *Hypothermia, acidosis,* and *coagulopathy*, each result from bleeding, and in trauma, the combination of these three conditions acting together is commonly known as the *triad of death* (**Fig. 5.36B**).[126] These three conditions share a complex relationship such that each condition can compound the other and increase mortality.[127,128] In brief, blood loss diminishes tissue temperature (i.e., hypothermia) and decreases oxygen/nutrient delivery to the tissue. Decreased substrate delivery causes the body

to anaerobically metabolize glucose for energy (i.e., lactic acidosis). Acidosis interrupts the coagulation cascade and reduces myocardial efficiency, which further reduces the oxygen delivery, hence triggering a deadly cycle. Bleeding also triggers a clotting response. Massive bleeding, however, exhausts the supply of clotting factors, leading to a consumptive coagulopathy that leads to more bleeding, worsening hypothermia, and deepening acidosis.

In addition, TBI alone, without systemic injuries, can result in coagulopathy.[129] Tissue factor (TF), also called factor III and thrombokinase, is the primary activating moiety for the extrinsic coagulation pathway. When TF is released into the circulation, it initiates the coagulation cascade and may eventually manifest as disseminated intravascular coagulation (DIC). Because of the rich TF content of brain tissue, severe head injuries are often complicated by DIC. Recent data suggest that head trauma also releases procoagulant-rich microparticles.[130] Finally, life-threatening hemorrhage frequently leads to *hypotension,* which contributes to hypothermia and acidosis. Hypotension is a very important independent predictor of prognosis in the trauma patient.[131] Hypotension is associated with an increased likelihood of synchronous abdominal injury and higher injury severity score, and it quadruples the risk of death, triples the risk of operative intervention, and doubles the risk of requisite ICU care.[132]

Injuries in modern warfare are often caused by explosion and related high-velocity penetrating shrapnel, leading to *noncompressible* bleeding, meaning that it cannot be treated by external compression or the application of tourniquets or topical dressings.[133,134] Non-compressible bleeding accounts for approximately 85% of preventable deaths on the battlefield, 80% of which include acute hemorrhage within the abdomen and chest.[135-137] Focused assessment with sonography in trauma (FAST) has assumed a key role in the rapid noninvasive assessment of thoracoabdominal trauma and assists in decreasing disposition time. In one recent study, sensitivities and specificities of FAST scans for blunt and penetrating trauma were 93% and 100% and 90% and 100%, respectively.[138] Corresponding values for detection of pneumothoraces were 85% and 100%. This study showed a valuable role for FAST in all traumas, particularly when injuries involved hemodynamic compromise. FAST scans improve diagnostic capabilities in comparison with simple hemodynamic assessments alone and are a valuable addition to the traumatologist's repertoire of bedside assessment tools.

Abdominal hemorrhage most frequently involves injury to the spleen, liver, or retroperitoneal vasculature. It is typically noncompressible because bleeding deep within the abdomen cannot be treated by external compression. Emergency surgical intervention is currently the only available method for treating abdominal hemorrhage. In combat, the resulting blood loss often leads to death from what would otherwise be potentially survivable wounds. Abdominal injuries from combat are often more complex and difficult to stabilize, as compared to their civilian counterparts. These injuries are particularly problematic in austere areas where military resources and infrastructure are at a premium, and extended evacuation times may occur due to persisting tactical threats.[139] Approximately 50% of deaths from abdominal hemorrhage are deemed preventable.[133] Combat fatalities due to abdominal hemorrhage can be primarily attributed to delays in hemorrhage control during transportation, highlighting the need for rapid, far-forward hemorrhage treatments.[140] Transport time to reach a hospital where surgery can take place varies but is

estimated to average 1 hour. In the setting of refractory bleeding, the transfusion of blood products to correct the coagulopathy can be ineffective and can also cause transfusion-related acute lung injury. Recent evidence suggests that damage control resuscitation with the use of recombinant factor VIIa can increase survival.[141,142]

In 2010, Defense Advanced Research Projects Agency (DARPA) launched its Wound Stasis System program with the objective of finding a technologic solution that could mitigate damage from internal hemorrhaging. A novel foam-based product was developed by Arsenal Medical, Inc. to treat life-threatening, noncompressible abdominal trauma. The polymeric foam develops in situ after the product solution is injected into the patient's abdomen, and the resultant foam conforms to the inside of the body cavity creating a tamponade effect to slow bleeding. This allows time for injured patients with abdominal bleeding injuries to be transported to where they can receive definitive surgical care during which the foam agent can also be removed. Preclinical data of this product in a swine model of lethal liver injury have shown a sixfold reduction in blood loss and a dramatic increase in 3-hour survival rates from 8% to 72%.[143] The foam is designed to be administered on the battlefield by a combat medic and is easily removed by doctors during surgical intervention at an appropriate facility. Removal of the foam takes less than 1 minute following incision. The surgeon removes the foam by hand as a single block, leaving only minimal amounts in the abdominal cavity and with no significant adherence of tissue to the foam. The foam system is durable, lightweight, and small in size, facilitating battlefield use. While the DARPA-funded program is focused on improving outcomes for injured soldiers with noncompressible hemorrhage, the novel foam-based technology may translate to civilian trauma.

LESSON 7: HYPERTHERMIA AND BURNS ARE MORE COMMON

Just as *hypo*thermia can be harmful in trauma, so can *hyper*thermia. Troops are often exposed to prolonged elevated environmental temperatures (temperatures in Iraq can reach 135°F [57°C]). These temperatures, in addition to wearing body armor, maneuvering heavy equipment, and other intense physical activities, frequently cause soldiers to become dehydrated. This potentially fatal combination can lead to hyperthermia and heat stroke.[144,145] After controlling for severity of illness, diagnosis, age, and complications, elevated body temperature is independently associated with longer ICU and hospital stays, higher mortality rates, and worse outcomes.[146] The mortality from heat stroke can approach 60%, and survivors may sustain permanent neurologic damage.[147] The brain (especially the Purkinje cells of the cerebellum) is very sensitive to hyperthermia.[148-149]

Heat can injure the brain in several ways:

1) Hyperthermia-induced vasoconstriction with resultant cerebral ischemia and oxidative damage
2) Induction of inflammatory cytokines associated with elevated ICP and decreased CBF
3) Increased leakage of endotoxin from the intestine into the systemic circulation
4) Direct cytotoxicity from the heat
5) Activation of coagulation with resultant microthrombosis
6) Increased hemoglobin extravasation after TBI[150]

Indeed, heat stroke resembles sepsis in many aspects. As usual, genetic polymorphisms can protect or predispose the individual to heat stroke.[151] In addition to encephalopathy, heat stroke can cause serious systemic complications such as rhabdomyolysis, ARDS, DIC syndrome, and multiple organ failure. Unique to this situation are the synergistic effects of TBI and elevated environmental temperatures that act together to facilitate and accelerate increases in core temperature, acidosis, and coagulopathy. *It can be said that hyperthermia, acidosis, and coagulopathy are as much a* lethal triad *in TBI as are hypothermia, acidosis, and coagulopathy in major trauma.*[144] The symptoms of heat stroke can occur during the first day but are actually more common on the second and third days following the insult, providing another reason for the delayed onset of some battlefield brain injuries. The imaging findings of heat stroke are listed in **Table 5.5** and one example showing the predilection for the cerebellum is shown in **Fig. 5.46**.[152–156]

TABLE 5.5 Imaging Findings in Heat Stroke

■ Loss of gray–white matter discrimination (cytotoxic edema)
■ DWI abnormalities within the cerebellar dentate nuclei and splenium of the corpus callosum
■ T2 hyperintensity within external capsules, lateral putamen, and paramedian thalamus
■ Contrast enhancement
■ Patchy cortical lesions in the frontal and parietal lobes
■ Hyperintense lesions in the cerebellum and delayed cerebellar atrophy
■ Bilateral hippocampal T2 hyperintensity (rare)

DWI, diffusion-weighted imaging.

"Heat Stroke"

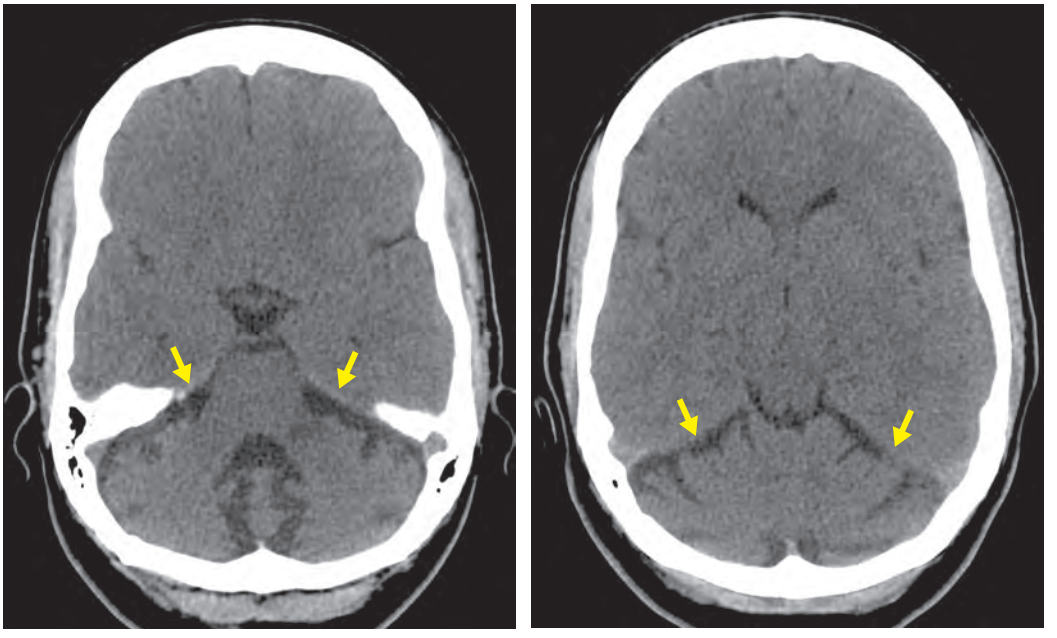


Figure 5.46. Heat Stroke. Axial noncontrast CT images of a 25-year-old male 3 months status-post an episode of severe hyperthermia (confusion, seizure, and body temperature of 104°F). Note severe diffuse cerebellar atrophy (*arrows*) with relative sparing of the cerebral hemispheres.

★**KEY POINT** Troops are at risk of heat stroke because of the potentially lethal combination of wearing heavy equipment, vigorous physical activity, hot and humid weather, and dehydration.

Because the skin is the largest organ of the body and the incendiary IED is the most common weapon of war, it is no surprise that burns are common in combat. Indeed, they comprise 5% to 10% of all casualties.[157] Of these, 20% are categorized as severe (i.e., involving >20% of total body surface area). In modern warfare, it is estimated that one injury in four is a burn.[158] Burns caused by the high temperature of an explosion or the burning of clothing typically involve the exposed parts of the body, including the face, neck, and hands (**Figs. 5.47, 5.53,** and **5.77**). Although many organ systems are affected by burn injury, the pulmonary system often sustains the most damage, and inhalation injury is a major contributor to mortality in thermally injured patients.

Burns associated with IED explosions are different from typical domestic burns. War and terrorist-related burn injuries are rarely seen in isolation. The wounds are deeper, more extensive, more complicated, and more often associated with inhalation injury compared with the typical accidental burns of civilian life.[159] In both settings, a compartment syndrome may develop due to capillary leak and soft tissue swelling, leading to elevations in pressures to the threshold that they threaten tissue perfusion. Escharotomies and fasciotomies may be necessary to prevent ischemic injury. Indeed, burns epitomize the inflammatory response. Anybody who has suffered sunburn knows all too well the rubor, calor, and dolor of inflammation. Now, multiply that feeling by

100 and you might approximate the excruciating pain of a blast burn injury. Temperatures from the explosive gases can reach 3,000°C (5,432°F) and result in fatal third-degree burns in victims close to the detonation. In nonblast burn patients, >90% will have SIRS at the time of death, and infection will be responsible for the fatal outcome in 46%.[160] Fortunately, many burn injuries that would have been fatal a decade ago are survivable due to rapid and proper treatment.[161] Nonetheless, survivors typically suffer long periods of hospitalization with notoriously disfiguring wounds.

It's important to remember that burns are a *distracting injury*. Thus, when these patients receive narcotics to treat their pain, the diagnosis of associated injuries, such as TBI, can be masked and made more difficult. In addition, the unique aspects of burn physiology and wound care associated with blast trauma can adversely impact the injured brain and complicate patient care.[162] For example, resuscitation is complicated by obligatory burn edema and the voluminous extravascular fluid shifts that result from thermal trauma. This can be very tricky because too little fluid resuscitation will result in cerebral hypoperfusion and too much will result in cerebral edema. Furthermore, burns are known to decrease myocardial contractility, increase the rate of venous thromboembolism, and prolong the duration of transport to a definitive treatment center. Soldiers with burn trauma are transported across three continents, with one stop in Germany, then to the burn center at Brooke Army Medical Center in San Antonio, Texas, with total ground and air transport times greater than 24 hours over 3 to 6 days.[163] Military burn casualties with head trauma are at increased risk for secondary TBI given the multiple complicated transfers. Thermal injuries have long-term effects, and up to 45% of combat *and* civilian burn patients suffer PTSD.[164,165]

IED Blast Burn Injury

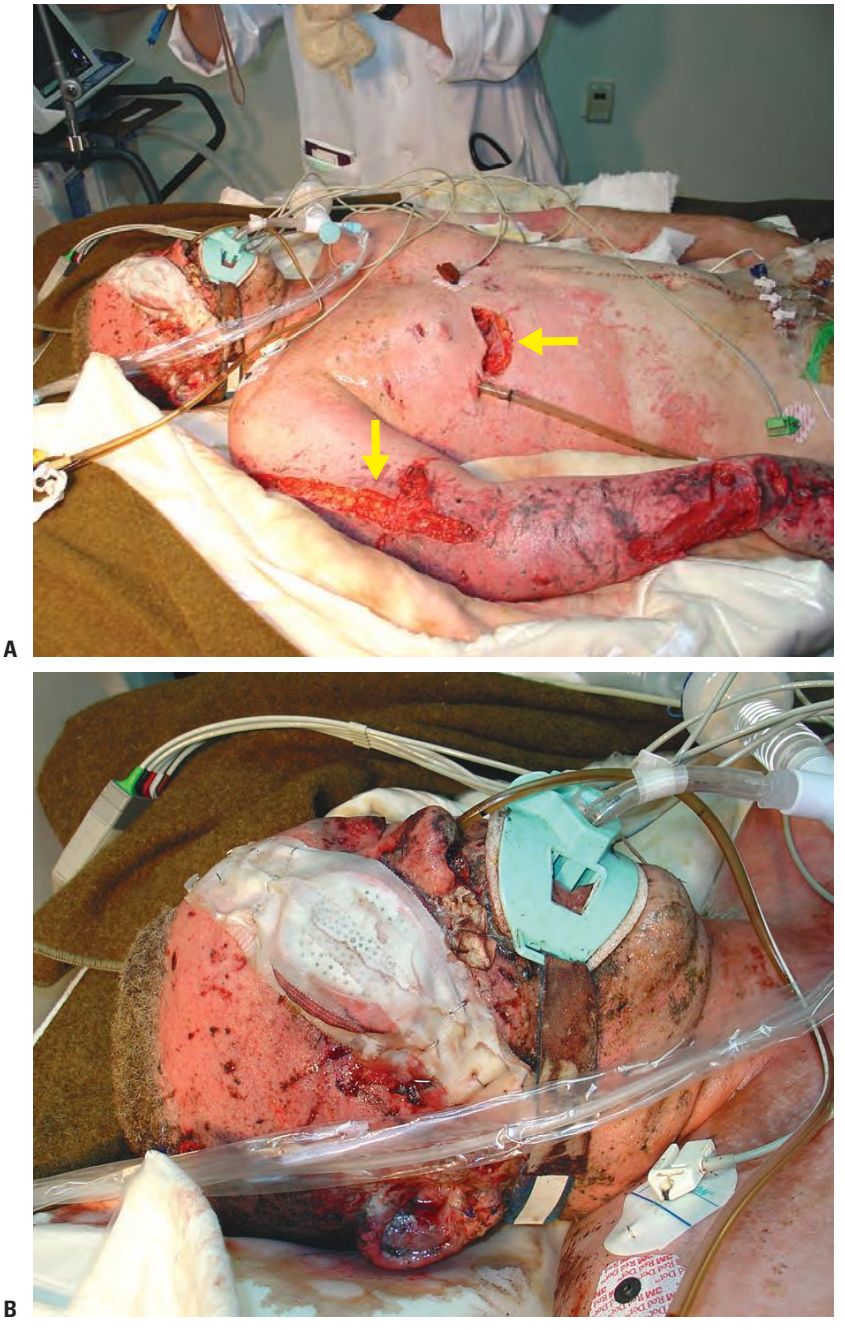


Figure 5.47. IED Blast Burn Injury. A. Note the characteristic distribution of combat trauma involving the upper extremity, skull base, and face. There is relative sparing of the chest due to body armor, but the soldier suffered blast lung trauma. He also lost vision in his right eye and underwent right upper extremity and chest wall escharotomies (*arrows*) for thermal injury. **B.** Close-up photograph of the soldier's face. Note the innumerable focal cutaneous shrapnel wounds.

★**KEY POINT** Military burn patients with TBI are at increased risk for *secondary* brain injuries given the multiple complicated transfers.

LESSON 8: ASSESSMENT OF THE TRUE EXTENT OF INJURY IN THE ACUTE SETTING IS MORE DIFFICULT

There are several reasons why the true extent of TBI injury incurred during combat may not be initially obvious to medical personnel. First, due to the extensive nature of the injuries, patients often have been treated for pain with analgesic and sedation medications before they arrive at the medical facility. These medications typically make it harder to complete an accurate neurologic assessment. Second, the patient (and physician) can be distracted by other injuries, such as an amputated limb. Third, current imaging technology, and specifically CT scanning, is not always able to detect the presence or extent of a TBI. Fourth, the physiologic and medical consequences of blast injuries (with or without burn injury) are unique and poorly understood. Blast injuries evolve over time and often don't manifest clinical signs or symptoms until 24 to 48 hours after injury. This has been documented in the lungs and bowel.[166–168]

In the brain, the blast wave not only induces an evolving injury in the parenchyma but delayed neurologic deterioration has also been documented secondary to blast-induced vasospasm.[169] Thus, with blast injuries, "what you see may not be what you get." The insidious and relentlessly progressive evolution of the injuries may culminate in devastating cellular effects and toxicities. Blast injuries warrant a high index of suspicion to enable early detection and interventions to ameliorate sequelae and complications. Early symptoms may be overlooked or disregarded in the face of other injuries. If blast-related injury is missed in battlefield triage, soldiers may return to combat prematurely. Early symptoms may be overlooked or disregarded in the face of other injuries. Unidentified blast TBI may be associated with a propensity for incurring additional injuries, and recovery from subsequent injury is slower following recurrent TBI.[19,170–172] Soldiers, like athletes, who have suffered two proximate injuries are up to nine times as likely to experience another TBI.

Soldiers with repetitive mild TBI predispose themselves to catastrophic second impact syndrome (SIS), also termed *dysautoregulation syndrome* (**Fig. 5.48**).[173,174] In SIS, devastating neurologic injury and even fatalities can occur following a mild TBI that occurs in the time window of ongoing postconcussive symptoms from a prior mild TBI. SIS is more common in young adults and is typified by the sudden onset of cerebral swelling with raised ICP. A force that would normally cause a mild TBI without obvious CT abnormality in an injury-naïve patient may result in a significant lesion on imaging in a patient who sustains injury

as part of a SIS. The pathophysiology of SIS is not understood. Awareness of delayed TBI complications such as SIS, pseudoaneurysm rupture, hydrocephalus, delayed infarction from vasospasm, and heat stroke can lead to preventative and therapeutic measures that can make the difference between good and poor functional recovery.

In acute TBI, the following CT findings can be helpful in assessing patient outcome:

TABLE 5.6 CT Findings in Acute Head Trauma that Are Associated with a Poor Prognosis

- Effacement of the basal cisterns
- Subarachnoid hemorrhage
- Intraventricular hemorrhage[a]
- Midline shift
- Subdural hematoma versus epidural hematoma (EDH)[b]
- Combination of intra- and extra-axial hemorrhages
- Focal versus diffuse injury
- Increased number (i.e., lesion burden) of intra-axial hemorrhages
- Large (>60 cm^3) size of the hemorrhage
- Location of the hemorrhage in the deeper structures of the brain
- Heterogeneity (mixed density) of the hemorrhage
- Hydrocephalus
- Corpus callosal injury

[a]In contrast to several studies in which the relation of intraventricular hemorrhage (IVH) to poorer outcome was mainly caused by the association with other predictors, the well-respected Rotterdam Study found IVH to be an independent predictor.[175]
[b]Although initially counterintuitive, the Rotterdam CT scoring system actually found the presence of an acute EDH to be a favorable prognostic finding.

1) Basal Cistern Effacement

Patients with obliterated perimesencephalic cisterns have long been known to have a worse outcome. In an early study by Toutant and colleagues,[176] effaced basal cisterns were associated with a fourfold risk of poor outcome, compared with those with preserved basal cisterns (controlling for similar GCS scores), and absent basal cisterns were associated with a mortality rate of 77%. In another early study by Teasdale and colleagues,[177] compression of the basal cisterns and the third ventricle closely correlated with an ICP >20 mm Hg, clinical signs of mesencephalon dysfunction, and a worse prognosis. Compression of the perimesencephalic cistern ipsilateral to the burden of injury has been associated with a worse outcome than compression of the contralateral cistern.[178] Presumably, contralateral cisternal effacement in the setting of an open ipsilateral cistern indicates a proportionate shift of the brain and brain stem structures to the contralateral side, whereas ipsilateral cisternal effacement represents a disproportionate shift of the cerebrum over brain stem structures that could lead to distortion of vital midline structures.

In a preliminary report from the National Traumatic Coma Data Bank (TCDB) pilot study on CT imaging, the ominous value of compressed or absent basal cisterns in severe head injury was further demonstrated.[179] Mortality rate when cisterns were absent was 77%, 39% when cisterns were compressed, and only 22% when cisterns were open. Subsequent studies have confirmed the important association between cisternal effacement and prognosis.[175,180,181] Compressed or absent basilar cisterns indicated a threefold risk of increased ICP and were found to be a strong CT predictor of 6-month mortality (associated with a twofold to threefold increase in mortality).

2) Traumatic Subarachnoid Hemorrhage

Numerous studies have described a significant correlation between the presence of SAH on CT and poor outcome.[180,182–186] One recent study showed that the extent of SAH was an independent risk factor for a number of cognitive domain deficits that occur following TBI.[187] SAH found in the perimesencephalic region is particularly ominous. Traumatic SAH is associated with a twofold increase in mortality, and hemorrhage in the basilar cisterns has a 70% positive predictive value for poor outcome. The presence of traumatic SAH is also correlated with the occurrence of secondary deterioration, and the degree and location of SAH have been reported to be associated with delayed ischemic symptoms caused by vasospasm. In addition, the greater the amount of SAH, the worse the prognosis.[188] A 40% incidence of traumatic SAH was reported in the U.S. TCDB population.[179] The presence of SAH also predicted an abnormal ICP, and the predictive value of SAH was shown to be additive to other CT scan parameters, such as the presence of abnormal cisterns, mass lesions, and midline shift. The outcome of patients with traumatic SAH was significantly worse than that of patients whose first CT scan did not show SAH. Unfavorable outcome was seen in 60% of SAH patients, as compared to 30% of patients without SAH. Finally, patients with traumatic SAH have a higher incidence of contusions, acute SDHs, IVH, and increased ICP signs.

3) Midline Shift

Shift of the midline structures as a valuable prognostic sign is somewhat controversial. Patients with diffuse cerebral injury (and a GCS score <8) typically demonstrate no significant midline shift, and yet they may often have poor outcomes. However, in the setting of a mass lesion, midline shift usually indicates increased ICP and, untreated, it is an indicator of poor clinical outcome. This association is somewhat complicated by the fact that when the shift is caused by intracranial hemorrhage, the latter also negatively impacts outcome. Nevertheless, most studies show that survival decreases as midline shift increases, and midline shift of >15 mm is a reliable prognosticator of poor outcome.[175,180,186] This is particularly the case when focal (i.e., non-TAI) intracranial injuries are evident. Athiappan and colleagues[189] found the prognostic value of midline shift varies with the type of intracranial lesion. It is more important in patients with a single contusion or intraparenchymal hematoma than for those with either multiple lesions or an extra-axial EDH or SDH. They also concluded that the presence of midline shift is better correlated with the type of pathology and GCS score, rather than the degree of midline shift taken alone.

In a retrospective study of 75 consecutive patients with head injury, Quattrocchi and colleagues[190] also found that the presence or absence of midline shift on the admission CT was prognostically significant. The presence of midline shift was associated with a poor outcome in 50% of cases, whereas the absence of midline shift was associated with a poor outcome in only 14% of cases. Significant predictive factors for poor outcome in this study were the presence of intracranial hemorrhage (34%), intracranial hemorrhage with midline shift (61%), and midline shift out of proportion to the extent of intracranial hemorrhage (88%). A more recent study concluded that the most important parameter for prediction of unfavorable outcome is the magnitude of the midline shift.[191] This parameter, as a continuous variable, was by itself a better predictor of morbidity and mortality than the Marshall CT score. The presence of midline shift has been incorporated into several neurosurgical treatment guidelines.[192] For example, an EDH less than 30 cm^3 with less than 15-mm thickness and less than 5 mm of midline shift may be managed nonoperatively in patients with GCS >8 without focal signs.[193]

By contrast, midline shift > 5 mm is alone, independent of the patient's GCS score, an indication to surgically evacuate an acute SDH.[194] Mathew and colleagues[195] recommended conservative management with a midline shift <10 mm in conscious patients, but Wong[196] recommended it only on those patients with a GCS score of 15. Wong found that a midline shift >5 mm in patients with a GCS score <15 was significantly related to conservative management failure due to exhaustion of the cerebral compensatory mechanisms within 3 days of injury. Although horizontal shift of the pineal gland is reported to correlate with the level of consciousness, one early study demonstrated that pineal shift is not of value in predicting restoration of consciousness after evacuation of the mass lesion.[179]

4) Subdural versus Epidural Hematoma

Many studies have shown that prognosis in patients with an EDH is much better than in those with an SDH or intracerebral hematoma.[185,197] Bricolo and colleagues[198]

postulated that mortality should approach zero in patients with an uncomplicated EDH. Although initially counterintuitive, the Rotterdam CT scoring system found the presence of an acute EDH to be a favorable prognostic finding. Further, some EDHs are clearly more indolent than other EDHs. Specifically, an EDH located anterior to the tip of the temporal lobe is particularly benign.[199] However, patients with both extra-axial and intra-axial bleeds tend to have a poor outcome.[200] Note that the Marshall CT classification does not permit any distinction on type of mass lesion nor does it include the presence of SAH or IVH.[201]

5) Focal versus Diffuse Injury

An important study by Gennarelli and colleagues[202] performed 30 years ago showed that focal lesions on CT were associated with a higher mortality than diffuse lesions. Patients with a GCS score of 6 to 8 and diffuse cerebral injury suffered 13% mortality, whereas patients with an identical GCS score and an SDH had a mortality of 36%. Similarly, a detailed study of patients with moderate head injury by Rimel and colleagues[203] demonstrated that as a patient's GCS score decreased from 12 to 9, the incidence of an intracranial mass increased. Similarly, patients with large contusions and low initial GCS scores are at risk for delayed deterioration.[204]

In a study by Richard and colleagues,[205] the combination of a hematoma with early brain swelling reduced the chance of full recovery in children. These early findings and those of others suggest that the presence of a focal intracranial mass lesion plays an important role in determining prognosis and that while somewhat counterintuitive, patients with diffuse injury (e.g., TAI) are more likely to survive than those with focal intracranial hemorrhage. For patients with TAI who survive, the extent of the initial axonal disconnection is a large determinant of their final outcome. Although an increased number of microbleeds (detected on MRI, not CT) adversely affects patient prognosis, the location of the microbleeds is also important. Initial studies suggest that lesion burden on susceptibility-weighted MRI correlates with clinical outcome, including duration of coma and long-term functional disability.[186,206–208]

6) Size of Intraparenchymal Lesions

Recent CT studies have shown a clear relationship between patient outcome and the size of the hematoma.[209] Intracranial hemorrhage has an approximately 80% positive predictive value for poor functional outcome, with worsening prognosis as the hematoma volume increases in size.[180] The correlation between hematoma volumes and midline shift are nearly colinear. In prognostic models, the analysis of hematoma volume and midline shift as continuous variables may be more informative over their use as dichotomous threshold values.[213] About half of patients with intracranial hemorrhage show enlargement of the lesion after hospital admission.[214,215] Of course, the timing of the initial CT scan likely influences the likelihood of hematoma expansion on serial CT and outcome prediction.

Guidelines and recommendations for the surgical evacuation of focal mass lesions have been published.[216] An acute SDH with a thicknesses >10 mm or midline shift >5 mm is usually evacuated, regardless of the patient's initial GCS score. Mathew and colleagues[195] proposed guidelines for the conservative management of traumatic acute SDHs. Their

criteria include a GCS ≥13, midline shift <10 mm, absence of CSF basal cisternal effacement, and absence of other associated intraparenchymal lesions. As mentioned previously, patency of the perimesencephalic cisterns is a protective finding and has been significantly correlated with a favorable outcome in patients conservatively managed, but hematomas greater than 10 mm are usually evacuated regardless of the status of the basal cisterns.[217] Similar results were found in the study by Kido and colleagues,[218] which showed a positive relationship between lesion size and the GCS score and between lesion size and serum catecholamine levels. Interestingly, previous research indicated that serum catecholamine levels reflect the GCS score and enabled prediction of the GOS score.[219,220]

7) Lesion Location

Hemorrhage location is also important for prognosis. In adults, posterior fossa hemorrhage is a predictor of poor outcome.[221] Small pediatric case series show that posterior fossa hemorrhage has been associated with fatal outcomes, but if the child survives, recovery and overall prognosis may be better than in adults.[200] *In general, the deeper the lesion, the worse the prognosis*. A landmark study by Levin and colleagues[222] classified lesions on imaging into (1) no lesion, (2) cortical, (3) subcortical, and (4) deep central gray/brain stem groups, according to the depth of the deepest parenchymal lesion. They showed that the depth of the brain lesion was directly related to severity of the patient's acute impairment of consciousness and inversely related to outcome. Lesko and colleagues[223] also observed that lesions of the brain stem are associated with a poor prognosis. Along this line, a difference in patient outcome was found between patients with a normal CT study versus an abnormal CT study, with 80% of patients with a negative study demonstrating a good outcome ($p = 0.0001$). An exception to this conclusion was noted in patients who had undetected brain stem lesions or those in whom secondary complications developed.

8) Hemorrhage Heterogeneity ("Spot Sign," "Swirl Sign")

Active bleeding or hyperacute bleeding may be identified on a noncontrast CT as a hypodense area within a hyperdense hematoma (**Fig 5.48**). These hypodense areas correspond to blood that has not yet clotted. Anemia, coagulation status, and time from injury to CT scan play a role in the appearance of blood on CT. Patients with heterogeneous (i.e., mixed-density) clots present earlier to the hospital, have poor GCS scores at admission, exhibit large clot volumes, have a high incidence of active bleeding at surgery, and have increased morbidity and mortality as compared with the patients with homogeneous hyperdense hematomas.[224] Contrast CT scans are often acquired immediately after CTA studies, and the presence of a *spot sign* on contrast CT is indicative of ongoing active bleeding and is an ominous predictor of poor outcome.[225-228]

9) Hydrocephalus

Multivariate logistic regression analysis indicates that hydrocephalus influences functional and behavioral outcome as well as the development of post-traumatic epilepsy.[209,229,230] Post-traumatic hydrocephalus affects about 50% of patients with severe TBI.

10) Injury to the Corpus Callosum

Injury to the corpus callosum has long been known to be significantly associated with an unfavorable prognosis. It is uncertain, however, whether the callosal injury merely serves as a marker of shearing injuries in more critical areas of the brain such that the poor outcome is related to the coexistence of TAI elsewhere in the brain.[231-233] Many studies of callosal lesions and prognosis may have been based on CT scan, which may, as compared to MRI, have underestimated the presence and extent of TAI.

Neurologic recovery is not reliably predicted from the pattern of injury identified on the initial CT study. One reason may be that relatively few studies have used a multivariate approach that explicitly takes into account the potential covariance among clinical and imaging predictive variables. A recent study by Yuh and colleagues[234] demonstrated that several predictors including midline shift, cistern effacement, SDH volume, and GCS were related to one another. Rather than being independent features, they concluded that their importance may be related to their status as surrogate measures of a more fundamental underlying clinical feature, such as the severity of intracranial mass effect. Another reason for the discrepancy between the CT findings and patient prognosis is attributed to CT's inability to demonstrate nonhemorrhagic white matter shearing injuries, brain stem pathology, and subtle superficial cortical lesions.

Gentry and colleagues[235] provided much of the groundwork for assessing the role of MRI in predicting patient prognosis after head injury. Their data, and many subsequent studies, concluded that MRI is significantly more sensitive than CT in detecting nonhemorrhagic and hemorrhagic (excluding SAH) traumatic lesions and that MRI more closely parallels a patient's final outcome. An inverse correlation was found between both the GCS and GOS and the number of shearing lesions. For example, although 80% of patients without shearing lesions demonstrated a good recovery, only 27% of patients with more than 10 shearing lesions had a good outcome. Not surprisingly, primary brain stem injury was also associated with a worse prognosis than if the patient demonstrated no abnormality within the brain stem. In addition, prognosis was not significantly correlated with the presence of isolated extra-axial collections unless they were associated with brain herniation.

Advances in outcome prediction may be possible with DTI because it is perhaps the most promising imaging tool to noninvasively characterize injury to the microstructure of the cerebral white matter, including brain regions that appear normal on conventional MRI.[29,30,236,237] Fiber tracking analyses have been related to global outcome and cognitive processing speed in the early postinjury time period. However, longitudinal data are needed to determine whether these relations between DTI and neurobehavioral outcome of TBI persist at longer follow-up intervals. In the nonacute setting, decreased brain volume has been associated with a poorer prognosis. Volumetric quantitation in TBI patients suggests that such measures might be predictive of cognitive outcome.[238,239] Bigler and colleagues[239] performed outcome studies focused on the volumes of the hippocampus and the temporal horn of the lateral ventricle. They found that, in the subacute phase after brain trauma, the volume of the temporal horn of the lateral ventricle correlated with intellectual outcome and that of the hippocampus with verbal memory function.

"Blast-Plus" TBI (Second Impact Syndrome)

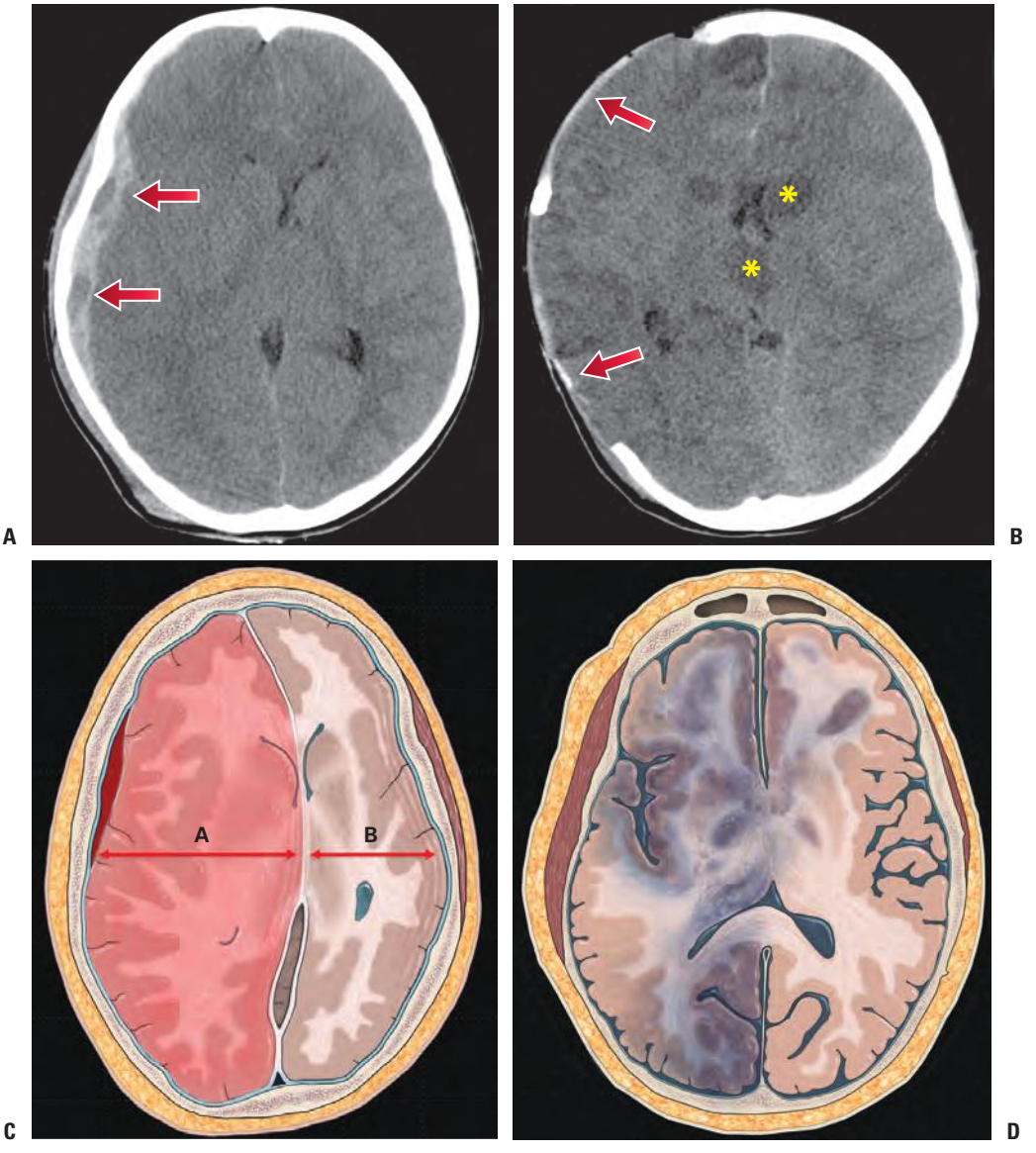


Figure 5.48. **IED Blast Trauma.** This 18-year-old soldier experienced *possible* LOC after an explosion. He had been exposed to prior IED blasts without LOC, and he described intermittent headaches to a fellow soldier. **A.** Noncontrast axial CT on admission demonstrates a right holohemispheric heterogeneous SDH (*arrows*) and a subtle decrease in gray–white matter differentiation. The intrinsic heterogeneity of the SDH suggests active extravasation and predicts expansion of the collection. **B.** The 24-hour postoperative CT shows interval development of multifocal nonhemorrhagic ischemic infarctions affecting the entire right hemisphere as well as the left caudate nucleus and paramedian thalami (*asterisk*). There is external herniation of the swollen brain through the craniectomy defect (*arrows*). **C.** Illustration of a different patient with a grossly similar admission CT, shows right hemispheric cerebral hyperemic swelling (note hyperemic right hemisphere **A** > left hemisphere **B**). **D.** Illustration of the brain several weeks later, shows multifocal ischemic injury. Unlike typical post-herniation infarction, SIS ischemia may not respect vascular territories. (Adapted with permission from Cantu R, Gean AD. Second impact syndrome and a small subdural hematoma: an uncommon catastrophic result of repetitive head injury with a characteristic imaging appearance. J Neurotrauma. 2010;27:1557–1564.)

★**KEY POINT** Although not diagnostic, this imaging appearance is characteristic of that found in the SIS: small isolated SDH without intra-axial hemorrhagic injury on admission CT that evolves into extensive bilateral nonhemorrhagic ischemic infarctions.

LESSON 9: FACIAL INJURIES ARE MORE COMMON AND MORE COMPLEX

The head and neck area comprises about 12% of total body surface area, but a retrospective analysis of 26 recent conflicts has shown a disproportionately higher number of injuries to the head and neck in the Iraq–Afghanistan conflict.[17,240,241] They are not only more common, but they are also more complex (**Figs. 5.49** through **5.52**). Kevlar helmets do not prevent projectiles from entering the cranium through the face, and it has been postulated that the enemy specifically targets the unprotected face of a soldier wearing body armor. The increase in numbers of maxillofacial injuries has also been attributed to increased urban warfare, that is, the confined area of an urban battlefield concentrates victims exposed to airborne foreign bodies from an explosion.

In World War II (WWII), 40% of troops with facial injuries died. This high mortality rate has been dramatically reduced to <1% in the current conflict through rapid evacuation and treatment of the wounded, the use of antibiotics, and the inclusion of an oral surgeon at forward-operating facilities. This is the first war in which rigid internal fixation is being applied to maxillofacial wounds on a large scale (**Figs. 5.51** and **5.52**). It is not yet known whether rigid internal fixation of maxillofacial war wounds has an effect on the incidence of infection, especially in comminuted fractures and avulsion defects of the mandible. Nonetheless, the following factors have been found to be useful in preventing infection of the region: early definitive treatment with debridement, irrigation, removal of readily retrievable foreign bodies, repair of hard and soft tissues, and institution of broad-spectrum antibiotics as soon as possible.[242]

Complex Maxillofacial Trauma (AK-47 Gunshot Wound)

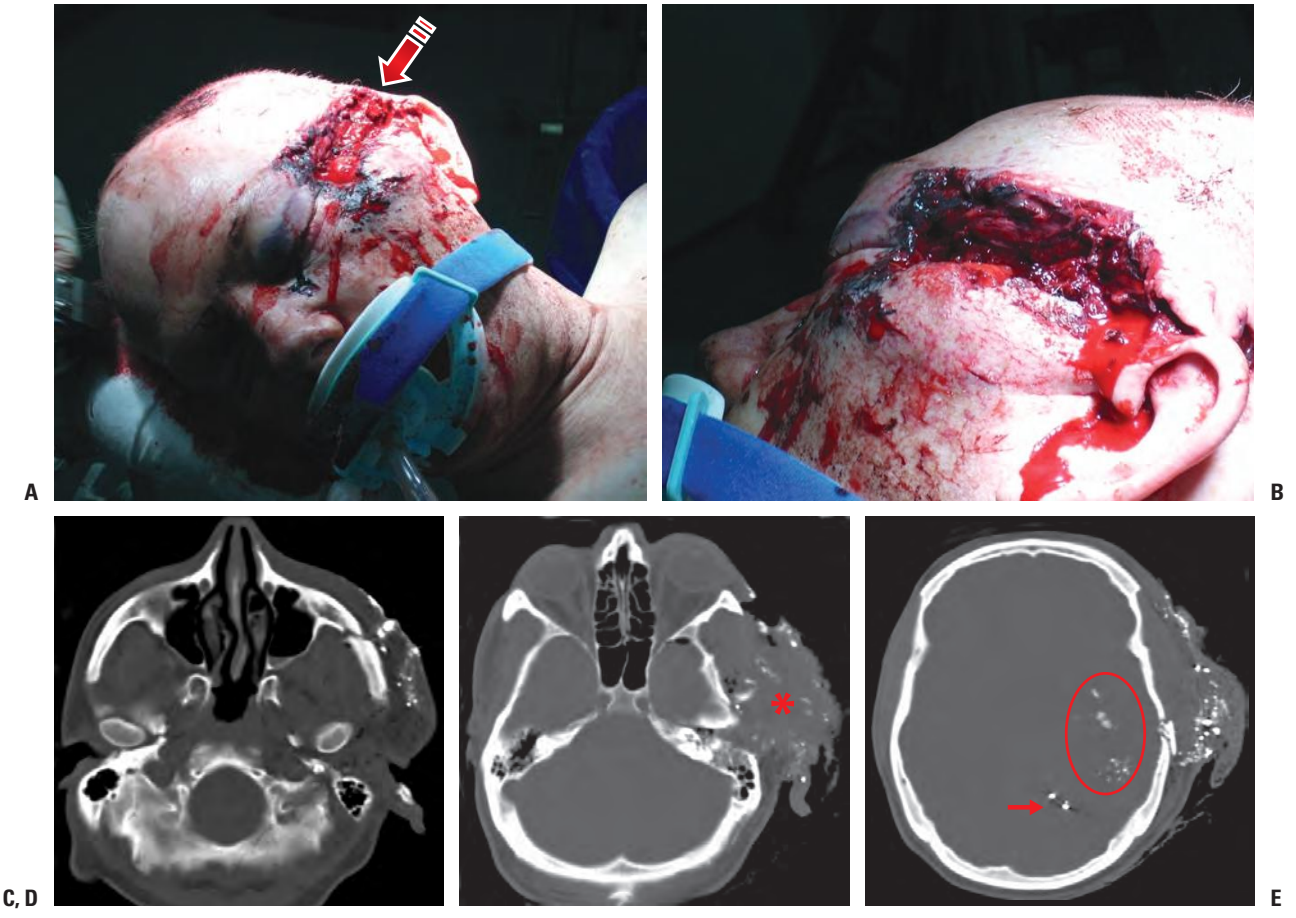


Figure 5.49. **Maxillofacial Ballistic Trauma. A, B.** Preoperative photographs demonstrate a severe left temporal craniofacial wound (*arrow*). **C–E.** Axial CT images show marked soft tissue swelling (*asterisk*) and a comminuted, compound temporal skull fracture with innumerable extracranial and intracranial radiopaque foreign bodies. Bullet fragments are displaced into the left occipital lobe (*arrow*) and bone fragments are displaced into the left temporal lobe (*circle*). Tangential and shallow wounds can be extremely mutilating, especially when caused by high-velocity, close-range AK-47 gunshot injuries.

★**KEY POINT** This case is similar to the ballistic keyhole fracture described in Figure 4.37 in that a tangential ballistic injury can cause significant intracranial injury.

The skeleton of the midface consists of an external shell of thin cortical bone supported by strong buttresses including the palate, zygomatic arch, zygomatic buttress, and lateral orbital walls. A strong blast wave impacting the midface may not damage these buttresses but can create severely comminuted fractures of the maxillary and ethmoid sinuses. Fractures of the orbital walls may directly damage the orbital soft tissue, and spalling and implosion of the sinuses can also contribute to the injury. Shearing forces can create a horizontal fracture of the mandibular body, and teeth may be transected at the cement-enamel junction by the blast wave.[243] Because these injuries are characteristically complex, securing an airway in such patients can be particularly challenging. The tongue falls back with mandible fractures. A free-floating maxilla can translate posteriorly, and displaced tooth fragments and/or foreign bodies can be aspirated. Furthermore, blood and rapidly evolving edema of the area can occlude the airway. Patients are rarely intubated nasally because of the potential for passage of the nasotracheal tube through a concomitant intracranial dural tear, accompanying the maxillofacial trauma. It is important for nasal septal hematomas to be immediately drained by incision and packing. Because the septal cartilage has no blood supply and receives all nutrients and oxygen from the perichondrium, an untreated septal hematoma may lead to a damaged septum and a saddle nose deformity. Disruption of the mucosal lining of the oral cavity can contaminate the deep structures of the face and neck with bacteria-laden saliva. However, the maxillofacial area recovers well, after conservative debridement, because of its rich vascular supply. There are no large muscle masses in the face, and thus, the risk of late cavitary necrosis and subsequent possible infection is considerably lower than with other types of combat injures.[244]

Complex Maxillofacial Injury (Blast Trauma)

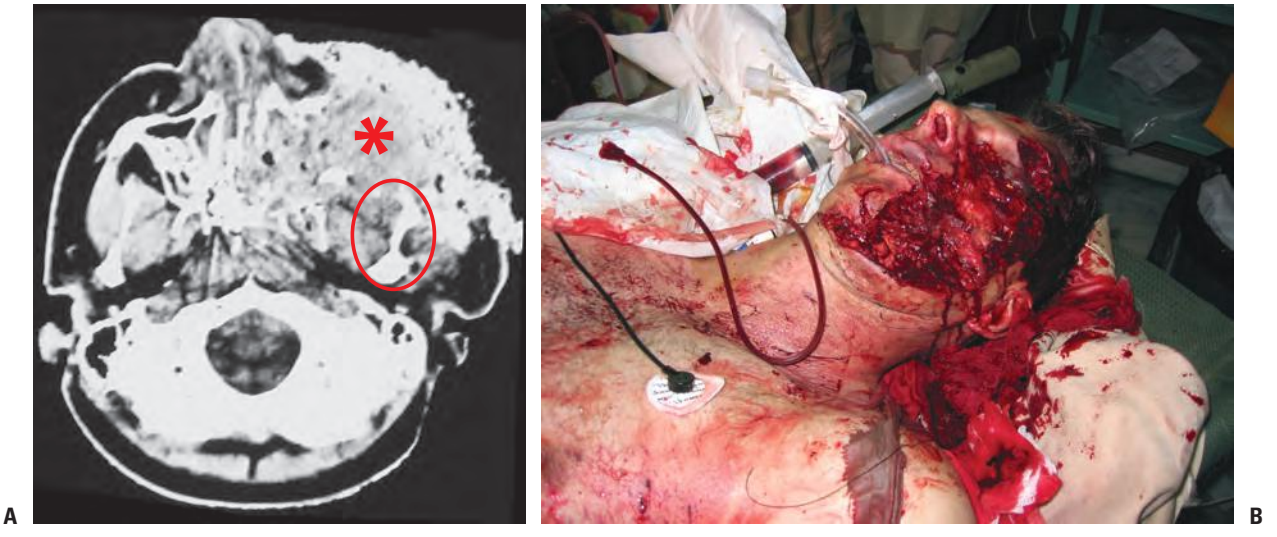
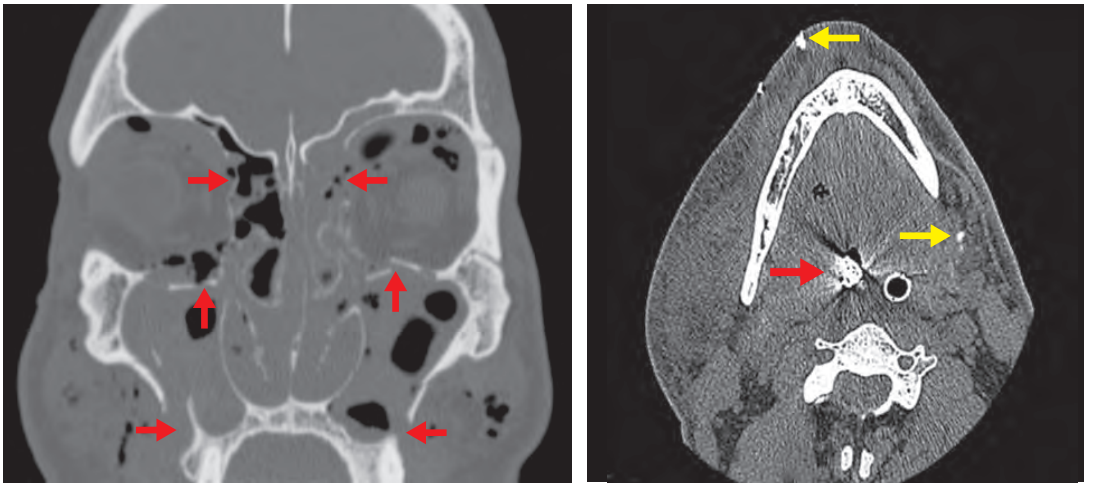


Figure 5.50. **Maxillofacial Trauma (IED Blast). A.** Noncontrast axial CT image of the same patient shown in **(B)** is limited by artifact, but nevertheless shows complete absence of discernible left facial structures (*asterisk*) other than a portion of the mandibular ramus (*circle*). The patient expired from hemorrhagic complications of polytrauma. **B.** Photograph of a soldier demonstrates mutilating facial soft tissue and osseous wounds characteristic of orbitofacial blast *degloving* injuries. (*Continued*)

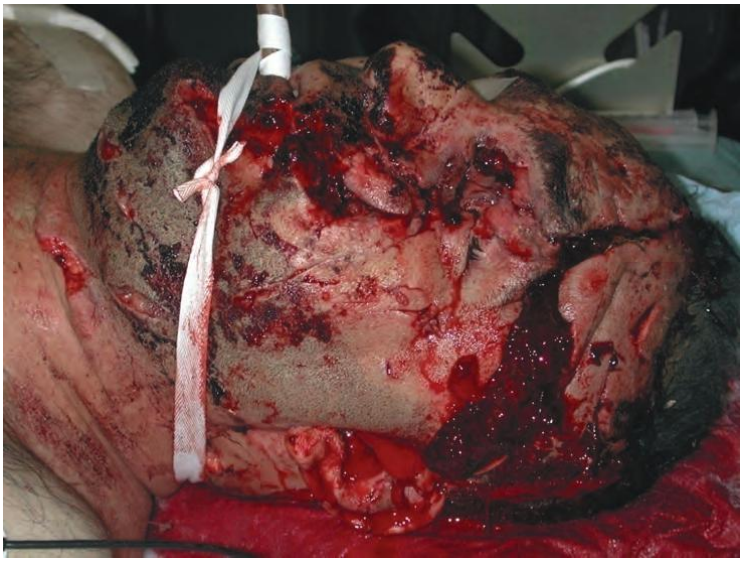


C D

Figure 5.50. (*Continued*) **C.** Coronal CT in another blast-injured patient illustrates innumerable bilateral midfacial fractures involving both orbits and the maxillary sinuses (*arrows*). Extensive orbital emphysema is noted, and there are bilateral superior orbital hematocysts present. **D.** Axial CT in the same patient shows small radiopaque densities within the right parasymphyseal skin and left submandibular region (*yellow arrows*) and a large metallic foreign body (shrapnel) embedded in the tongue (*red arrow*). There is marked swelling of the right masseter and hemifacial soft tissues. The airway is occluded and the patient is intubated.

★**KEY POINT** Facial trauma is more common in combat than in civilian trauma, and the injuries are more complex. Helmets do not protect from projectiles entering the cranium frontally through the face, and it has been postulated that the unprotected face of a soldier wearing body armor is not only exposed but specifically targeted by the enemy (See also Figure 3.22).

Complex Maxillofacial Injury (Blast Trauma)



A

Figure 5.51. **Complex Facial Trauma (Blast Injury). A.** Photograph of a patient status-post facial blast trauma demonstrates disproportionate involvement of the face due to protective body armor. There are innumerable irregular facial lacerations, including involvement of the left eye, ear, mouth, and nose. The patient is intubated. (*Continued*)

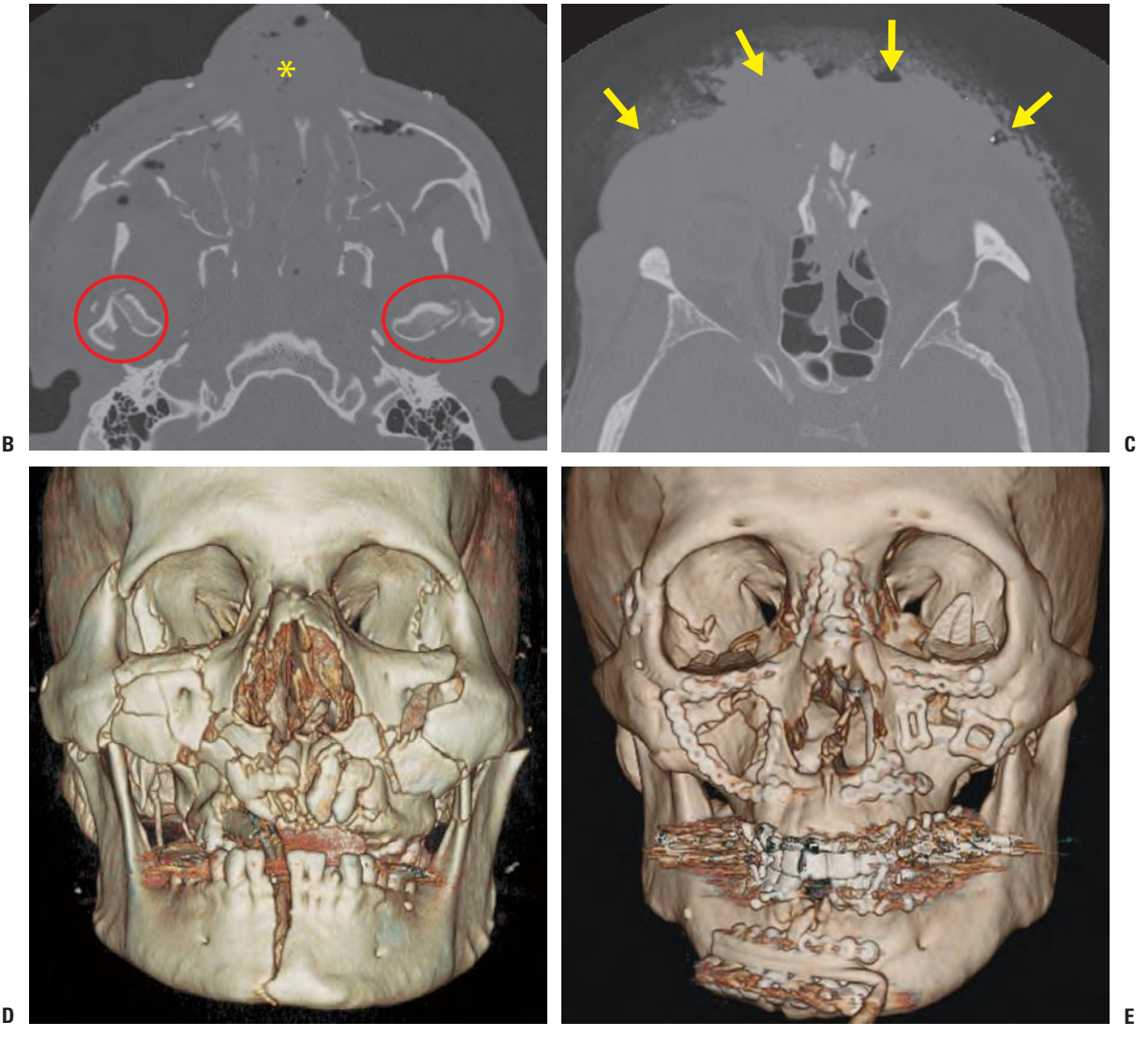


Figure 5.51. (*Continued*) **B, C.** Axial CT images in a different patient status-post complex facial blast trauma show fractures of all walls of the maxillary sinuses, nasal bones, nasal septum, pterygoid plates, and mandible (*circles*). Note extensive nasal (*asterisk*) and periorbital soft tissue swelling (*arrows*) with several punctate radiopaque foreign bodies. The patient also had bilateral globe rupture. The airway is occluded. **D.** Coronal reformatted 3D CT shows a midfacial "smash" injury with extensive comminuted fractures of the maxilla and mandible. The maxillary dentition has been completely disrupted. **E.** Postoperative 3D CT in a different patient with remarkably similar injuries illustrates the complex surgical reconstruction of panfacial trauma. Open reduction and internal fixation (ORIF) using multiple miniplates and maxillomandibular fixation was performed.

Mandible Blast Injury

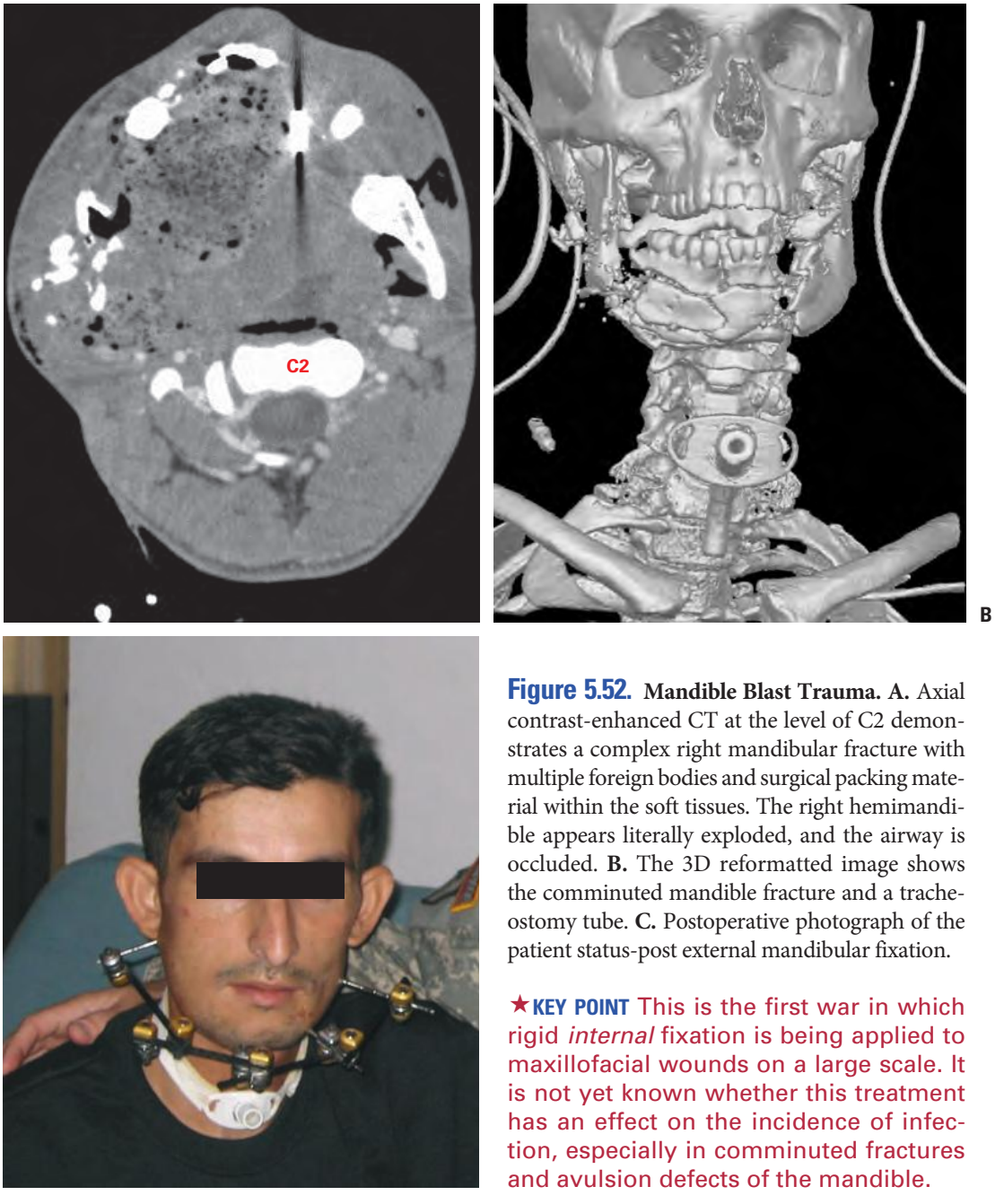


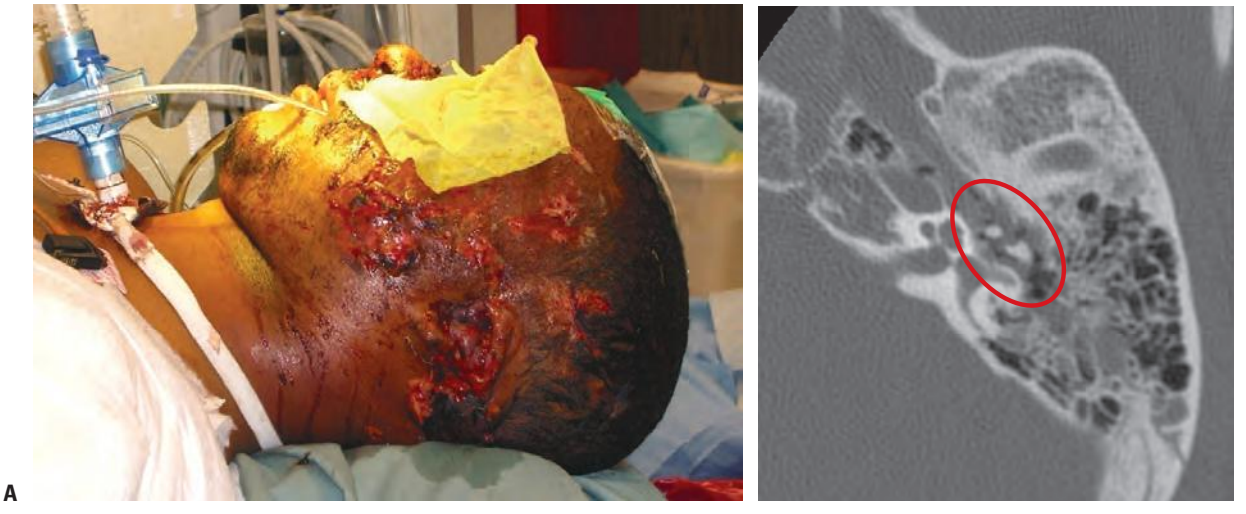
Figure 5.52. **Mandible Blast Trauma. A.** Axial contrast-enhanced CT at the level of C2 demonstrates a complex right mandibular fracture with multiple foreign bodies and surgical packing material within the soft tissues. The right hemimandible appears literally exploded, and the airway is occluded. **B.** The 3D reformatted image shows the comminuted mandible fracture and a tracheostomy tube. **C.** Postoperative photograph of the patient status-post external mandibular fixation.

★**KEY POINT** This is the first war in which rigid *internal* fixation is being applied to maxillofacial wounds on a large scale. It is not yet known whether this treatment has an effect on the incidence of infection, especially in comminuted fractures and avulsion defects of the mandible.

Hearing loss in combat is not surprising given that the function of the auditory system is to collect and amplify low-intensity pressure waves to transduce sound. Damage to the auditory system is far more common in combat than in civilian trauma because of the blast mechanism. Indeed, a ruptured tympanum, especially the pars tensa, is the most common sequelae of primary blast injury, followed by pulmonary injury and intestinal injury (as emphasized earlier, we don't yet know where BINT fits into this list).[245] In the Oklahoma City bombing, 35% of survivors reported auditory injury.[246] Of the survivors exposed to the 2003 bomb blast in Mumbai, India, 90% of patients had symptoms referable to auditory injury (hearing loss in 75% and tinnitus in 38%).[247] Of these patients, 77% had tympanic membrane perforations, in which 37% were bilateral. In a large retrospective study of U.S. service members injured in Iraq and Afghanistan, 16% of explosion-injured patients demonstrated symptomatic tympanic membrane perforation.[248]

Fortunately, the tympanic membrane frequently heals spontaneously within a few weeks. More extensive perforations may require tympanoplasty. The ossicular chain can be damaged by direct displacement of the ossicles (**Fig. 5.53**) or, more commonly, by severe distortion of the tympanic membrane at the attachment of the malleus. Perilymphatic fistula, damage to sensory structures on the basilar membrane of the cochlea, cholesteatoma (due to forceful distribution of squamous epithelium), and serous otitis are other causes for combat-related hearing loss.[249] Otalgia, tinnitus, vestibular dysfunction with vertigo, and bleeding from the external ear canal are additional complications of blast trauma. The observation that there is a significant association between barotraumatic tympanic perforation and concussive brain injury suggests that physicians who are treating blast survivors with tympanic membrane perforation need to have a high index of suspicion for concomitant brain injury.

Complex Maxillofacial Injury (Blast Trauma)



A B

Figure 5.53. IED Blast Trauma with Hearing Loss. A. Photograph of a soldier shows extensive 2° and 4° blast trauma. There is destruction of the left auricle from both shrapnel and burn injury. A tracheostomy tube is present because he also suffered blast lung injury. **B.** Axial CT through the left temporal bone demonstrates partial opacification of the mastoid air cells and incudomalleal dislocation (*circle*).

In studies of TBI resulting from noncombat trauma, anywhere from 20% to 65% of individuals with TBI have impaired olfaction.[250,251] The likelihood of loss of olfactory function following a TBI increases with the severity of the injury. The incidence of olfactory loss from a head injury may be higher in combat trauma. In a recent report of combat veterans, 52% with a history of mild TBI had impaired olfaction.[252] That study showed that *the most sensitive tool for detecting residual brain dysfunction on neurologic examination is olfactory testing*. Head injury usually impairs olfaction by shearing the olfactory nerves at the cribriform plate. Bruising of the anteromedial aspect of the frontal lobes can also compromise olfaction (**Fig. 5.54**).[253] MRI studies of civilians with post-traumatic anosmia have demonstrated damage to the olfactory bulbs and tracts, subfrontal cortex, and temporal lobes.[254,255] Importantly, individuals often do not recognize that they have impaired olfaction following TBI.[256] Testing for olfaction is a good way to identify recent and remote TBI.[257] In noncombat trauma, improvement in olfactory dysfunction due to head injury occurs in approximately one-third of patients, worsening of function evolves in another 20% to 25%, and the deficit stays stable in the remainder.[250,258] If recovery occurs, it usually does so in the first 6 months to 1 year following injury. Olfactory dysfunction is also correlated with an increased incidence of PTSD.[259,260]

"Blast-Plus" TBI (Post-traumatic Anosmia)

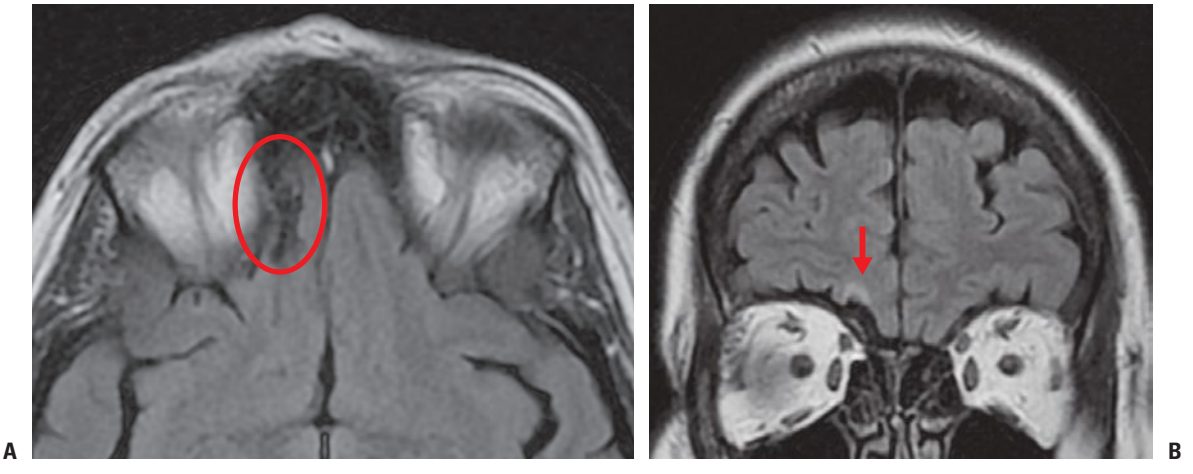


Figure 5.54. **A.** Axial and **(B)** coronal FLAIR MRI of a 21-year-old soldier status-post IED blast trauma who presented with chronic headaches, memory problems, and anosmia. Olfaction dysfunction after TBI often results from shearing and disruption of the fine and delicate olfactory fibers that pass through the cribiform plate and connect the receptors in the nose with the olfactory bulbs. For this reason, orbitofrontal lesions may be a marker for olfactory dysfunction following TBI. Note absence (i.e., cystic encephalomalacia) of the right medial orbital gyrus (*circle*). A small focus of T2 hyperintensity is seen adjacent to the encephalomalacia (*arrow*). The gyrus rectus is normal bilaterally. There is also diffuse volume loss for a patient this age.

★**KEY POINT** Olfactory dysfunction is frequently overlooked in combat victims and is correlated with a higher incidence of residual TBI and PTSD.

Unfortunately, while the ocular surface is only 0.1% of the total body surface area, it accounts for a disproportionately high percentage of explosive injuries.[261,262] Among the World Trade Center explosion survivors in 2001, 26% had ocular injuries.[263] A meta-analysis of blast injury occurring over the last 50 years revealed ocular injury in nearly 30% of blast survivors.[264] This is in stark contrast to typical civilian trauma. For example, ocular injuries are sustained in up to 80% of combat TBI victims compared with 3% in civilian TBI.[265] In addition, ocular injuries have increased in the current wars in comparison to prior conflicts.[266] Of all hospital admissions from the battlefield, 10% have ocular injuries, 64% of which are open globe and 13% of which required enucleation.[267] It should be remembered that the eyes and ocular adnexa are exposed to the same blast forces that cause TBI and can be expected to undergo similar destructive forces including compression, coup, contrecoup, shear, stress, and deceleration. The orbit is also a primary entry portal for intracranial penetrating foreign bodies (**Figs. 5.55, 5.56,** and **5.65**). Blast eye injuries are usually bilateral and often result in multiple ocular foreign bodies. Occasionally, the primary blast force can rupture the globe. A globe weakened by prior trauma or surgery (e.g., cataract) is at higher risk for damage from a primary blast injury. Because of the young age of our soldiers, globe rupture is a devastating injury following blast trauma.

War zone orbital and ocular injuries include complex corneo-scleral and lid lacerations, hyphema, serous retinitis, traumatic cataract, dacryops/dacrocystocele, orbital compartment syndrome (due to retrobulbar hemorrhage and/or orbital emphysema), orbital hematocyst, carotid cavernous sinus fistula, globe rupture (with and without intraocular foreign body), and orbital fracture (**Figs. 5.56** through **5.63**). The overall incidence of post-traumatic orbital emphysema with orbital fractures is 61%.[268] Because air in the orbit expands at high altitudes, air transport of patients with orbital emphysema requires special altitude restrictions to minimize the risk of orbital compartment syndrome. Flying at a lower altitude, however, puts the aircraft at risk for attack by insurgent firepower. Medial wall blow-out fractures are more often associated with orbital emphysema than are inferior orbital blow-out fractures. The bony lattice of the ethmoid labyrinth provides structural support to the medial walls. Therefore, although the lamina papyracea of the medial wall is thinner than the floor of the orbit, medial wall blow-out fractures occur less commonly than inferior blow-out fractures. Medial wall blow-out fractures are, however, frequently seen in combination with fractures of the orbital floor.

Orbital and ocular injuries were more common in early battlefield conflicts, but the introduction of safety goggles has decreased the incidence of these injuries over the years (**Fig. 5.64**). Unfortunately, soldiers can be noncompliant with the wearing of eye protection because it promotes dust and sweat accumulation, thereby interfering with the quality and range of vision. Because survival in a hostile fire zone requires high visual function, eye armor is sometimes intentionally removed. The face is also a prime target for sniper fire.[269] Terrorism and military injuries cause greater ocular damage and present with more severe visual acuity impairment than civilian injuries.[270] In the civilian setting, the eye is usually disrupted with a single rupture site and the globe remains largely intact. In an explosive injury, multiple projectiles strike the eye at high velocity and shred the ocular and adnexal tissue, often making repair impossible. Ocular injury is particularly common with landmine blast victims. The typical landmine casualty will have *peppering* of the face by small fragments

(mainly soil and grit) and bilateral penetrating ocular injuries. Frequently, ocular injuries from landmines are caused by small particles, rather than by large pieces of shrapnel, as seen with grenade or rocket injuries. With ocular injury caused by small fragments, the globe may be preserved.

Factors associated with a poor prognosis and globe loss include: wound >10 mm, injuries from blunt objects such as a BB (sharp foreign bodies cause less ocular disruption), location of a foreign body beyond the anterior segment, afferent pupillary defect, organic foreign bodies, and an initial visual acuity less than 10/200.[271] Janković and colleagues[272] showed a statistically significant correlation between admission within 12 hours of injury and improved postoperative visual acuity. However a more recent study by Ehlers and colleagues[273] found no significant association between time to surgical intervention and outcome.

Currently, soldiers with eye injuries are aeromedically evacuated and the mean time to primary repair is within hours of the injury.[274] Patients with open globe injuries are initially treated with *Patch and Evac* using a rigid eye shield (not a pressure patch). Flight evacuation to definitive medical care can risk further orbital damage from expansion of intraorbital air, introduced by the trauma. To avoid this, the pilot may need to fly at a lower altitude than usual. Planes flying at altitudes less than 5,000 ft are vulnerable to being attacked, so evacuation of patients with orbital injuries can be a difficult balance.

Two potential delayed complications of a ruptured globe include sympathetic ophthalmia and endophthalmitis. Sympathetic ophthalmia is a well-known, albeit rare, immune-mediated, inflammatory condition resulting in the fellow eye being blinded after an open globe injury. It typically develops <3 weeks postinjury, and 90% of cases occur by first year. Early enucleation of the wounded eye has made this complication extremely rare. In one recent study, the risk of sympathetic ophthalmia was found to be 0.3% (2 out of 660 patients).[275] Ehlers and colleagues[273] found the risk of endophthalmitis after an open-globe injury to be 4% (4 out of 96 patients). Organic foreign bodies tend to be more highly associated with endophthalmitis and sympathetic ophthalmia because they are more prone to infection or inflammatory reaction.[276] Interestingly, two recent studies of injured patients in Iraq found no cases of endophthalmitis or sympathetic ophthalmia despite a high incidence of delayed foreign body removal.[277,278] Lack of complications in combat patients may be due to the prompt closure of an injured globe performed in a theater hospital near the battlefield and the expedient use of broad-spectrum antibiotics. Fortunately, the rate of enucleation during wartime has decreased substantially over the last century. In World War I (WWI), it was as high as 50%. In WWII, it dropped to 40%, then 30% in the Korean War, and 20% during the Vietnam War. Currently, primary removal of the eye is performed in approximately 13% of combat ocular injuries.[279]

The Orbits Are the Main Entry Site to the Brain in Blast Trauma

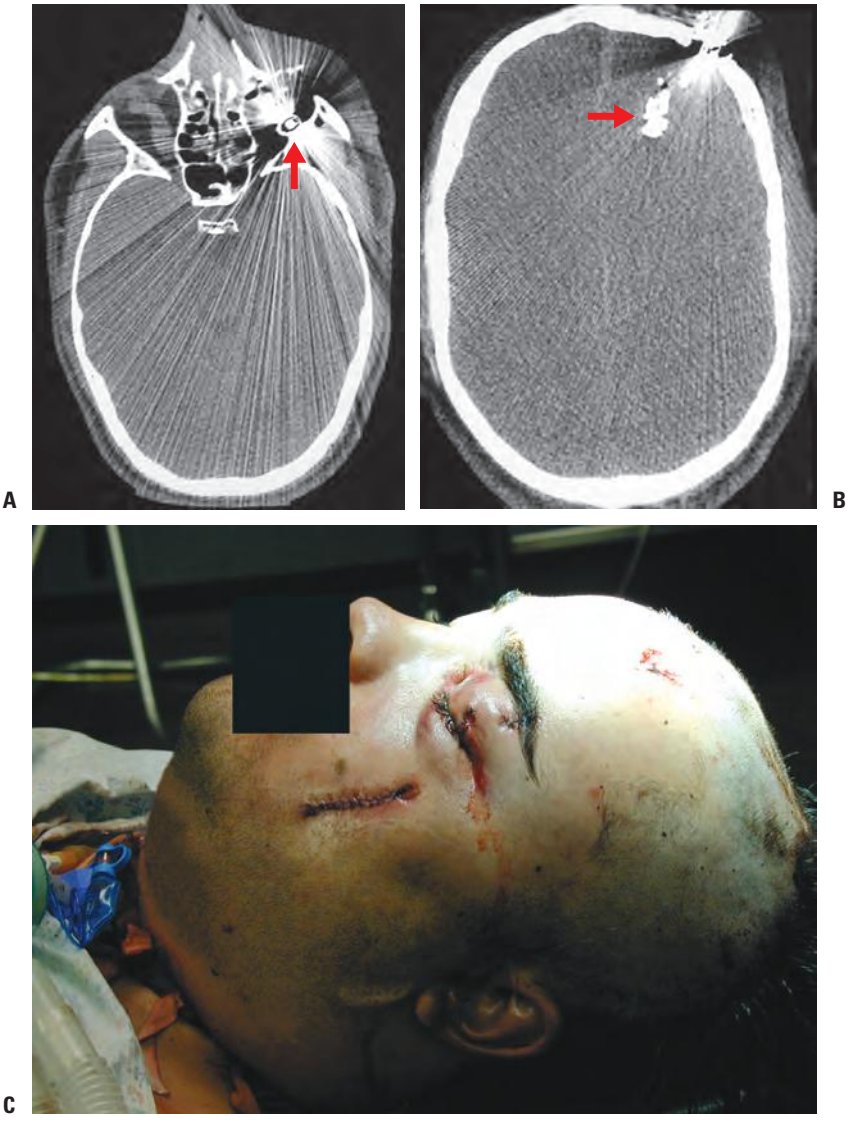


Figure 5.55. Orbitocranial Blast Trauma. A. Preoperative axial CT through the orbits demonstrates a large metallic foreign body within the left orbit causing severe streak artifact (*arrow*). **B.** CT image at the level of the centrum semiovale shows intracranial bone fragments from a second penetrating injury (*arrow*). Note complete effacement of the convexity sulci and loss of the gray–white matter differentiation, consistent with severe hypoxic–ischemic encephalopathy. **C.** Postoperative photo of the terrorist attack victim demonstrates the orbital entrance wound and a left facial penetrating injury.

★**KEY POINT** Penetrating orbitocranial foreign bodies are a particular problem in combat and in terrorist attacks.

Bilateral Penetrating Ocular Injury (IED Explosion)

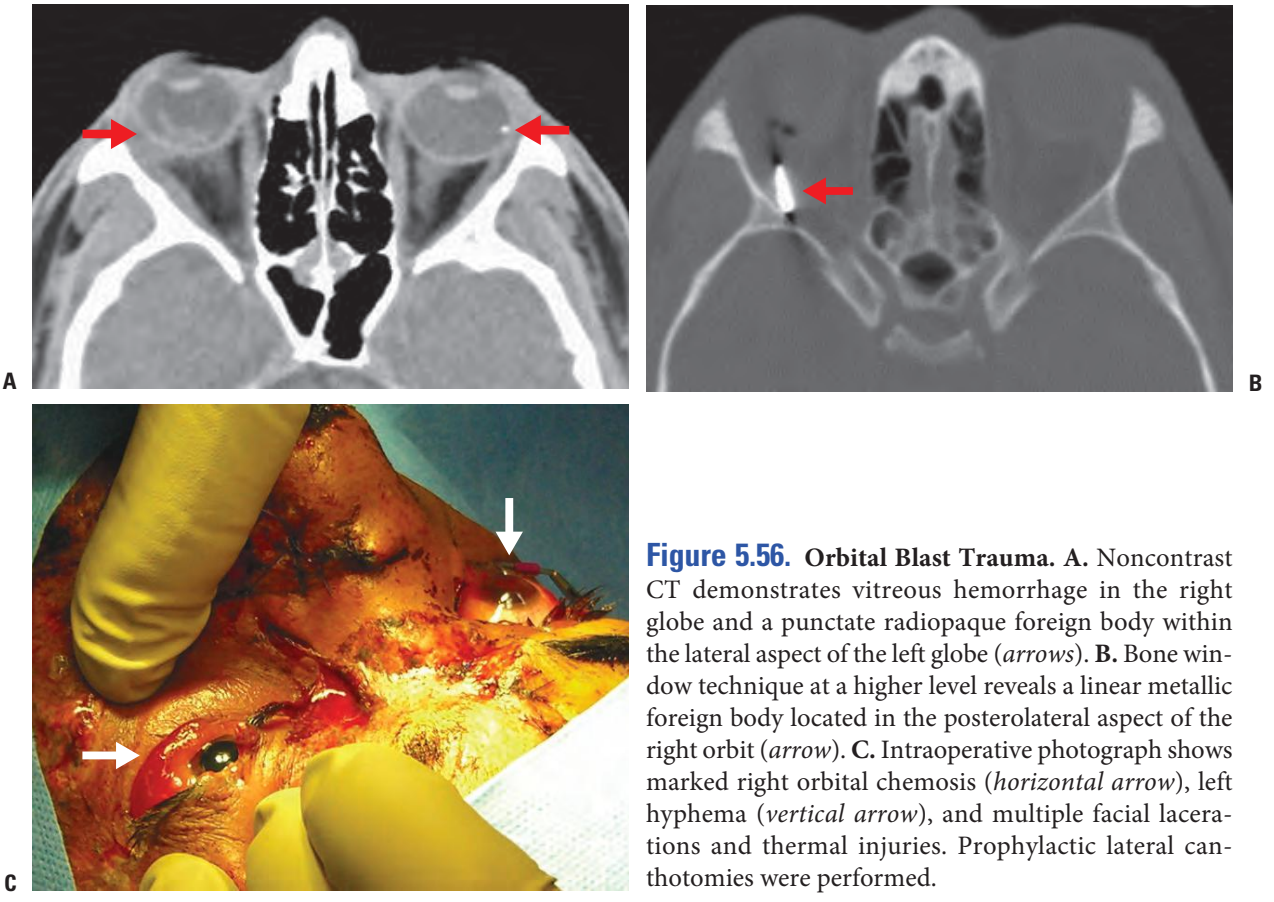


Figure 5.56. Orbital Blast Trauma. A. Noncontrast CT demonstrates vitreous hemorrhage in the right globe and a punctate radiopaque foreign body within the lateral aspect of the left globe (*arrows*). **B.** Bone window technique at a higher level reveals a linear metallic foreign body located in the posterolateral aspect of the right orbit (*arrow*). **C.** Intraoperative photograph shows marked right orbital chemosis (*horizontal arrow*), left hyphema (*vertical arrow*), and multiple facial lacerations and thermal injuries. Prophylactic lateral canthotomies were performed.

Unilateral Blunt Orbital and Ocular Trauma

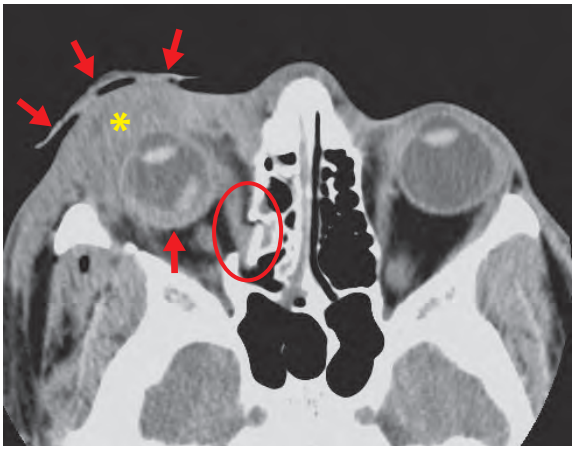


Figure 5.57. Globe Rupture, Medial Orbital Blowout Fracture, and Medial Rectus Entrapment. Axial CT demonstrates abnormal density within the posterior right globe (*red arrow*), consistent with an acutely ruptured globe (also known as an *open globe*). In this patient, the size of the globe is only minimally decreased, and the contour remains spherical. There is extensive periorbital soft tissue swelling (*asterisk*) and a linear area of soft tissue density overlying the orbit (*red arrows*) that represents a rigid protective shield to protect against prolapse of the internal contents of the eye. Note also a fracture of the medial orbital wall with displacement of the medial rectus into the fracture defect (*circle*).

★**KEY POINT** Other CT findings that suggest a ruptured globe include intraocular air, scleral discontinuity, deformity of globe contour, and intraocular foreign bodies.

Orbital and Ocular Trauma (Orbital Hematocyst)

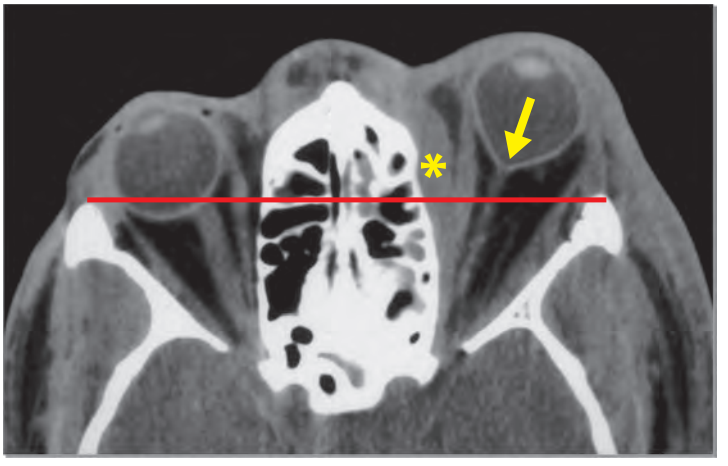


Figure 5.58. Orbital Hematocyst and Globe Tethering. Axial CT demonstrates well-defined soft tissue density located between the lamina papyracea and medial rectus muscle (*asterisk*), consistent with an orbital hematocyst. The orbital hematocyst usually results from injury to the subperiosteal vessels of the orbital wall resulting in focal hemorrhage. Thus, an orbital hematocyst is a form of subperiosteal hematoma. Note proptosis and tenting of the posterior aspect of the left globe (*arrow*). The mean age of patients with an orbital hematocyst is 17 years. With advancing age, the periosteum becomes more adherent to the orbital walls and the incidence of the hematocyst decreases.

★**KEY POINT** Globe tenting/tethering is an ominous imaging sign of increased intraorbital pressure and forewarns impending ischemic optic neuropathy.

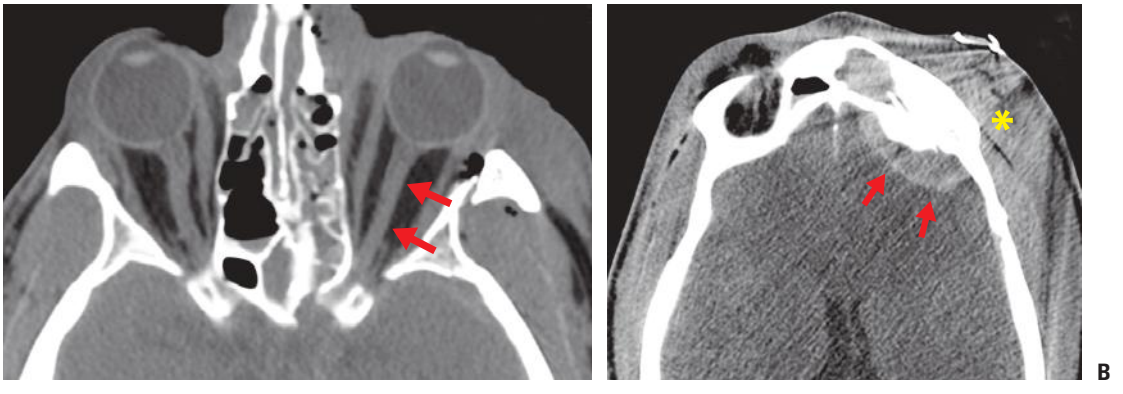


Figure 5.59. Orbital Hematocyst Secondary to Direct Extension from an Intracranial Epidural Hematoma. **A.** Axial CT demonstrates abnormal straightening of the left optic nerve sheath complex (*arrows*); compare with the normal lax right optic nerve. The shape of the left globe is only minimally deformed. **B.** Intracranial CT image shows an inferior frontal EDH (*arrows*) that, on contiguous levels, was observed to have dissected into the superior aspect of the orbit. Extensive periorbital and frontal subgaleal soft tissue swelling is also noted (*asterisk*).

★**KEY POINT** Prior to globe tenting/tethering, the optic nerve sheath complex loses its normal slack redundancy. Thus, straightening of the optic nerve is one of the earliest imaging manifestations of increased intraorbital pressure. The orbital hematocyst is also known as the "EDH of the orbit" because it represents a well-defined hematoma located between the orbital wall and the orbital periosteum. Unlike this case, it is usually seen without an associated intracranial EDH.

Ocular Trauma (Globe Rupture)

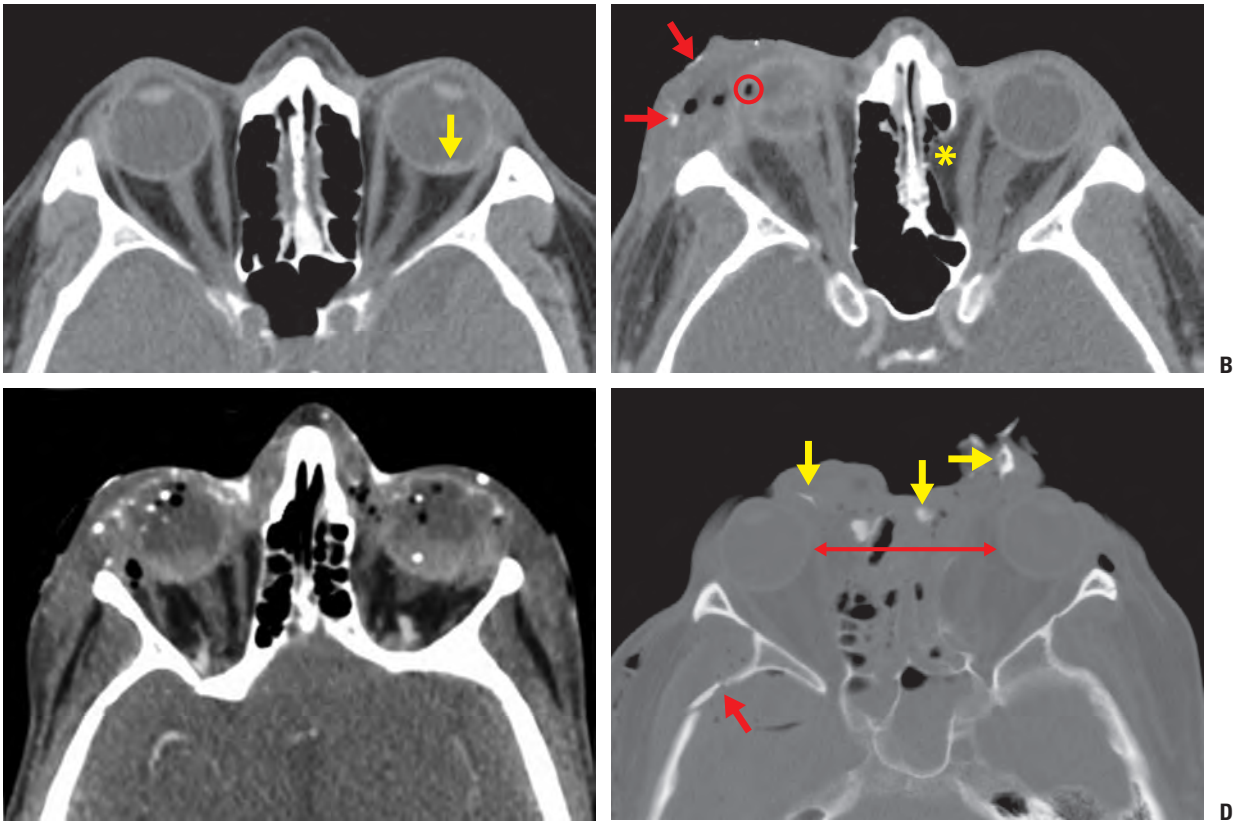


Figure 5.60. **Globe Rupture (Blast Injury). A. Subtle globe rupture.** Axial CT demonstrates subtle irregularity of the posterior aspect of the left globe (*arrow*). Although the shape of the left globe is preserved, the patient's intraocular pressure was abnormally decreased. **B. Obvious globe rupture.** Contrast-enhanced CT through the orbit shows a small, misshapen right globe with extensive periorbital soft tissue swelling, several radiopaque periorbital foreign bodies (*arrows*), and intraocular air (*circle*). Note the remote left medial orbital blow-out fracture (*asterisk*). **C. Bilateral globe rupture.** Contrast-enhanced axial CT demonstrates multiple tiny fragments and air within the right and left globes. Neither globe was salvageable. (Courtesy of Les Folio, DO, MPH, FAOCR, Col [ret], USAF, MC.) **D. Bilateral globe rupture and naso-orbitoethmoid (NOE) complex fracture.** Axial CT viewed at bone windowing shows an NOE fracture with resultant hypertelorism (note the increased intercanthal distance [*double-headed arrow*]). There are numerous periorbital radiopaque foreign bodies identified (*yellow arrows*) and a fracture of the right middle cranial fossa (*red arrow*).

★**KEY POINT** Factors associated with a poor prognosis and globe loss include: wound >10 mm, injuries from blunt objects such as a BB (sharp foreign bodies cause less ocular disruption), location of a foreign body beyond the anterior segment, afferent pupillary defect, organic foreign bodies, and an initial visual acuity less than 10/200. Two potential delayed complications of a ruptured globe include sympathetic ophthalmia and endophthalmitis.

Orbital Trauma (Orbital Emphysema)

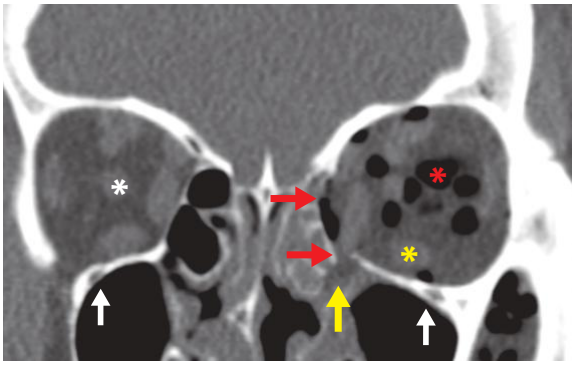


Figure 5.61. Orbital Blow-out Fracture. Coronal CT image shows a left medial wall blow-out fracture extending into the orbital floor (*red arrows*). Note elongation of the medial rectus muscle into the fracture defect and herniation of orbital fat into the maxillary sinus (*yellow arrow*). Compare to the normal right medial rectus muscle. The patient also demonstrated clinical evidence of muscle entrapment. The inferior rectus muscle is normal (*yellow asterisk*). Extensive intraconal and extraconal orbital emphysema (*red asterisk*) surrounds the optic nerve, which is barely discernible; compare with the normal right optic nerve (*white asterisk*). Inferior orbital blow-out fractures typically occur more laterally through the inferior orbital foramen (*white arrows*).

★ **KEY POINT** Because air in the orbit expands at high altitudes, air transport of patients with orbital emphysema requires special altitude restrictions. Flying at a lower altitude, however, puts the aircraft at risk for attack by insurgent firepower.

Orbital Trauma (Optic Canal Fracture)

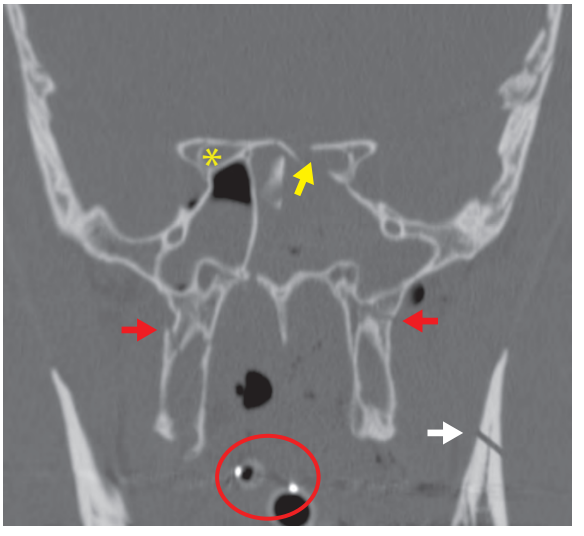


Figure 5.62. Optic Canal Fracture. Coronal reformatted CT shows a fracture through the left optic canal (*yellow arrow*); compare with the normal right optic canal (*asterisk*). Both sphenoid sinuses are opacified with blood. There are bilateral fractures of the pterygoids plates (*red arrows*) and a nondisplaced left mandibular fracture (*white arrow*). The patient is intubated and a nasogastric tube is present (*circle*).

★ **KEY POINT** When intraocular and intracranial causes of post-traumatic vision loss have been ruled out, visual impairment is usually due to direct optic nerve injury or interference with the blood supply to the optic nerve. The majority of optic nerve injuries occur at the level of the intracanalicular portion of the optic nerve.

Carotid-Cavernous Fistula (Blast Injury)

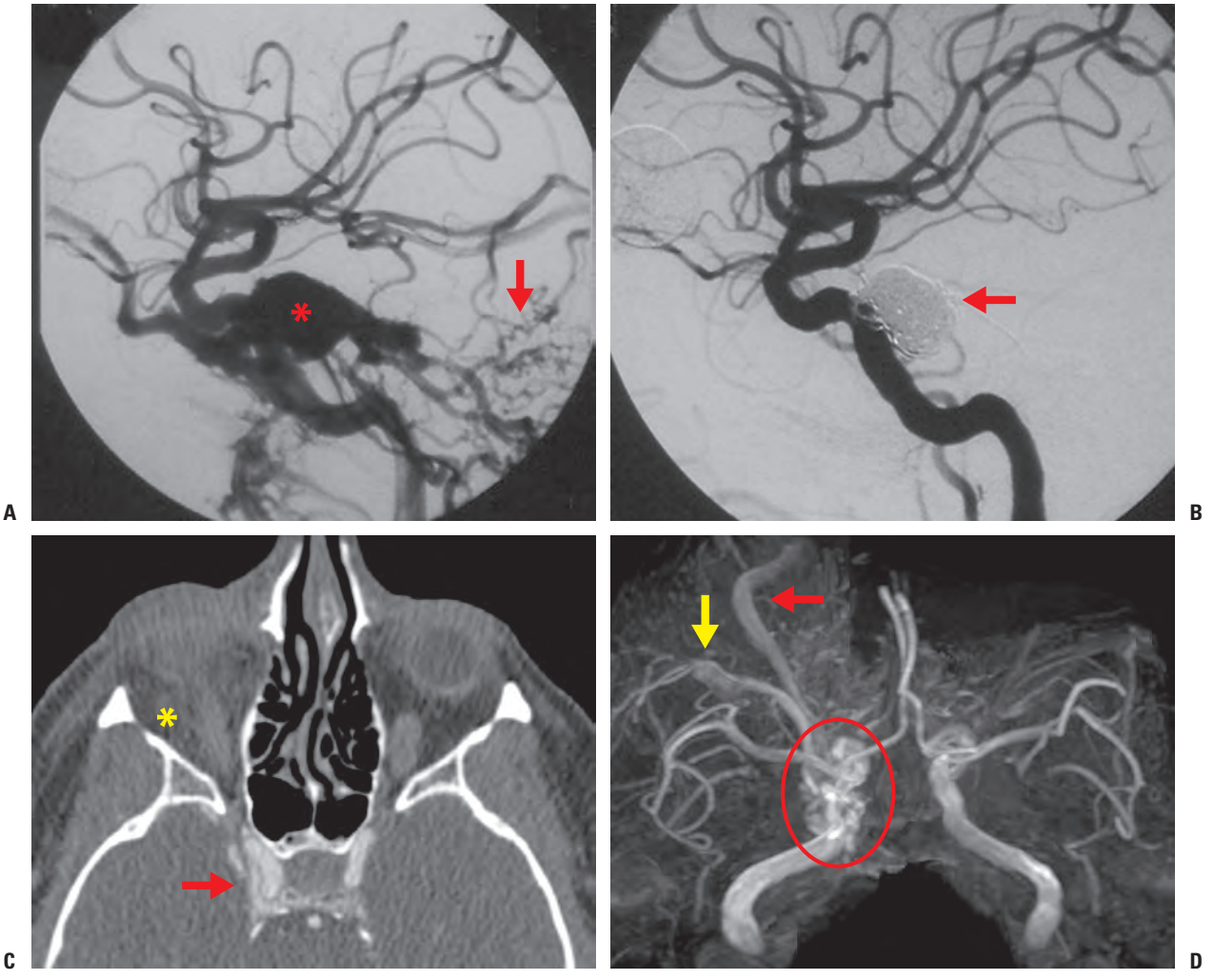
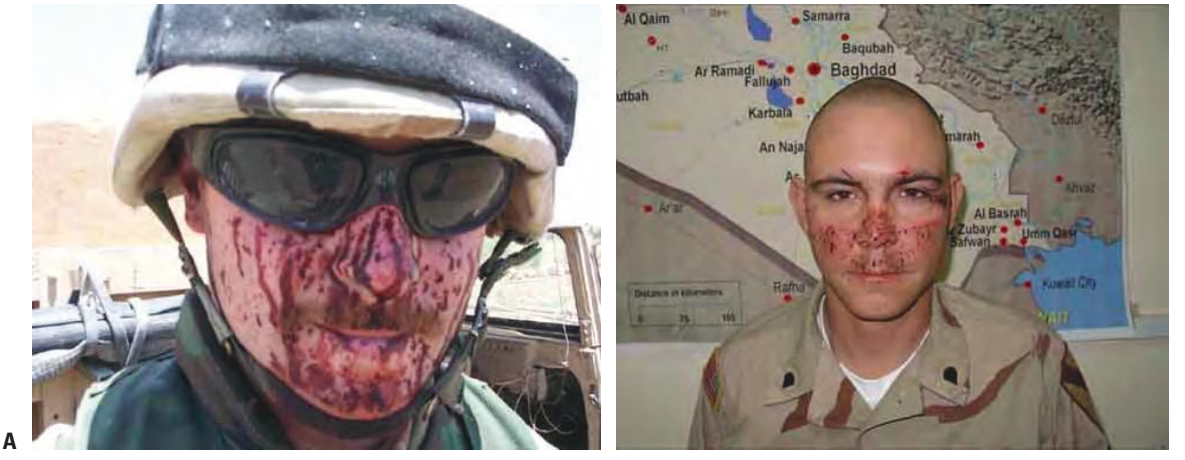


Figure 5.63. Carotid-Cavernous Fistula. This 25-year-old platoon leader, whose vehicle was struck by a rocket-propelled grenade, presented with a right sixth cranial nerve palsy several weeks after the attack. **A.** Lateral view from an ICA catheter angiogram obtained in the early arterial phase demonstrates a large pseudoaneurysm at the proximal portion of the cavernous ICA segment (*asterisk*). There is abnormal cortical venous reflux into posterior fossa veins (*red arrow*) via the superior petrosal vein and engorgement of the ophthalmic veins (*yellow arrow*), consistent with venous hypertension. **B.** Postembolization injection shows obliteration of the carotid–cavernous fistula (CCF) following placement of multiple embolic coils (*arrow*). The cranial nerve palsy completely resolved and the officer returned to full duty at 6 months without limitations. (Courtesy of Rocco A. Armonda, MD, Col [ret] MC, USA.) **C.** Axial CT angiogram image in a different patient demonstrates asymmetric contrast enhancement of the right cavernous sinus, mild right proptosis, and stranding of the retrobulbar fat, all characteristic imaging manifestations of a CCF. **D.** Collapsed view from a 3D time-of-flight (TOF) MR angiogram demonstrates the typical imaging findings of a traumatic high-flow, direct CCF: enlargement and abnormal flow-related enhancement of the right superior ophthalmic vein (*red arrow*) and sphenoparietal sinus (*yellow arrow*) and asymmetric flow-related enhancement of the cavernous sinus (*circle*).

★**KEY POINT** Urgent treatment for a traumatic CCF is indicated for patients with cortical venous drainage, decreased visual acuity, rapidly progressive proptosis, and intracranial hemorrhage.

Importance of Protective Eyewear in Combat

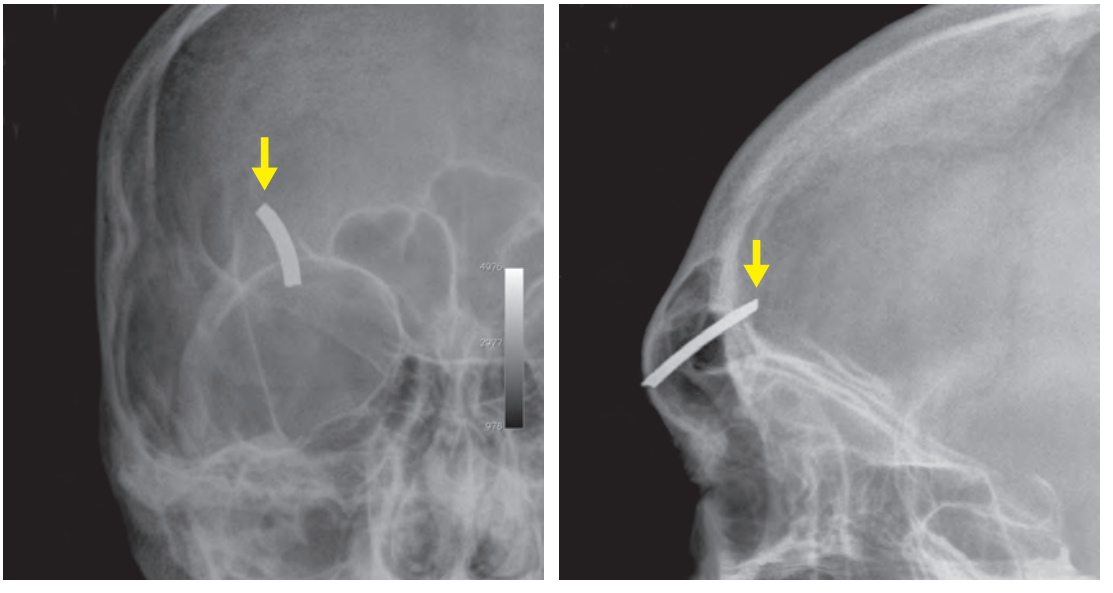


A B

Figure 5.64. **A.** Photograph of a soldier immediately following an IED explosion. **B.** Photograph of the soldier after wound cleaning illustrates the typical peppering of the face by innumerable blast foreign bodies with relative sparing of the orbits because of protective goggles.

★**KEY POINT** The enforced use of safety devices in combat, such as eye protection, has reduced the incidence of war-related eye injuries.

Importance of Protective Eyewear in the Garden



A B

Figure 5.65. This 26-year-old gardener presented to the ED claiming "something flew into my face while weed whacking." **A.** AP and (**B**) lateral radiographs show a curvilinear foreign body superimposed over the right orbit (*arrow*). Intracranial penetration cannot be excluded. (*Continued*)

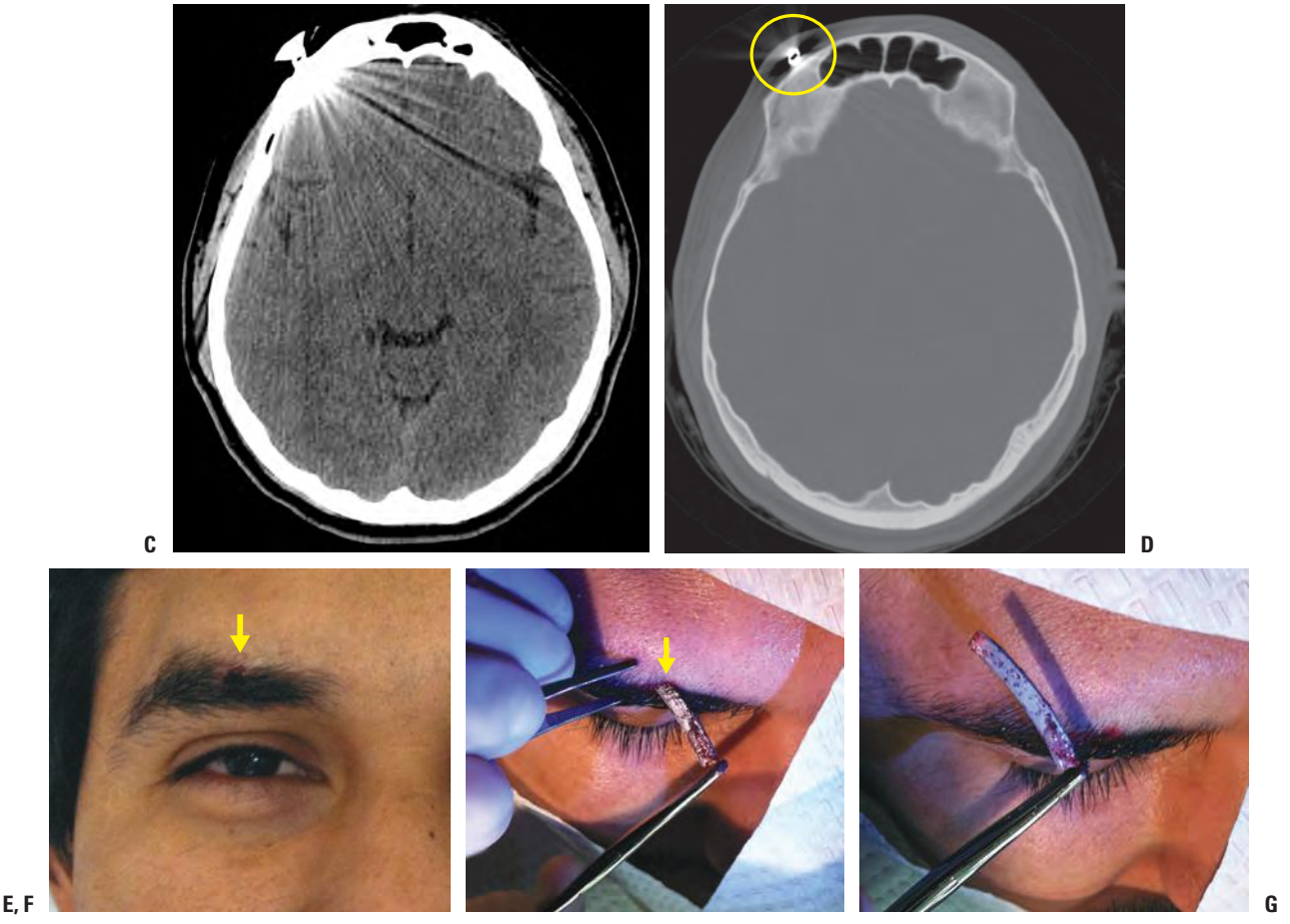


Figure 5.65. (*Continued*) **C.** Axial CT image viewed at brain window technique is limited by metallic streak artifact, which obscures the right frontal lobe; intracranial hemorrhage cannot be excluded. **D.** Bone window technique confirms the extracranial location of the foreign body (*circle*). **E.** Photograph of the patient shows a tiny entry wound (*arrow*). **F.** Removing the foreign body (*arrow*). **G.** Close-up photograph of the foreign body following surgical removal.

★**KEY POINT** Clinical manifestations of a potentially catastrophic penetrating injury can be extremely subtle (see **Figs. 5.72** and **5.73**).

LESSON 10: STROKE AND CEREBROVASCULAR INJURIES ARE MORE COMMON

One of the most striking differences between combat and civilian trauma is the higher incidence of neurovascular injuries in combat trauma.[134,167,280,281] Nearly 30% of troops presenting with severe head trauma suffer neurovascular injury. In addition, the incidence is greater in this war as compared to prior conflicts. The reasons for the increase in observed neurovascular trauma includes improved far-forward care (immediate resuscitation and early cranial decompression) resulting in improved survival rates in conjunction with improved diagnostic imaging with improved sensitivity for detection of vascular injuries. This is the first war in which a dedicated neurointerventionalist was on site at the combat support hospital.

There are a number of causes for ischemic infarction in our otherwise young and healthy soldiers (**Table 5.7**). First and foremost, the increased incidence of penetrating and blast injuries can *directly* injure the vessels of the head and neck and result in *vasospasm* (**Figs. 5.20, 5.66,** and **5.67**), vessel *laceration* (**Fig. 5.68**), *fistulization* (**Fig. 5.63**), *dissection* (**Figs. 5.69** to **5.71**), and *pseudoaneurysm* formation (**Figs. 5.24** and **5.72** to **5.74**). In one cohort of blast-injured patients who underwent cerebral angiography, 35% of patients

TABLE 5.7 Causes of Stroke in Combat

- Vascular dissection/laceration/fistulization
- Pseudoaneurysm
- Blast-induced cerebral vasospasm
- Drowning
- Hypoxemia
- Hypotension
- Cardiovascular derangement (blast-induced apnea, bradycardia, hypotension)
- Blast-induced dysautoregulation (coupled with hypoxic–ischemic injury)
- Post-herniation infarction
- Hyperthermia (heat stroke)
- Air embolism
- Deep venous thrombosis (DVT) in a patient with a patent foramen ovale
- Fat embolism
- Cortical vein/dural sinus thrombosis
- Meningitis

had a pseudoaneurysm and 47% had cerebral vasospasm.[169] The more severe the injury and the presence of SAH increased the likelihood of these abnormalities. In another more recent study using transcranial Doppler (TCD) ultrasonography, 65% of veterans with penetrating TBI had evidence of post-traumatic vasospasm, and 14% of veterans with closed TBI had signs of vasospasm.[282]

Compared to blunt force trauma, explosive trauma causes vasospasm more often and even *without* concomitant SAH.[283] The onset of blast-induced vasospasm is earlier than in civilian TBI, and it is often even seen acutely. Because of this high percentage of vascular injury, an aggressive screening process consisting of early CT and cerebral angiography performed on arrival to a stateside hospital is recommended. From a diagnostic perspective, vasospasm can sometimes mask the angiographic detection of pseudoaneurysms. It is unclear why cerebral vasospasm is so common in combat. As mentioned earlier, one cellular response to BINT is the disruption of integrins, which causes blood vessels to constrict when abruptly stretched. Vascular perturbations such as reduced expression of endogenous nitric oxide or other vasodilators and increased factors such as endothelin-1 contribute to vasoconstriction.[284] Vasospasm leads to reduced cerebral blood flow and microthrombosis, which result in further ischemic injury. In addition, the effect of vasospasm is exacerbated by systemic hypoxia and hypotension, both of which commonly occur in TBI.

The true incidence of traumatic pseudoaneurysms is unknown. The reported incidence varies widely, ranging from 3.2% in civilian penetrating TBI[285] to 42% of patients with GSWs and missile head injuries.[286] Traumatic pseudoaneurysms should be suspected in TBI patients with delayed intracranial hemorrhage, unexplained major arterial bleeding during hematoma evacuation, significant traumatic SAH, orbitofacial and pterional injuries, and the presence of penetrating fragments, especially if they cross midline or transverse into another dural compartment.[287] Patients present with a variety of signs and symptoms, which depend in part on the aneurysm location and the extent of other traumatic injury. Delayed intracranial hemorrhage (typically 2 to 3 weeks) is common and is associated with a mortality rate as high as 50%.[288] Although traumatic pseudoaneurysms have traditionally been treated by open surgery or managed conservatively, Cohen and colleagues[289] concluded that endovascular therapy offers a valuable alternative to surgery, allowing early aneurysm exclusion with excellent results. They emphasized that surgical manipulation can be very difficult in patients with edematous brain resulting from trauma and that surgical clipping of a traumatic aneurysm carries a high incidence of aneurysm rupture owing to traumatic dissection of the arterial wall. Surgical identification of peripheral aneurysms can also be challenging. In addition, pseudoaneurysm situated on arteries supplying eloquent brain areas usually requires surgical bypass before surgical or endovascular sacrifice of the parent artery. Stents may play an increasingly important role in the management of this condition. Rapid advances in stent technology may eventually make it feasible to reconstruct the vessel intraluminally and avoid vessel sacrifice.

There is consensus in the literature that a traumatic pseudoaneurysm is associated with a poor prognosis and should be diagnosed as soon as possible. Cohen and colleagues[289] adopted a policy of performing early screening angiography on TBI patients with cranial base fractures in the area of the carotid canal and penetrating injuries to the pterional area, MCA candelabra, and crossing midline structures. Although technically advanced CTA can be an excellent noninvasive modality for

detecting cerebral vasospasm and pseudoaneurysms, a negative result should not prevent further investigation, especially when evaluating smaller peripheral arterial vessels and when evaluating arterial segments adjacent to bone or metal fragments.[290,291] Therefore, invasive catheter angiography is still required when a traumatic pseudoaneurysm is suspected. Additionally, early angiography can miss delayed aneurysm formation, and repeat angiography may be indicated in selected cases.[292,293] MRI is contraindicated in most blast injuries because of the presence of ferromagnetic fragments. It can be helpful in other penetrating injuries, such as those due to a wooden object or in stab wounds, when the penetrating object was withdrawn at the time of injury.[294,295]

Blast-Induced Cerebral Vasospasm

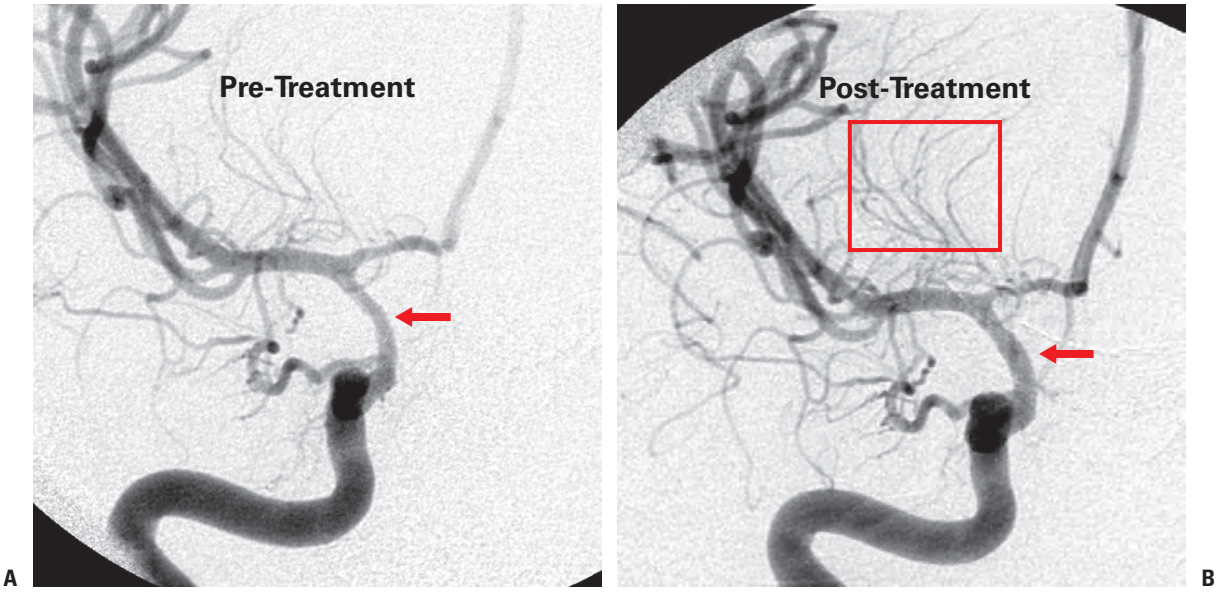


Figure 5.66. Vasopasm (IED Blast Injury). AP views from a right internal cerebral artery catheter angiogram pre- (**A**) and post-angioplasty (**B**) + intra-arterial nicardipine injection show interval increase in ICA vessel caliber (*arrows*) and improved visualization of the lenticulostriate vasculature (*box*). (Courtesy of Rocco Armonda, MD, Col [ret], MC, USA)

★**KEY POINT** Post-traumatic vasospasm most commonly occurs in the intradural segment of the distal ICA. Compared to blunt force trauma, explosive trauma causes vasospasm more often and even *without* concomitant SAH. In addition, the onset of blast-induced vasospasm occurs earlier than in civilian TBI. Vasospasm leads to reduced cerebral blood flow and microthrombosis which result in further ischemic injury. Finally, the effect of vasospasm is exacerbated by systemic hypoxia and hypotension, both of which commonly occur in TBI.

Multivessel Injury (IED Blast Trauma)

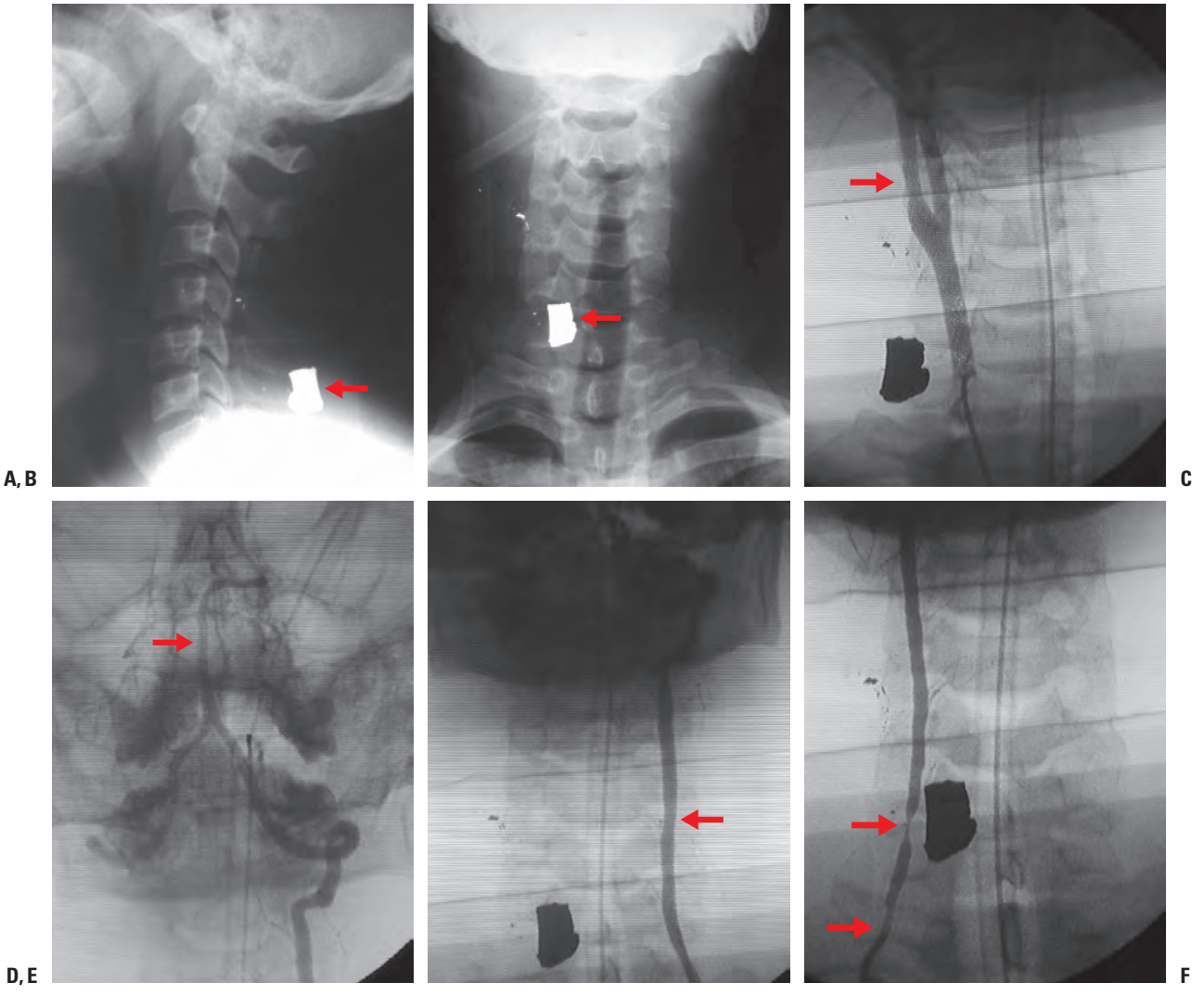
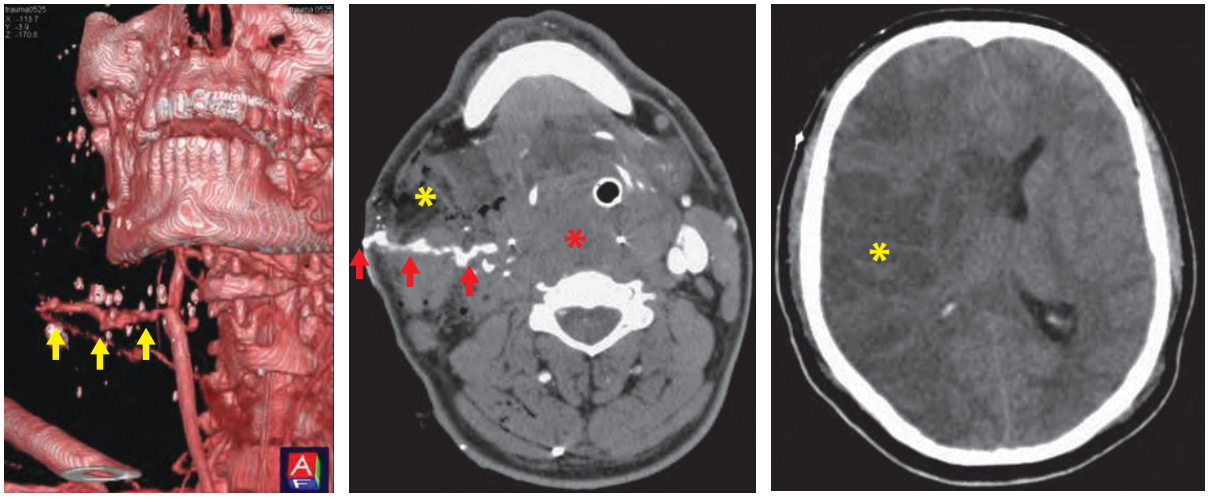


Figure 5.67. **Vasospasm (IED Blast). A.** Lateral and **(B)** AP plain radiographs of the cervical spine demonstrate a large radiopaque foreign body in the posteromedial soft tissues of the neck (*arrow*). **C.** AP view from a right common carotid artery catheter angiogram reveals mild caliber irregularity of the proximal ICA (*arrow*). **D.** Left vertebral injection shows normal filling of the distal vertebral artery and basilar artery (*arrow*). **E.** AP view of the proximal vertebral artery shows minimal caliber irregularity. **F.** AP view following injection of the right vertebral artery shows severe segmental narrowing of the proximal vertebral artery, ipsilateral to the projectile (*arrows*).

★**KEY POINT** Vascular irregularity may be due to vasospasm, compression by a surrounding hematoma, or a vascular dissection.

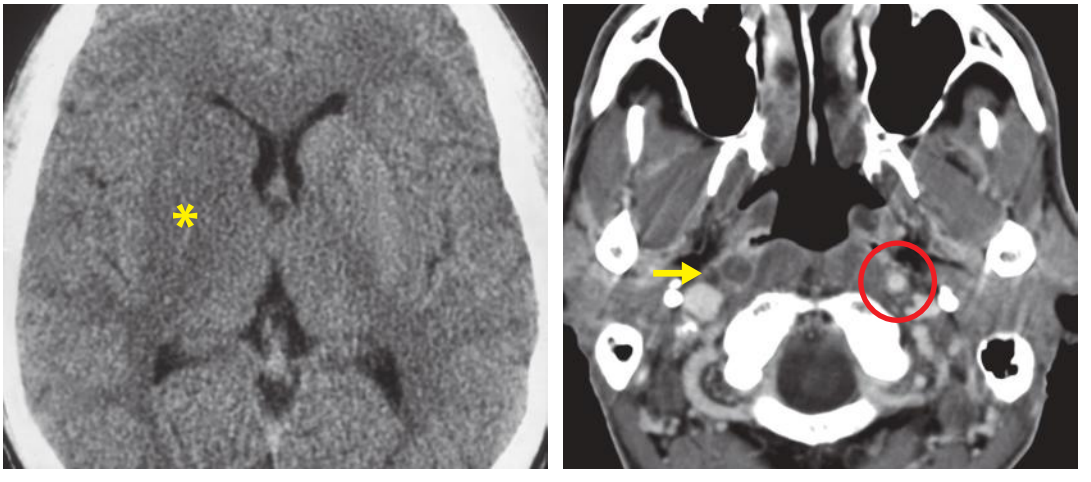
Vascular Laceration and Middle Cerebral Artery Infarction (IED Explosion)



A, B C

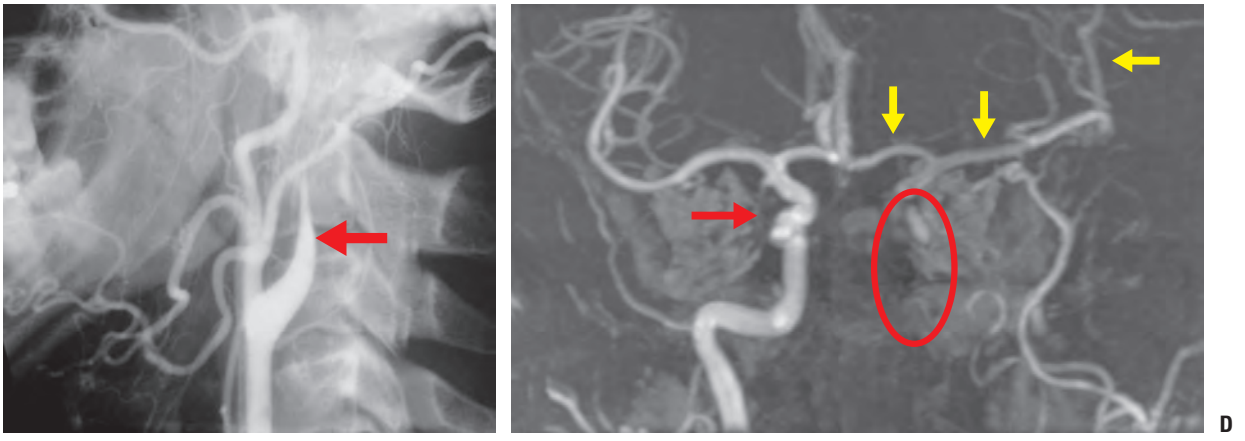
Figure 5.68. Cerebral Infarction due to Traumatic Vascular Laceration. A. Volume-rendered CTA shows active extravasation at the level of the carotid bifurcation (*arrows*). Numerous small foreign bodies are noted within the soft tissue of the face and neck. **B.** Axial CTA image demonstrates complete occlusion of the airway (*red asterisk*), soft tissue hemorrhage and air within the right sternocleidomastoid muscle and submandibular region (*yellow asterisk*), and a linear tract of active contrast extravasation extending to the skin surface (*arrows*). **C.** Noncontrast CT demonstrates a large nonhemorrhagic right MCA ischemic infarction (*asterisk*).

Ischemic Infarction and Traumatic Vascular Dissection



A B

Figure 5.69. Vascular Dissection. A. Noncontrast axial CT demonstrates subtle low density within the right caudate head and lentiform nucleus (*asterisk*), consistent with an acute ischemic infarct involving the lenticulostriate vessels. **B.** Delayed contrast-enhanced CT shows a normal left ICA (*circle*) and an enlarged, nonenhancing right ICA (*arrow*). The increase in size of the right ICA is due to the combination of the nonenhancing true lumen plus the surrounding nonenhancing dissecting subintimal hematoma. Subtle peripheral enhancement of the vessel wall represents the adventitia. (*Continued*)

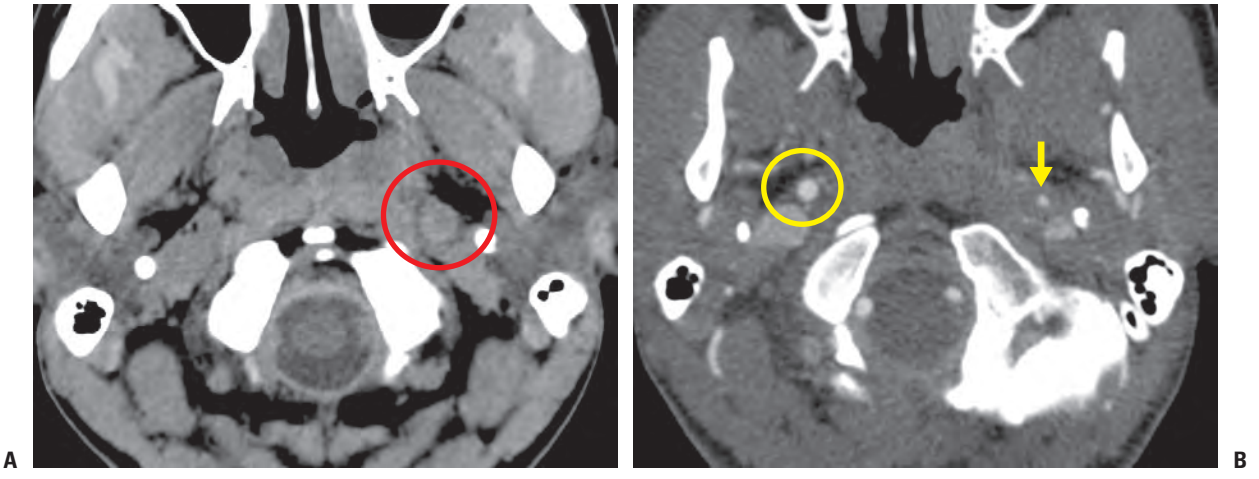


C D

Figure 5.69. (*Continued*) **C.** Lateral unsubtracted view from a right common carotid artery catheter angiogram shows the typical "rat-tail" occlusion of an acute vascular dissection (*arrow*). **D.** AP view from the intracranial TOF maximum intensity projection magnetic resonance angiography (MRA) demonstrates absence of flow-related enhancement of the right ICA (*circle*). Note the patent but asymmetrically decreased flow-related enhancement of the right hemispheric vasculature (*yellow arrows*). A prominent right external carotid artery (ECA) branch is also evident (*red arrow*).

★**KEY POINT** The *delayed* contrast-enhanced CT is helpful in differentiating a completely occluded artery from slow filling of a markedly narrowed arterial lumen. Thus, if the CTA source images suggest complete occlusion, a *delayed* CT exam through the vessel should be obtained.

Vascular Dissection (Ischemia without Infarction)



A B

Figure 5.70. **Vascular Dissection (Ischemia without Infarction). A.** Noncontrast CT image obtained prior to the dedicated CTA shows an increase in size of the left ICA (*circle*). **B.** Axial CTA source image shows a decrease in caliber of the residual lumen of the left ICA (*arrow*). The surrounding dissecting hematoma is identified as the circumferential gray area surrounding the narrowed enhancing portion; compare with the normal right ICE (*circle*). (*Continued*)

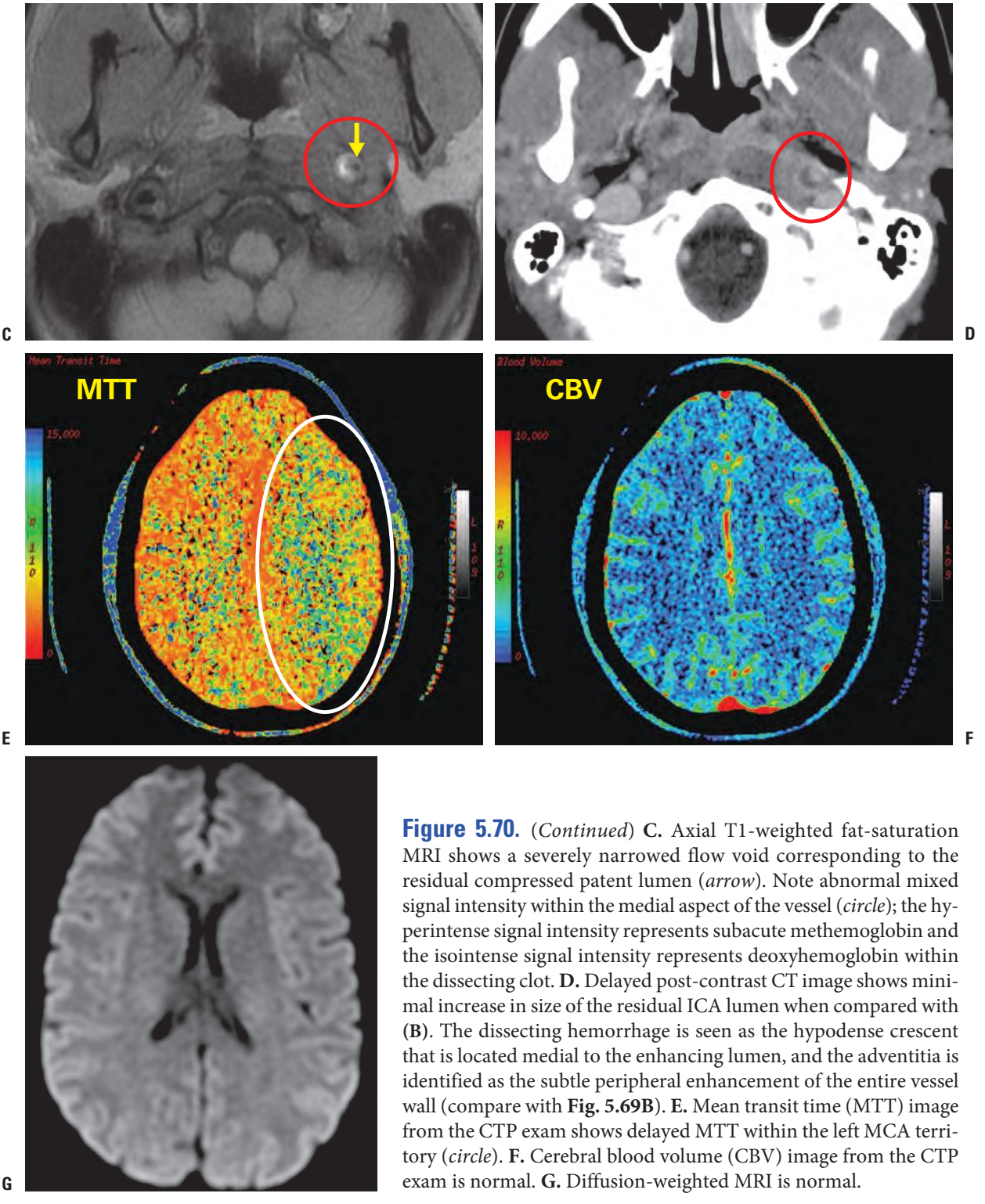


Figure 5.70. (*Continued*) **C.** Axial T1-weighted fat-saturation MRI shows a severely narrowed flow void corresponding to the residual compressed patent lumen (*arrow*). Note abnormal mixed signal intensity within the medial aspect of the vessel (*circle*); the hyperintense signal intensity represents subacute methemoglobin and the isointense signal intensity represents deoxyhemoglobin within the dissecting clot. **D.** Delayed post-contrast CT image shows minimal increase in size of the residual ICA lumen when compared with **(B)**. The dissecting hemorrhage is seen as the hypodense crescent that is located medial to the enhancing lumen, and the adventitia is identified as the subtle peripheral enhancement of the entire vessel wall (compare with **Fig. 5.69B**). **E.** Mean transit time (MTT) image from the CTP exam shows delayed MTT within the left MCA territory (*circle*). **F.** Cerebral blood volume (CBV) image from the CTP exam is normal. **G.** Diffusion-weighted MRI is normal.

★**KEY POINT** The above findings are consistent with left MCA *tissue at risk* caused by decreased flow within the left ICA from the compressed true lumen in the neck. More commonly, however, vascular dissections cause ischemic injury via distal embolization of thrombus that forms at the site of intimal injury.

Vascular Dissection (Preserved Luminal Caliber)

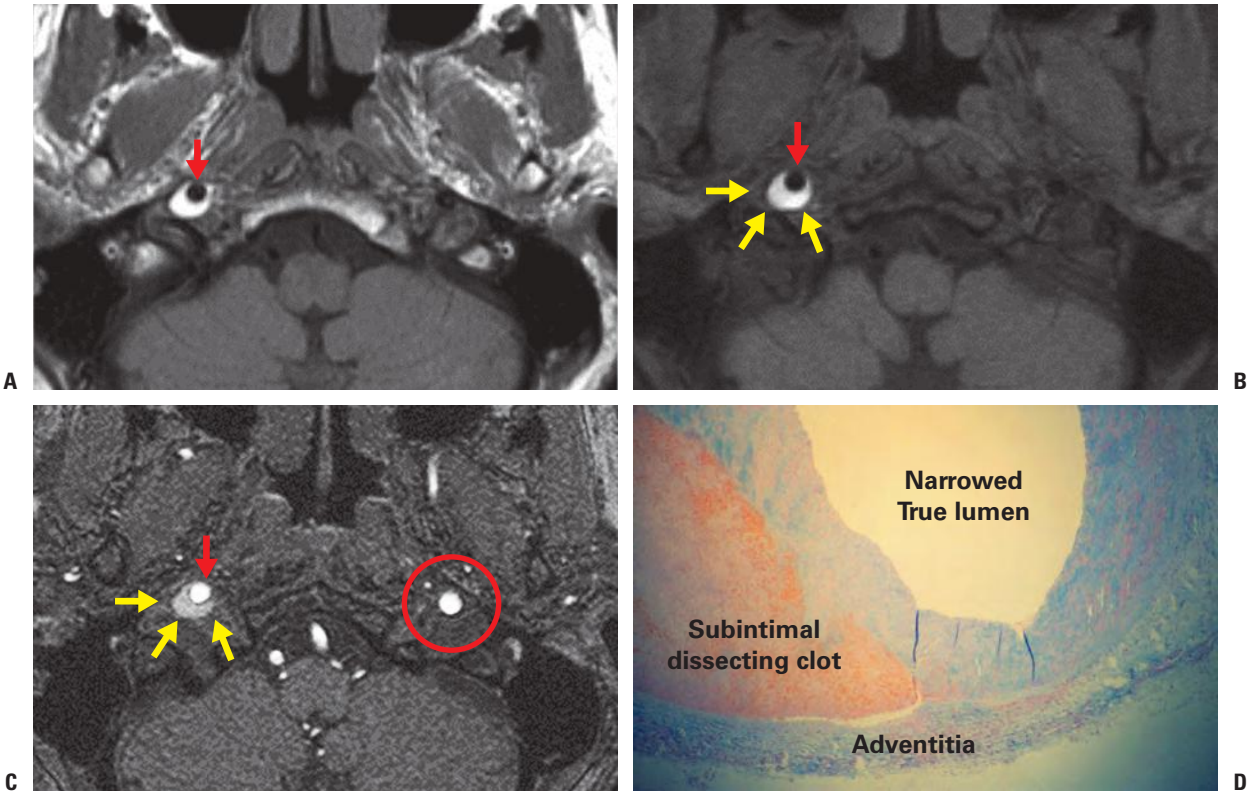


Figure 5.71. **Traumatic Vascular Dissection (Blunt Civilian Trauma). A.** Axial T1-weighted MRI shows the characteristic crescent sign of a subacute vascular dissection (*circle*). The true lumen of the ICA is identified as the well-defined, round signal void that is surrounded by an abnormal crescent of T1 hyperintensity, the latter of which represents methemoglobin within the dissecting hemorrhage. The adventitia is seen as the thin black line surrounding the vessel—compare with the pathology specimen in **(D)**. **B.** Fat-saturated, T1-weighted MRI increases the conspicuity of the hyperintense crescent sign (*yellow arrows*) eccentric to the ICA flow void (*red arrow*). **C.** Axial source image from the MRA shows an increase in diameter of the ICA, but the true lumen is only a small portion of this (*red arrow*). The crescent sign is less conspicuous on this sequence (*yellow arrows*). Compare with the normal caliber of the left ICA (*circle*). **D.** Pathology specimen in a different patient demonstrates the characteristic dissecting subintimal clot causing narrowing of the true lumen (Pathology courtesy of Howard Rowley, MD.)

★**KEY POINT** This case illustrates how a vascular dissection can be missed on catheter angiography if the size of the true lumen is preserved. This occurs more commonly when the dissection is subadventitial. Subintimal dissections cause abnormalities of the true lumen and are usually evident on conventional angiography. MRI provides direct visualization of the dissecting hematoma and the signal void of the true lumen, and, therefore, it has an added advantage over conventional angiography, which is capable of visualizing only the patent lumen.

Pseudoaneurysm and Delayed Migration of a Foreign Body

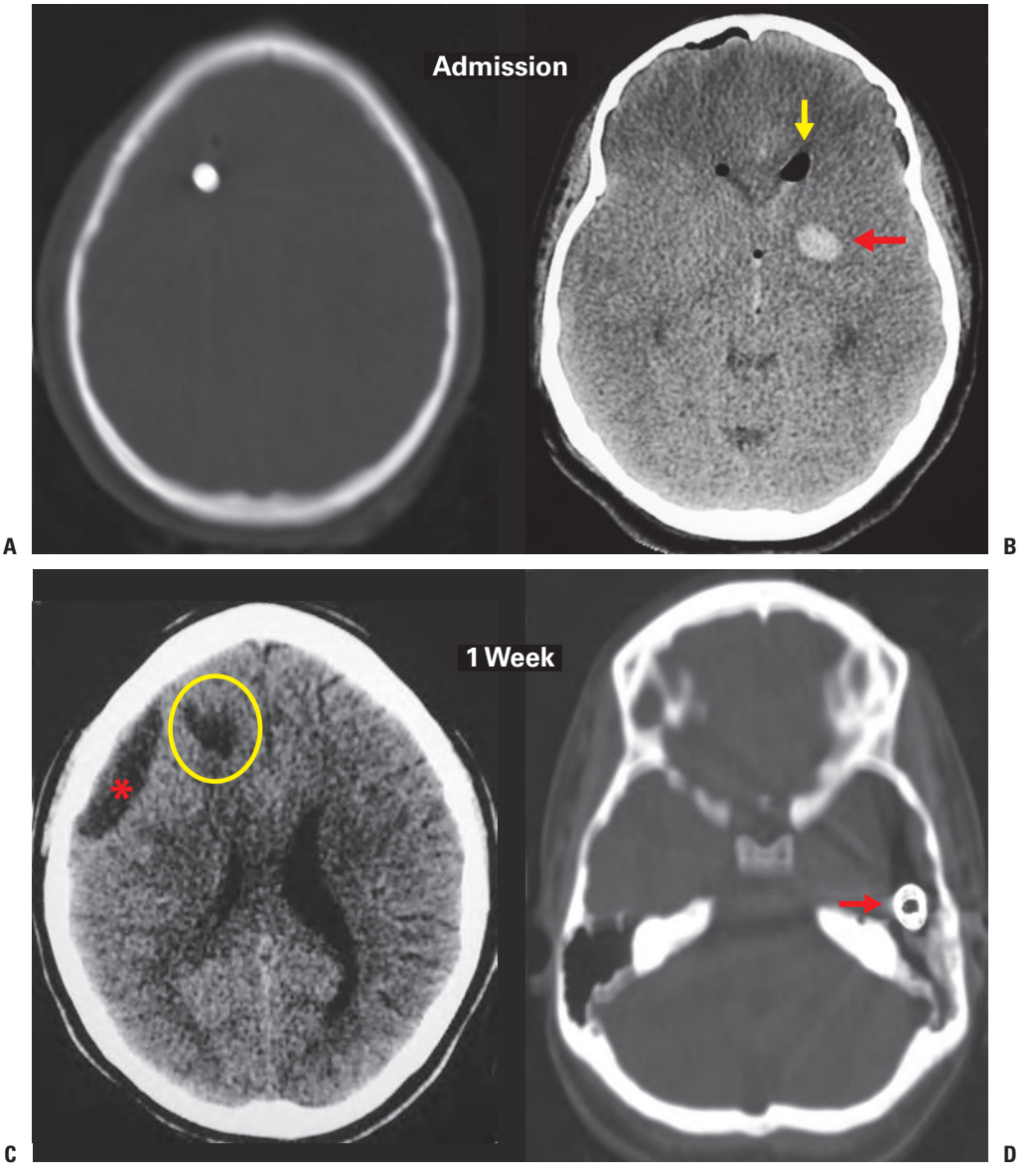


Figure 5.72. **Missile Migration.** This 16-year-old girl presented with a GCS = 7 following a car bomb explosion. A very small entry site was noted immediately anterior to the left tragus **(J)**. **A.** Admission CT image viewed at bone windowing demonstrates a large round metallic foreign body within the right frontal lobe. *There were no additional foreign bodies.* **B.** Admission CT image viewed at brain windowing shows a left putaminal hemorrhage (*red arrow*), which was located along the direct trajectory of the projectile (i.e., it entered the left temporal bone, crossed the midline anterosuperiorly, and ultimately terminated in the right frontal lobe). There is loss of the normal gray–white matter differentiation, which is primarily due to technical artifact, but we cannot exclude superimposed cerebral swelling. **C.** One-week follow-up CT shows a small right frontal extra-axial collection (*asterisk*) and a focal area of low attenuation in the region of the previously noted projectile (*circle*). **D.** Image at a lower level from the same study viewed at bone windowing shows the new location of the foreign body adjacent to the left temporal bone, that is, it had "fallen down" through the original wound canal to end up adjacent to the entry site. (*Continued*)

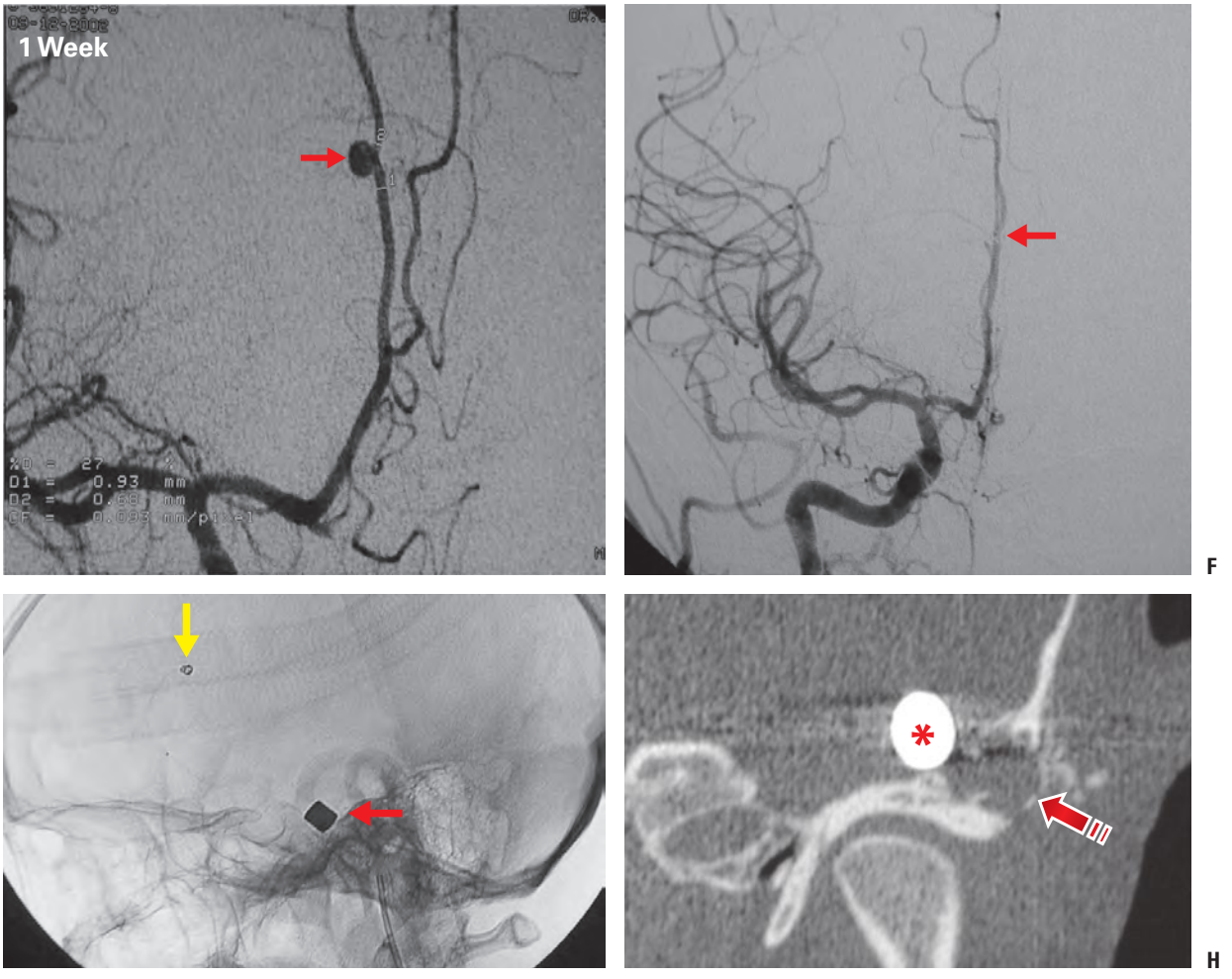


Figure 5.72. (*Continued*) **E.** Oblique frontal view from the cerebral angiogram at 1 week after injury shows a traumatic pseudoaneurysm of the right pericallosal artery (*arrow*). **F.** Postembolization angiogram in the AP view shows occlusion of the pseudoaneurysm (*arrow*). **G.** Lateral unsubtracted view from the procedural angiogram for embolization demonstrates a microcatheter and endovascular coils within the right pericallosal artery pseudoaneurysm (*yellow arrow*). Note also the left temporal projectile (*red arrow*) **H.** Coned-down high-resolution coronal CT image through the left temporal bone shows a comminuted fracture of the mastoid at the entry site with the adjacent foreign body (*asterisk*). (*Continued*)

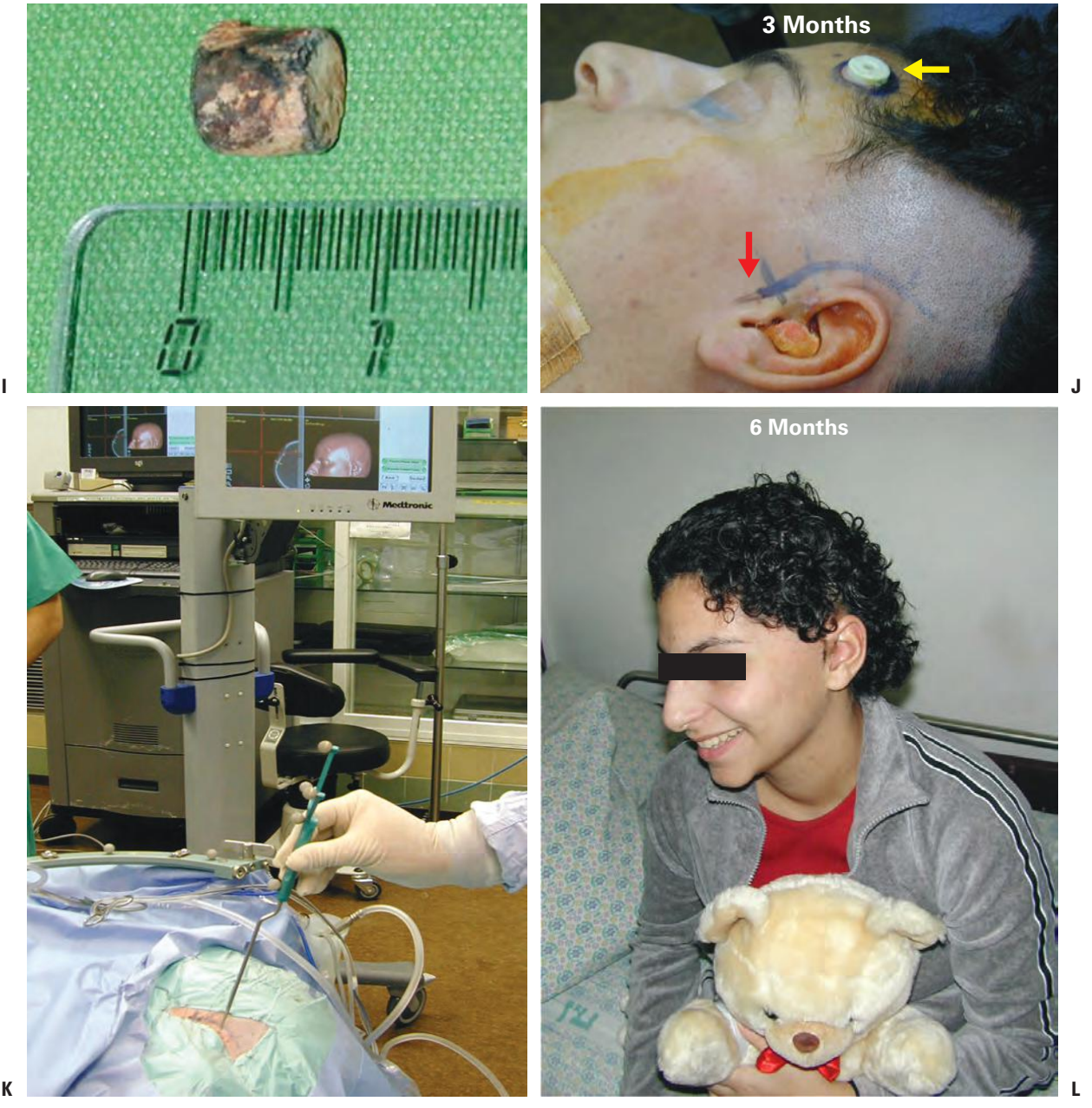


Figure 5.72. (*Continued*) **I.** Photograph of the surgically removed foreign body. **J, K.** Three-month follow-up photographs during intraoperative image guidance for surgical removal of the left temporal foreign body show a tiny residual scar at the entry site (*red arrow*) and the fiducial marker for stereotactic removal (*yellow arrow*). **L.** Six-month follow-up photograph of the patient. (Courtesy of Guy Rosenthal, MD, Hadassah Hospital, Jerusalem, Israel.)

★**KEY POINT** This case illustrates three important teaching points: (1) The liberal use of cerebral angiography in blast injury is often warranted, especially if the projectile crosses the midline, (2) the entry site wound can be amazingly subtle (see also **Figs. 5.65** and **5.73**), and (3) projectiles can migrate over time.

Dissection/Pseudoaneurysm (Hand Grenade Injury)

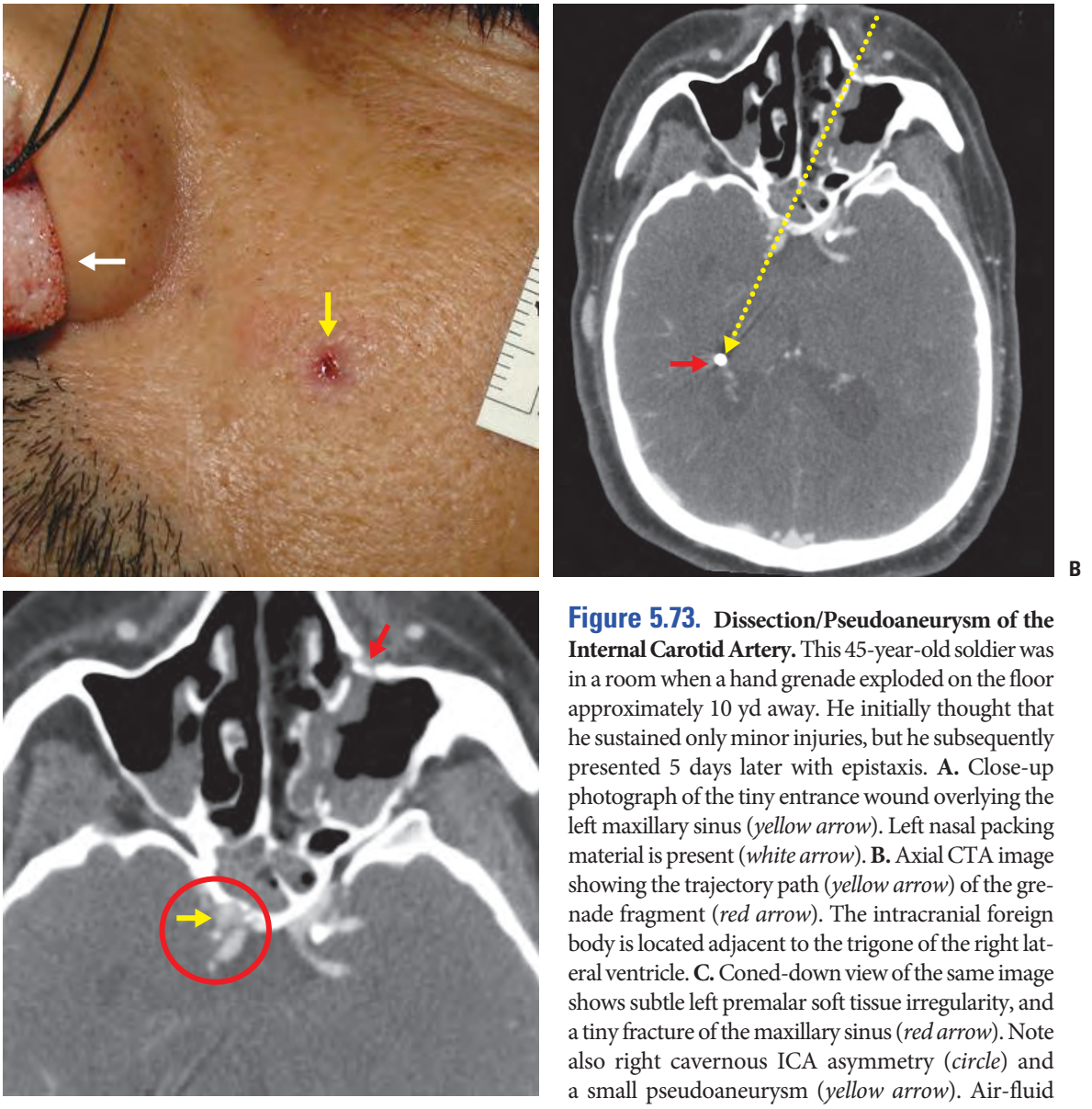
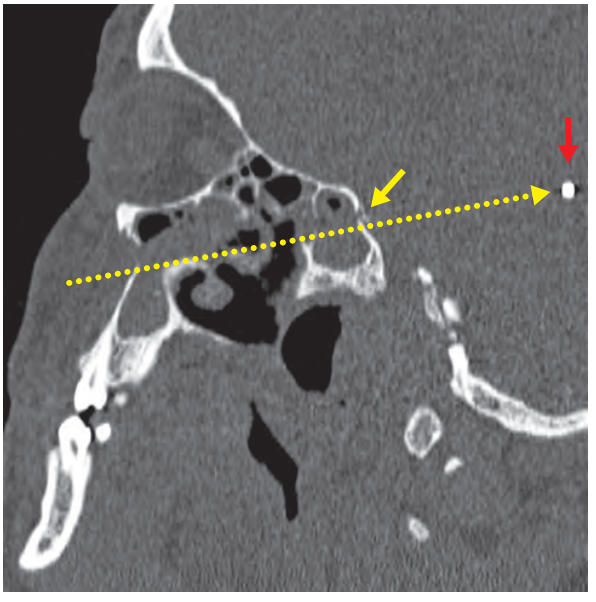
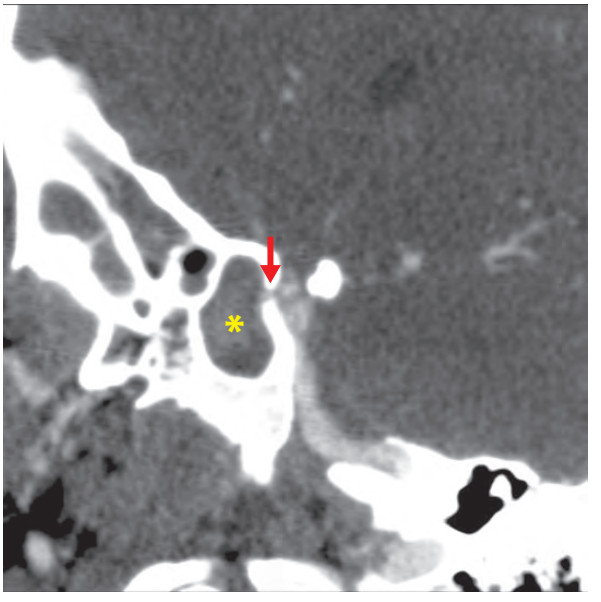


Figure 5.73. Dissection/Pseudoaneurysm of the Internal Carotid Artery. This 45-year-old soldier was in a room when a hand grenade exploded on the floor approximately 10 yd away. He initially thought that he sustained only minor injuries, but he subsequently presented 5 days later with epistaxis. **A.** Close-up photograph of the tiny entrance wound overlying the left maxillary sinus (*yellow arrow*). Left nasal packing material is present (*white arrow*). **B.** Axial CTA image showing the trajectory path (*yellow arrow*) of the grenade fragment (*red arrow*). The intracranial foreign body is located adjacent to the trigone of the right lateral ventricle. **C.** Coned-down view of the same image shows subtle left premalar soft tissue irregularity, and a tiny fracture of the maxillary sinus (*red arrow*). Note also right cavernous ICA asymmetry (*circle*) and a small pseudoaneurysm (*yellow arrow*). Air-fluid levels in both maxillary sinuses and sphenoid sinuses are consistent with blood. (*Continued*)



D



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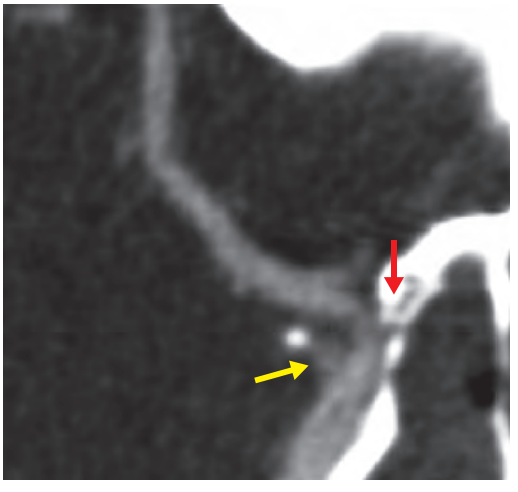

F

Figure 5.73. (*Continued*) **D.** Parasagittal reformatted CT of the face demonstrates the fragment trajectory through the maxillary and sphenoid sinuses (*dotted arrow*). The intracranial metallic fragment is again identified (*red arrow*). Note the small chip fracture of the posterior wall of the sphenoid sinus (*yellow arrow*). **E.** Parasagittal localizer image from the curved planar reformatted CTA shows hemorrhage within the sphenoid sinus (*asterisk*) via the defect in the posterior wall of the sphenoid sinus (*arrow*). **F.** Curved multiplanar reformatted CTA image demonstrates subtle narrowing of the cavernous segment of the ICA in proximity to the posterolateral wall of the sphenoid sinus. A small pseudoaneurysm (*yellow arrow*) and thin intimal flap (*red arrow*) are identified. (Modified with permission from Willson TJ, Folio L. Severe epistaxis from an intracranial vascular bleed from grenade injury. Mil Med. 2008;172:934–935).

★**KEY POINT** This case illustrates three important teaching points: (1) The entry site of a projectile can be remarkably subtle on physical exam, (2) delayed vascular complications of blast injuries are a common problem, and (3) curved multiplanar reformatted CTA can assist in identifying both the trajectory of the projectile and injury to the vessel wall.

Traumatic Pseudoaneurysm

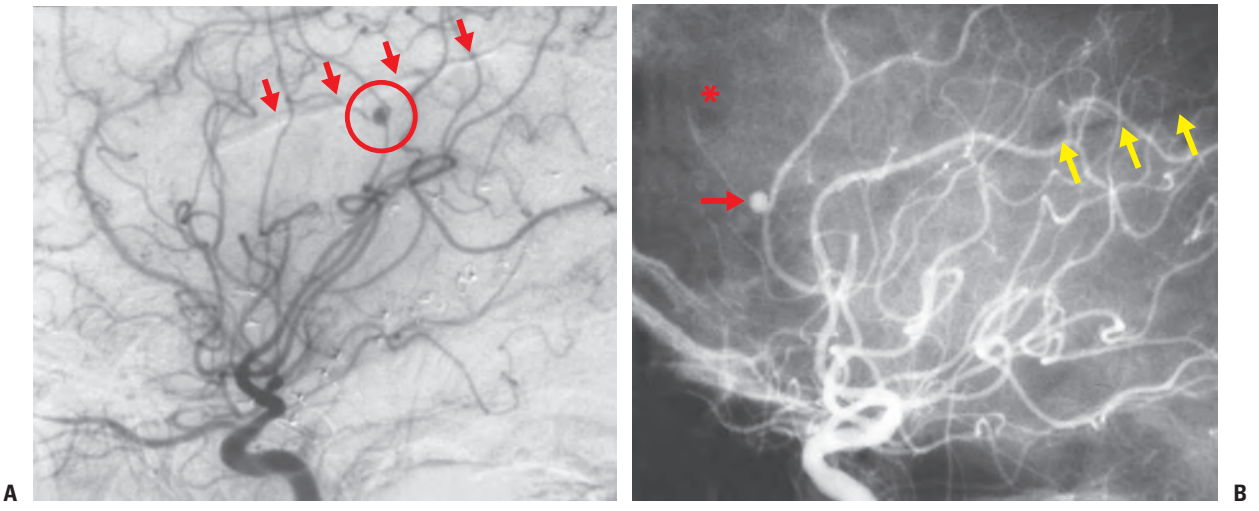

Figure 5.74. Traumatic Pseudoaneurysm. A. Lateral view from an ICA catheter angiogram shows a linear skull fracture (*arrows*) with an underlying pseudoaneurysm of a peripheral branch of the middle cerebral artery (*circle*). **B.** Lateral view from an unsubtracted catheter angiogram in a different patient shows a linear skull fracture (*arrows*) and a pseudoaneurysm of the callosomarginal artery. Note the relative area of hypovascularity in the frontal region (*asterisk*) consistent with an intracranial hematoma seen on CT.

Second, infarction following penetrating blast injuries can be due to *foreign body or air embolization* (**Figs. 3.9B** and **5.75** to **5.77**). Foreign body emboli gain access to the vascular system by direct propulsion or erosion into the vessel lumen. A high clinical suspicion for foreign body embolization is warranted when fragments are adjacent or within vascular structures.[296] Arterial embolization is more often symptomatic than venous embolization, but *paradoxical* emboli (i.e., passage of a foreign body from the venous to the arterial system by communication through a right-to-left shunt) are also well described.[297] This shunt can occur in up to 10% of the population via a patent foramen ovale, ventricular septal defect, atrioventricular septal perforation, or arteriovenous fistula. Of note, intracranial foreign bodies can also embolize into the extracranial circulation. Although not available in prior conflicts, endovascular snare removal of missile emboli is now possible in the war zone.[298] Blast injuries can also disrupt pulmonary alveoli and cause air to enter the pulmonary veins, resulting in air embolism to the brain and spinal cord. Air embolism is also a well-known complication of mechanical ventilation in blast lung injury. Air emboli can affect any organ, but the most serious consequences occur after cerebral and coronary air emboli. These are assumed to be the cause of some immediate deaths on the battlefield.[299] Third, *cardiovascular derangement* from the blast explosion can cause apnea, bradycardia, and hypotension and result in diffuse hypoxic–ischemic injury, large vessel territory infarction, or border-zone (i.e., watershed) infarction (see **Fig. 5.78**). Blast-induced cerebrovascular *dysautoregulation* can potentiate these effects (see **Fig. 5.48**). Fourth, severe brain swelling is associated with blast brain injuries, leading to ICPs so high that the brain vasculature is directly compressed and results in *herniation infarction* (**Fig. 5.15**). Fifth, massive *blood loss* from a traumatic amputation and/or coagulopathy can result in hypotensive ischemic infarction.

Foreign Body Embolus (IED Blast Injury)

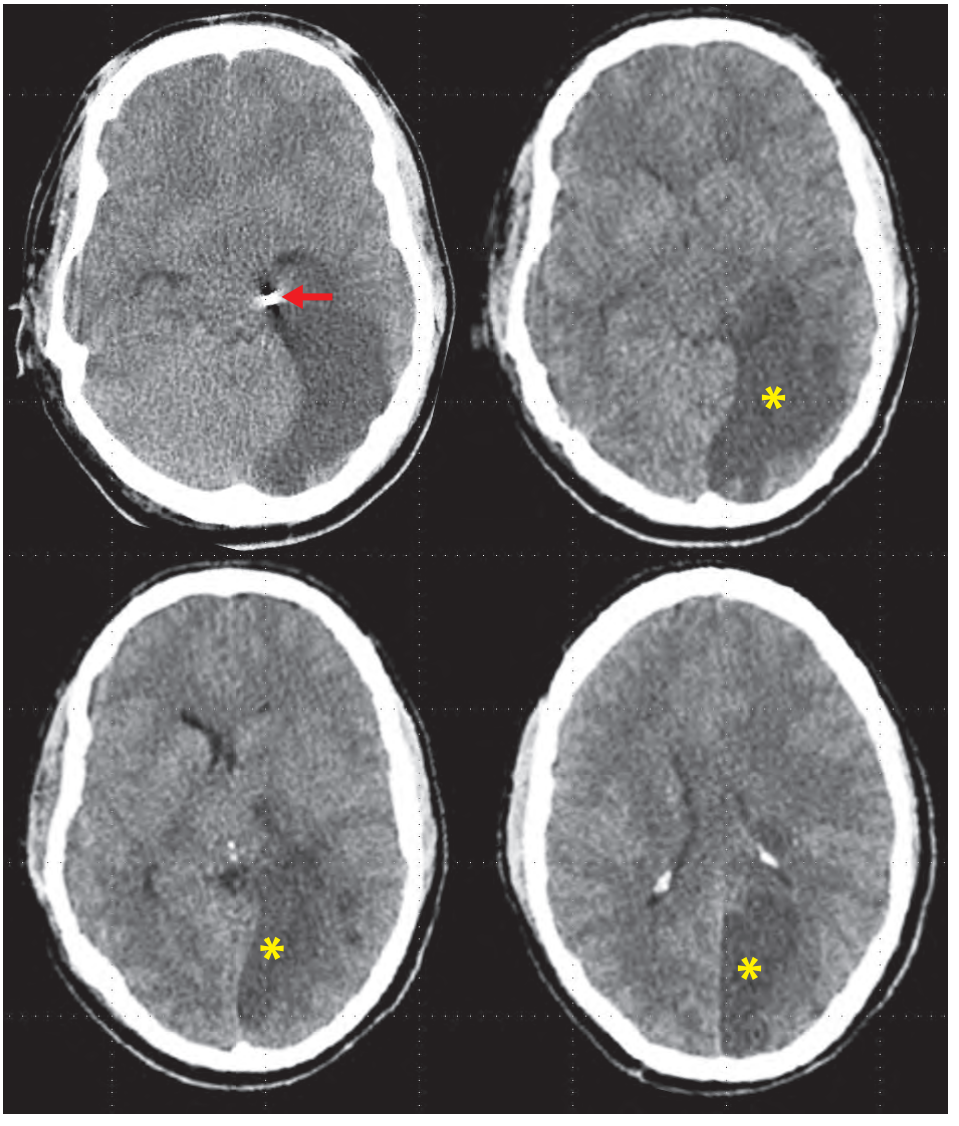

Figure 5.75. Embolic Infarction (IED Blast with Metallic Fragment Embolus). This 19-year-old soldier arrived to the Forward Surgical Team awake and alert with shrapnel to the neck and left upper extremity. On postinjury day 2, he developed aphasia and right hemiparesis. The noncontrast axial CT images demonstrate a metallic foreign body lodged in the region of the proximal left posterior cerebral artery (*arrow*). The metallic embolus resulted in a nonhemorrhagic ischemic infarction of the brain supplied by the posterior cerebral artery (*asterisk*) and the posterior limb of the internal capsule.

Intraluminal Thrombus (IED Explosion)

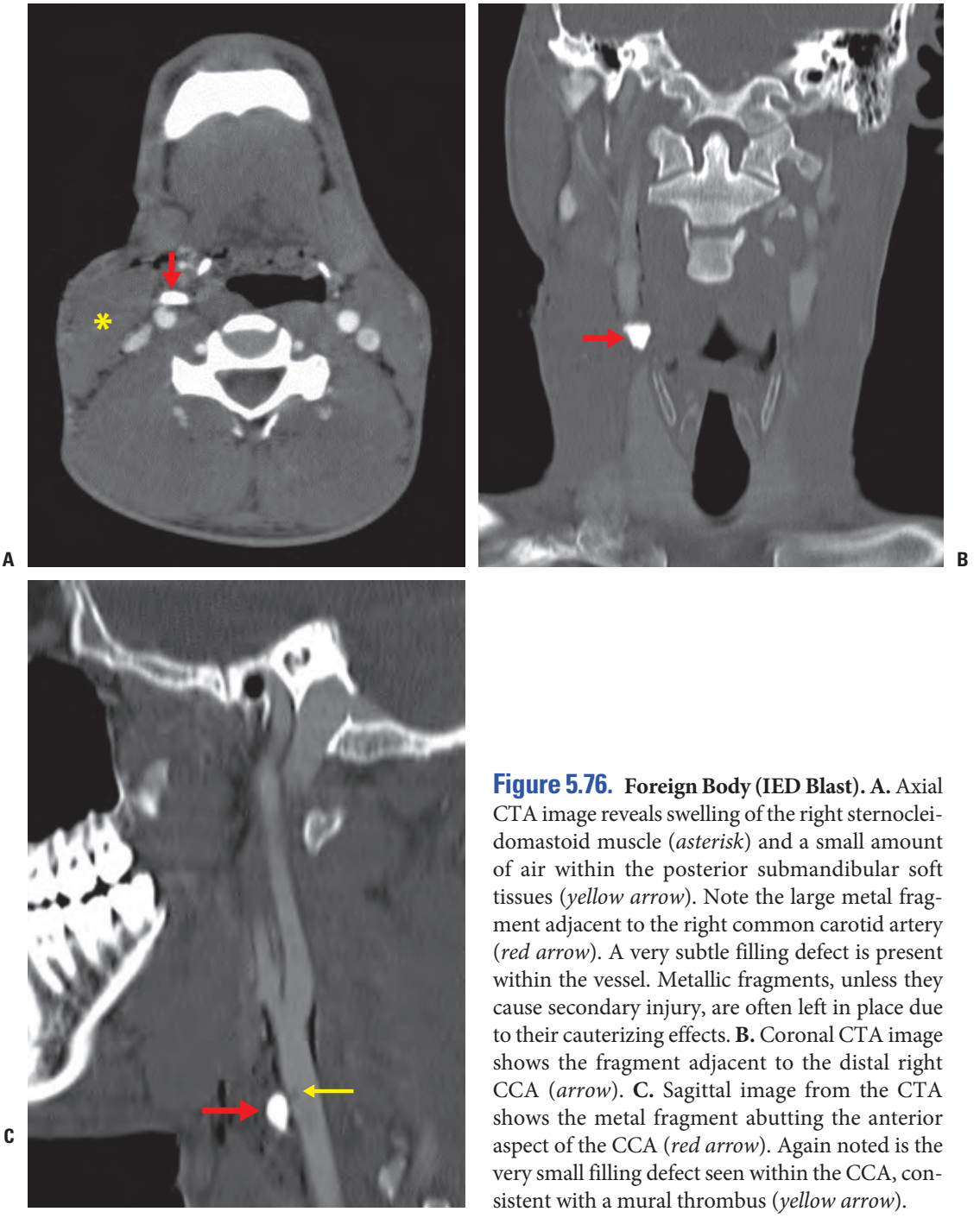

Figure 5.76. Foreign Body (IED Blast). A. Axial CTA image reveals swelling of the right sternocleidomastoid muscle (*asterisk*) and a small amount of air within the posterior submandibular soft tissues (*yellow arrow*). Note the large metal fragment adjacent to the right common carotid artery (*red arrow*). A very subtle filling defect is present within the vessel. Metallic fragments, unless they cause secondary injury, are often left in place due to their cauterizing effects. **B.** Coronal CTA image shows the fragment adjacent to the distal right CCA (*arrow*). **C.** Sagittal image from the CTA shows the metal fragment abutting the anterior aspect of the CCA (*red arrow*). Again noted is the very small filling defect seen within the CCA, consistent with a mural thrombus (*yellow arrow*).

Foreign Body Embolus (IED Blast Injury)

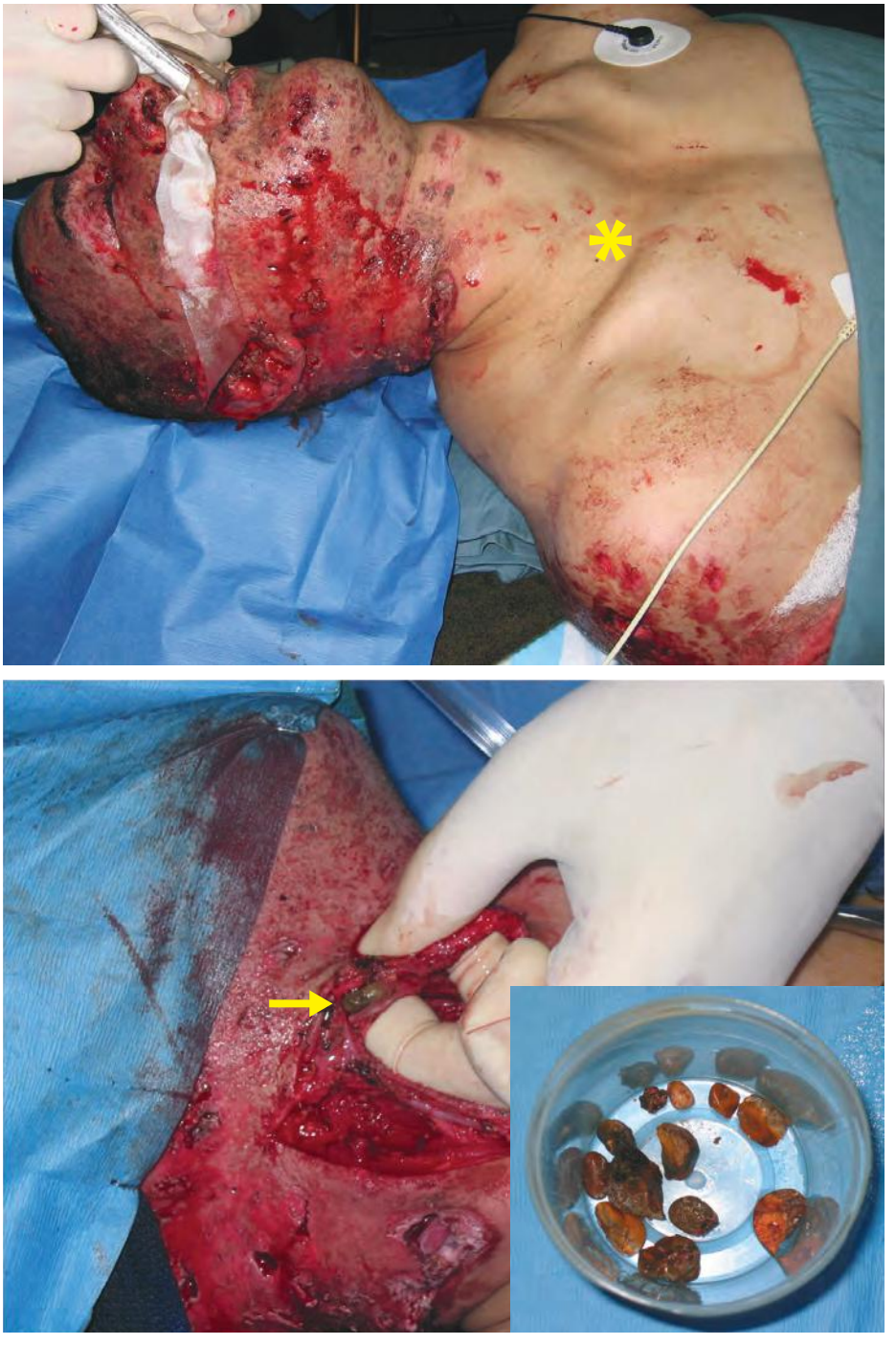

Figure 5.77. Foreign Body Embolus (IED Explosion). These two photographs illustrate three key findings common to IED blast injuries: (1) innumerable small penetrating wounds, many of which are contaminated with small rocks (*arrow* and *inset*); (2) preferential injury to the face (and shoulders) with relative sparing of the chest (*asterisk*); and (3) thermal injury superimposed on the penetrating wounds.

Sixth, difficult extrication and prolonged evacuation from an overturned vehicle in rivers such as the Tigris and Euphrates rivers can result in *drowning* (**Figs. 5.78** and **5.79**). Although the heavy body armor (>50 lb) is invaluable for protecting the soldier from penetrating trauma, it can feel awkward and limit a soldier's mobility. Constrained like a turtle, the soldier may have difficulty moving freely. Seventh, amputations can predispose the soldier to *cerebral fat emboli* (**Fig. 5.80**). Fat embolism can also cause hypoxic–ischemic brain injury due to pulmonary compromise. Hypoxic–ischemic injury may also arise from neurogenic pulmonary edema resulting from overactivation of central sympathetic mechanisms in the medulla.

Hypoxic–Ischemic Encephalopathy (IED Explosion)

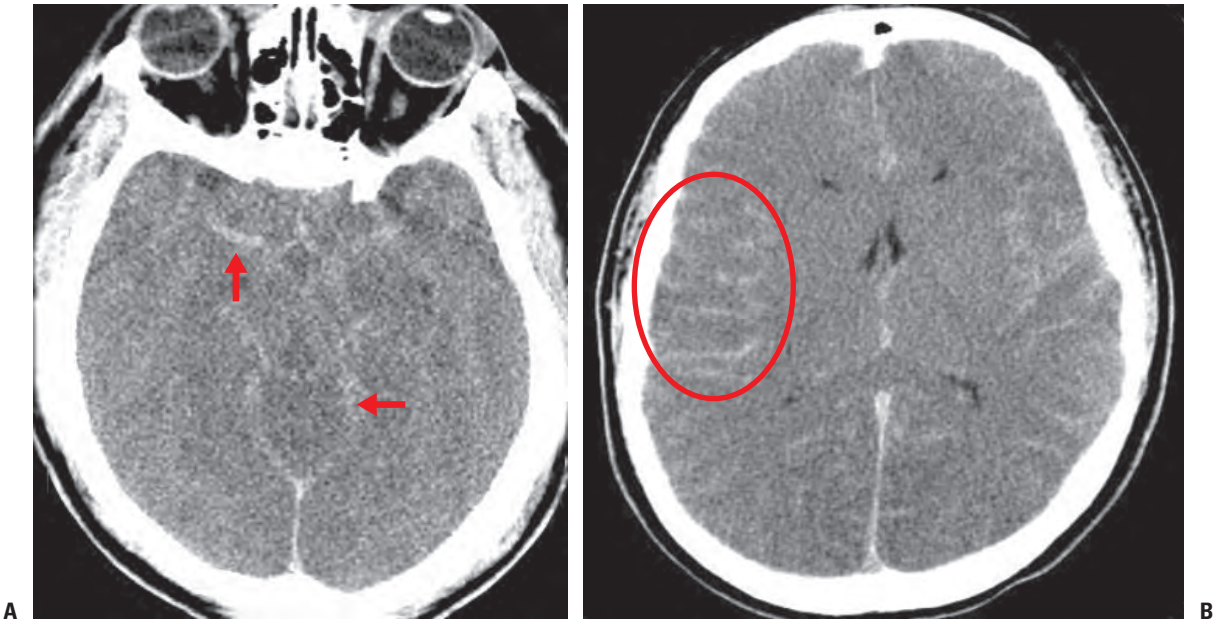

Figure 5.78. Hypoxic–Ischemic Encephalopathy and Pseudo–Subarachnoid Hemorrhage. A. Noncontrast axial CT images at the level of the midbrain and **(B)** basal ganglia show complete effacement of sulci and cisterns. There is loss of distinction among the deep gray nuclei, white matter, and cortex. The vascular structures and dural reflections appear abnormally dense (*arrow, circle*), which may mimic acute SAH and subdural hemorrhage.

★**KEY POINT** The hyperdensity in pseudo-SAH is thought to be due to a combination of venous stasis, loss of low-density CSF, and increased contrast appearance between the hyperdense sluggish vasculature against the hypodense ischemic parenchyma.

Drowning (Prolonged Extrication from Humvee Rollover into the Tigris River)

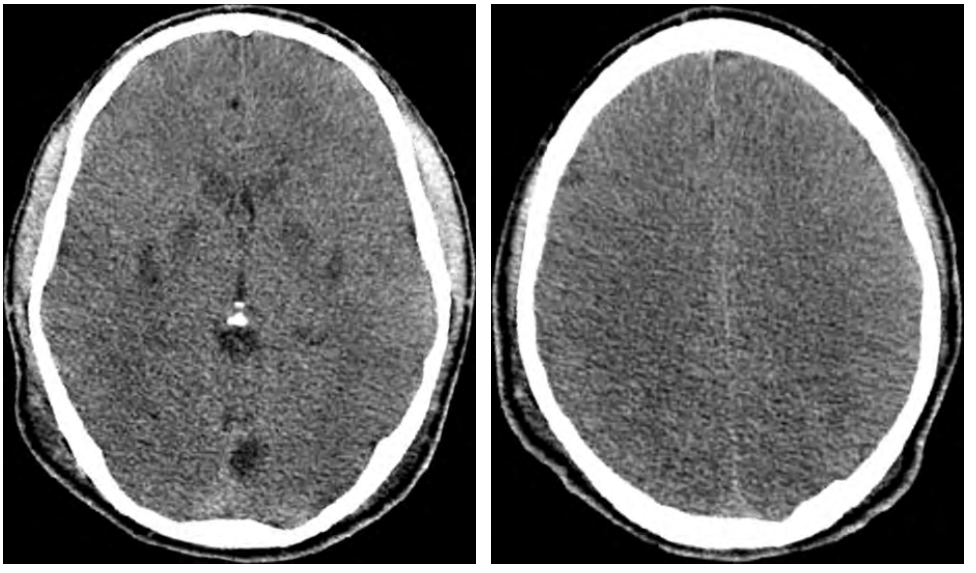

Figure 5.79. Anoxic Brain Injury. This 23-year-old soldier presented with pulseless electrical activity (PEA) and a GCS = 3 after a roadside bomb capsized his vehicle into the river. Noncontrast axial CT images reveal diffuse loss of gray–white differentiation, complete effacement of the cerebral sulci and cisterns, and bilateral symmetric low attenuation lesions of the caudate, globus pallidus, and posterolateral putamen.

★**KEY POINT** In cases of profound asphyxia, the caudate nuclei and putamina are affected more significantly than the thalami.

Cerebral Fat Embolism (Humvee Rollover with Femoral Fractures)

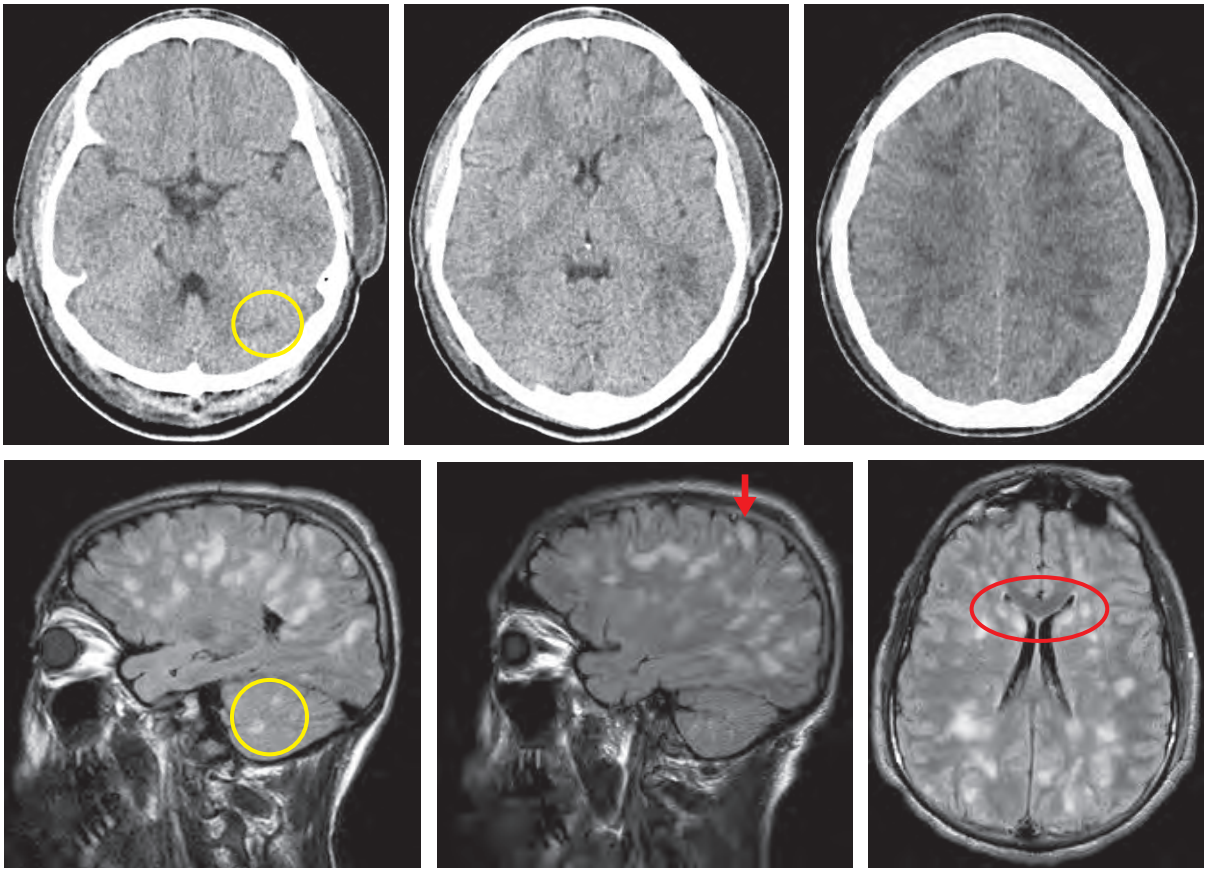

Figure 5.80. Cerebral Fat Embolism. Noncontrast CT images **(top row)** demonstrate numerous nonhemorrhagic low attenuation areas within the subcortical white matter. Note the focus of low attenuation within the left cerebellar hemisphere (*circle*). A frontotemporal subgaleal hematoma is also noted (*arrow*). FLAIR MRI **(bottom row)** performed 48 hours later strikingly demonstrates the multifocal white matter T2 hyperintense lesions, consistent with cerebral fat emboli.

★**KEY POINT** Note involvement of the cerebellum (*yellow circles*), basal ganglia (*red circle*), and cortical gray matter (*arrow*). The location of traumatic lesions in these three locations can help differentiate cerebral fat emboli from TAI.

Eighth, *paradoxical embolic infarction* can be seen in troops due to their increased incidence of deep venous thrombosis (DVT)/pulmonary emboli. Risk factors in our troops for deep vein thrombosis include dehydration, sepsis, and prolonged air-evacuation with constraining straps. Ninth, quaternary blast injuries can result in *hypoxemia* in the setting of an inhalational burn, both thermal and chemical (e.g., chlorine). Tenth, cerebral infarction can be associated with *meningitis* resulting from a dural tear with CSF leakage or from implanted clothing and/or debris from a penetrating TBI. A CSF fistula is associated with a 20-fold higher risk of intracranial infection.[300] Finally, as described earlier, heat stroke can occur in combat. The CT and MRI findings of heat stroke reflect the direct thermal injury, hypoxic ischemic injury, and coagulation abnormalities leading to tissue infarction.

LESSON 11: COMBAT TBI PATIENTS ARE PARTICULARLY VULNERABLE TO SECONDARY TBI

Blast victims are at particular risk for developing secondary TBI. Recall that brain injury occurring at the moment of the initial trauma is called primary TBI and is irreversible. This initial injury then sets in motion a series of deleterious biochemical processes called secondary TBI that worsen patient outcome. Secondary TBI occurs most often in the first 24 hours after the injury; it is potentially preventable. Indeed, inhibiting or reversing these processes has been the goal of trauma and critical care physicians for years. Unfortunately, the care of the polytrauma patient by definition involves managing multiple injured body systems simultaneously. In the carefully controlled environment of a civilian hospital, prevention of secondary TBI is usually manageable.

On the unpredictable battlefield, however, there are numerous reasons why combat casualties are vulnerable to secondary TBI. First, combat wounds often occur in a dirty environment, and casualty data show infections occur more frequently than in other forms of trauma. The incidence of meningitis in this population has been reported to be 9.1%, and the incidence of meningitis in patients with a concomitant CSF leak is nearly 26%.[23] Because of the complex craniofacial penetrating injuries, CSF rhinorrhea is a common problem in combat trauma. Second, amputations are an inevitable consequence of improved body (torso) armor, and controlling blood pressure in the setting of extreme blood loss is challenging. The massive fluid resuscitation that occurs in the field can have both positive and negative effects on the injured brain. Specifically, because the injured *young* brain is particularly prone to cerebral dysautoregulation, excess fluid can exacerbate intracranial hypertension, whereas hypotension can result in cerebral ischemia. Tailored fluid resuscitation to maintain adequate CBV and cerebral perfusion is crucial. Third, as mentioned previously, burns occur more often in combat, and the unique and stringent demands of their management can complicate the care of the other body systems, including the brain. Fourth, systemic hyperthermia is known to exacerbate damage to an already injured brain. Fifth, the staged continuum of care described previously is, by its very design, associated with multiple patient transfers, and each transfer sets up the potential risk for hypotension and other secondary insults. Impaired cerebral perfusion can have a dramatic effect on the injured brain—*a single episode of hypotension can double patient mortality.*[301] Decreased cerebral oxygenation can also result from concomitant pulmonary trauma resulting in hypoxemia, hypocapnia, and metabolic acidosis. As described earlier, the lungs are

vulnerable to primary blast injury, pulmonary embolism, and aspiration pneumonia. Finally, injuries caused by the first-degree overpressure blast wave tend to evolve over time. Unlike blunt and penetrating injuries, the injury from the primary impact is not initially obvious. It sometimes takes 24 hours for the blast injury to declare itself. In a purist sense, this is not really a secondary injury because it is actually the normal evolution of the primary injury. However, its delayed nature complicates the treatment of other injuries and thus, puts the soldier at risk for additional secondary TBI.

LESSON 12: POST-TRAUMATIC STRESS DISORDER (PTSD) IS MORE COMMON FOLLOWING COMBAT THAN FOLLOWING CIVILIAN TRAUMA

"The War Inside"

In 1933, U.S. President Franklin D. Roosevelt said, "The only thing we have to fear is fear itself." He was commenting on the economic future of the United States, but he could have easily been referring to the unreasonable, overgeneralized fear of PTSD. *PTSD is a very normal reaction to a very abnormal situation*. Because of this definition, physicians often prefer the term "post-traumatic stress" (PTS) rather than PTSD. The description of the syndrome started in the second part of the 19th century. Since then, different names have been used: railway spine, commotio cerebri, shell shock, traumatic neurosis, war neurosis, battle fatigue, concentration camp syndrome, and rape trauma syndrome.[302] By definition, PTSD is an anxiety disorder characterized by three symptoms: avoidance, reexperiencing, and hyperarousal. It can be found in both the *Diagnostic and Statistical Manual of Mental Health Disorders, 5th edition* (DSM-V) of the American Psychiatric Association and in the last *International Classification of Diseases* (ICD-10) released by the World Health Organization (WHO).[303] PTSD is a disorder of extinction, or otherwise put, a disregulation in the unlearning of fear responses. We learn fear easily, and extinction is the brain's way of weeding out maladaptive associations. Patients with PTSD have difficulty correctly identifying stimuli that could be threatening. There are two subtypes of PTSD: *dissociative* PTSD and *hyperaroused* or *reexperiencing*.[304,305] The less common dissociative subtype is characterized by overmodulation of affect, whereas the more common, undermodulated type is characterized by intrusions and emotional hyperarousal. The two types represent a different reaction to the acute trauma response and use different neuroanatomic pathways (as shown via functional neuroimaging). In both cases, however, it is believed that the traumatic experiences precipitate stress responses (corticosteroid neurotoxicity) and traumatic memory encoding through limbic, autonomic, and neuroendocrine dysregulation.[306–308]

Who Gets It?

Although most adults have experienced traumatic events through their lifetime, relatively few develop PTSD.[309] Studies have shown that, unrelated to the traumatic event, additional risk factors for developing PTSD include younger age at the time of the trauma, lower socioeconomic status, lack of social support, and some premorbid personality characteristics.[310] Women are at higher risk than men (both in combat and in civilian trauma), especially women with preexisting anxiety. PTSD is more common in veterans

with blast-related TBI compared with those with nonblast exposure.[311] Individuals without religious beliefs appear to be at greater risk for developing PTSD.[312] Experiencing trauma multiple times is associated with an increased frequency of PTSD-associated symptoms—this may be particularly relevant to our troops who are exposed to multiple tours or multiple events within the same tour.[313,314] It is also relevant to victims of child and domestic abuse.

Comorbidity of PTSD with other psychiatric disorders is high, compounding symptom severity and social dysfunction. For example, approximately 40% of PTSD subjects diagnosed in the acute post-traumatic period also meet major depressive disorder criteria, and up to 95% of trauma victims found to have PTSD over their lifetime have a history of major depression. More surprising is the association of PTSD with bipolar disorder; patients with bipolar disorder are at greater risk for developing PTSD than those without.[315] Individuals exposed to violence during their childhood also develop PTSD more frequently than unexposed persons.[316]

Chronic PTSD can manifest in different clinical forms. The reexperiencing syndrome can appear long after the traumatic event. It may follow a relatively asymptomatic latency period that can sometimes last for years. Even though the majority of people with acute stress disorder subsequently develop PTSD, the current data indicates that many people can develop PTSD without initially displaying acute stress disorder. Available twin and family studies indicate that PTSD is at least moderately heritable, with approximately 30% of variance accounted for by genetic factors.[317,318] To date, several genetic components (single nucleotide polymorphisms) for PTSD have been identified that may explain this risk.[319] The level of trauma exposure interacts with risk allele count, such that PTSD is increased in those with higher risk allele counts and higher trauma exposures. Thus, the individual's genetic makeup contributes to the magnitude of the PTSD response.[320–323]

Why Do We Care So Much?

Most importantly, the occurrence of PTSD following both combat and civilian trauma can significantly disrupt an individual's life. The financial cost of PTSD and depression among service members is estimated to exceed $6 billion in the first 2 years following deployment alone.[324] According to U. S. Department of Defense's casualty website, more than 2,700 military personnel have committed suicide since 2001, but that number does not include National Guard and reserve troops not on active duty. Suicides among active personnel reached a historic high in 2012: 350 service members died of self-inflicted wounds, far exceeding American combat deaths in Afghanistan and more than double the number of reported suicides from a decade ago.[325,326] Among veterans, the number who die from suicide has remained relatively stable over the past decade (averaging about 22 per day). The percentage of the nation's daily suicides committed by veterans was 21% in 2010.[327] It is noteworthy that the risk of suicide is increased among military personnel with TBI, and this risk increases with the number of head injuries a soldier experiences.[328] We aren't sure how concussion might contribute to suicide risk, but recent studies of retired NFL players have shown that depressive symptoms were more common in players who had suffered concussions during their career as compared to those who had not experienced concussions. The epidemic of military suicides is perplexing—more men than women kill themselves; 90% of suicides involved enlisted men, not officers; 75% of suicides involved personnel who did not attend college; and suicide is high even among those who did not fight.

PTSD substantially increases the risk for age-related diseases, such as cardiovascular, autoimmune, and neurodegenerative diseases, along with early mortality.[329–331] PTSD is associated with an increased risk of metabolic syndrome and thus an elevated risk for cardiovascular disease and diabetes.[332] PTSD promotes early telomere shortening by increasing cell turnover and promoting the release of reactive oxygen species that damage telomeric DNA via oxidative stress.[333] Individuals with PTSD often demonstrate dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, increased sympathetic nervous system activation, and elevated inflammatory activity resulting in stress-related elevations in cortisol and catecholamines. Indeed, the psychological toll of deployment may be disproportionately high compared with physical injury. Finally, PTSD is also associated with numerous socioeconomic costs incurred by unemployment, divorce, substance abuse, domestic violence, and homelessness.

How Common Is PTSD in Combat?

Although stress has always been a part of combat, it seems to be particularly common in the recent conflict. Over 300,000 troops (i.e., >20% of all soldiers) returning from Iraq and Afghanistan may suffer from mental health conditions.[334,335] Of those who suffered an LOC, up to 44% met criteria for PTSD. It is estimated that up to 45% of patients with burn injuries are affected with PTSD.[165] In addition, the frequency of psychiatric problems is increasing while the rates for other medical diagnoses remain constant.[336] These victims are added to the >500,000 existing Vietnam veterans with chronic PTSD who incur estimated disability costs of $4.3 billion per year.[337] The increase in PTSD in the current war is thought to result from increased troop survival of formerly lethal injuries and the concomitant remembrance of psychologically horrific events that are associated with battlefield trauma.[338] The vast majority of these soldiers have been exposed to a specific traumatic, combat-related situation, such as being attacked or ambushed, seeing dead bodies, handling of remains, killing of enemy, being shot at, seeing fellow soldiers and friends die or be seriously injured, and a sense of helplessness at stopping violent situations, especially when cruelty is involved. Besides these specific acute events, troops are exposed to the chronic threat of being attacked, heat (up to 130° F), sand, insomnia, and the separation from home and family.

Chemical hazards are another threat, and they were particularly problematic in the Persian Gulf War (code-named Operation Desert Storm). Gulf War veterans were exposed to a wide variety of pesticides and herbicides, including organophosphates, acetylcholinesterase inhibitors, and binary nerve agents. Although the conflict lasted only 7 months (August 1990 to February 1991), over 25% of the nearly 700,000 veterans have developed a symptom complex of pain, fatigue, headache, gastrointestinal, bladder, and other functional nociceptive and interoceptive complaints, termed *Gulf War illness*. An interesting recent imaging study using DTI in these veterans demonstrated abnormal white matter diffusivity in the right inferior frontooccipital fasciculus.[339] The right inferior frontooccipital fasciculus links multiple cortical regions that are involved in the perception of fatigue and pain that are symptom constructs in the diagnosis of Gulf War illness. The researchers postulate that the neuropathologic abnormalities in axons underlying the increased diffusivity on DTI may account for the most prominent symptoms of Gulf War illness and that this imaging finding may be a potential biomarker for Gulf War illness.

As is the case with TBI, the scope of the PTSD problem is likely much greater than is

currently appreciated. One of the reasons for this underestimation is the lack of a sensitive diagnostic test. Another reason is the pervasive problem of stigma in the military and society as a whole. Like the professional football player struggling with a concussion, to the average civilian with life stressors and challenges, there is a common fear in those who seek help for mental problems. In the military, many fear they will be perceived as weak and that acknowledging a need for help will damage their military career. Indeed, over half of all soldiers surveyed in one study said that leadership would treat them differently if they sought counseling. In addition to increased troop survivability, soldiers are at a higher risk of developing PTSD in the current war because they are subject to more frequent deployments of greater duration and with shorter rest periods in between. Interestingly, compared to moderate and severe TBI, mild TBI (which is far and away the most common type of combat TBI) is more commonly associated with an increased risk of PTSD.

PTSD Also Occurs in Civilian Trauma

The *battlefield of the mind* is not limited to war and terrorism. Exposure to civilian trauma such as rape, natural disasters, child and domestic abuse, and even car accidents has been linked to the development of PTSD.[310] The prevalence of PTSD after rape has been estimated to be 55%.[309] In one recent prospective study of patients with major trauma, the prevalence rate of PTSD was 25% 1 year after trauma and 20% after 2 years.[340] The prevalence of PTSD in a large cohort of World Trade Center tower survivors 2 to 3 years after the September 11, 2001 terrorist attacks was estimated at 15%.[341] The prevalence of PTSD after an acute myocardial infarction has also been estimated to be 15%.[342] Even veteran responders with medical expertise deployed to disaster areas may experience significant psychological effects from exposure to the tragic circumstances in up to 20% of cases.[343] In the general population, the estimated lifetime prevalence of PTSD is 7.8% in adult Americans. In the United States, it is the third most common anxiety disorder. The prevalence of PTSD around the world is estimated to range between 2% and 15%, whereas the prevalence in high-risk groups is reported to vary from 3% to 58%.[344] As emphasized earlier, the risk for developing PTSD is influenced not only by the particular traumatic event but also by preexisting genotypic factors that regulate the expression of genes related to the serotoninergic system and the adrenocorticotropic axis.

The Symptoms of PTSD Overlap with TBI

Both PTSD and TBI manifest with disinhibition, emotional lability, impulsivity, decreased social regulation, fatigue, depression, irritability, sleep disorders, difficulty concentrating, difficulty switching between two tasks, slowed thinking, depression, and short-term memory problems. Symptoms of headache and dizziness are more characteristic of TBI than PTSD. In contrast, terror and intrusive nightmares are characteristic of PTSD and not TBI. Of course, the soldier may struggle with both issues, as TBI and PTSD are frequently comorbid in the war setting.[345,346] The onset of symptoms of PTSD involving motor vehicle accidents and combat-related stress can be *delayed* in up to 10% of cases.[347,348] It is speculated that damage to the PFC by mild TBI may compromise the ability of the victim to engage in positive coping strategies needed to manage the aftermath of psychological trauma.[349]

Although the incidence of PTSD is increased in patients with TBI, it is important to remember that PTSD can occur without having suffered blunt or penetrating brain injury. Recent animal studies have shown that exposure to blast acoustics alone, *without* TBI, can increase anxiety and trigger specific behavioral and molecular changes.[350–352] In these cases, direct exposure to blast overpressure triggered a gliotic response, subcellular changes in the molecular organization of neurons, and cellular death.

Imaging Applications Are in Their Infancy in Psychiatry, in General, and PTSD in Particular

Currently, PTSD is impossible to visualize with CT and cannot be reliably diagnosed by MRI. Certain structural changes have been reported to be associated with PTSD, including atrophy of the hippocampus, parahippocampal gyrus, amygdala, orbitofrontal cortex, and anterior cingulate cortex (ACC).[320,353–356] There are two hypotheses about the cause of brain atrophy in PTSD. As mentioned earlier, one explanation for the reduced volume is the neurotoxicity caused by elevated glucocorticoids, reduced BDNF, and the inhibition of the regeneration of damaged brain tissue.[357] The second hypothesis is that people who have a smaller hippocampus at birth are genetically at higher risk for developing PTSD.[358] In addition, several studies suggest that childhood abuse may contribute to hippocampal volume loss in patients who develop PTSD at a later stage in their lives.[359] In similar volumetric MRI studies, Kasai and colleagues[307] found that combat-exposed twins with PTSD had significantly lower gray matter density of the pregenual ACC, as compared to combat-exposed twins without PTSD. This suggests that PTSD causes volume loss in the ACC. Taken together, results from these and other studies imply that volume loss can both result from PTSD and predispose the individual to the development of PTSD. Such duality of cause and effect complicates studies of PTSD/complicates our understanding of PTSD. Ultimately, longitudinal studies in populations regularly exposed to traumatic events are needed to discern whether PTSD causes volume loss in specific areas of the brain or whether smaller volumes in these brain regions are present before the trauma and predispose to the development of PTSD. In addition to volume changes, structural changes have also been correlated with PTSD. A recent DTI study found a reduction in FA in the frontal white matter tracts of veterans with impulsivity and suicidality, who had also had a history of mild TBI.[360] This suggests that post-traumatic microstructural changes in the brain may predispose individuals to frontal-limbic dysfunction, leading to behavioral disinhibition and possibly even suicide. *Functional* neuroimaging in PTSD is very much a work in progress, but several interesting findings have emerged. As mentioned previously, most fMRI studies use some form of symptom provocation paradigm, but more recently alterations in brain functioning may be probed using fMRI in patients during resting conditions (i.e., *default-mode network connectivity*).[49] The functional neuroimaging observations in PTSD are briefly listed in the following text.

Advanced Neuroimaging in PTSD

1) Functional MRI Findings in PTSD

- ***Increased activation of the amygdala***, both at rest and with provocation.[361–363] The amygdala is an essential component of the brain circuitry that assigns emotional significance and produces appropriate behavioral responses to external stimuli, especially when responding to fear and fear conditioning. It is also integral to emotional learning, both pleasant and unpleasant.[364] These responses are characterized

by activation of the autonomic system: freezing, potentiated startle, release of stress hormones, and changes in blood pressure and heart rate. The amygdaloid complex, located in the medial temporal lobe, is structurally diverse and comprises approximately 13 nuclei. These are further divided into subdivisions that have extensive intranuclear and internuclear connections. There are several sources of sensory information to the amygdala, including the PFC, perirhinal cortex, and hippocampus. In addition, the amygdaloid nuclei have widespread efferent projections to the cerebral cortex, brain stem, and hypothalamus, the latter of which has a major influence on the coordination of ingestive, reproductive, and defensive behaviors.

It has been known for over a century that the temporal lobe, including the amygdala, is involved in emotion. In 1888, Brown and Schafer[365] described *taming* in the monkeys associated with temporal lobe retraction. Klüver and Bucy[366] elaborated on this finding by characterizing a collection of emotional disturbances caused by temporal lobe damage, which became known as Klüver–Bucy syndrome. Monkeys with such temporal lobe lesions exhibited an absence of anger and fear, increased exploration, visual agnosia, hyperorality, hypersexuality, and loss of social interactions. Subsequent work has shown that lesions restricted to the amygdala produce many of these effects including a loss of fear and anger, increased exploration, and hyperorality. The reduced fear and anger, or taming effect, of amygdala lesions is seen in many animal species. Although amygdala damage in humans rarely results in the full-blown Klüver–Bucy syndrome, it is associated with emotional deficits, including loss of the recognition of fear in others.[367]

fMRI studies have shown that the severity of PTSD symptoms is directly correlated with increased blood flow in the amygdala. Diminished *habituation* of fearful versus happy responses has also been observed in PTSD patients. Interestingly, most PTSD patients show amygdala hyperactivity, and post-traumatic damage to the amygdala in combat veterans has been shown to be *protective* for the development of PTSD.[368] Similarly, TBI lesions located in the right limbic system (i.e., the right cingulum and hippocampus) have been found to inhibit the development of PTSD manifestations.[369] Perhaps these structures are critical for reexperiencing the trauma. Indeed, in the past, psychosurgical lesions in this area were found to benefit patients with severe anxiety.[370] Note that although most PTSD patients show amygdala hyperactivity, the less common, dissociative subtype does not. Animal studies are limited by the inability of nonhuman subjects to tell us how they feel. Even with human subjects, our understanding of the amygdala and its role in emotion is hampered by the abstract nature of emotion itself.

- ***Decreased activation of the dorsolateral prefrontal cortex.***[371] The dorsolateral prefrontal cortex (dlPFC) is one of the three regions of the PFC, along with the ventromedial prefrontal cortex (vmPFC) and the orbitofrontal cortex (OFC). Whereas the vmPFC is largely ascribed to *emotional* or *affective* functions, the dlPFC is primarily associated with *cognitive* or *executive* functions. The normal dlPFC inhibits emotional responses via reciprocal connections with the ventral PFC. It also helps us properly execute fear responses. In addition, it has been well established that the PFC works in conjunction with the hippocampus to form new memories. PFC activity is not only involved in the creation of memories but also in preventing unwanted memories from surpassing threshold for behavioral recognition. fMRI studies have demonstrated the recruitment of the dlPFC during the regulation

of negative emotion through reappraisal/suppression strategies. Cognitive reappraisal of an adverse event is associated with an increase in dlPFC signal and a decrease in the OFC, amygdala, and hippocampus (**Fig. 5.81**). Thus, the dlPFC is involved in actively reducing anxiety. Decreased dlPFC function may predispose the patient to PTSD such that they are less likely to engage in cognitive coping strategies that promote adjustment to events. Interestingly, adult patients with PTSD show marked improvement with treatment with transcranial magnetic stimulation over the right dlPFC.[372] In addition, treatment with omega-3 polyunsaturated fatty acids has been reported to enhance hippocampal neurogenesis and to reduce PTSD symptoms.[373]

- ***Decreased activation of the ventromedial prefrontal cortex.*** The right vmPFC has an inhibitory influence on the emotional limbic system. It modulates the response to a stressful situation by inhibiting the amygdala to fearful cues and thus plays an important role in the extinction of fear conditioning. It does so by modulating the HPA axis and autonomic response system. When prefrontal activation is decreased, the amygdala does not receive sufficient inhibitory feedback, resulting in higher autonomic arousal and exaggerated responses as manifest in patients with PTSD. The more severe the symptoms, the more significant the task-related hypometabolism (decreased rCBF) signal changes. Of note, however, the Vietnam Head Injury Study showed that damage to the vmPFC was actually protective against later development of PTSD.[374] This was postulated to be due to a lack of insight into the stressor rather than a lack of amygdala control. Similar to transcranial magnetic studies of the dlPFC, a recent study showed that fear extinction can be facilitated by repeated medial PFC deep transcranial magnetic stimulation in PTSD patients resistant to standard treatment.[375]

Functional MRI in a Memory Suppression

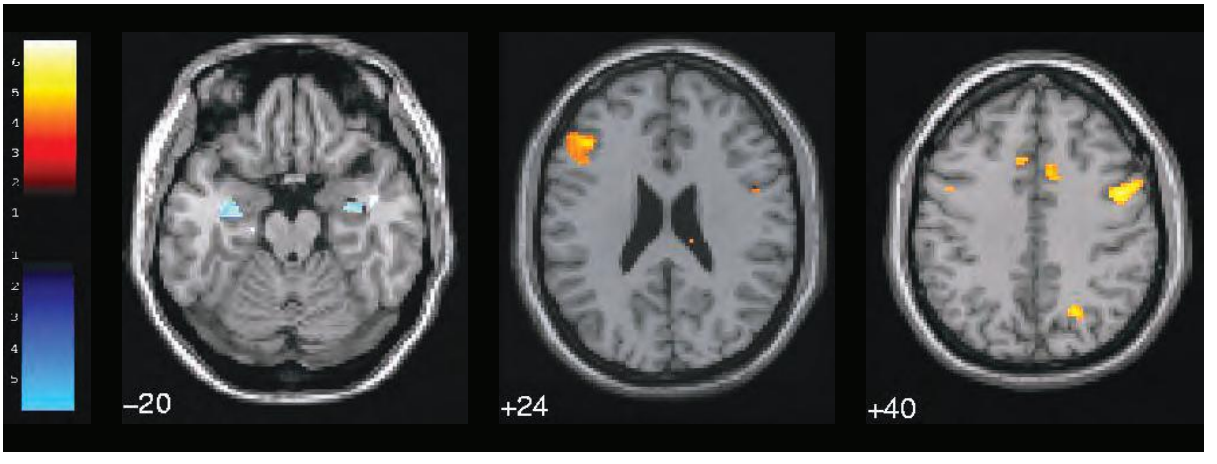

Figure 5.81. Is it Possible to Erase Memories? This fMRI study using a memory suppression paradigm in healthy volunteers demonstrates decreased activation of the hippocampus (*blue areas*) and increased activation of the dlPFC and anterior cingulate cortex bilaterally, as well as the right lateral premotor cortex (*yellow areas*). In layman's terms, this shows that when we try to suppress a memory, we are actually doing something very active and engaging a distinct network of the brain that is trying to keep the unwanted memories at bay. (Courtesy of Michael Anderson, Cambridge.)

- ***Increased activation of the anterior insula.***[376] The insula participates in the intensity interpretation of stimuli. When the insula is activated, individuals become more sensitive to adverse possibilities. Given the role of the insula in interoception, as well as evidence indicating abnormal anterior insular function in anxious patients, this region has been proposed to serve a crucial role in proneness to anxiety.[377]
- ***Increased activation of the fusiform gyrus.***[378–380] The fusiform gyrus is part of the visual cortex of the occipital lobe. It is known to be involved in the perception of emotions to facial stimuli. Patients with PTSD may attempt to dispel visual images of upsetting events to a greater extent than patients without PTSD.
- ***Decreased activation of the striatum*** such that reward-related stimuli appear to be attenuated.[381] Interestingly, a recent fMRI study in healthy participants showed that if the threat of pain is associated with potential reward, activation of the striatum and ACC decreased.[382] These findings have led to the suggestion that the loss of appreciation of pleasure and an overexpectation of pain (i.e., an altered balance of pain/pleasure integration) may underlie many symptoms of PTSD.[383]
- ***Increased autonomic arousal.***[384] This finding is consistent with the hypervigilant state of PTSD patients. The autonomic dysregulation is hypothesized to be due to hypoactivation of the medial PFC, which elicits a sympathoexcitatory response.

2) Positron Emission Tomography (PET) and Single Photon Emission Computed Tomography (SPECT) Findings in PTSD

- ***Increased flow/activation of the amygdala.***[385] This is similar to the fMRI findings cited previously.
- ***Reduced activation of the hippocampus*** during memory-related tasks (not conclusive as studies using tasks with emotional content report inconsistent findings).[386]
- ***Decreased activation of the dlPFC and anterior cingulate cortex.***[384,387] The anterior cingulate gyrus is part of a system that orchestrates the autonomic, neuroendocrine, and behavioral expression of emotion. It is associated with regulating affective arousal networks, and more specifically, the extinction of fear responses. The ACC is part of the executive control network that works with the dlPFC to inhibit inappropriate behavior. Note that an abnormal increase in activation of top–down cognitive-appraisal neural structures, such as the dorsal ACC and medial PFC, may contribute to arousal dysregulation via promoting hypervigilance.
- ***Increased resting metabolic activity of the anterior cingulate cortex.***[388,389] In a fascinating study involving veterans with twins, veterans with PTSD and their unexposed co-twins had significantly higher resting regional cerebral glucose metabolic rates in the ACC compared with veterans without PTSD and their unexposed co-twins. Resting metabolism in the ACC in unexposed co-twins was positively correlated with combat exposure severity, PTSD symptom severity, and alcohol use in their exposed twins. The study concluded that enhanced resting metabolic activity in the ACC appears to represent a familial risk factor for developing PTSD after exposure to psychological trauma.
- ***Decreased activation of the thalamus.***[390] All sensory information (except for olfaction) is routed through the thalamus to the cerebral cortex. Thus, the thalamus is often referred to as the sensory gateway to the cortex. High levels of arousal during traumatic experiences have been hypothesized to lead to altered

thalamic sensory processing, which in turn results in a disruption of the transmission of sensory information to the frontal cortex, cingulate gyrus, amygdala, and hippocampus.

- ***Decreased functional connectivity associated with working memory difficulties.*** PTSD patients are characterized by more activation in the bilateral inferior parietal lobes and the left precentral gyrus and less activation in the inferior medial frontal lobe, bilateral middle frontal gyri, and right inferior temporal gyrus, as compared to control groups without PTSD.[391]
- ***Induction of panic attacks and decreased metabolism*** within the prefrontal, temporal, and parietal cortices following yohimbine (a drug that stimulates norepinephrine production) administration.[392]

3) Magnetoencephalography (MEG) Findings in PTSD

- ***Synchronous neural interactions.***[393–395] MEG is an FDA-approved, noninvasive technique used to measure magnetic fields generated by small intracellular currents in neurons. Synchronous neural interaction (SNI) is a special type of MEG test that assesses the functional interactions among neural populations derived from MEG recordings. The presence of SNIs in MEG recordings has been able to differentiate PTSD patients from healthy control subjects. Thus, proponents of the SNI test believe that it is a functional neuromarker for PTSD.

4) Magnetic Resonance Spectroscopy (MRS) Findings in PTSD

- ***Decreased N-acetylaspartate–to–creatine ratio*** *in the basal ganglia, right medial temporal lobe, dlPFC, ACC, and possible increased Cho in the hippocampus and ACC.*[396–398] Decreased NAA has also been noted in patients with a generalized anxiety disorder following a previous traumatic event.[399] These studies do not answer the question of whether the NAA changes were preexisting or a consequence of trauma, rather than due to PTSD per se.

In summary, wounded soldiers who would have likely died in previous conflicts now survive but with significant emotional injury. Although our understanding of the psychological morbidity associated with war is in its infancy, recent neuroimaging studies are slowly revealing the underlying cortico-limbic neurocircuitry of PTSD. The abnormalities are observed initially as a functional metabolic abnormality that may later evolve to a structural abnormality. The imaging findings reflect the underlying psychobiologic circuits involved in emotions related to pleasure, fear, anger, and attention (i.e., hyperarousal of the amygdala and insufficient control by the PFC). Although these studies are promising and provide insight into the neural substrates of PTSD, the results must be taken cautiously, as not all PTSD studies concur. Explanations for the inconsistent results include methodologic variations between studies, the complexity of PTSD symptomatology, the presence of comorbidities, and the heterogeneity of responses to trauma, with variations related to genetic and environmental influences. Moreover, because no two traumatic events and no two individuals are the same, considerable variability arises in correlating the nature and severity of the trauma with the extent of emotional disability from PTSD. Reproducible and reliable imaging findings to diagnose PTSD and validated methods for clinical use are still lacking. The results from current advanced neuroimaging techniques are promising but have not been robustly validated for universal clinical application.

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CHAPTER 6

Transferring Lessons to the Home Front

With the risk of terrorist attacks increasing worldwide, not to mention the apocalyptic threat of large-scale mass casualty incidents (MCIs) such as that which recently occurred in Japan, it is imperative that we learn from our combat colleagues. Home front disasters provide a reminder to health care providers of the importance of planning and preparedness for hospitals to function optimally during and after a catastrophic event. In 2005 alone, the U.S. Department of State reported approximately 11,000 international terrorist attacks.[1] In the United States, between 1983 and 2002, there were 36,110 bombing incidents,[2] including the attacks of September 11, 2001. The magnitude and intensity of the attack on the World Trade Center made it the worst human-made disaster in the history of the United States, with almost 3,000 fatalities, 71,000 jobs lost, and labor and capital losses reaching $36 billion in the months following the attacks.[3] In Israel, the incidence of terrorist attacks is much greater.[4] Over half of the victims, usually civilians, are injured in explosions, often by suicide bombers. Explosions are the most common deliberate weapon of terrorism because of their potential to quickly and inexpensively inflict considerable harm and devastation on large groups of people. When these events occur, they place tremendous strains on medical resources, and they present unique diagnostic, triage, and management challenges for physicians that are much different from their normal daily workload.[5,6]

LESSONS THAT SHOULD BE APPLIED TO CIVILIAN TRAUMA

Combat and Terrorist Injuries Are Different from Civilian Injuries

It is important that physicians become familiar with the spectrum of injuries inflicted by blasts and explosions. As illustrated earlier, these

injuries are different from civilian trauma, and thus they are unfamiliar to the vast majority of physicians. Current explosive devices used in bombings combine the primary blast effect with both blunt impact trauma and penetrating trauma. The objects placed in these bombs more frequently penetrate the skull and injure the brain and the cerebral vasculature, as compared to the wounding potential of weapons found in civilian trauma. In particular, cerebral swelling, vasospasm, burn injury, and delayed deterioration are common problems with blast brain injury. Surface wounds from fragment injuries and high-kinetic energy bullets are much more common in war and terrorist attacks than in typical civilian trauma. Physical recovery is also confounded by emotionally traumatic events. Understanding these crucial peculiarities is critical for the management of blast casualties, and they present especially difficult management issues to the patient with traumatic brain injury (TBI). Although physicians and neuroscience researchers have only scratched the surface regarding the precise mechanisms of the pathologic and functional sequelae of blast-induced neurotrauma (BINT), war and terrorism are forcing a reluctant study of all dimensions of blast trauma.

Combat and Terrorist Injuries Are More Severe than Civilian Injuries

The wounding capacity of modern weapons has increased exponentially since the time of the American Revolution.[7] Combat and terror-related injuries are more severe than non–terror-inflicted injuries.[8] Victims require a more prolonged hospital stay, incur a higher hospitalization cost, and have a higher mortality rate than victims of any other form of trauma.[9] In a recent analysis of data from the Israel Center for Disease Control, bombing casualties affected younger age groups with higher Injury Severity Scores (ISS >16, 30% vs. 10% for other trauma), increased immediate mortality (as high as 29% for closed space bombings), greater in-hospital mortality rates (6% vs. 3% for other trauma), more frequent need for surgical intervention, longer hospital length of stays, and greater demands on intensive care resources.[10,11] In general, **major trauma** (or polytrauma) is defined as an ISS >15. Peleg[12] found similar numbers for terrorist victims, with 30% having ISS >16, 53% requiring surgical procedures, 23% requiring an intensive care unit (ICU) stay, and 20% having a hospital length of stay greater than 14 days, and all measures were increased as compared with those for victims of other causes of trauma, such as car accidents or gunshot wounds. Thus, bomb victims have higher hospital resource utilization than victims of any other trauma. As illustrated previously, bomb blast casualties may present with primary blast injury, such as *blast lung*, and may also suffer from penetrating glass shards, traumatic amputation, inhalation of toxic detonation products, deafness, and burns. In general, 30% of terrorist victims suffer TBI, which is much higher than civilian trauma, and a relatively large proportion of patients (18%) injured in explosions suffer a moderate or severe TBI.[13] Note that *injuries caused by suicide bombers in the civilian population tend to be more severe than those suffered in combat due to the lack of protection, unpredictability of the attack, and the fact that civilian victims are not as physically conditioned and medically fit prior to injury.*[14]

Blast Brain Injuries and Post-traumatic Stress Disorder (PTSD) Are More Common than Previously Thought

Prior wars have brought attention to these injuries, and this war is no exception. As discussed previously, new insights into the incidence,

treatment, and imaging manifestations of combat post-traumatic stress disorder (PTSD) are shedding light on this problem. It is now well accepted that residual PTSD and TBI symptoms are more prevalent in personnel exposed to blast TBI than to blunt TBI.[15] This may be particularly problematic in terrorist attacks and accidental explosions where victims are unprotected by body armor and are caught off guard. In addition, one key insight is the discovery that PTSD is turning out to be more common in nonblast civilian TBI than previously reported.

One of the Rare Benefits of War Is the Advancement in Neurotrauma Care that Occurs

War forces us to develop higher standards of trauma care and pass them on to succeeding generations. Even Hippocrates wrote, "He who would become a surgeon should join an army and follow it." Second century Greek physician Galen honed his skills not only in the sanctuary of Asclepius, god of healing, but also as physician to the gladiators. More recently, Dr. William Mayo said, "Medicine is the only victor in war."[16] Perhaps war's greatest contribution to medicine is the chance to run public health experiments on a grand scale under exigent circumstances. Evidence-based medicine drives medical decision making in the modern era, and this large, unique database has taught us a lot.[17,18] Like previous conflicts, the wars in Iraq and Afghanistan have fostered improvements in training, systems, evacuation, resuscitation, wound care, and surgery techniques. More specifically, medical advances that have been accelerated by war and terrorism that can be applied to civilian trauma care at home include:

1) **Advancements in resuscitation strategies** and management of the **complex polytrauma** patient. For example, our approach to treating blood loss has evolved over the decade. The restoration of circulating blood volume has changed from saline or starch-based volume expanders to fresh whole blood or packed cell transfusion augmented by fresh frozen plasma and platelets.
2) **Improved understanding of the role of decompressive craniectomy** in the setting of brain swelling.
3) **Increased familiarity and improvement in advanced neuromonitoring devices.**
4) **Improved medical care in austere environments.** Many of these lessons will be relevant to MCIs and natural disasters suffered abroad. A recent review of global injury showed that approximately 80% of severe TBIs occur in austere environments, defined as regions lacking in prehospital response teams and advanced care in an ICU.[19]
5) **Dramatic increase in funding for blast injury research.** In contrast to stroke, heart disease, HIV/AIDS, and cancer, funding for TBI research has been disproportionately lacking until now.
6) **Infection control advances** (e.g., rapid diagnostic strategies for infection; antimicrobial prophylaxis; improved understanding of nosocomial infections, particularly the *Acinetobacter* species; increased experience with contaminated wounds).
7) **Insight into the incidence and treatment of cerebral vasospasm** (e.g., neurointerventional radiologist on site in the war zone). To monitor traumatic vasospasm, transcranial Doppler (TCD) ultrasonography is routinely used by the U.S. Army and is now only beginning to be embraced in civilian hospitals. Because of data acquired from wartime TBI, the role of TCD is also now being used to assess patients who have undergone a decompressive craniectomy.

Some of these patients suffer prolonged atmospheric pressure on their brain because of the cranial defect, and TCD can be helpful to evaluate the cerebral blood flow dynamics.

8) **Refinement of damage control resuscitation guidelines** (e.g., in this conflict, the benefit of vascular shunting has allowed temporizing stabilization of a vascular injury until the physiologic effects of the trauma have improved to allow definitive repair).

9) **Improved burn and wound care technology** (e.g., the use of the wound vacuum-assisted closure [VAC] technique is new in this war).

10) **Better understanding of the role and treatment of abdominal blast injury** leading to intra-abdominal hypertension and its downstream effects on intracranial pressure.

11) **Increased experience with the reconstruction of complex craniofacial trauma.**

12) **New paradigms of prehospital care** with advances in combat medic training, better tourniquet and hemostat bandages, anesthetics, and rapid access to blood products. This data can be translated to paramedic guidelines in civilian trauma.

13) **Development of improved protective gear** that can be worn by police bomb squads or even by civilians in terrorist targeted areas. Knowledge of new concepts in protective gear and devices can be translated to athletic equipment in professional and nonprofessional sports activities at home.

14) **Development of gear that can *lighten the load* of the warfighter.** Electrically powered external devices, designed to increase mobility of those disabled by trauma, may be applicable to spinal cord–injured civilians and veterans.

15) **Improved combat communication.** This is the first war utilizing teleradiology, computerized patient tracking, and a web-based patient registry. In addition, the novel concept of a weekly video-teleconference, as described, may be applicable to rural trauma systems in civilian practice or to domestic MCI scenarios.

16) **Better guidelines for triaging large numbers of simultaneously injured casualties.** This will be helpful in future terrorist attacks as well as natural disaster MCIs.

17) **Increased database for clinical care guidelines.** This is the first war to use the Joint Theater Trauma Registry (JTTR) to provide follow-up on the use and effectiveness of established clinical care guides. It mimics the civilian trauma system outcome success and has translated into improved morbidity and mortality on the battlefield.

18) **Development of new tools, such as the surface wound mapping (SWM) database and the surface wounding analysis tool (SWAT) software.** These have been successfully implemented to describe combat injury, mortality, and distributions of wounds and associated internal injuries. They are being used in quality of care assessments and to direct resources for optimal care, training activities, and research. They are also providing additional data for those researching improvements in personnel/vehicle protection, injury outcomes (both immediate and over time), resource management, functional impairment, and long-term planning.

Every Physician (and Department) Needs to Plan *in Advance*

MCIs caused by terrorist attacks and natural disasters should be viewed as a predictable surprise. One must treat a large number of casualties in a short period of time. For example, in the 2004 Madrid commuter train bombing, there were more than 2,000 casualties, and the closest hospital received 272 patients in less than 2 hours. As such, MCIs create particular challenges for any medical system and can quickly overwhelm local facilities.[20,21] Fortunately, the Boston Marathon bombing occurred in a city that is home to seven trauma centers and multiple world-class hospitals. Boston emergency medical services (EMS) personnel wisely distributed casualties among the area's trauma centers, so each one received a manageable number of victims.[22,23] Other "fortunate" aspects of the Boston MCI include (1) the bombing occurred at a major event where large numbers of police, security, and EMS personnel were already deployed; (2) the bombing occurred on a state holiday, so the city's operating rooms and other clinical services were running at less than full capacity; (3) the attack happened before the 3 pm shift changes at area hospitals, so a full complement of staff and two shifts of health care providers were on site at each facility; and (4) the detonation occurred outside. As discussed earlier, blasts that occur inside produce more primary blast injuries because surrounding walls concentrate blast waves. In addition, the absence of structural collapse facilitated the swift extrication of victims.

Although the initial triage of the wounded is funneled to the emergency department (ED), a busy radiology department needs to be capable of responding with rapid throughput of patients and the capacity to quickly make room for newly arriving casualties.[24] Patients with TBI usually have the highest priority for computed tomography (CT) scanning as they may require immediate lifesaving operations. Review of the Israeli experience showed that nearly 40% of admitted patients were sent to the CT scanner directly from the ED. Although initially thought to be efficient, in practice this in fact created a bottleneck in the flow of patients from the ED to definitive treatment.[25,26] Thus, triage through the radiology department needs to consider not only getting patients in and scanned but also disposition of patients from radiology back to the ED, to the operating room, or to ward and ICU care settings, in an expeditious and safe fashion.

Radiologists are critical members of the first-line team of physicians managing an MCI. The unpredictable wave of casualties, unusually high stress environment, and the different kinds of injuries mandates a reorganization or, in most cases, a freeze of the routine clinical workflow.[27] Every radiology department should have a disaster plan, *and the staff should be familiar with it*.[28,29] Remember, "Time is Brain." In the event of an MCI, each physician should be easily reachable and should know his or her expected role in advance. Has your department performed practice drills? Do you know *your* role?

Learning about the National Response Framework (NRF) and the local Incident Command System will also allow physicians to understand the larger picture at the national, state, and regional levels.[30] In brief, whether it's an overturned school bus, a hotel fire, an earthquake, or a terrorist attack, *all disasters are managed locally through the on-scene incident commander*. Do *you* know who that is at your facility? Should the event overwhelm the local emergency operations center (EOC), then the state EOC will become involved. Should the disaster continue to escalate, the governor will request assistance from the federal government by declaring a state of emergency and request federal assistance. In response, the

President of the United States may also declare a state of emergency, which allows the federal government to provide financial and material assistance through the Federal Emergency Management Agency (FEMA).[31]

The triage of MCI patients is based on the principle of "the greatest good for the greatest number."[32,33] This model divides casualty response needs into urgent, immediate, delayed, minimal, or expectant (i.e., victims whose survival is unlikely even in the presence of adequate resources and care). The role of medical imaging in the workup of these patients, either in sporadic events or as part of an MCI, including terror-related attacks, has been described.[34] In brief, at the moment of notification, all available personnel should be called in via pager, text messaging, or cellular telephone. The use of social media such as Facebook and Twitter is becoming more common because many can be notified at the same time. Adequate communication such as a *walkie-talkie* may be more practical in the hospital. Senior radiologists should be present at the CT console for rapid on-the-spot viewing and prompt identification of injuries. These individuals should be designated in advance to help facilitate rapid evaluation and casualty throughput. Senior radiologists should work with the ED physicians and trauma personnel to decide who truly needs CT scanning and in what order. They should prioritize the efficient use of all available imaging techniques to ensure optimal triage, diagnosis, and treatment of casualties.[35] Identification of critical life-threatening injuries and major problems can be best communicated through tiered reports—initial *wet* preliminary interpretations are made immediately and left with/on the patient to be acted upon as soon as resources for the next stage of care become available. Final reports or completed interpretations for the permanent record are secondary backups to the primary record. They should also be appended to the patient. Both patient flow and care may be greatly improved with tiered reporting of radiology results.[36]

Depending on the scale of the MCI, routine clinical protocols may need to be abbreviated. Streamlined MCI imaging protocols and treatment algorithms should be prepared in advance and readily accessible. This is especially important because, as noted earlier, the first bottleneck in patient management occurs at the CT scanner. In addition, a second wave of casualty imaging should be anticipated because some patients may have been inappropriately triaged in the field to hospitals without trauma facilities.

To summarize, the unpredictable nature of MCI makes preparation key. Planning and practice for these unexpected scenarios, whether from terror, natural disaster, or large-scale accidents, are absolutely necessary. Therefore, in order to utilize hospital resources efficiently, specific imaging protocols for managing the sudden surge of patients should already be in place and all radiology staff should know their assigned roles in advance. In addition, radiologists should be familiar with the unique spectrum of injuries inflicted by explosive munitions, which are often different, more severe, and polytraumatic than those encountered in typical civilian trauma.

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INDEX

Note: Page numbers followed by followed by *f*, indicate a figure and those followed by *t*, a table.