Ali Salim · Carlos Brown · Kenji Inaba · Matthew J. Martin
Editors

Surgical Critical Care Therapy

A Clinically Oriented Practical Approach

Springer

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Editors
Ali Salim
Brigham and Womens's Hospital
Harvard Medical School
Boston, MA, USA

Carlos Brown
Dell Medical School
University of Texas at Austin
Austin, TX, USA

Kenji Inaba
Division of Trauma Surgery, Rm C5L100
University of Southern California
Los Angeles, CA, USA

Matthew J. Martin
Trauma and Emergency Surgery Service
Legacy Emanuel Medical Center
Portland, OR, USA

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Preface

The field of surgical critical care is constantly expanding, evolving, and undergoing rapid change. Providers are experiencing increasing volumes of complex surgical cases and clinically challenging postoperative patients from a wide variety of surgical subspecialties. With the introduction of new technologies, less invasive surgery, balanced resuscitation strategies, and an aging population, updating and communicating improved care techniques for the critically ill surgical patient are crucial. As critical care providers, our goal is to deliver optimal, evidence-based care supported by relevant policies and data; however, there is no comprehensive source that provides concise and practical guidance to surgical intensivists and multidisciplinary ICU team members.

The *Surgical Critical Care Therapy* textbook will provide a comprehensive, state-of-the-art review of the field and will serve as a valuable resource for clinicians, surgeons, and researchers with an interest in surgical critical care. The chapters focus on the management of common problems and critical decision-making scenarios that arise in the Surgical Intensive Care Unit. For example, several well-designed randomized prospective trials have recently altered the way we care for surgical patients presenting with traumatic brain injury, hemorrhagic shock, acute respiratory distress syndrome, and sepsis. The protocols, care bundles, guidelines, and checklists that show improved process measures and patient outcomes will be discussed in detail throughout the book.

We hope that this textbook will help guide patient management and stimulate future investigative efforts. Each chapter is written by widely recognized and established experts in the field who share numerous tips and wisdom gained over the course of their careers. We also believe that this textbook will become an invaluable resource for residents preparing for their in-service exams or the critical care portions of their general surgery board exams and for all fellowship-trained intensivists who are taking the surgical critical care board examinations.

We wish to thank the professional editorial efforts of Springer and to acknowledge our peers and family members for their support throughout this project. Without the help of so many, this project could not have been brought to fruition.

Austin, TX, USA — Carlos Brown
Los Angeles, CA, USA — Kenji Inaba
Tacoma, WA, USA — Matthew J. Martin
Boston, MA, USA — Ali Salim

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Contributors

Craig R. Ainsworth Burn Intensive Care Unit, Burn Center, US Army Institute of Surgical Research, Brooke Army Medical Center, JBSA Fort Sam Houston, TX, USA

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

Hasan B. Alam Department of General Surgery, University of Michigan, Ann Arbor, MI, USA

Deidra Allison Dell Seton Medical Center at University of Texas, Surgical Intensive Care Unit, Austin, TX, USA

Jared L. Antevil Division of Cardiothoracic Surgery, Department of Surgery, Walter Reed National Military Medical Center, Bethesda, MD, USA

Reza Askari Department of Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

Trauma, Burn, and Surgical Critical Care, Brigham and Women's Hospital, Boston, MA, USA

Jayson Aydelotte Department of Surgery and Perioperative Care, Dell Medical School at the University of Texas-Austin, Austin, TX, USA

Asad Azim Department of Surgery, University of Arizona, Tucson, AZ, USA

Chad G. Ball Department of Surgery, University of Calgary, Foothills Medical Center, Calgary, AB, Canada

Jessica Ballou Oregon Health and Science University, Portland, OR, USA

Brittany K. Bankhead-Kendall Dell Medical School, University of Texas at Austin, Austin, TX, USA

James M. Bardes Division of Trauma, Emergency Surgery and Surgical Critical Care, Los Angeles County and Univeristy of Southern California Medical Center, Los Angeles, CA, USA

Galinos Barmparas Department of Surgery, Division of Acute Care Surgery and Surgical Critical Care, Cedars-Sinai Medical Center, Los Angeles, CA, USA

Brian C. Beldowicz Department of Surgery, Division of Trauma, UC Davis Health, Acute Care Surgery and Surgical Critical Care, Sacramento, CA, USA

Elizabeth Benjamin Division of Trauma, Emergency Surgery and Surgical Critical Care, Los Angeles County and Univeristy of Southern California Medical Center, Los Angeles, CA, USA

Andrew C. Bernard Department of Surgery, University of Kentucky, Lexington, KY, USA

Robel Beyene Division of Trauma and Surgical Critical Care, Vanderbilt University Medical Center, Nashville, TN, USA

Bishwajit Bhattacharya Section of General Surgery, Trauma and Surgical Critical Care, Department of Surgery, Yale School of Medicine, New Haven, CT, USA

Walter L. Biffl Scripps Memorial Hosptial La Jolla, San Diego, CA, USA

David W. Bliss Department of Surgery, Keck School of Medicine of the University of Southern California, Division of Pediatric Surgery, Children's Hospital Los Angeles, Los Angeles, CA, USA

Karen J. Brasel Department of Surgery, Division of Trauma, Critical Care, and Acute Care Surgery, Oregon Health and Science University, Portland, OR, USA

Stacy Brethauer Digestive Disease and Surgery Institute, Cleveland Clinic, Cleveland, OH, USA

Eric Bui Department of Surgery, UCSF East Bay – Highland Hospital, Oakland, CA, USA

Marko Bukur Department of Surgery, Bellevue Hospital Center, New York, NY, USA

Division of Trauma and Surgical Critical Care, New York University School of Medicine, New York, NY, USA

Samuel Cemaj Department of Surgery, University of Nebraska Medical Center, Omaha, NE, USA

Julietta Chang Massachusetts General Hospital, Department of General Surgery, Boston, MA, USA

Aaron M. Cheng University of Washington, Department of Surgery, Division of Cardiothoracic Surgery Co-Director, Cardiothoracic ICU, University of Washington Medical Center Director, Thoracic Surgery, Harborview Medical Center, Seattle, WA, USA

Kevin K. Chung Department of Medicine, Brooke Army Medical Center, JBSA-Fort Sam Houston, TX, USA

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

David J. Ciesla Department of Surgery, University of South Florida College of Medicine, Tampa, FL, USA

Mark Cipolle Department of Surgery – Surgical Critical Care, Christiana Care Health System, Newark, DE, USA

Damon Clark University of Southern California Keck School of Medicine, Keck Hospital, Norris Cancer Hospital and LAC-USC, Department of Surgery, Acute Care Surgery and Surgical Critical Care, Los Angeles, CA, USA

Christine S. Cocanour Department of Surgery, Division of Trauma, UC Davis Health, Sacramento, CA, USA

Ellie Cohen Hennepin County Medical Center in Minneapolis, Minneapolis, MI, USA

Christos Colovos Department of Surgery, Cedars-Sinai Medical Center, Los Angeles, CA, USA

Charles H. Cook Division of Acute Care Surgery, Trauma, Surgical Critical Care, Harvard Medical School, Boston, MA, USA

Zara Cooper Department of Trauma, Burn, and Surgical Critical Care, Brigham and Women's Hospital, Boston, MA, USA

Ben Coopwood Department of Surgery, Dell Medical School, University of Texas at Austin, Austin, TX, USA

Martin A. Croce University of Tennessee Health Science Center, Memphis, TN, USA

Bruce A. Crookes Department of Surgery, General Surgery Division, Medical University of South Carolina, Charleston, SC, USA

Daniel G. Cuadrado General and Thoracic Surgery, Department of Surgery, Madigan Army Medical Center, Tacoma, WA, USA

Mitchell J. Daley Department of Pharmacy, Dell Seton Medical Center at the University of Texas, Austin, TX, USA

Benjamin L. Davis Division of Acute Care Surgery, University of Arkansas for Medical Sciences, Little Rock, AR, USA

Kimberly Davis Section of General Surgery, Trauma and Surgical Critical Care, Department of Surgery, Yale School of Medicine, New Haven, CT, USA

Marc de Moya Division of Trauma, Acute Care Surgery, Medical College of Wisconsin, Froedtert Hospital, Milaukee, WI, USA

Joseph J. DuBose Division of Vascular Surgery, David Grant Medical Center, Travis AFB, CA, USA

Jeremiah J. Duby Department of Pharmacy Services, UC Davis Health, Sacramento, CA, USA

Touro University, College of Pharmacy, Vallejo, CA, USA

UCSF School of Pharmacy, San Francisco, CA, USA

Alexander L. Eastman Department of Surgery, Division of Burns, Trauma, and Critical Care, University of Texas Southwestern, Dallas, TX, USA

Matthew J. Eckert Department of Surgery, Madigan Army Medical Center, Joint Base Lewis-McChord, Tacoma, WA, USA

Alexandra Edwards Department of Surgery, University of Kentucky, Lexington, KY, USA

Margaret K. M. Ellis Department of Anesthesiology and Perioperative Medicine, Oregon Health and Science University, Portland, OR, USA

Charity H. Evans Department of Surgery, University of Nebraska Medical Center, Omaha, NE, USA

Paul M. Evans Division of Acute Care Surgery, Department of Surgery, Grady Memorial Hospital, Atlanta, GA, USA

Peter Fagenholz Division of Trauma/Acute Care Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

Stephen A. Fann Department of Surgery, General Surgery Division, Medical University of South Carolina, Charleston, SC, USA

Dina M. Filiberto University of Tennessee Health Science Center, Memphis, TN, USA

Damon Forbes Pulmonary & Critical Care Service, Womack Army Medical Center, Ft. Bragg, NC, USA

Jamie Golden Department of Surgery, Keck School of Medicine of the University of Southern California, Division of Pediatric Surgery, Children's Hospital Los Angeles, Los Angeles, CA, USA

Eduardo Gonzalez Department of Surgery, University of Colorado, Denver, CO, USA

Daniel Grabo Department of Trauma, Acute Care Surgery and Surgical Critical Care, West Virginia University, Morgantown, WV, USA

Daniel L. Gramins Division of Cardiovascular & Thoracic Surgery, Sulpizio Cardiovascular Center, UC San Diego Health System, La Jolla, CA, USA

Amar Gupta Department of General Surgery, Foothills Medical Center, Calgary, AB, Canada

Adil H. Haider Center for Surgery and Public Health (CSPH), Brigham and Women's Hospital, Boston, MA, USA

David A. Hampton R Adams Cowley Shock Trauma Center, University of Maryland Medical Center, Baltimore, MD, USA

Wendy F. Hansen Department of Obstetrics and Gynecology, University of Kentucky College of Medicine, Lexington, KY, USA

Alyssa Fitzpatrick Harlow Center for Surgery and Public Health (CSPH), Brigham and Women's Hospital, Boston, MA, USA

Elliott R. Haut Department of Surgery, Anesthesiology/Critical Care Medicine (ACCM), and Emergency Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Johns Hopkins University Bloomberg School of Public Health, Baltimore, MD, USA

Thomas J. Herron Department of Surgery, University of South Florida College of Medicine, Tampa, FL, USA

Michael Hernon Department of Surgery, Boston University School of Medicine, Boston Medical Center, Boston, MA, USA

Sameer A. Hirji Department of Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

Athena Hobbs Department of Clinical Pharmacy, Baptist Memorial Health Care & The University of Tennessee Health Science Center – Memphis, Memphis, TN, USA

Emily K. Hodge Department of Pharmacy, Dell Seton Medical Center at the University of Texas, Austin, TX, USA

John B. Holcomb Department of Surgery, McGovern Medical School, University of Texas Health Science Center at Houston (UTHealth), Houston, TX, USA

Cindy H. Hsu Department of Emergency Medicine and Surgery, University of Michigan, Ann Arbor, MI, USA

Aaron R. Jensen Department of Surgery, Keck School of Medicine of the University of Southern California, Division of Pediatric Surgery, Children's Hospital Los Angeles, Los Angeles, CA, USA

Christian Jones Department of Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Bellal Joseph Department of Surgery, University of Arizona, Tucson, AZ, USA

Gregory J. Jurkovich Department of Surgery, Division of Trauma, UC Davis Health, Acute Care Surgery and Surgical Critical Care, Sacramento, CA, USA

Kyle J. Kalkwarf Department of Surgery, McGovern Medical School, University of Texas Health Science Center at Houston (UTHealth), Houston, TX, USA

George Kasotakis Department of Surgery, Boston University School of Medicine, Boston Medical Center, Boston, MA, USA

Andrew S. Kaufman General Surgery Resident, Department of Surgery, Walter Reed National Military Medical Center, Bethesda, MD, USA

Edward Kelly Harvard Medical School, Division of Trauma, Burns, and Surgical Critical Care, Department of Surgery, Brigham and Women's Hospital, Boston, MA, USA

Uzer Khan Department of Surgery, Division of Trauma, Acute Care and Critical Care, Ruby Memorial Hospital, WVU Medicine, Morgantown, WV, USA

Andrew W. Kirkpatrick Regional Trauma Services, Departments of Surgery, Critical Care Medicine, University of Calgary, Calgary, AB, Canada

Lisa M. Kodadek Department of Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA

John Kuckelman Department of Surgery, Madigan Army Medical Center, Joint Base Lewis-McChord, Tacoma, WA, USA

Eric J. Ley Department of Surgery, Cedars-Sinai Medical Center, Los Angeles, CA, USA

George Paul Liao Department of Surgery, Division of Acute Care Surgery and Surgical Critical Care, Cedars-Sinai Medical Center, Los Angeles, CA, USA

Tarek Madni Department of Surgery, Division of Burns, Trauma, and Critical care, University of Texas Southwestern, Dallas, TX, USA

Darren J. Malinoski Department of Surgery, Oregon Health & Science University, Portland, OR, USA

Daniel Margulies Section of Trauma, Emergency Surgery & Surgical Intensive Care and Professor of Surgery, Cedars-Sinai Medical Center, Los Angeles, CA, USA

University of California at Los Angeles David Geffen School of Medicine, Los Angeles, CA, USA

Matthew J. Martin Trauma and Emergency Surgery Service, Legacy Emanuel Medical Center, Portland, OR, USA

Robert G. Martindale Department of Surgery, Oregon Health & Science University, Portland, OR, USA

Maher M. Matar Department of Surgery, Division of General Surgery, The Ottawa Hospital, Ottawa, Ontario, Canada

Paul B. McBeth Departments of Surgery and Critical Care Medicine, Foothills Medical Centre, Calgary, AB, Canada

Nicolas Melo Department of Surgery, Division of Acute Care Surgery, Cedars-Sinai Medical Center, Los Angeles, CA, USA

Maureen Mercante Department of Surgery – Surgical Critical Care, Christiana Care Health System, Newark, DE, USA

Ernest E. Moore Department of Surgery, University of Colorado Denver, Denver, CO, USA

Department of Surgery, Denver Health, Denver, CO, USA

Hunter B. Moore Department of Surgery, University of Colorado, Denver, CO, USA

Filip Moshkovsky Department of Surgery – Surgical Critical Care, Christiana Care Health System, Newark, DE, USA

Philip S. Mullenix Division of Cardiothoracic Surgery, Department of Surgery, Walter Reed National Military Medical Center, Bethesda, MD, USA

Stephanie Nitzschke Department of Trauma, Burn, Surgical Critical Care & Emergency General Surgery, Brigham and Women's Hospital, Boston, MA, USA

Kathleen O'Connell Department of Surgery, University of Washington, Seattle, WA, USA

E. Charles Osterberg University of Texas – Dell Medical School, Austin, TX, USA

Douglas R. Oyler Department of Pharmacy, University of Kentucky Chandler Medical Center, Lexington, KY, USA

Erin Palm Los Angeles County, University of Southern California Medical Center, Los Angeles, CA, USA

Caroline Park Department of Acute Care Surgery, LAC-USC Medical Center, Los Angeles, CA, USA

Charles S. Parsons Department of Surgery, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA

Dave D. Paskar Divisions of General Surgery Trauma & Critical Care Medicine, University of Toronto, Toronto, ON, Canada

Danielle Pigneri Department of Surgery, Division of Trauma, UC Davis Health, Sacramento, CA, USA

Priya S. Prakash Department of Surgery, Division of Trauma and Acute Care Surgery, Pritzker School of Medicine, University of Chicago, Chicago, IL, USA

R. Lawrence Reed II Revenue Cycle Services, IU Health, Department of Surgery, IU Health Methodist Hospital, Indianapolis, IN, USA

Trista D. Reid Division of General and Acute Care Surgery, UNC Department of Surgery, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Patrick M. Reilly Department of Surgery, Division of Trauma, Surgical Critical Care, and Emergency Surgery, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, USA

Peter Rhee Division of Acute Care Surgery, Department of Surgery, Grady Memorial Hospital, Atlanta, GA, USA

Sandro B. Rizoli Department of Trauma & Acute Care Surgery, St. Michael's Hospital, University of Toronto, Toronto, ON, Canada

Dusten T. Rose Department of Pharmacy, Dell Seton Medical Center at the University of Texas, Austin, TX, USA

Evan Ross Department of Surgery, The University of Texas Medical Branch, Galveston, TX, USA

Javid Sadjadi Department of Surgery, University of California San Francisco East Bay, Oakland, CA, USA

Joseph V. Sakran Division of Acute Care Surgery, Emergency General Surgery, The Johns Hopkins University, Baltimore, MD, USA

Ali Salim Division of Trauma, Burn, Surgical Critical Care and Emergency General Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

Mitchell B. Sally Department of Surgery, Oregon Health & Science University, Portland, OR, USA

Pablo G. Sanchez University of Pittsburgh, Department of Cardiothoracic Surgery, Division of Lung Transplant and Lung Failure, University of Pittsburgh Medical Center, Associate Director of Lung Transplant and ECLS, Pittsburgh, PA, USA

Jack Sava Department of Trauma, MedStar Washington Hospital Center, Washington, DC, USA

Martin A. Schreiber Division of Trauma, Surgical Critical Care, and Acute Care Surgery, Oregon Health and Science University, Portland, OR, USA

Anupamaa Seshadri Department of Surgery, Brigham and Women's Hospital, Boston, MA, USA

David A. Spain Department of Surgery, Stanford University, Stanford, CA, USA

Nicole A. Stassen Department of Surgery, Acute Care Surgery Division, University of Rochester Medical Center, Rochester, NY, USA

Deborah M. Stein R Adams Cowley Shock Trauma Center, University of Maryland Medical Center, Baltimore, MD, USA

Ian J. Stewart Combat Casualty Care Research, Clinical Investigation Facility, David Grant USAF Medical Center, Travis AFB, CA, USA

Aaron Strumwasser Los Angeles County, University of Southern California Medical Center, Los Angeles, CA, USA

Sharven Taghavi Department of Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

James M. Tatum Department of Surgery, Cedars-Sinai Medical Center, Los Angeles, CA, USA

Pedro G. R. Teixeira Department of Surgery and Perioperative Care, University of Texas at Austin, Dell Medical School, Austin, TX, USA

Marc D. Trust Department of Surgery and Perioperative Care, University of Texas at Austin, Dell Medical School, Austin, TX, USA

Jeffrey S. Upperman Department of Surgery, Keck School of Medicine of the University of Southern California, Division of Pediatric Surgery, Children's Hospital Los Angeles, Los Angeles, CA, USA

Tarsicio Uribe-Leitz Center for Surgery and Public Health (CSPH), Brigham and Women's Hospital, Boston, MA, USA

Gary Vercruysse Director of Emergency General Surgery, University of Michigan, Ann Arbor, MI, USA

Gregory P. Victorino Department of Surgery, University of California San Francisco East Bay, Oakland, CA, USA

Elizabeth Warnack Department of Surgery, Bellevue Hospital Center, New York, NY, USA

Division of Trauma and Surgical Critical Care, New York University School of Medicine, New York, NY, USA

Alison Wilson WVU Critical Care and Trauma Institute, Department of Surgery, Ruby Memorial Hospital, WVU Medicine, Morgantown, WV, USA

Franz S. Yanagawa Department of Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Daniel Dante Yeh Department of Surgery, Ryder Trauma Center, Miami, FL, USA

Traumatic Brain Injury

1

Asad Azim and Bellal Joseph

Introduction

Traumatic brain injury (TBI) is a non-degenerative, non-congenital disruption of brain function from an external force that leads to a permanent or a temporary impairment of cognitive and/or physical functions—it may or may not be associated with a diminished or altered state of consciousness. The external forces that create the injury may be the result of a variety of insults, including acceleration or deceleration, compression, penetrating objects, and complex mechanisms like blast injuries. TBI is the leading cause of death and disability among trauma patients. According to an estimate, about 2.5 million TBIs occur every year. Of those, about 50,000 people die, and approximately 80,000–90,000 survivors suffer severe lifelong neurological disabilities [1]. The external cause of injury ("mechanism of injury") associated with TBI varies with age and demographics. Males aged 0–4 have the highest rates of TBI-related visits, whereas adults aged 75 years and older have the highest rate of TBI-related hospitalizations and deaths (1). Falls are the leading mechanism of injury of TBI, accounting for 40% of all TBI-related emergency department (ED) visits (2). They cause more than half (55%) of all TBIs among children aged 0–14 years and 81% of all TBIs among adults aged 65 years and older. The second leading mechanism of injury is unintentional blunt trauma, accounting for 15% of all TBI-related ED visits (1). Motor vehicle collisions and assaults are the third and fourth leading mechanisms of injury, accounting for 14% and 10% of TBI-related ED visits, respectively [2].

A. Azim (✉) · B. Joseph
Department of Surgery, University of Arizona, Tucson, AZ, USA
e-mail: asadazim@surgery.arizona.edu

Types of Primary Injuries

Various types of primary TBI are summarized below.

- *Subdural Hematoma (SDH)*: SDH is the most common type of traumatic brain lesion and occurs in about 20–40% of severely head-injured patients. SDH originates in the space between the dura and the arachnoid matter of the meninges [3]. It results from damage and tearing of cortical bridging veins, which drain the cerebral cortical surface into the dural venous sinuses. The presentation can be acute, subacute, or chronic. Patients have variable loss of consciousness (LOC). On CT imaging, SDH appears to be crescent-shaped. It tends to be associated with underlying cerebral injury and thus usually has a poor prognosis [4].
- *Epidural Hematoma (EDH)*: EDH is a form of intracranial bleed between the dura mater and the inner table of the skull. It results from tearing of arterial dural vessels, i.e., middle meningeal artery. The most common site is temporal, where the bone is very thin and susceptible to fracture. On CT imaging, EDH appears to be lenticular-shaped. EDH is usually due to skull injury rather than brain injury, although brain injury certainly can occur with them. Morbidity and mortality associated with EDH is primarily due to the mass effect from the hematoma, which, if left unchecked, can lead to brain herniation [5].
- *Subarachnoid Hemorrhage (SAH)*: SAH results from disruption of small pial vessels between the subarachnoid and the pia mater of the meninges. Trauma is the most common cause of SAH. Patients with traumatic SAH have 70% higher risk of developing cerebral contusion and 40% higher risk of developing subdural hematoma [6]. SAH is a marker of the severity of TBI. The positive predictive value of SAH (>1 cm) for poor outcome is 72–80%. On CT imaging, SAH appears as hyperattenuating material filling the subarachnoid space [7].
- *Intraparenchymal Hemorrhage (IPH)*: This is a form of intracerebral bleed in which there is bleeding within the brain parenchyma. IPH, along with cerebral edema, may

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disrupt and compress adjacent brain tissue, constituting an immediate medical emergency. On CT imaging, IPH appears as the accumulation of blood within different intracranial spaces, most commonly as a lobar hemorrhage [8].

- *Intraventricular Hemorrhage (IVH)*: IVH refers to bleeding into the ventricular system of the brain, where cerebrospinal fluid is produced and circulates toward the subarachnoid space. It commonly results from an intracerebral hemorrhage with ventricular reflex. On CT imaging, blood appears as hyper-dense material in the ventricles that is best seen in the occipital horns. Blood in ventricular system also predisposes these patients to post-traumatic hydrocephalus. IVH is also a marker of severity of injury and is associated with adverse outcomes [9].
- *Cerebral Contusion*: Contusion is bruising of brain tissue often caused by a blow to the head. When this happens, the blood-brain barrier loses its integrity, thereby creating a heterogeneous region. This type of lesion usually occurs in coup or contrecoup injuries. It manifests in cortical tissue and can be associated with multiple microhemorrhages and small vessels that leak into brain tissue. The most common regions of the brain affected are the frontal and anterior temporal lobes. Cerebral contusions often take 12–24 h to evolve and may be absent on an initial head CT scan [10].
- *Cerebral Concussion*: This is the most common type of TBI. It occurs with a head injury caused by acceleration/deceleration forces or contact forces. It can result in rapid-onset, short-lived impairment of neurological function that resolves spontaneously. Concussions are a clinical diagnosis as there are no CT scan findings associated with it. The key signs and symptoms of a concussion are confusion and amnesia [11].
- *Diffuse Axonal Injury (DAI)*: A DAI is the most common and devastating type of TBI, resulting from extensive damage to white matter tracts over a widespread area. This injury develops from traumatic shearing forces that occur when the head is rapidly accelerated or decelerated. DAI is commonly seen in motor vehicle collisions and shaken baby syndrome. The sites frequently involved in DAI are the frontal and the temporal lobes. CT imaging usually appears normal. Newer imaging modalities, such as diffusion tensor imaging, are more sensitive than a standard MRI for detecting a white matter tract injury [12].

Secondary Brain Injury

Secondary brain injury is a consequence of pathological processes set in motion at the time of primary insult. Mechanism behind secondary brain injury is complex. It is purposed that it is due to the liberation of proinflammatory cytokines and chemicals as result of primary injury that leads to cerebral edema neuronal death and disruption of the blood-brain barrier [13]. The common pathways that contribute to this damage are the liberation of excitatory amino acids, platelet-activating factors, and oxygen free radicals and ubiquitous nitric oxide radicals [14]. While little can be done to limit primary injury, the main goals of current TBI management strategies are targeted at limiting secondary brain injury. With recent advances and better understanding of cellular and biochemical functions, it has become more clear that inadequate blood flow and substrate delivery result in exacerbation of secondary injury [15]. Hence, ensuring adequate nutritional supply and avoiding hypoxia and hypotension can help limit secondary brain injury and enhance neuronal recovery [16].

Emergency Management

History and Physical Examination

A history and physical examination should be obtained, including the events preceding a trauma, a description of the actual event, and complete description of the patient's neurological status. History of medications as well as medications given in the prehospital setting should be determined. Special attention should be paid to medications with the ability to alter the neurological examination, including sedatives or psychopharmacologics, paralytics, atropine (for cardiac resuscitation), and other mydriatics (for evaluation of ocular trauma). Primary and secondary surveys should be performed thoroughly evaluating for systemic injuries. Open lacerations and a vigorous scalp hemorrhage may lead to hypovolemia.

Neurological Assessment

An accurate neurological examination is necessary in order to make a correct diagnosis as well as to plan appropriate treatment strategies. The exam may be limited or altered by age, language, sedative or paralytic medication, alcohol intoxication, or illicit drug abuse. It is crucial to monitor trends that appear in neurological examinations overtime because they fluctuate based on the patient's improving or declining condition. The accuracy and completeness of a neurological exam is based on the alertness and cooperativeness of the patient. The extent of the examination must be tailored to each patient's neurological ability.

- *Pupillary Response*: Documenting pupillary abnormality is important, and it has a high diagnostic and prognostic utility [17]. Pupillary asymmetry is defined as a difference of >1 mm between the pupils. A dilated pupil is defined as a diameter of a pupil >4 mm. A fixed pupil shows no response to bright light. Orbital trauma,

hypotension, and hypoxia are common causes of pupillary dilation. Hypoxia and hypotension should be corrected before herniation can be excluded as a cause of pupillary dilation. Orbital trauma can be ruled out by using direct and consensual response for each pupil.

- *Glasgow Coma Scale (GCS)*: An important component of a primary survey is to obtain an accurate GCS. It has become the standard for the objective measurement of the severity of a TBI. A GCS assesses a patient's neurological status based on three components: motor function, verbalization, and eye opening (Table 1.1). A patient who is neurologically intact can receive a maximum score of 15, and the most severely injured patient can get a minimum score of 3. If the patient is intubated, the verbal component is given a score of "q," and the overall score is annotated with a "T." A GCS 13–15 defines a mild TBI—such patients are usually awake and have no focal deficits. A GCS 9–12 is considered a moderate TBI, in which patients have altered sensorium and focal neurological deficits. Patients with a GCS 3–8 have a severe TBI. Usually, they will not follow commands, and they fit the criteria of comatose state [17].

Airway, Breathing, and Circulation

Clinicians should adhere to the basic principles of trauma resuscitation, including rapid assessment and maintenance of an airway, breathing, and circulation [18]. The maintenance of an unobstructed and clear airway is of the utmost importance as hypoxia is the most critical factor leading to adverse outcomes in TBI patients. A multicenter trial has shown that mortality rises by 17% in patients that experience hypoxic episodes following a TBI [19]. Regarding patients with a GCS <9, guidelines recommend that skilled personnel should intubate them by rapid sequence induction. During intubation, the cervical spine should be considered injured until proven otherwise, and it must be protected.

Once the airway is secured, the patient must be ventilated appropriately to maintain normocarbia ($PaCO_2$ 35–40 mmHg). Monitoring of oxygen saturation and capnography is recommended in severely injured patients to avoid unrecognized hypoxemia or changes in ventilation. A study of 11,000 TBI patients showed that both hypo- and hypercarbia were associated with increased mortality in TBI patients [20]. In patients with signs of brain herniation, transient hyperventilation may be an option.

Hypotension is a major secondary brain insult. Studies have shown that even a single episode of hypotension is associated with a dramatic increase in mortality in TBI patients [21]. It should be treated with appropriate fluid resuscitation and blood products to achieve euvolemia. Recent studies have shown that maintaining systolic blood pressure above 100 mmHg is associated with decreased mortality and better neurological outcomes in TBI patients [22].

Table 1.1 Glasgow Coma Scale

Score	Motor	Verbal	Eye opening
6	Obeys command	–	–
5	Localizes to pain	Oriented	–
4	Withdraws to pain	Confused	Spontaneously
3	Flexes arm	Words/phrases	To voice
2	Extends arm	Makes sounds	To pain
1	No response	No response	Remain closed

Radiological Assessment

Computed Axial Tomography (CT) Scan CT scan remains the investigation of choice for patients presenting with head trauma. In a single, rapid pass, without patient repositioning, scans of the head, neck, chest, abdomen, and pelvis can be performed. Additionally, administration of contrast also allows for a CT angiogram reconstruction in order to evaluate vasculature of the head and neck. CT scan findings after trauma include SDH, EDH, SAH, IPH, IVH, contusions, hydrocephalus, cerebral edema or anoxia, skull fractures, ischemic/infarction (if >12 h old), and mass effect resulting in midline shift. Indications for an initial posttraumatic head CT scan include GCS ≤14, unresponsiveness, focal deficit, amnesia for the injury, altered mental status, and signs of basilar skull fracture [23].

Magnetic Resonance Imaging (MRI) MRI scans have better parenchymal resolution and can evaluate infarction, ischemia, edema, and DAI. An MRI is also helpful to determine a ligamentous injury of the spine or a traumatic cord injury. It is generally performed after the initial trauma evaluation and resuscitation have been completed. MRIs have limited availability, slower image acquisition time, image interference by monitoring devices, and a greater cost. Although their use in the initial assessment of trauma is not routinely recommended because intracranial surgical lesions seen on an MRI can also be identified on a CT scan [24], their use in the ICU setting can play a crucial role in evaluating DAI.

Intensive Care Unit Management

Monitoring

Blood Pressure Systolic blood pressure (SBP) plays a critical role in a secondary brain injury cascade after a severe TBI. TBI patients admitted with a systolic blood pressure of less than 85 mmHg have mortality rates as high as 35%, compared to only 6% in patients with a higher SBP [19]. Autoregulatory vasodilation plays a critical role in maintaining cerebral perfusion. After disruption of cerebral

autoregulation, which is a common event following severe TBI, cerebral perfusion relies on SBP. Hence, a low SBP leads to cerebral ischemia, which is recognized as the single most important secondary insult. In order to decrease mortality and improve clinical outcomes following a TBI, SBP should be maintained at ≥100 mmHg for patients 50–69 years old or at ≥110 mmHg for patients 15–49 years old or over 70 years old (5).

Intracranial Pressure (ICP) The concept of intracranial pressure is based on the Monro-Kellie hypothesis. Assuming that the skull is a closed space, the hypothesis states that there is a balance between brain, blood volume, and CSF. Increase in the volume of one constituent (e.g., cerebral edema) or an addition of a constituent (i.e., hemorrhage or tumor) mandates a compensatory decrease in other constituents in order to maintain ICP. The management of raised ICP varies greatly in clinical practice, and there are inconsistent reports about the utility of ICP monitoring on clinical outcomes and survival of TBI patients. According to the recently updated Brain Trauma Foundation (BTF 4th Edition 2016) guidelines, ICP monitoring should be performed in all salvageable patients with severe TBI (GCS 3–8) and an abnormal head CT, a normal head CT scan with a SBP of ≤90 mmHg, posturing, or age ≥40 years [25]. Studies have shown that treating ICP above 22 mmHg is recommended to reduce overall mortality [26]. Moreover, management of severe TBI using information from ICP monitoring is associated with reduced in-hospital and 2-week post-injury mortality. A vast majority of patients with severe TBI meet the criteria for ICP monitoring based on these guidelines. However, only a small subset of these patients receives ICP monitoring based on institutional guidelines. A prospective multicenter controlled trial performed in Ecuador demonstrated that there is no difference in clinical outcomes in patients who underwent ICP monitoring compared to those who were managed with an established protocol of neuroimaging and clinical examination [27]. Medical management remains the standard of care for elevated ICP, with a possible role for ICP monitoring and operative intervention in a subset of patients. However, further studies are required to better define subset of patients requiring ICP monitoring.

Cerebral Perfusion Pressure Monitoring (CPP) A traumatically injured brain is at a high risk of a local cerebral ischemia around the area of primary insult as well as global ischemia due to loss of cerebral circulation. In such a situation, maintaining adequate cerebral perfusion is of prime importance. CPP is defined as the pressure gradient across the cerebral vascular bed between blood inflow and outflow. It is calculated as the difference between mean arterial pressure (MAP) and ICP. Studies have shown that a CPP of less than 50 mmHg is associated with a high risk of cerebral ischemia and secondary brain injury. The BTF guidelines recommend a target CPP value between 60 and 70 mmHg for improved survival and favorable outcomes [25]. TBI management includes CPP monitoring in the "bundle" of care; however, the impact of CPP-based management of TBI patients remains unclear. There is some evidence which suggests that the management of TBI patients' using information from CPP monitoring is associated with 2-week post-injury mortality.

Treatment

Hyperosmolar Therapy

An injured brain is highly susceptible to secondary ischemia from either systemic hypotension or diminished cerebral perfusion (attributable to intracranial hypertension, cerebral edema, and inflammation). The objective of hemodynamic therapy in TBI is to ensure adequate brain perfusion and to keep intracranial pressure within normal limits. There are various methods for controlling ICP; however, one of the key pharmacological interventions is hyperosmolar therapy [24–29]. Such therapies reduce ICP by two distinct methods. One commonly accepted mechanism is via establishment of an osmolar gradient across the blood-brain barrier, with the gradient favoring the flow into the circulation. Another mechanism, which explains the rather more rapid action of osmolar agents, is improvement in the rheology of the blood due to plasma expansion as well as decreased hematocrit, which leads to decreased viscosity and more efficient cerebral blood flow (CBF). It is believed that the two most commonly utilized hyperosmolar agents, that is, hypertonic saline and mannitol, utilize both mechanisms [29].

- *Mannitol*: Mannitol is a naturally occurring sugar alcohol used clinically for its osmotic diuretic properties. It has been accepted as an effective tool for reducing intracranial pressure. Although there has never been a randomized comparison of mannitol with a placebo, both the BTF and the European Brain Injury Consortium identify level II and III evidence to support its use for the treatment of intracranial hypertension after a TBI. Mannitol can be administered as a bolus in response to raised ICP or as a continuous drip in a prophylactic fashion [29]. Studies have shown that bolus infusion is superior to continuous therapy; however, a difference of opinion still exists concerning the two modes of administration. Although mannitol plays a vital role in controlling ICP in severe TBI patients, its eventual diuretic effect is undesirable in hypotensive patients, and appropriate monitoring and aggressive fluid resuscitation are required to replenish fluid loss and to maintain SBP within target limits. Clinicians should be cautious, however, because mannitol