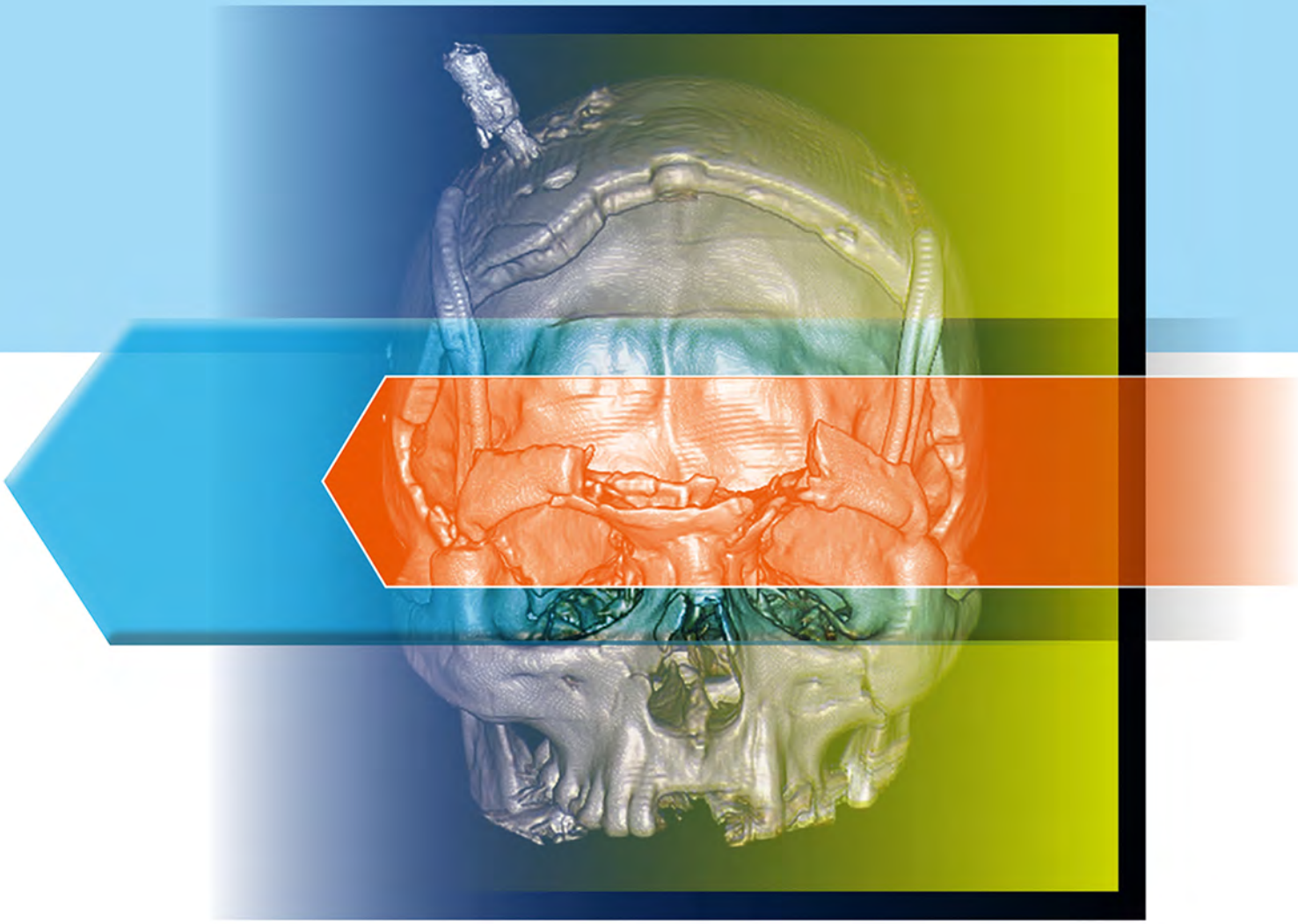
Neurotrauma and Critical Care of the Brain

Second Edition

Jack Jallo
Christopher M. Loftus



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Neurotrauma and Critical Care of the Brain

Second Edition

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107 illustrations

Thieme
New York • Stuttgart • Delhi • Rio de Janeiro

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Library of Congress Cataloging-in-Publication Data

Names: Jallo, Jack, editor. | Loftus, Christopher M., editor.
Title: Neurotrauma and critical care of the brain / [edited by] Jack Jallo, Christopher M. Loftus.
Description: Second edition. | New York : Thieme, [2018] | Includes bibliographical references and index.
Identifiers: LCCN 2018008641| ISBN 9781626233362 (print) | ISBN 9781626233409 (eISBN)
Subjects: | MESH: Brain Injuries, Traumatic–diagnosis | Brain Injuries, Traumatic–therapy | Critical Care–methods
Classification: LCC RC387.5 | NLM WL 354 | DDC 617.4/81044–dc23
LC record available at https://lccn.loc.gov/2018008641

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Thieme Publishers Rio de Janeiro, Thieme Publicações Ltda.
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Av. Nilo Peçanha, 50 – Sala 2508
Rio de Janeiro 20020-906 Brasil
+55 21 3172-2297 / +55 21 3172-1896

Cover design: Thieme Publishing Group
Typesetting by DiTech Process Solutions

Printed in The United States of America by
King Printing Company, Inc.

5 4 3 2 1

ISBN 978-1-62623-336-2

Also available as an e-book:
eISBN 978-1-62623-340-9

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Foreword

There is no greater pleasure for an academic than to see his student follow in his footsteps and ultimately to surpass him (I must admit some mixed feelings about the latter!). I am therefore delighted to have the privilege of writing this brief foreword to a book that my former resident Jack Jallo, MD, has co-edited with Chris Loftus, MD. This book brings together many of the current thought leaders in the field of traumatic brain injury and by doing so provides us with an easy-to-access and valuable resource.

While it is true that we do not yet have a single agent that has been proven to improve the outcome from traumatic brain injury, there is little doubt that the outcomes from this common and often devastating condition have improved substantially over the past three decades. In the 1970s the mortality associated with severe TBI—even treated in some of the best centers—was approximately 50 percent. Several current series report mortalities of 30 percent or less. Furthermore, the quality of neurologic recovery among the survivors is also better.

These dramatic improvements can only be ascribed to a combination of factors, including the introduction of seat belts and air bags, better rescue squads, more effective monitoring technologies, earlier CT scanning, prompt evacuation of intracranial hematomas, the growth of trauma centers, neurocritical care, and neurorehabilitation, and the effect of evidence-based management guidelines. It is highly unlikely that any single drug will exceed the cumulative effect of these diverse interventions. While it remains important to continue the search for agents that can modulate the many biochemical cascades that are set in motion by traumatic brain injury, it is important to use the many tools that we already have available to us.

The diverse disciplines that impact the care and outcome of the head-injured patient are concisely presented in this beautiful volume. It will no doubt serve as a very helpful starting point for the newcomer to the field, as well as a convenient source of up-to-date information for the seasoned neuro-traumatologist.

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Jallo and Loftus, Neurotrauma and Critical Care of the Brain, 2nd Ed. (ISBN 978-1-62623-336-2),

Preface

Brain and spinal cord injuries have devastating impacts on patients, their families, and our communities. As the ability to treat neurotrauma continues to improve, health care providers must focus not only on limiting the immediate damage of these complex injuries, but also on optimizing the long-term outcome for those affected by them.

An update of this text is necessary given the considerable advancements in the field of brain and spinal cord injury. Since the first edition published almost a decade ago, the guidelines for traumatic brain injury have been updated and significant research in the role of ICP management and decompressive craniectomy has been published. Additionally, there has been increasing emphasis on the role of critical care management in spinal cord injury.

This text is intended to serve as both a substantive and a rapid reference, as the information in each chapter is distilled into summarizing tables. We retained the book structure of the first edition; early chapters focus on the science underlying daily practices and acute care and critical care management, followed by chapters on nonacute care, outcomes, and socioeconomics. This edition retains the emphasis on critical care and further expands on this content. We also review the updated guideline recommendations.

It is our hope that this text will continue to serve as an important tool for all involved in the care of these patients, including bedside nurses, house staff, emergency physicians, intensivists, and surgeons. It is by our best efforts that these most vulnerable patients are best served.

Acknowledgments

In an undertaking such as this, there are many people to thank, as this is truly a collaborative effort. I wish to first thank all the contributors for their time and effort. Without them this text would not be possible. I understand that an undertaking such as this strains already busy schedules. I also want to acknowledge the staff at Thieme for their patience and support in making this text possible, especially Sarah Landis and Timothy Hiscock.

This endeavor would not be possible without the training and education provided me by many mentors over the years. I am forever indebted to them. Most importantly, none of this would be possible without the support of my family.

Thank you.

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Jallo and Loftus, Neurotrauma and Critical Care of the Brain, 2nd Ed. (ISBN 978-1-62623-336-2),

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Jallo and Loftus, Neurotrauma and Critical Care of the Brain, 2nd Ed. (ISBN 978-1-62623-336-2),

1 Brain Trauma and Critical Care: A Brief History

Nino Stocchetti and Tommaso Zoerle

Abstract
This chapter describes progressive changes in the medical and surgical approach to traumatic brain injury (TBI). First we illustrate the attempts to surgical treatment of blunt and penetrating head injuries caused by combats. During the First and Second World Wars, military medicine incorporated fundamental concepts, from early intervention to asepsis, that improved the discouraging results of delayed surgical treatment with intractable infections. Then we summarize improvements in central nervous system exploration, from intracranial pressure measurement (and then monitoring) to a more complete understanding of intracranial pathophysiology, as developed in neurosurgery, neuroanesthesia, and with revolutionary imaging tools such as the CT (computed tomography) scan. The birth of intensive care, based on supported ventilation, accurate and systematic monitoring, and specialized personnel, is described. Concurrently, renewed interest in TBI led to large, multicenter observational studies. These became possible when standardized scales for severity and outcome measurement were broadly used worldwide. The predominant nihilistic attitude toward the most severe cases changed when data on aggressive and tailored medical treatment, combined with neurosurgery, were published. These studies demonstrated the improvements in the outcome of TBI patients and set the standard for modern TBI management. This chapter describes how TBI care has evolved, with special focus on how critical care has become an integral part of TBI treatment.

Keywords: traumatic brain injury, critical care, neurosurgery, neuroradiology, history

1.1 Introduction

Today the clinical pathway for severe traumatic brain injury (TBI), from rescue to rehabilitation and discharge, seems straightforward. Normalization of perfusion and oxygenation, rapid transport to a neurotraumatologic center, identification and evacuation of intracranial masses, intracranial pressure (ICP) monitoring and treatment, early rehabilitation, etc., are considered standard, and supported by internationally approved guidelines (even if the published evidence is weak).[1]

The severe patient, suffering from a harsh insult to the brain, is managed in the intensive care unit (ICU) by a team of different specialists, using a sophisticated technological armamentarium for diagnosis (ultrasound, computed tomography [CT] scans, magnetic resonance imaging [MRI], etc.), monitoring (ICP, brain tissue oxygenation, microdialysis, hemodynamic support, etc.), and therapy (artificial ventilation, temperature management, artificial nutrition, etc.).

What appears standard today, however, has really only developed quite recently (in the last 50 years), and is still tumultuously evolving. This chapter describes how TBI care has evolved, with special focus on how critical care has become an integral part of TBI treatment.

This historical review is based mainly on references published in English. Contributions in other languages, especially if appearing in journals not listed in PubMed, may have been missed.

1.2 Brain Trauma and Military Surgery

TBI was a common problem during combat, and TBI treatment was the realm of military surgery for millennia. Skull fractures and impaired consciousness as consequences of trauma were described, and trepanation was performed, as part of Hippocratic medicine. Early interventions (within the first 3 days after injury) were recommended, with the aim of "exiting blood," most likely a form of hematoma evacuation.[2]

Penetrating brain injuries became extremely frequent with the introduction of firearms, and a structured approach to TBI was described at the end of the 18th century in a manual by a military surgeon in the revolutionary American army.[3] The *Plain Concise Practical Remarks on the Treatment of Wounds and Fractures*, published in 1775 by Dr. Jones, focused on scalp wounds and depressed skull fractures. The manual stressed the usefulness of early, or prophylactic, trephination. The algorithms presented in the manual were limited to a strictly surgical approach, even if symptoms related to brain damage, and particularly to concussion, were identified. In the absence of antiseptic measures, results were profoundly worsened by infectious complications.

A fundamental step forward was the identification of neurological symptoms, rather than skull fractures, as an indication for surgery. Percival Pott (1713–1788) was the first to state strongly that the neurological status, not just fractures, should be the indication for trephination.[4]

With time, military medicine incorporated the progress of anesthesia and surgery made in civilian life, including the development of neurosurgery as a separate specialty, at the beginning of the 20th century. Antisepsis was progressively, though not smoothly, accepted after Joseph Lister published "On the Antiseptic Principle in the Practice of Surgery" in 1867.[5]

During the First World War, pioneers of neurosurgery, such as Harvey Cushing, served in the British and U.S. armies, offering TBI patients the most advanced treatment available at the time. Adequate and definitive management was only possible in specialized hospitals, where anesthesia, blood pressure measurement, fluoroscopy, antisepsis, and high-quality surgery were provided by trained neurosurgeons. Mortality was reduced from 54 to 29%.[6,7]

During the Second World War, care for the injured was provided by a better organized care system, using standardized instrumentation, blood transfusions, improved anesthesia, and antisepsis. Specialized treatment for head injuries was promoted by the Oxford group led by Sir Hugh Cairns, who created mobile (motorized) neurosurgical units at the battle front. The

Jallo and Loftus, Neurotrauma and Critical Care of the Brain, 2nd Ed. (ISBN 978-1-62623-336-2),

first mobile unit was deployed in North Africa; ambulances evolved into "motorized operating theaters," providing prompter surgical care. Each unit was staffed by a neurosurgeon, a neurologist, and an anesthesiologist.[8]

The debate regarding the benefits of early versus delayed surgery was fierce, but evidence accumulated in favor of prompt treatment. Sir Hugh Cairns also contributed to TBI prevention by promoting the use of protective helmets for motorcycle dispatch riders. His research contributed to the use of crash helmets by both military and civilian motorcyclists.[9]

The experience accumulated during wartime led to the publication of large series of cases. Detailed analysis of complications after injury and surgery (infection, seizures, and neurological morbidity) was made available to the English-speaking scientific community. The body of knowledge accumulating for TBI treatment during the Second World War, and the obstacles to the free circulation of people and ideas, was among the reasons for the creation of the Journal of Neurosurgery. The first editorial note stated: "Since the outbreak of war in 1939, there has been less interchange between British and American neurosurgeons than before," motivating the publication of an English journal to improve communication of ideas and opinions.[10]

The main—or only—possible TBI treatment, however, was surgery. There were no specific therapies for TBI. A fatal outcome was expected for severe, comatose cases, while less severe patients were kept in a quiet, dark environment, to relieve headache. Luminal and morphine were used for restless cases.[11] Mortality was around 50% for severe patients, and the number of surviving veterans after TBI increased. Even after successful acute treatment, they required lengthy care before returning to normal life. The need for and the encouraging results of rehabilitation after injury became clear, thanks to the seminal work of Dr. Howard Kessler and others.[9]

1.3 Brain Trauma Since the Second World War (1945–1980)

Interest in TBI declined after the Second World War. The general feeling was that severe cases were not amenable to successful treatment, in a sort of self-fulfilling prophecy. Comatose patients were lying in hospitals, usually in the neurosurgical ward, with a clinical course, almost unavoidably fatal, involving hyperthermia, tachycardia, decerebrate posture, and pneumonia. Most of these features were felt to derive from brainstem herniation, and, as such, not treatable.

However, patients were ultimately dying because of respiratory failure, and the concept of preventing/treating respiratory complications was proposed by a few clinically focused surgeons. Prevention of vomiting and avoidance of oral feeding, for instance, were identified as useful and attainable goals. Then other targets were proposed: airways protection by tracheostomy and tracheal suction, attention to normal oxygenation, maintenance of fluid balance, sedation with a lytic cocktail (chlorpromazine, promethazine, pethidine, and levallorphan), and intravenous and enteral nutrition. This medical treatment was proposed in combination with "routine burr-holes, for excluding surface blood collections" in an article published in Lancet in 1958.[12] Maciver described 26 patients managed in Newcastle, United Kingdom, with this innovative approach: their mortality was 38%, compared to 70 to 77% of historical controls. Despite the promising results, however, these new ideas were not widely accepted, or applied. Still in 1964, the opinion of W. Ritchie Russell, an authoritative Oxford University neurologist, concerning TBI was very negative: "... already some completely hopeless cases are being kept alive, and nobody hopes for more success in that direction."[13]

This pessimistic attitude was challenged by sort of a trauma epidemic: with motorization, road traffic and road traffic accidents were increasing, accompanied by an overwhelming load of injuries, including severe TBI. Concomitantly, major changes were taking place in several areas: technological advances in intracranial diagnosis, the birth of intensive care with artificial respiratory support, ICP monitoring, and therapies for brain edema.

The most important change, however, was a shift in the medical community. A few innovators changed the overall approach to TBI, and established the principles that shape TBI therapy today, as described in the following sections.

1.4 Improvements in the Diagnosis of Intracranial Lesions

The possibility of imaging the intracranial vasculature by injecting radio-opaque contrast material into the brain vessels (brain angiography) was introduced in 1927 by the Portuguese neurologist Egas Moniz. Angiography could identify compression or displacement of the cerebral vasculature attributable to expanding hematomas, and greatly improved diagnostic capabilities. After the Second World War, several centers adopted this technique, with direct puncture of carotid and brachial arteries by neurosurgeons, who then interpreted the radiological findings. Gradually, a specialized branch of radiology devoted to the nervous system developed.

In October 1971, the first patient underwent a CT scan, heralding a revolution in imaging: masses compressing the brain became directly visible. For years, however, the machines were extremely rare and costly, restricted to major academic centers; as a consequence, the CT scan became widely used only in the 1980s.[14]

The standard diagnostic approach, until CT scans were adopted everywhere, was based on neurologic observation combined with skull X-ray, to exclude fractures, a fundamental risk factor for surgical expanding lesions. In case of fractures, closer observation and further diagnostic procedures were used, such as angiography if CT scan was not available. This approach made early detection, and earlier treatment of expanding intracranial lesions, possible.

1.5 Improvements in Pathophysiological Understanding: Cerebrospinal Fluid Pressure

The biological basis of ICP regulation, as a function of intracranial volumes, was described by the Scottish anatomist and

surgeon Alexander Monro (1733–1817) and his student George Kellie (1758–1829) in the late 18th century. The clinical symptoms related to elevated ICP were described in 1866 by Leyden, and this discovery disclosed high ICP (HICP) as a common consequence of various pathologies, including brain tumors and TBI.

Jonathan Hutchinson (1886), a senior surgeon for the London Hospital, made the important observation of ipsilateral pupillary dilatation with middle meningeal artery hemorrhage. The understanding of the localizing significance of neurological signs associated with compressive mass lesions increased remarkably.[6]

The central role of HICP as a cause of neurological worsening became evident in 1901 with the publication of the "Cushing triad" (bradycardia, systolic arterial hypertension with increased pulse pressure, irregular respiratory pattern), interpreted as a consequence of brain compression. More precisely, Jackson in 1922 identified brainstem compression as the cause of the Cushing findings.

In 1891, the first ICP measurements by lumbar puncture were published Quinke.

The lumbar puncture disclosed the risk of raised ICP after TBI but was not viable for continuous measurement and did not reflect the supratentorial pressure if the ventricular space was not communicating with the spinal subarachnoid space.

Continuous access to cerebrospinal fluid (CSF) was offered by external ventricular drainage (EVD). First performed in 1744 by Claude-Nicholas Le Cat, EVD was eventually introduced into clinical practice with a refined technique and better materials in 1960. The addition of manometry to the drain by Adson and Lillie in 1927 allowed accurate measurement of CSF pressure, opening up the possibility of continuous ICP recording.[15]

In 1951, in a French journal Guillaume and Janni reported their pioneering experience with continuous ICP measurement. In 1953, data on continuous ICP measurement in various pathologies was also published by Ryder in the United States.[16] In 1960, the Swedish neurosurgeon Nils Lundberg reported a large series of patients with brain tumors in whom ICP was monitored through EVD.

Then, the Lundberg experience on measuring ICP was extended to TBI patients, and his first publication on this topic described 30 cases successfully monitored in 1965.[17]

Control of ICP, with surgical and/or medical therapies, became a measurable and attainable target. Interest in this new parameter boomed, both in Europe and the United States. In 1972, Mario Brock and Herman Dietz, innovative German neurosurgeons, organized the first international ICP symposium in Hannover, Germany, where 64 papers were presented, both experimental and clinical.[18]

Two years later, 132 papers were submitted to the second symposium in Lund.

Together with accumulating clinical experience, a better theoretical understanding of ICP dynamics was gained from animal experiments (in Rhesus monkeys) by Thomas Langfitt. He demonstrated an exponential ICP rise in response to progressive additions of water to an intracranial balloon.[19] The ICP pressure-volume curve was further analyzed by Antony Marmarou, who published a model of the intracranial system that formed the basis for determining intracranial elastance.[20]

1.6 Medical Treatment of Raised Intracranial Pressure: Brain Edema

Brain swelling and water accumulation in the injured brain (edema) as causes of HICP were known to pathologists and neurosurgeons from direct observation. The only possible therapies, however, were limited: Quinke used repeated CSF lumbar taps to lower ICP, while Cushing promoted surgical decompression as a method for relieving the swollen brain.[6] In 1919, however, Weed explored the ICP response to different fluids in cats. Intravenous water infusion raised ICP (measured with manometry through the atlanto-occipital ligament), while hypertonic sodium lowered it. For the first time, a pharmacological treatment against brain edema was offered.[21] Temple Fay and colleagues in Philadelphia introduced hypertonic saline to reduce ICP in 1921, and reported its use in head trauma in 1935.[22]

After initial enthusiasm, however, the evidence that the beneficial effects of hypertonic solutions were short lasting, while side effects could be frequent and life-threatening (renal failure, cardiovascular complications, seizures), precluded the widespread adoption of osmotic therapies.

In 1954, urea was proposed as an anti-edema compound, based on experimental work on ICP in monkeys. Two years later, the first report on 26 patients treated with urea was published.[23] Urea, however, was difficult to prepare and store, not stable in solution, and caused venous irritation. After 1960, mannitol became the preferred osmotic drug.[22]

1.7 Improvements in Pathophysiological Understanding: Neuroanesthesia

The young Harvey Cushing, at that time a second-year medical student, was asked to administer ether to a patient, in preparation for surgery. The patient died before the surgical procedure began. This lesson was well taken; in promoting modern neurosurgery Cushing always stressed the importance of a skilled anesthesiologist at his side.[6]

Neurosurgery expanded dramatically after the Second World War, with new techniques, procedures, and equipment. Central to this expansion was highly specialized interest in neuroanesthesia, which required techniques for intraoperative control of brain swelling, using hyperventilation, negative end-expiratory pressure, and osmotic drugs. The delicate interaction of systemic hemodynamic and respiratory parameters with intracranial homeostasis had an immediate, sometimes dramatic, effect on the behavior of the brain exposed for tumor and vascular surgery. The cerebral vasoconstriction induced by hypocapnia, demonstrated in man by Gotoh in 1965,[24] had been used intraoperatively years before.[25] Hypothermia, first used for other indications in 1938, was used for brain aneurysm repair in the 1950s.[26]

In 1961, a group of U.S. anesthesiologists established the Commission on Neuroanesthesia, sponsored by the World Federation of Neurology; in 1965, a Neuroanesthesia Traveling Club of Great Britain and Ireland was founded. A large amount of knowledge accumulated rapidly. The first textbook of neuroanesthesia was published by Andrew Hunter in 1964.[27]

Close cooperation between neurosurgeons and anesthesiologists was obviously essential in the operating room. Interestingly, this cooperation extended to research and to the foundation of the first scientific associations. The concepts developed for intraoperative management could also be applied to the postoperative period. The study of CSF physiology, for instance, with a special focus on acid–base balance, was applied to comatose patients after surgical hematoma evacuation.[28] Hypothermia, hyperventilation, and hypothermia were soon tested for ICP control outside the operating room.

1.8 A Common Language and Large International Series

In the 1970s, special interest on head injury was cultivated in the Institute of Neurological Sciences in Glasgow, Scotland, by a group of brilliant neurosurgeons led by Brian Jennet. At a time of obscure, unstructured, and often confusing definitions (coma carus, decerebrate posture, etc.), a standardized, pragmatic approach to the neurological examination was needed. The Glasgow Coma Scale (GCS) was published in 1974, offering a simple complement to classic neurologic examination. This responsiveness scale was easy to use for monitoring trends, and to exchange information. Within 4 years, the GCS had been proposed worldwide for a standardized assessment in TBI. By assigning a number to each response for the three components of the scale (eye opening, verbal response, motor response), the patient's performance could be ranked, creating a GCS score.[29,30]

One year later, the Glasgow Outcome Scale summarized the possible outcome after injury in five broad, but clearly defined, categories.[31] A common language for evaluating severity and results thus became available, allowing larger studies among cooperating centers.

The first big data collection, with standardized terminology and classification, reported on 700 severe TBI cases (coma lasting at least 6 hours) in three countries (Scotland, Netherlands, and United States). Differences in the organization of care and in management details were documented, but with no differences in mortality (50% in each center). This finding could be interpreted in a rather nihilistic way, suggesting that the intensity or quality of care did not affect the outcomes across centers. This, however, was not the conclusion of the study.[32] On the contrary, the methodology developed for this international data collection was proposed for the critical appraisal of innovative, and potentially improved, methods of care.

In the United States in 1977, the National Institute of Neurological Disorders and Stroke started up a Traumatic Coma Data Bank (TCDB) with a pilot phase (581 patients) and a full phase (1,030 patients). The full phase started enrollment in 1984 and completed follow-up in 1988.[33] Mortality in closed head injury was 38%. Besides suggesting improved outcomes, this data collection allowed seminal observations on ICP, CT scan classification, outcome determinants, etc.[34,35,36,37]

1.9 The Birth of Intensive Care Medicine

Difficult postoperative cases have been of concern from the beginning of modern neurosurgery. Dandy in 1932, at Johns Hopkins Hospital, concentrated the sicker neurosurgical patients in a special three-bed unit where more observation and care could be provided. However, not much therapy was available; in particular no means to support ventilation or perfusion.

Artificial positive pressure ventilation through tracheotomy for respiratory support was probably first attempted in the 1940s, by a Danish physician named Clemmesen, for treating patients with barbiturate poisoning. This concept, however, was applied largely in Copenhagen, Denmark, during and after the poliomyelitis epidemic in 1952/1953. Thanks to the intuition of a young anesthesiologist, Bjorn Ibsen, mortality was impressively reduced (from 92 to 25%) by protecting the airways with tracheostomy and supporting ventilation, using rubber bags squeezed by volunteering medical students.[38]

In 1948, machines delivering intermittent positive pressure had already been used in Los Angeles for polio patients by Albert Bower, working with the biomedical engineer Ray Bennett. These machines were first used to supplement intermittent negative pressure "iron lungs," and then went through a complex process of technical refinement. Data on this approach to polio were published in 1950, and was known by Ibsen who, however, resorted to manual ventilation. Over the next few years, the first artificial positive pressure ventilators entered the market.[39]

It is important to note that mortality was reduced not only by ventilatory support but also through a structured approach. Systematic data collection of arterial pressure and other physiologic data, an embryonal monitoring system, was implemented; sedation or anesthesia with barbiturates was used to facilitate ventilation and bronchial suction; continuous, skilled nursing was maintained around the clock.[40]

Indications for intensive treatment exploded rapidly, outside the polio epidemic. Trauma, hemorrhagic shock, tetanus, various forms of respiratory failure, intoxications, etc., were all indications for intensive care unit (ICU) admission.[41] General ICUs were opened in all major hospitals in the 1950 to 1960s. The specific organization of each ICU, and its staffing, depended on the local situation. In London, an ICU to treat patients with neuromuscular diseases was opened in 1954. The Mayo Neuroscience ICU opened in 1958 with combined neurosurgical and neurological expertise. A cooperative effort by neurologists, anesthesiologists, and neurosurgeons led to the neurologic/neurosurgical ICU at the Massachusetts General Hospital in Boston.

The body of knowledge related to the specific problems of neuro-ICU accumulated rapidly. The first textbook on neurocritical care (entitled "Neurological and Neurosurgical Intensive Care") was published by Alan Ropper and Sean Kennedy in 1983. The journal Critical Care Medicine hosted a permanent neurocritical care section in 1993; 2 years later, the Society of Critical Care Medicine established a neuroscience section. In

2002, the Neurocritical Care Society was founded in San Francisco by a small group of neurointensivists. In 6 years, the Neurocritical Care Society gained nearly 1,000 members from around the world.

1.10 Aggressive Surgical and Medical Care for Head Injured Patients

In 1972, Donald Becker, a young neurosurgeon in Richmond, VA, challenged the concept that therapy could not substantially influence outcomes after severe TBI. He managed all severe TBI in his institution with a combination of surgical and medical treatment. Milestones were early diagnosis of surgical masses, ICP monitoring and therapy, artificial ventilation, sedation, and normothermia. CT scans became available only in the last 9 months of this 4-year study. Previously diagnosis was based on pneumoencephalography and/or angiography. Mortality in the first 160 patients was 30%, with an impressive rate (60%) of favorable outcomes.[42]

The findings from the first international data collection in three countries,[31] where therapy seemed relatively unimportant, were strongly questioned. No direct comparison was possible—the patients in Richmond had different baseline characteristics, and were younger, for instance—but aggressive treatment in the ICU seemed beneficial even for the most severe cases, lowering mortality without increasing permanent severe disability or vegetative status. The basic hypothesis of this work was that secondary brain damage played an important role in worsening outcome, and that this secondary damage could be prevented or attenuated by intensive medical treatment. The initial data were reinforced in a second series of 225 cases published by the Richmond group in 1981.[43]

The strategy of a combined (surgical and medical) approach to intracranial hypertension was advocated by H. Shapiro before the Richmond paper, but without specific reference to TBI. His concept was that appropriate monitoring and treatment could only be provided in a specialized ICU, like the neuro-ICU he was directing in Philadephia.[44]

In 1979, L. Marshall in San Diego published his results on 100 severe TBI, confirming 60% of favorable outcomes at 3 months. Prevention and treatment of medical complications in the ICU was acknowledged as a plausible explanation for these positive results.[45] There were concerns about this approach, however, because ICU was costly, beds were limited, and futile therapies could improve survival but at the expense of prolonged and severe disability.[46] Despite opposition, however, in the next few years a paradigm of intensive treatment, centered on respiratory and hemodynamic support, ICP monitoring and therapy, temperature control, early nutrition and physiotherapy, etc., became standard for TBI.

A systematic review of the literature documents an impressive reduction in mortality from 1970 to 1990, probably connected with ICP monitoring and aggressive intensive care.[13]

1.11 Lessons Learned, and New Problems

TBI research has expanded impressively, with more than 87,000 articles on the subject listed in PubMed (search "Traumatic Brain Injury," August 2016). There were more than 27,000 articles on ICP in the same database at the same date. Almost 1,000 articles on ICP have been published yearly in the last 5 years. New challenges, such as blast injuries, are emerging.[47]

TBI treatment has changed dramatically in the last 50 years, moving from pioneer experiments to an accepted standard, as indicated in international guidelines.[1] These specify the prevention and correction of secondary insults during TBI acute treatment, which require an intensity of monitoring and therapy that can only be achieved in an ICU. While the usefulness of single modalities, such as ICP monitoring, or interventions like hypothermia has been questioned, the concept that severe TBI must be treated in the ICU is universally accepted.[48,49]

The modern neuro-ICU can call on a wide range of monitoring technologies, integrated in multimodal systems, and requires the cooperation of experts from several different fields (intensivists, anesthesiologists, neurosurgeons, neuroradiologists, bioengineers, computer specialists, physicists, etc.).

The backbone of intensive care, however, remains the diligent work at the bedside by skilled nurses and dedicated doctors, applying all technological advances wisely to achieve goals, such as adequate brain perfusion and oxygenation, identified in the last two centuries, but made measurable in the last few decades.

The main lesson of this brief historical review is that every single step forward very often resulted from the patient work of many people, intelligently understood and applied by a few pioneers.

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2 The Epidemiology of Traumatic Brain Injury in the United States and the World

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Abstract

Although traumatic brain injury (TBI) is a major cause of death and disability worldwide, quality epidemiological data that may allow us to compare findings or to fully understand the multiple factors that contribute to this preventable condition are scarce or lacking. A systematic review of the European TBI literature found that the combined rate of TBI hospitalization and death in the 23 countries that met the inclusion criteria was approximately 235 per 100,000. The authors also found that it was difficult to reach consensus on all epidemiological findings across the studies because of critical differences in methods employed in the reports. In the United States, the Centers for Disease Control and Prevention (CDC) has reported that the total combined rate for TBI-related emergency department (ED) visits, hospitalizations, and deaths has reached 823.7 per 100,000 (available at http://www.cdc.gov/traumaticbraininjury/index.html).

In this chapter, we intend to describe the current epidemiology and prevention of TBI in the United States and the world. For this purpose, we have used publicly available data disseminated by the CDC and researchers worldwide.

Keywords: traumatic brain injury, head injury, epidemiology, prevention, review, incidence, prevalence, severity, external cause, outcomes

2.1 Introduction

Preventing traumatic brain injury (TBI) worldwide requires that public and clinical health practitioners and partners have standard clinical and epidemiological definitions and a clear understanding of the factors that contribute to this condition. Data on these factors, however are are scarce or lacking.[1,2,3]

2.2 Definition

Even in 2016, no universally accepted standard definition for TBI exists. For diagnostic purposes, clinicians use a constellation of signs and symptoms as well as laboratory and imaging criteria to identify cases of TBI. Other researchers, including epidemiologists, operationalize these clinical definitions to identify cases of TBI from databases coded using codes of the International Classification of Disease (ICD).[4]

2.2.1 Clinical Definition

According to the Common Data Elements (CDE) Project, TBI is an alteration in brain function, or other evidence of brain pathology, caused by an external force (described at https://www.commondataelements.ninds.nih.gov/tbi.aspx#tab=Data_Standards). Examples of these forces include blows, falls, sudden acceleration or deceleration of the head, and blast waves resulting from explosions. The CDE project is an international effort to develop a common definition and datasets for TBI research so that information is consistently captured and recorded across studies.

Brain injuries range from mild TBI or concussion to coma and even death. Mild TBI or concussion presents with headache, confusion, dizziness, poor concentration, disorientation, nausea/vomiting, disturbances of hearing or vision, loss of memory (often limited to the timeframe immediately surrounding the injury), lethargy, impairment or loss of consciousness (LOC) for ≤ 30 minutes, or seizures.[4,5] These symptoms may be transient, and their absence at the time of examination does not rule out TBI. Thus, patient history is a critical component of diagnosis.[1,2,4,5] Objective signs of TBI include skull fractures, neurologic abnormalities, altered consciousness, or intracranial lesions.[1,2,4,5,6]

2.2.2 International Classification of Disease-Based Definitions

To track TBI, Centers for Disease Control and Prevention (CDC) mainly relies on ICD-coded vital statistics and on administrative/billing records (▶ Table 2.1, ▶ Table 2.2, ▶ Table 2.3) issued for services rendered to patients in medical facilities.[7,8,9] These definitions are imperfect, but their usefulness for research and surveillance purposes warrant their inclusion into even the most sophisticated classification systems.[7,10,11,12]

ICD-9-CM (ICD, Ninth Revision, Clinical Modification)-Based TBI Morbidity Definition

From 1995 to October 2015, researchers in the United States have used a CDC definition based on ICD-9-CM codes to identify cases of TBI from ICD-9-CM-coded medical administrative/billing databases[7,8,9,13] (▶ Table 2.1). Injury mechanism (e.g., falls), location of injury (e.g., home), and intentionality of the injury

Table 2.1 Centers for Disease Control and Prevention (CDC) ICD-9-CM-based surveillance definition for traumatic brain injury (TBI) related morbidity

ICD-9-CM Code	Description
800.0–801.9	Fracture of the vault or base of the skull
803.0–804.9	Other and unqualified multiple fractures of the skull
850.0–854.1	Intracranial injury, including concussion, contusion, laceration, and hemorrhage
950.1–950.3	Injury to optic nerve and pathways
995.55	Shaken infant syndrome
959.01	Head injury, unspecified

Source: Marr and Coronado 2004.[7]

Table 2.2 Proposed Centers for Disease Control and Prevention (CDC) ICD-10-CM surveillance definition for traumatic brain injury (TBI) morbidity

ICD-10-CM code	Description
S02.0, S02.1–[a]	Fracture of skull
S02.8, S02.91	Fracture of other specified skull and facial bones; unspecified fracture of skull
S04.02, S04.03–, S04.04–	Injury of optic chiasm; injury of optic tract and pathways; injury of visual cortex
S06–	Intracranial injury
S07.1	Crushing injury of skull
T74.4	Shaken infant syndrome

Source: A surveillance case definition for traumatic brain injury using ICD-10-CM. National Association of State Head Injury Administrators (NASHIA). Webinar, September 17, 2015. Available at: https://www.nashia.org/pdf/surveillance_tbi_case_definition_23Sep2015_cleared.pdf.
[a]"–" indicates any fourth, fifth, or sixth character. Seventh character of A or B for S02.0, S02.1–, S02.8, and S02.91. Seventh character of A for S04.02, S04.03–, S04.04–, S06–, S07.1, and T74.4

can also be determined using ICD-9-CM's external cause of injury codes or E-codes. CDC has defined a set of E-code groupings to standardize reporting of those external causes.[7,14]

ICD-10-CM-Based TBI-Related Morbidity Definition

The use of ICD-10-CM has been required in the United States since October 2015.[15,16,17] This update contains approximately five times as many diagnostic codes as the ICD-9-CM system.[13,15,16,17] CDC's TBI Surveillance Definition Workgroup led by Victor Coronado developed an ICD-10-CM-based definition[16] to be used in the United States (▶ Table 2.3).

ICD-10-CM includes greater detail than the comparable ICD-9-CM codes. For example, code S06.8 includes codes for injuries to the intracranial portion of the internal carotid artery, more categories for describing loss of consciousness, etc. To ease the ICD-9-CM to ICD-10-CM transition, CDC has prepared general equivalence maps (GEMs) and a code-to-code reference dictionary for ICD-9-CM and ICD-10-CM[16] (available at http://www.cdc.gov/nchs/icd/icd10cm.htm).

ICD-9-CM to ICD-10-CM Transition Challenges

The implementation of the proposed ICD-10-CM TBI definition poses some challenges. For example, this process should evaluate the sensitivity, positive predictive value, and the impact of excluding ICD-10 CM code S09.90 (*unspecified injury of head*), which is the equivalent to ICD-9-CM code 959.01 (*head injury unspecified*), one of the most commonly reported TBI ICD codes in the United States since its implementation in 1999. The criteria to exclude code S09.90 is based on a study[10] that found that 75.3% records coded with 959.01 in EDs did not meet the clinical criteria for TBI (S09.90). The exclusion of this code may lead to a decreased number of reported cases of TBI in the United States. Also, ICD-10-CM codes that are not currently proposed as indicative of TBI will need to be identified and evaluated.[13,16,17]

Table 2.3 Centers for Disease Control and Prevention (CDC) ICD-10 based surveillance definition for traumatic brain injury (TBI) related mortality

ICD-10 Code	Description
S01.0-S01.9	Open wound of the head
S02.0, S02.1, S02.3, S02.7–S02.9	Fracture of the skull and facial bones
S04.0	Injury to optic nerve and pathways
S07.0, S07.1, S07.8, S06.0-S06.9	Intracranial injury
S07.9	Crushing injury of head
S09.7-S09.9	Other unspecified injuries of head
T01.0[a]	Open wounds involving head with neck
T02.0[a]	Fractures involving head with neck
T04.0[a]	Crushing injuries involving head with neck
T06.0[a]	Injuries of brain and cranial nerves with injuries of nerves and spinal cord at neck level
T90.1, T90.2, T90.4, T90.5, T90.8, T90.9	Sequelae of injuries of head

Source: Faul M, Xu L, Wald MM, Coronado VG. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002–2006. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2010.
[a]For consistency with the World Health Organization (WHO) standards for surveillance of central nervous system injury, these codes are included here. However, these codes are not used in the United States; in the United States, nosologists are instructed to assign separate ICD-10 codes for the injury to the head and the injury to the neck.

ICD-10-Based TBI-Related Mortality Definition

▶ Table 2.3 includes the CDC-recommended ICD-10-based definition to identify cases of TBI-related death from ICD-10 coded death certificates in the United States. This definition has been used since 1999.[18]

2.2.3 Traumatic Brain Injury Severity

Brain injuries range from mild TBIs or concussions to coma and even death.

Mild Traumatic Brain Injury or Concussion

This condition, often defined as an injury to the brain presenting with a Glasgow Coma Scale (GCS) score of 13 to 15,[4,8] is the most common type of TBI reported every year in outpatient settings. Mild TBI represents approximately 75 to 95% of all TBI-related medical encounters in the United States civilian[4,19,20] and military[21] populations. While some consider concussion a subset of mild TBI, CDC has described concussion as simply another name for mild TBI.[4,22]

Moderate Traumatic Brain Injury

Moderate TBIs are injuries to the brain presenting with a GCS of 9 to 12.[8,23] These injuries are more likely than cases of mild TBI to have positive findings on computed tomography (CT) scans, and are more likely to lead to negative outcomes, including death.[23,24] Moderate TBIs are more likely to be associated with diffuse axonal injury and correlated with decreased sensory integration.[25,26,27,28] TBI in this range have a stronger correlation with intracerebral hemorrhage, which has poor prognostic outcomes.[29]

Severe Traumatic Brain Injury

This condition includes injuries to the brain presenting with a GCS of 8 or less.[8,20,30] While these injuries account for a small proportion of overall TBI, they are often associated with worse acute prognostic outcomes than mild or moderate TBI and are correlated with more severe sequelae and lower odds of recovery.[20,31,32] In addition to acute comorbidities such as respiratory distress and cerebral ischemia, survivors of severe TBI often experience neuropsychiatric sequelae related to memory and learning, which can linger for years.[31,33,34]

2.3 Traumatic Brain Injury Surveillance

CDC defines public health surveillance as "the ongoing and systematic collection, analysis, and interpretation of outcome-specific data for use in the planning, implementation, and evaluation of public health practice and the timely dissemination of findings to those who make decisions".[35] National and local surveillance systems to study the epidemiology of TBI are therefore crucial to decrease the incidence and outcomes of this potentially preventable condition.

2.3.1 Measuring the Incidence of Traumatic Brain Injury in the United States

No unique system exists in the United States to track the incidence and the determinants that contribute to TBI. In the United States, very few TBI surveillance systems are based on medical review and abstraction; an example of such system is the non-ICD-coded Consumer Product Safety Commission's National Electronic Injury Surveillance System-All Injury Program (CPSC NEISS-AIP) sponsored by CDC.[36]

Data Sources

ICD-9-CM- and ICD-10-CM-Coded Administrative Databases

These include data from national surveys conducted by the National Center for Health Statistics (NCHS) and the National (Nationwide) Healthcare Cost and Utilization Project (HCUP) (described at http://www.cdc.gov/nchs/dhcs/index.htm and https://www.hcup-us.ahrq.gov/databases.jsp, respectively).

Other Non-ICD-Coded Sources

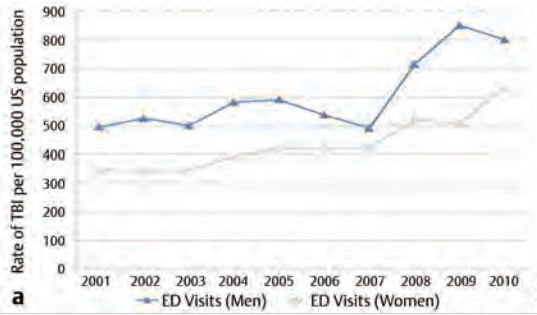
CDC uses the NEISS-AIP (available at http://www.cdc.gov/ncipc/wisqars/nonfatal/datasources.htm) to study the incidence of sports and recreation (SR) related TBI. NEISS-AIP is a national probability sample of hospital-based EDs in the United States and its territories. Patient information is abstracted from medical records resulting from every nonfatal emergency department (ED) visit involving an injury or poisoning associated or not with consumer products.[36]

2.3.2 Measuring the Long-Term Consequences of Traumatic Brain Injury

Data related to the long-term consequences of TBI (i.e., impairment and disability) in the United States are limited and dated. The two national-level estimates currently cited in the literature were extrapolated from two CDC-sponsored follow-up studies of hospitalized TBI survivors conducted in Colorado in the late 1990s and in South Carolina in the early 2000s.[4,37,38] These extrapolations suggest that 3.2 to 5.3 million persons were living with a TBI-related disability at the time of those studies.[4,37,38] However, because the incidence of TBI in the states varies widely (http://www.cdc.gov/injury/stateprograms/indicators.html), the utility of these estimates is limited; moreover, they do not account for TBI survivors who were not hospitalized or did not seek medical care.[1,2]

2.4 Gaps and Limitations in Traumatic Brain Injury Surveillance in the United States

Although CDC provides periodic updates on the national incidence of TBI in the United States, many limitations exist.[1,2] First, because TBI estimates in the United States are based on de-identified ICD-coded data, researchers are able to only describe the number of TBI-related hospitalizations or ED visits; therefore, these systems do not allow studying multiple TBI-related hospitalizations or visits from the same patient for the same injury or other additional TBIs. Second, these systems do not account for persons who do not seek care or seek care in facilities not under surveillance. Third, these databases do not contain information on the injury event itself, the circumstances of the injury, or information on military survivors. Fourth, small sample size in some of these systems preclude the production of reliable yearly estimates. Fifth, these systems lack uniform collection methods to capture, for example, race and ethnicity, and a significant proportion of the external causes of injury. Sixth, CDC has funded only 20 of the 50 states in the United States to produce state-level TBI incidence estimates; these states, like the national surveys, also rely on ICD-coded administrative/billing data sharing the same limitations as the other national systems. Even the NEISS, a system that uses medical record review and abstraction, has limitations[36]; for example, small sample size, lack of specific TBI-related diagnostic codes, and lack of information surrounding the injury event. Although other organizations gather sports-related information, they target organized sports only and selected populations like high



Year	Men	Women
2001	494.6	349.3
2002	525.8	345.1
2003	500.8	348.4
2004	582.4	393.2
2005	590.9	421.6
2006	538.1	421.4
2007	491.6	424.3
2008	714.1	521.2
2009	850.9	508.1
2010	800.4	633.7

Fig. 2.1 **(a)** Rates of traumatic brain injury related emergency department visits by sex: United States, 2001 to 2010. **(b)** Raw numbers for ▶ Fig. 2.1a. (These images are provided courtesy of National Hospital Ambulatory Medical Care Survey: United States, 2001–2010 (Emergency Department Visits). Available at: http://www.cdc.gov/traumaticbraininjury/data/rates_ed_bysex.html. Accessed May 12, 2016.)

schools or colleges and may not routinely collect an athlete's concussion history, use of personal protective equipment (e.g., helmets), and the circumstances of an injury. Limitations also affect reporting TBI-related deaths[18]; for example, the number of death certificates with inaccurate or incomplete documentation of cause-of-death information cannot be quantified; therefore, the total number of TBI deaths may be over- or underestimated[18]; moreover, little is known about the accuracy of reported circumstances and causes of injury-related deaths.[18]

2.5 The Burden of Traumatic Brain Injury in the United States and the World

2.5.1 The Incidence of Traumatic Brain Injury in the United States

Using multiple data sources, CDC has estimated that the total combined rates per 100,000 for TBI-related visits to EDs, hospitalizations, and deaths have increased from 2000 to 2010 (http://www.cdc.gov/traumaticbraininjury/index.html). These combined rates increased slowly from 521.0 in 2001 to 615.7 in 2005, and gradually decreased to 566.7 in 2007. In contrast, from 2008 to 2010, these rates rapidly reached 823.7 per 100,000.

Traumatic Brain Injury Related Visits to Emergency Departments

Cases of TBI treated and released from the EDs represent approximately 70 to 80% of all reported TBI cases in the United States annually.[1,2,9]

By Sex

On average, every year during 2001 to 2010, the rates of TBI hospitalization per 100,000 population were higher in men than in women (▶ Fig. 2.1). From 2001 to 2010, these rates increased for men (from 494.6 to 800.4, respectively) and women (from 349.3 to 633.7, respectively).[39] These increases, however, were steeper from 2007 to 2010 (▶ Fig. 2.1); among men, they increased 63% (from 491.6 to 800.4, respectively), and in women, they increased 49% (from 424.3 to 633.7, respectively).[39] Additional research suggests that this latter trend may be most pronounced among young individuals participating in sports and recreational activities.[36,39,40,41]

Table 2.4 Annual average age-adjusted rates per 100,000 population for traumatic brain injury (TBI) related visits to outpatient departments and to office-based physician offices, by year: United States, 1995 to 2009

Period	Age-adjusted rates per 100,000 population		
	Outpatient department[a]	Office-based physician visits[b]	Total
1995–1997	42.6	234.6	277.2
1998–2000	38.1	305.0	343.1
2001–2003	36.7	204.0	240.7
2004–2006	35.2	306.8	342.0
2007–2009	28.1	352.3	380.3

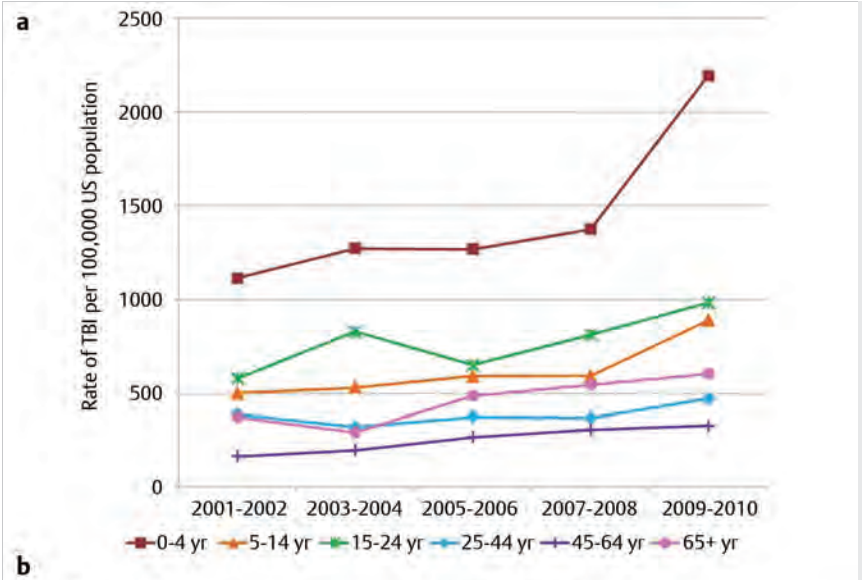
[a]Data for outpatient department visits were obtained from CDC's (Centers for Disease Control and Prevention) National Hospital Ambulatory Medical Care Survey for TBI alone or TBI in conjunction with other injuries or conditions. Persons who were admitted to hospital or referred to emergency department were excluded.
[b]Data for office-based physician visits were obtained from CDC's National Ambulatory Medical Care Survey (NAMCS) for TBI alone or TBI in conjunction with other injuries or conditions. Persons who were admitted to hospital or referred to emergency department were excluded.
Source: Coronado et al 2012.[42]

By Age Group

From 2001–2002 through 2009–2010, 0- to 4-year-olds had the highest rates of TBI-related ED visits per 100,000 population of any age group, with almost twice the rate of those in the next highest age group (i.e., 15- to 24-year-olds; ▶ Table 2.4). For periods 2001–2002 through 2009–2010, these rates increased for all age groups, but were especially high among 0- to 4-year-olds whose rates increased greater than 50% from 1,374.0 during 2007 to 2008 to 2,193.8 during 2009 to 2010 (▶ Fig. 2.2). The observed rises in ED incidence did not necessarily reflect increases in severity; over the same period (2007–2010), rates of both hospitalization and mortality have remained constant.[39]

By External Cause

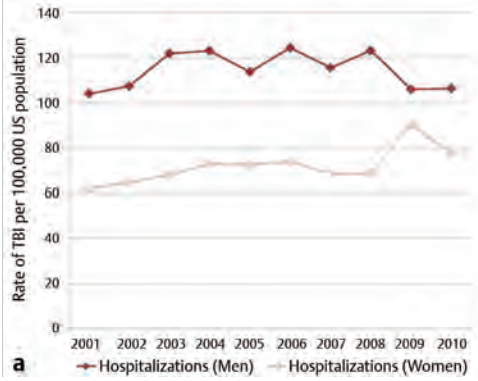
In EDs, the external causes of TBI vary by age group (http://www.cdc.gov/traumaticbraininjury/data/dist_ed.html). In these



b

Period	Age group in years					
	0-4	5-14	15-24	25-44	45-64	65+
2001-2002	1112.6	498.8	576.9	388.3	164.8	373.1
2003-2004	1272.3	529.8	827.5	320.3	197.3	293.3
2005-2006	1268.3	591.4	648.3	373.0	267.0	485.8
2007-2008	1374.0	590.2	811.3	366.7	307.4	544.7
2009-2010	2193.8	888.7	981.9	470.0	328.2	603.3

Fig. 2.2 **(a)** Rates of traumatic brain injury related emergency department visits per 100,000 population by age group and reporting period: United States, 2001–2002 to 2009–2010. **(b)** Raw numbers for ▶ Fig. 2.2**a**. (These images are provided courtesy of National Hospital Ambulatory Medical Care Survey: United States, 2001–2010 (Emergency Department Visits). Available at: http://www.cdc.gov/traumaticbraininjury/data/rates_ed_byage.html. Accessed May 20, 2016.)



a

b

Year	Men	Women
2001	104.0	62.1
2002	107.3	64.7
2003	121.9	68.2
2004	123.0	72.9
2005	113.7	72.6
2006	124.3	73.8
2007	115.5	68.6
2008	123.1	68.6
2009	105.0	90.4
2010	106.3	77.6

Fig. 2.3 **(a)** Rates of traumatic brain injury related hospitalization per 100,000 population by age group and reporting period: United States, 2001–2002 to 2009–2010. **(b)** Raw numbers for ▶ Fig. 2.3**a**. (These images are provided courtesy of National Hospital Discharge Survey: United States, 2001–2010 (Hospitalizations). Available at: http://www.cdc.gov/traumaticbraininjury/data/rates_hosp_bysex.html. Accessed May 20, 2016.)

settings, falls are the leading mechanism of TBI in those aged 0 to 4 (72.8%) and ≥ 65 years (81.8%). TBIs resulting from being struck by/against an object (34.9%) and falls (35.1%) account for the majority of TBIs in 5- to 14-year-olds. Among 15- to 24- and 25- to 44-year-olds, the proportions of TBI-related ED visits due to assaults, falls, and motor vehicle trauma (MVT) events are nearly equal within and across these age groups.

TBI-Related Visits to Outpatient Departments and to Office-Based Physician Offices

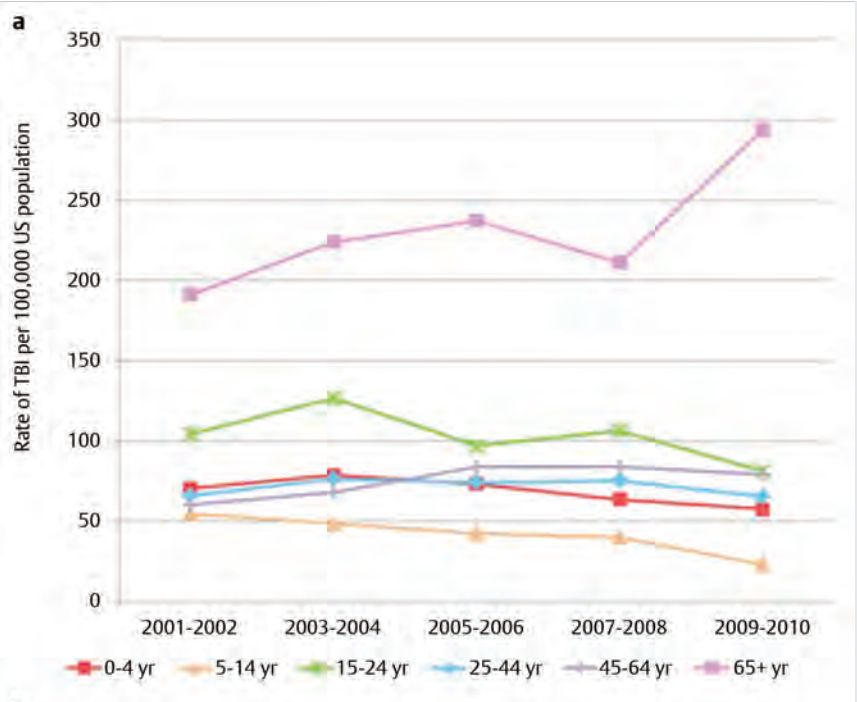
Data on incidence of TBI treated at outpatient departments (ODs), Office-Based Physician Offices (O-BPOs), and other non-ED outpatient facilities represent important knowledge gaps in TBI epidemiology. A study found that the average annual rate of TBI visits to ODs significantly decreased from 42.6 per 100,000 population during 1995 to 1997 to 28.1 per 100,000 population during 2007 to 2009 ($p = 0.010$; ▶ Table 2.4).[42] In contrast, the average annual rate of TBI per 100,000 population treated in O-BPOs increased nonsignificantly from 234.6 during 1995 to 1997 to 352.3 during 2007 to 2009.[42]

Traumatic Brain Injury Related Hospitalizations

Research suggests that approximately 12% of the estimated total of nonfatal TBI-related visits to EDs, ODs, and OB-POs are hospitalized.

By Sex

On average, every year, during 2001 to 2010, men have had higher rates of TBI-related hospitalizations per 100,000 population than women (▶ Fig. 2.3). Among males, these rates slightly



b

Period	Age group in years					
	0-4	5-14	15-24	25-44	45-64	65+
2001-2002	70.3	54.5	104.1	65.9	60.1	191.5
2003-2004	78.7	48.5	126.6	76.4	67.9	224.2
2005-2006	73.3	42.5	97.1	74.2	83.7	237.5
2007-2008	63.4	40.0	106.5	75.2	83.9	211.4
2009-2010	57.7	23.1	81.2	65.3	79.4	294.0

Fig. 2.4 **(a)** Rates of traumatic brain injury related hospitalization per 100,000 population by age group and reporting period: United States, 2001–2002 to 2009–2010. **(b)** Raw numbers ▶ Fig. 2.4**a**. (These images are provided courtesy of National Hospital Discharge Survey: United States, 2001–2010 (Hospitalizations). Available at: http://www.cdc.gov/traumaticbraininjury/data/rates_hosp_byage.html. Accessed May 20, 2016.)

increased from 2002 to 2009 but remained relatively unchanged in 2001 (104.0) and in 2010 (106.3; ▶ Fig. 2.3). In contrast, in women, these rates increased by 20%, from 62.1 in 2001 to 77.6 in 2010.

By Age Group

Between periods 2001 to 2002 and 2009 to 2010, the rates of TBI hospitalization per 100,000 population decreased for all persons ≤ 44 years of age; in contrast, these rates increased almost 25% for 45 to 64 (from 60.1 to 79.4, respectively) and greater than 50% for ≥ 65 year olds (from 191.5 to 294.0, respectively; ▶ Fig. 2.4). The increases in the latter group were largely due to a 39% increase between 2007 to 2008 and 2009 to 2010. Among 5- to 14-year-olds, these rates fell greater than 50% from 54.5 in 2001 to 2002 to 23.1 per 100,000 in 2009 to 2010. Falls are the most commonly reported cause of hospitalized TBI, representing approximately 23% of TBI-related hospitalizations, especially among older adults (aged ≥ 65 years) and ≤ 5 year olds.

By External Cause

In the settings, the external causes of TBI vary by age group (http://www.cdc.gov/traumaticbraininjury/data/dist_hosp.html). As with the ED, falls account for the majority of TBI-related hospitalizations in 0- to 4-year olds (46%) and in ≥ 65 (38%) year olds. TBI-related hospitalizations due to MVT-related crashes increase through age 44 years before decreasing beginning at ages 45 to 64 years. Young adults (15- to 24-year-olds) have the highest proportion of TBI-related hospitalizations due to MVT-related events (33%).

Traumatic Brain Injury Related Mortality

TBI comprise nearly half of all injury-related deaths in the United States.[43]

By Sex

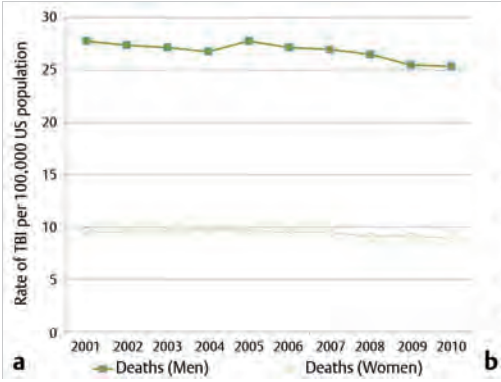
In general, each year from 2001 to 2010, men had more than twice the rate of TBI-related deaths per 100,000 population than women (▶ Fig. 2.5). From 2001 to 2010, however, these rates decreased for both men (from 27.8 to 25.4, respectively) and women (from 9.6 to 9.0, respectively; ▶ Fig. 2.5).

By Age Group

Between 2001 to 2002 and 2009 to 2010, the rates of TBI-related death per 100,000 population decreased for ≤ 44 year olds, remained relatively stable for 45- to 64-year-olds, and increased from 41.2 to 45.2 for ≥ 65 year olds (▶ Fig. 2.6).

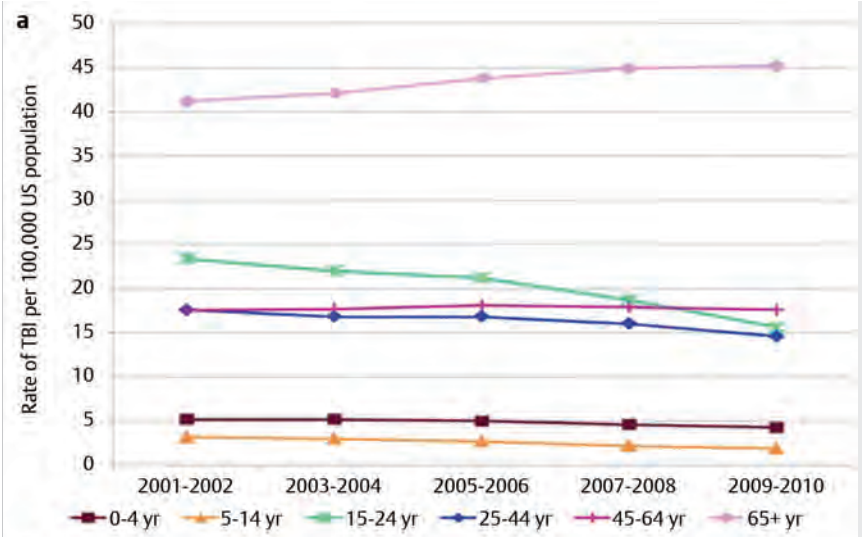
By External Cause

The external causes of TBI-related death vary by age group (http://www.cdc.gov/traumaticbraininjury/data/dist_death.html). In 0- to 4-year-olds, they are primarily associated with assaults



Year	Men	Women
2001	27.8	9.6
2002	27.4	9.5
2003	27.2	9.6
2004	26.8	9.8
2005	27.8	9.7
2006	27.2	9.5
2007	27.0	9.5
2008	26.5	9.1
2009	25.5	9.1
2010	25.4	9.0

Fig. 2.5 **(a)** Rates of traumatic brain injury related deaths per 100,000 population by sex and year: United States, 2001 to 2010. **(b)** Raw numbers for ▶ Fig. 2.5**a**. (These images are provide courtesy of National Vital Statistics System Mortality Data: United States, 2001–2010 (Deaths). Available at: http://www.cdc.gov/traumaticbraininjury/data/rates_deaths_bysex.html. Accessed May 20, 2016.)



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	0-4 yr	5-14 yr	15-24 yr	25-44 yr	45-64 yr	65+ yr
2001-2002	5.2	3.2	23.4	17.6	17.5	41.2
2003-2004	5.2	3.0	22.0	16.8	17.7	42.1
2005-2006	5.0	2.7	21.2	16.8	18.1	43.8
2007-2008	4.6	2.2	18.7	16.0	17.9	44.9
2009-2010	4.3	1.9	15.6	14.6	17.6	45.2

Fig. 2.6 **(a)** Rates of traumatic brain injury related deaths per 100,000 population by age group and year: United States, 2001 to 2010. **(b)** Raw numbers for ▶ Fig. 2.6**a**. (These images are provide courtesy of National Vital Statistics System Mortality Data: United States, 2001–2010 (Deaths). Available at: http://www.cdc.gov/traumaticbraininjury/data/rates_deaths_byage.html. Accessed May 20, 2016.)

(42.9%) and MVT-related crashes (29.2%). MVT-related crashes account for a majority of TBI-related deaths (55.8%) in youth (5- to 14-year-olds) and almost half (47.4%) in young adults (15- to 24-year-olds). Falls account for the majority (54.4%) of TBI-related deaths in adults 65 years of age and older. Research has found that the rates of TBI-related mortality are bimodal and vary in cause by age, peaking among those aged 20 to 24 years (23.6 per 100,000) with firearms and MVT-related crashes as major mechanisms of injury, and again among individuals aged 65 years and older (24.5–103.8 per 100,000), when falls become a major contributor to injury; fully one-third of all TBI-related deaths occur among older adults.[18,44] Overall, the most common mechanisms of injury among TBI-related deaths are firearms (6.4 per 100,000), MVT-related crashes (5.8 per 100,000), and falls (3.1 per 100,000).[18]

Traumatic Brain Injury by Age Group

Age-specific rates for TBI-related ED visits are highest among young children and adolescents, while TBI-related hospitalization and death rates are highest among older adults, who are especially vulnerable to falls.[9,42,44] Between 2002 and 2006, children age ≤ 14 years accounted for over 470,000 TBI-related ED visits, 35,000 hospitalizations, and 2,100 deaths; during the same period, older adults (i.e., persons aged ≥ 65 years) accounted for 140,000 TBI-related ED visits, 81,000 hospitalizations, and 14,000 deaths.[9] Most of the TBI-related ED visits among young people occur in children age 0 to 4 years; these patients presented with a rate of over 1,200 visits per 100,000 population, while the rate among those age 5 to 9 years was 530 per 100,000.[9] Persons aged 55 to 64 years had the lowest

rate of TBI treated in EDs, totaling only 198 visits per 100,000—a rate approximately 84% lower than that of 0- to 4-year-old children.[9,20] Falls were the leading cause of injury among all age groups except those aged 15 to 34 years, where MVT-related injuries were more common.

Traumatic Brain Injury by Severity

Measuring the incidence of TBI by severity is difficult as information to assess and determine injury severity is not captured in most of used databases. Based on existing reports and approximations, mild TBIs are the most common form of TBI, accounting for between 75 and 95% of all TBI-related ED-related visits.[1,2,4,18,20] Moderate TBIs are less common, with ED incidence estimates ranging from 2.1 to 24%,[20,23] though initial severity assessments, including GCS, changed substantially within the first 6 hours after presentation.[23] Severe TBIs are estimated to account for between 3.5 and 21% of TBI-related ED presentations, though these account for a majority of TBI-related deaths.[9,18,20]

External Causes of Traumatic Brain Injury

Falls

Falls are a prominent cause of TBI-related morbidity and mortality, especially among older adults and very young children. While in the general U.S. population, falls account for approximately 38% of TBI-related ED visits, 23% of hospitalizations, and 17% of TBI-related deaths; in contrast, among older adults they account for 76% of TBI-related ED visits, 65% of hospitalizations, and 43% of deaths.[9,18,44] It is expected that the burden of fall-related injuries (including TBI) will grow as the U.S. population continues to age. Advanced age–related and fall-related TBI association is likely due to a combination of the normal aging process (including impaired balance and reaction time) and an increased likelihood for comorbidity and polypharmacy.[45] In children ages 0 to 4 years, falls account for 42% of TBI-related hospitalizations.[9] Fall-related mortality is approximately 50% higher among men than among women among all age groups, though this disparity grows to 350 to 500% among individuals aged 15 to 55 years.[18]

Motor Vehicle Traffic Related Crashes

In the general U.S. population, MVT-related TBI account for approximately 16% of TBI-related ED visits, 21% of hospitalizations, and 32% of TBI-related deaths each year, ranking these injuries among the top causes of TBI-related mortality nationwide.[9] Adolescents and young adults are at especially high risk of these injuries, as 58% of all MVT TBI-related ED visits and 46% of deaths occur among these groups. Adolescents aged 15 to 19 years, the single highest-risk group, have MVT-related TBI hospitalization and death rates more than double the national average (46.2 vs. 19.4 per 100,000 and 6.3 vs. 2.6 per 100,000, respectively). As with other mechanisms of TBI-related injury, males are more commonly affected by MVT TBI than females, though the mortality rate ratio varies from 1.2 among the very young to 3.1 among 20-to 24-year-olds and those aged 85 years and older.[18]

Sports and Recreation Related Traumatic Brain Injury

SR-related TBI are common in young males and represent a growing public health problem in the United States.[36] Nationwide, these injuries account for an estimated 285,000 ED visits each year, and approximately 70% of these occur among individuals age 19 years or younger.[36] Patients seen in EDs for SR-related TBI are twice as likely to be male. Among males, American football and bicycling are the activities with more TBIs; while bicycling, playground activities, and soccer are most common among females. SR-related TBI involving off-road vehicles represent the single highest risk group for severe TBI, followed by equestrian sports; these injuries, along with those resulting from bicycling, are more commonly hospitalized after initial ED presentation than injuries resulting from other SR activities.[36,46]

Assault-Related Traumatic Brain Injury

These type of TBIs represent approximately 11% of all TBI-related ED visits and deaths nationwide.[9] Individuals aged 20 to 24 years are at substantially higher risk for these injuries; their assault-related TBI rates are more than three times higher than the national average (161 vs. 50 per 100,000); rates were similarly high for hospitalizations (10 vs. 5 per 100,000) and deaths (5 vs. 2 per 100,000). These observations largely reflect age-specific patterns among males, as a 2006 analysis showed that the highest incidence of assault-related TBI among females occurs in individuals age 0 to 4 years.[47] Males, however, are more likely than females to suffer assault-related TBI across all age groups, and exhibit an overall age-adjusted rate of injury over six times higher than their female counterparts (12 vs. 2 per 100,000).[47]

Suicide- and Homicide-Related Traumatic Brain Injury

TBI suicides and homicides are overwhelmingly firearm related. In 2011, CDC reported that over 96% of TBI suicides and homicides were firearm related.[18] This study showed that rates of both firearm-related TBI suicide and homicide remained relatively stable at approximately 4.7 and 1.4 per 100,000, respectively, since 1999. Racial disparities among firearm-related TBI suicide and homicide rates, however, are striking; firearm-related TBI suicide rates in 2007 were lowest among Hispanics at 2.0 per 100,000, followed by non-Hispanic Blacks at 2.1 per 100,000, American Indian/Alaska Native populations (AI/AN) at 3.7 per 100,000, and highest among non-Hispanic Whites at 5.7 per 100,000. Disparities in the rates of firearm-related TBI homicide were also wide: non-Hispanic Whites had the lowest rate at 0.6 per 100,000, followed by AI/AN at 1.1 per 100,000, Hispanics at 1.5 per 100,000, while the rate among non-Hispanic Blacks was highest at 4.8 per 100,000.

Risk Factors

Age

Age is an important correlate for TBI incidence. TBI-related ED visits are highest among children younger than 5 years, adolescents, young adults, and ≥ 65 year olds.[1,2,9,18] TBI ED visits are

most common among the 0- to 4-year-old group, whose rates are nearly 2.7 times higher than the U.S. average (1,256 vs. 468 per 100,000). Rates of TBI hospitalizations follow a similar distribution pattern, although hospitalizations are more common among ≥ 75 and 15- to 19-year-olds (339 and 120 per 100,000, respectively). TBI-related deaths are most common among ≥ 75 and 20- to 24-year-olds (57.0 and 24.3 per 100,000 population, respectively), while the average rate across all ages is 17 per 100,000. TBI-related deaths are rare among the young, as rates for those younger than 15 years are less than 5 per 100,000.[9]

Sex

Overall, TBIs are more common among men than among women. Males represent as many as 77% of TBI-related ED visits among persons aged 10 to 14 years, and as few as 36% of visits among those aged 75 years and older.[9] Hospitalizations exhibit a similar pattern, peaking at 79% male in the 20- to 24-year-age group, but only 39% male among those aged 75 years and older. TBI-related deaths are most common among men of all ages; fully 81 to 82% of TBI deaths among 20- to 34-year-olds are male, though this proportion drops to 58 to 59% among those younger than 10 years and those older than 74 years. Among fatal TBI, external mechanism of injury differed substantially by sex. Overall, the most common cause of fatal TBI among men was firearm injury (11 deaths per 100,000), while among women, MVT-related injuries were more common (3.5 deaths per 100,000).[22] Striking disparities are seen among firearm-related TBI deaths in the oldest adults, where rates among men are nearly 35 times higher than those of women (32.4 vs. 0.9 per 100,000).

Race/Ethnicity

While the majority (78%) of TBI-related ED visits occur among Whites, population-specific rates are 38% higher among Blacks than among Whites (619 vs. 448 per 100,000); American Native/Alaska Native/Asian/Pacific Islander populations (AN/A/PI) exhibit still lower rates (335 per 100,000).[9] ED visits are most common among children younger than 5 years across all races, though the ratios of these rates to the race-specific all-age averages varied from 2.5 among Whites to 3.4 among AN/A/PI populations. Age-adjusted TBI-related death rates are highest among Whites (17.7 per 100,000), followed by Blacks (17.3 per 100,000) and AN/A/PI populations (11.2 per 100,000). The distribution of death rates, however, varies substantially by race. Among Whites, TBI-related deaths account for 28%, compared with 37% among AN/A/PI populations and 47% among Blacks.

Recurrent Traumatic Brain Injury

Increasing evidence suggests that a single TBI can produce long-term gray and white matter atrophy, precipitate or accelerate age-related neurodegeneration, and may even increase the risk of developing dementia, symptoms similar to Parkinson's disease, and motor neuron disease.[21,48] In the past, research focused on mild TBI in young adults or TBI in SR revealed a link between the number of TBIs incurred and cognitive impairment,[49,50,51] or the increased risk of experiencing new TBIs,[51] or the occurrence of persistent postconcussion symptoms (PCS),[51] or the rare and controversial second impact syndrome.[52,53] associated with massive cerebral edema[54] and death.[55] A meta-analysis[56] focused on the impact of having ≥ 1 mild TBI found that the overall effect on neuropsychological functioning was not significant; its follow-up component, however, revealed that recurrent mild TBI was associated with poorer performance on measures of delayed memory and executive functioning. More recently, a population-based study of recurrent TBI in New Zealand[57] found that approximately 10% of TBI cases presented ≥ 1 recurrent TBI within the year after initial index injury. In this study, males, people younger than 35 years of age, and those who had experienced a TBI before their index injury were at highest risk of recurrent TBI. Persons with recurrent TBI had significantly increased PCS that tended to be more frequent and severe at 1 year, compared to persons with one TBI only. There was no difference in overall cognitive ability and disability between those with one TBI only and those with recurrent TBI.

Most catastrophic outcomes are, however, reported in the literature of recurrent TBI especially in contact sports. Recent research suggest that even mild TBI can increase the risk of later-life cognitive impairment and neurodegenerative disease, especially if the injuries are recurrent.[49,58,59] Recurrent TBIs of disparate severity have been associated with various dementias[60,61,62] and among athletes practicing contact sports to a tauopathy-labeled chronic traumatic encephalopathy (CTE).[63,64,65,66] Recurrent TBI is also probably linked to a reduced age of onset for Alzheimer's disease (AD).[67,68] Brain autopsies of athletes in various sports with CTE have found tau-immunoreactive neurofibrillary tangles and neuropil threads,[59,68] suggesting that pathological processes similar to AD may be involved. Repetitive mild TBI can provoke the development of CTE, a tauopathy. McKee et al[21] have found early changes of CTE in four young veterans of the Iraq and Afghanistan conflict who were exposed to explosive blast and in another young veteran who was repetitively concussed. Four of these five veterans with early-stage CTE were also diagnosed with posttraumatic stress disorder (PTSD). Advanced CTE has been found in veterans who experienced repetitive neurotrauma while in service and in others who were accomplished athletes.[21] Mild cognitive impairment (or insipient dementia) and self-reported memory problems were more common among football players who reported more than three concussions than those who reported none.[49,58,69] The possible link between mild TBI and CTE or early dementia has implications for military service members (SMs) and veterans as approximately 233,000 TBIs have been officially reported between 2000 and 2012 (www.dvbic.org/tbi-numbers.aspx), nearly 80% of which are mild.[70]

Behavioral and Environmental Factors

Alcohol and Drugs

The behavioral risk factors of TBI are common to most types of injury. Alcohol use has been associated with up to seven times greater risk of falls among adults of all ages; alcohol use specifically among the elderly, for whom falls are the single most important cause of TBI, may further the odds of a fall-related hospital admission.[30,71,72] Alcohol has similarly been identified as a risk factor for injuries ranging from gender-related violence to high school sports-related TBI.[73,74] Furthermore, individuals suffering TBI under the influence of alcohol are four times more

likely to suffer recurrent TBI than those suffering non-alcohol-related TBI.[75] Use of illicit drugs and/or alcohol has been independently associated with MVT-related injuries and all-cause trauma, and represent substantial independent risk factors for serious injury.[76,77]

Use of Protective Equipment

Helmets have been shown to dramatically reduce TBI severity and improve related outcomes in a variety of circumstances. Helmet use while cycling is associated with nearly 50% reduction in health care–related costs; accordingly, a North Carolina law requiring helmet use for motorcyclists was shown to prevent approximately 200 hospital admissions and save an estimated $10 million in 2011 alone.[78,79] Military helmets used as recently as the Vietnam War, while protective from shrapnel and debris from shelling, were unable to offer protection against bullets and other forms of injury.[80] The advancement of helmet technology, however, including the development of Kevlar, resulted in substantially improved protection for combatants in recent conflicts in Iraq and Afghanistan and a substantial reduction of the number of casualties and injury severity resulting especially from blunt forces.[81,82,83] The mandatory use of helmets in college and high school–level American football in 1978 and 1980, respectively, drastically reduced the number and severity of reported head injuries; repeated mild TBI and subconcussions continue to be an issue of significant concern in the sport.[84]

Comorbidities and Prescription Drugs

Comorbidities of several types have been associated with risk of TBI. Falls, the top mechanism of TBI among older adults, are more likely among individuals with a variety of neurologic, endocrine, and cardiovascular diseases.[85,86,87,88,89] Individuals suffering from conditions that impair or substantially change gait, lower extremity proprioception or sensation, or vision are also at high risk.[90,91,92,93,94,95] Polypharmacy and the introduction of new medications, especially those affecting blood pressure, have long been associated with increased risk of falls and subsequent injury, especially among the elderly.[96,97,98] Recent research, however, has suggested that at appropriate doses, certain types of antihypertensive can actually reduce the odds of a fall.[99,100] In addition to increases in risk of TBI, patients on anticoagulant medications (so-called blood thinners) are at elevated risk of developing post-TBI hemorrhages, which can substantially complicate both the clinical picture and prognosis.[101,102,103]

Traumatic Brain Injury in the U.S. Military

TBI is a significant health issue affecting U.S. SMs and veterans. SMs are increasingly deployed to areas where they are at risk for experiencing blast exposures from improvised explosive devices (IEDs), suicide bombers, land mines, mortar rounds, and rocket-propelled grenades. These and other combat-related activities put military SMs at increased risk for sustaining a TBI. Data from the Defense and Veterans Brain Injury Center (http://dvbic.dcoe.mil/dod-worldwide-numbers-tbi) indicates that from 2000 to the first quarter of 2016, 347,962 TBIs were reported among U.S. SMs by the Department of Defense (DoD) worldwide, including the continental United States (▶ Table 2.5); of these, 58.4% were reported by the U.S. Army, 13.6% by the U.S. Navy, 13.7% by the U.S. Air Force, and 14.3% by the U.S. Marines (http://dvbic.dcoe.mil/dod-worldwide-numbers-tbi). Overall, 82.3% of all these injuries were mild TBIs (▶ Table 2.5). During the 2001 to 2011 conflicts in Afghanistan and Iraq and other war theaters around the globe, the high rate of TBI- and blast-related concussion events resulting from combat operations directly impacted the health and safety of SMs.[81,82,83] During that period, the overall annual numbers of TBI progressively increased from approximately 12,407 in 2002 when the war operations started to 32,907 in 2011 when the war-related deployment started to decrease (http://dvbic.dcoe.mil/dod-worldwide-numbers-tbi). These numbers declined from 30,801 in 2012 to 22,637 in 2015 (▶ Table 2.5). Not all of these injuries, however, were battle related. A study of US Army soldiers deployed to Iraq and Afghanistan from September 11, 2001, through September 30, 2007, who were hospitalized due to TBI found 2,898 of these cases; of these, almost half of all TBIs were non-battle-related.[104] In this study, 65% of severe TBIs resulted from explosions; and the overall rates per 10,000 soldier-years of TBI hospitalization were 24.6 for Afghanistan and 41.8 for Iraq. Although rates of TBI hospitalization rose over time for both campaigns, in Iraq, U.S. soldiers with TBI experienced 1.7 times higher hospitalization rates and 2.2 times higher severity than U.S. soldiers in Afghanistan.

Active duty and reserve SMs are at increased risk for sustaining a TBI compared to their civilian peers (http://dvbic.dcoe.mil/about/tbi-military). This may result from several factors, including the specific demographics of the military; in general, young individuals aged 18 to 24 years are at greatest risk for TBI (http://dvbic.dcoe.mil/about/tbi-military). In the Veterans Administration (VA) system, TBI and the need for increased resources to provide health care and vocational retraining for individuals with a diagnosis of TBI have become major focuses as SMs transition to veteran status. Veterans sustain TBIs throughout their life span, with the largest increase as they enter into their 70 s and 80s; these TBIs often result from falls and are associated to high levels of disability (http://dvbic.dcoe.mil/about/tbi-military).

Traumatic Brain Injury in Special U.S. Populations

Traumatic Brain Injury in Rural United States

Data from the 1991 to 1992 Colorado TBI surveillance system revealed that the combined average annual age-adjusted rates of hospitalized and fatal TBI per 100,000 population was significantly higher in rural areas than in urban areas (172.1 vs. 97.8).[105] Similarly, TBI mortality in rural areas was almost twice than those in urban areas (33.8 vs. 18.1).[105] Prehospital TBI mortality per 100,000 population was 10.0 in urban areas and 27.7 in rural areas. Although dated, these findings may reflect issues related to access to acute health care that may still impede care in the United States. People in rural areas travel two to three times further for specialty care, have fewer medical visits even when community resources are available, and have less access to medical specialists.[106,107] Often, primary care physicians are the single source of care of persons with TBI-related disability in rural areas, and these are less likely to have

Table 2.5 Number of US military service members diagnosed with traumatic brain injury worldwide by year and injury severity: 2000 to first quarter 2016

Year	Penetrating (%)	Severe (%)	Moderate (%)	Mild (%)	Not classifiable (%)	Number total
2000	2.5	1.6	14.8	65.5	15.5	10,958
2001	2.5	1.6	14.5	71.4	10.0	11,619
2002	1.9	1.3	11.7	77.7	7.4	12,407
2003	2.2	1.4	11.2	80.1	5.1	12,815
2004	2.5	1.1	11.0	82.4	3.0	14,468
2005	2.1	1.2	10.6	82.9	3.1	15,530
2006	2.0	1.3	9.8	85.1	1.8	17,036
2007	1.7	1.0	9.9	85.3	2.0	23,218
2008	1.6	0.9	7.1	80.8	9.5	28,538
2009	1.8	1.2	6.8	83.0	7.2	28,958
2010	1.1	0.9	6.7	85.9	5.4	29,442
2011	1.3	1.1	6.1	83.5	8..0	32,907
2012	0.8	0.9	6.1	85.0	7.2	30,801
2013	0.7	0.7	7.2	83.5	7.9	27,646
2014	0.7	0.7	8.6	83.4	6.6	25,093
2015	0.6	0.7	11.9	82.5	4.3	22,637
2016 (1st quarter)	0.4	0.5	13.0	86.0	0.1	4,592
Totals	1.4	1.0	9.0	82.3	6.3	347,962

Source: Defense Medical Surveillance System (DMSS), Theater Medical Data Store (TMDS) provided by the Armed Forces Health Surveillance Branch (AFHSB). Prepared by the Defense and Veterans Brain Injury Center (DVBIC). Available at: http://dvbic.dcoe.mil/dod-worldwide-numbers-tbi and http://dvbic.dcoe.mil/files/tbi-numbers/DoD-TBI-Worldwide-Totals_2000–2016_Q1_May-16–2016_v1.0_2016–06–24.pdf.

received advanced training in the long-term management of TBI.[108]

Post-TBI care and rehabilitation are also a concern in rural areas. The estimated prevalence of TBI-related disability is higher in these areas than in urban and suburban areas (24% of TBI disability is rural, vs. 15% urban and 14% suburban).[109] Rural areas in the United States have fewer long-term rehabilitation facilities and community-based services to support independent living after a TBI.[107] Persons affected by TBI who are enrolled in vocational rehabilitation services in rural geographical areas are more likely to discontinue services and have considerably worse employment outcomes when compared with vocational rehabilitation clients in urban areas (7 vs. 24%, respectively).[110]

Traumatic Brain Injury in Institutionalized Persons (e.g., Prisons, Juvenile Detention Centers)

At the end of 2014, approximately 1.9 million people in the United States were incarcerated.[111] TBI prevalence in this population is high, as 25 to 87% of inmates report having experienced a TBI[112,113,114,115]; in the general U.S. population, this number is approximately 1%.[42] Unfortunately, these prison-related studies often fail to address how and when incarcerated individuals experience TBI or elucidate the circumstances surrounding the injury. Prisoners with history of TBI also often experience severe depression and anxiety,[113] substance use disorders,[116,117,118] anger,[119] homelessness,[120] or suicidal ideation and/or attempts.[119,121] Elevated rates of TBI[122,123] and/or physical abuse[123,124,125] have been reported in children and teenagers later convicted of a variety of crimes. History of TBI in male prisoners is strongly associated with perpetration of domestic and other kinds of violence.[126] Addressing the problem of TBI in prisons may require routine screening for TBI,[127,128] alcohol, and substance abuse as well as appropriate treatment for these conditions.[129,130]

Estimated Prevalence of Traumatic Brain Injury in the United States

Currently, no ongoing surveillance of TBI-related disability exists in the United States. The only nationally representative estimates of TBI-related disability were derived from extrapolations of cross-sectional state-level estimates of lifetime TBI-related disability in Colorado and South Carolina. Using these dated databases, it has been estimated that the number of persons living with the long-term consequences of TBI in the United States ranges from 3.2 million[37,38] to 5 million people.[4] Data describing the epidemiological and clinical characteristics of TBI survivors in the United States are needed to monitor the trends and to meet the medical and societal needs of these populations.