

# TBI graphing may serve to expand/focus rehabilitation use for rmTBI cases

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## Abstract

Repeated mild traumatic brain injury (rmTBI) causes significant harm/loss in the lives of many sufferers and communities worldwide, yet the use of rehabilitation intervention is rare for this group. This analysis examines the possible benefits of using TBI graphing as a transformative tool for implementing rehabilitation strategies for those with suspected or known rmTBI. As an example, TBI graphing was used to examine the life of Charles J. Whitman (hereafter denoted as CW). Data points were created from public information sources and graphed in relation to time to assist in visualizing relationships and progressions. CW's TBI graph demonstrated a direct correlation between the timing of potential TBIs and the start and progression of sequelae. Reverse chronology (used to create the graph) also uncovered numerous factors that could have contributed to secondary injuries and worsening of sequelae. With the ability to make complex TBI relational markers visible, the use of TBI graphing may be a useful tool in expanding/focusing the role of rehabilitation for rmTBI sufferers by demonstrating need, assisting in integrated

multidisciplinary plan development, monitoring impact, and providing a possible warning of harmful sequelae progression and/or degenerative conditions to aid in intervention.

## Introduction

Due to the brain's potential regenerative and neuroplastic properties, utilization of appropriate rehabilitation treatments is often effective following a TBI. Moderate-to-severe TBI patients are traditional candidates for intervention; however, the majority of occurring TBIs are considered mild and these sufferers are not usually slated for rehabilitation for numerous reasons. Even though the first mTBI has less potential to lead to long-term consequences, when the issue is compounded with additional mTBIs sustained over time (rmTBI), there is a greater likelihood of experiencing life-altering impairments and neurodegenerative diseases, such as chronic traumatic encephalopathy (CTE) [1,2] and possibly Alzheimer's [3,4]. Research consistently indicates that with each mTBI an individual experiences, the threat of a negative outcome significantly increases [5] through cognitive, emotional, behavioral, and/or physical dysfunction linked to a broad range of sequelae. It has also been shown that each experienced TBI escalates the risk of incurring another [6]. As often the case with mTBI and rmTBI, functional deficits may not appear until days, weeks, months, or even years later. Injury dynamics and timing, damage from secondary phases [7], genetic predispositions [8], environmental influences [9], adrenaline/drug masking, and provoked degenerative mechanisms may be some major

### HIGHLIGHTS

- TBI graphing may make complex TBI relational markers more visible.
- Results may provide an opportunity to better view sequelae trends, relationships, and progressions.
- Graphing may demonstrate rehabilitation need and impact.

### KEYWORDS

TBI graphing, reverse chronology, traumatic brain injury rehabilitation, TBI aggravators, repeated mild traumatic brain injury, rmTBI, Whitman, UT tower shooter, chronic traumatic encephalopathy, CTE

factors for delayed development, surfacing, and chronicity. Thus, each individual's unique blueprint and circumstances make how brain injuries manifest and progress differ from person to person and not be fully predictable.

Despite many rmTBI sufferers remaining fairly functional for long periods of time, others may eventually develop impairments that hinder desired/needed performance. In worst-case scenarios, rmTBI sequelae can put sufferers, and those around them, at risk [10]. The link between rmTBIs and increased violent behavior is well-documented [11,12]. Since rmTBIs may lead to increased aggression [13,14], disinhibition [15], worsening cognitive function [16,17], addictions [18], and other adverse effects, it is apparent why a large portion of the prison population has been afflicted with brain injury [19,20].

Even with the monumental ramifications rmTBIs have globally, the use of rehabilitation is typically only targeted toward moderate-to-severe TBI patients due to the presence of obvious injury. The subtle, slow, and sometimes inconsistent development/appearance of rmTBI sequelae can make detection and the realization for need difficult. (Unrelated factors inducing symptoms that resemble rmTBI must also be considered [21].) By the time detrimental issues are noticeable within an rmTBI sufferer, possible rehabilitation may become more difficult to attain and less effective due to the progressive and degenerative nature of the cause [22]. It is estimated that 90% of mTBI and concussion cases examined by emergency departments (in the U. S.) result in negative CT scans (i.e., uncomplicated) [23], but over 65% of this set may still have functional deficits within 3 to 6 months after injury [24]. In a 2019 cohort study involving over 1,100 mTBI patients, 53% reported impairments even a year after injury [25] and only three lifetime mTBIs have been associated with persistent cognitive deficits [26]. Further observations found four or more mTBIs led to a decline in working memory, attention, and processing speed. Despite such lasting effects, even suspected uncomplicated mTBI cases will typically not participate in formal rehabilitation services. With growing evidence demonstrating that enduring rmTBI sequelae resemble moderate-to-severe injuries [27] and may drive neurodegenerative conditions like CTE, more focus should be placed on increasing societal brain injury awareness and researching/implementing potential methods of care and intervention for this group. These goals are not without significant challenges.

Since the general population is mostly unaware of how easily the brain can be injured, what problems injuries can cause over time, and what can be done to mitigate damage, it is believed that many mTBIs go unreported, undiagnosed, and undetected. When sequelae become a hindrance later, sufferers tend not to associate the issues with the earlier potential brain injuries and may not even mention the possibility when finally seeking medical or psychological assistance. Due to lack of TBI knowledge and experience, some healthcare professionals may also overlook and fail to consider it when diagnosing potential problems. The differing characteristics of each vulnerable population (e.g., children, senior adults, homeless individuals, domestic violence victims, military service members, correctional facility inmates, and impact sports participants) create their own set of treatment challenges, as well. In other situations, stigmas surrounding brain injuries may deter someone from seeking help. An injured brain may also be unable to recognize its own deficits (anosognosia) [28]. Regardless of the reasons that prevent rehabilitation intervention, demonstrating need may be an important first step in changing perceptions and removing barriers.

The expanding and changing elements of rmTBI sequelae can also present challenges for rehabilitators. Assessment tools can be extremely valuable, but they have limitations [29]. Assessments tend to focus on identifying dysfunctions at a particular moment in time, so problems can be missed if they surface later. Whether initial or administered at intervals, they also do not guarantee accurate analyses/diagnoses since modes can be limited in scope and interpreted (or administered) inappropriately. Results may also be influenced by transient factors (possibly not associated with TBI) and/or not reflect the true nature of the injury. Since most mTBIs and rmTBIs go initially untreated, assessments administered when treatment is finally sought may not provide a full picture of the sufferer's situation.

By reverse chronicling life events, converting them into data points, and graphing the time relationships in correlation with injuries, a window is created to visually observe and analyze trends and discover contributions from possible TBI aggravators. With sequelae start and progression (and aggravators) made clearer, rehabilitators may have a new tool to better determine need, plan, implement, monitor, and alter/halt strategies. Results may also illustrate rehabilitation need to apprehensive sufferers, their support network, and

those involved in their healthcare. To examine the possible benefits of this process, the case of CW is used as an example.

CW (DOB: 06/24/1941) was killed by two police officers on August 1, 1966, ending his over 90-minute fatal shooting spree from the observation deck of the University of Texas at Austin (UT-Austin) tower. In addition to those murdered and wounded at (and adjacent to) the campus, CW stabbed his mother and wife to death hours earlier. Due to unusual circumstances and it being the first-of-its-kind tragedy, the investigation involved local, state, and federal authorities. A commission of experts was formed by the governor of Texas to also analyze the cause of CW's actions. As a result, every aspect of his life was examined and tremendous amounts of data were collected and archived. Commission members suspected a biological causal factor, but brain injury was not considered possibly due to limited knowledge of the time.

## Methods

Collected publicly available data, applicable to the research, was arranged in reverse chronological order of occurrence (from death to birth) and color coded to assist in visualizing relationships and progressions. Examples of chronicled information included (but were not limited to): data in medical/health records; prescribed medications; known illegal drug intake; injuries/accident reports; educational records/grades/reports; disciplinary actions (military and nonmilitary); driving infractions; language changes, observations, and changes in thought processes analyzed through personal writings; witness testimonies regarding academic performance, health, and behavior; diet changes; and more. Each used data point was labeled with the known (or best estimated) occurrence date, described, and highlighted with important information/research concerning that entry. Data was collected from: the Dolph Briscoe Center for American History (on site at UT-Austin); the Austin History Center (correspondence and electronic file transfers); National Personnel Records Center; newspaper/magazine articles; books; verified and reliable online resources; and correspondence and/or telephone interviews with five individuals who knew CW personally.

The data primarily consisted of records created by the military, Federal Bureau of Investigation, Austin Police Department, Bureau of Drug Abuse and Control, CW's personal collection, and other

government/law enforcement agencies. To facilitate accuracy, primary sources were used and cross-referenced when possible. Relevant data with only one source was included in the chronology if deemed reliable.

Conflicting data was tested using various methods. If no resolution for relevant conflicting data was found, it was included and noted. Irrelevant conflicting data was omitted. Cognitive, behavioral, and emotional incidents were included if they appeared to be atypical with known information. Results of the reverse chronology were analyzed to determine possible correlations with available TBI and secondary injuries research/case studies. In a few instances, potential subjective witnessing and the lack of full context for each incident needed to be considered; however, sources unknown to each other provided corroborating information for some events. Data points that were associated with known TBI sequelae were used to create the graph in relation to time (Fig. 1).

## Results

Potential TBIs occurred most likely when CW was physically abused during childhood (pediatric abusive head trauma (AHT)), involved in a serious motorcycle accident (possibly two) and two vehicular rollover accidents (one resulting in unconsciousness for several hours), hit in the head with a pugil stick, struck by lightning through a telephone line, and assaulted with severe blows to the head at the ages of 16 and 23 (Table 1).

The first rollover accident occurred in a Corvette convertible and the second in a military jeep that likely did not have a roll bar or hard top. As a lifelong hunter and rifleman in the U. S. Marine Corps (6 years), CW was also exposed to possible repeated blast pressure injuries, which is also tied to a higher risk of CTE [32]. It was reported that he was involved with martial arts, which is considered a contact sport susceptible to mTBI injuries. Despite so many potential TBI events, doctors did not believe CW had a brain injury since there were no changes in his "speech, gait, face, or hand movements" [33]. The knowledge of that time only associated TBI with these limited observable indicators.

When applicable life events were graphed, prominent sequelae trends became visible near the ages of 16, 17, 18, 20, 22, and 25. At 16, CW was severely assaulted by his father and nearly drowned in a pool. Shortly after the incident, a doctor had to make a drainage incision for an infection on the left

side of his face. Approximately 3 months later, CW underwent an appendectomy (for acute appendicitis). Trauma has been identified as a possible cause of appendicitis [34]. Another surgery followed to remove adhesions that had formed on the colon [35]. At 17, the beginning of chronic headaches and a decline in math/language skills followed a motorcycle accident that resulted in hospitalization. At 18, the start of lifelong digestive problems coincided with a harsh hit the head during “boot camp.” At 20, approximately 14 months after the lightning strike, CW entered an environment that required an elevated level of cognition for success. The difficulties (seen on the graph) may have existed earlier, but this is when a witness was able to observe the dysfunction. Several months later, the first reports of aggression and assault followed the first rollover accident.

At 22, CW was the driver of a military jeep that lost control and rolled down a 25-foot embankment. A witness reported that he lapsed into unconsciousness for several hours (a criterion of moderate TBI) after lifting the vehicle off a passenger who was trapped underneath. Not long after, significant cognitive changes become apparent and a persistent feeling that “something is not right” develops. From the time of the probable moderate TBI until the day of the tragedy, at the age of 25, a smaller trend occurs near the time of a major head assault by several fellow Marines.

Interestingly, results from the autopsy and commission investigation recorded numerous physical anomalies that have links to long-term inflammation, which support the link between rmTBIs and sequelae start/progression seen in the graph. The most controversial and debated find during the autopsy was an approximately 2 cm x 1.5 cm x 1 cm glioblastoma (GBM) located near the right amygdala [33] that was estimated to have begun growing between shortly after his 24<sup>th</sup> birthday and the spring before the tragedy. At the time, investigating doctors dismissed the impact the tumor may have had on CW’s actions; however, research since then has shown possible links to altered behavior (even at that size) [36,37] and TBI injury sites [38]. Commission members believed CW would have died from the tumor within a year. The timing of the tumor certainly hints that it may have been a contributing factor to CW’s violent end, but reverse chronology unexpectedly revealed the presence of numerous TBI aggravators at the time of the tragedy, as well, including: a sustained amphetamine addiction [39], exposure to high ambient temperatures for an extended period of

time [40-44], blood loss [45], high stress [46,47], and sleep deprivation [48-55]. With an apparent degenerative condition (as highlighted by the GBM) and the unusual presence of so many factors that can impact brain functioning, it suggests a compounding scenario that evolved passed his ability to restrain. Some additional observations included brownish tint of white matter regions (possible white matter rarefaction, damage, or demyelination) [56-58], slight flattening of convolutions, and much thinner than normal skull thickness, which are also indicators of potential long-term inflammation (with rmTBI as a possible cause). The nature and rapid rotational acceleration (RA) component of CW’s vehicular accidents especially increased the probability of diffuse axonal injury (DAI) and white matter damage [59]. Since the brain is tethered, however, studies have shown that even tangential forces applied to the head may result in some RA. Therefore, “angular acceleration of the skull is not required to cause angular acceleration of the brain” [60]. Even without RA, DAI is possible with excessive linear forces. Chronic inflammation can also impact axon sheaths and change brain morphology, including cortical gray matter volume [61], potentially affecting CW’s convolutions.

A study of similar males near that time period found the mean skull thickness to be 7.7 mm (frontal), 6.5 mm (upper parietal), 7.5 mm (lower parietal), and 5.7 mm (occipital) [62]. His skull was measured between 2 and 4 mm. Chronic inflammation from sustaining rmTBIs throughout a lifetime could be such a cause. Left untreated, adverse effects and functional deterioration from posttraumatic hydrocephalus (PTH) can result [63]. Neuroinflammation is considered a driving force for TBI-related neurodegeneration (TBI-ND) [64,65]. The commission also noted other clues of chronic inflammation such as “slight mineralization of blood vessels [66] in the globus pallidus, an occasional lymphocyte in perivascular spaces [67], subpial amyloid bodies [68], and slight periventricular gliosis [69] in the occipital regions.” The autopsy doctor recorded that pale brownish-yellow matter was found in CW’s proportionally large liver and his “relatively large” kidneys had “some congestion along the borderline of the two layers.” Liver and kidney inflammation have been linked to TBI, as well [70,71]. (Any changes in the liver and kidneys were not reported by the commission.) For the purpose of determining and implementing rehabilitation protocols, TBI graphing and reverse chronology results (as seen in CW’s case in Fig. 1) may provide an opportunity to collectively observe injury and

sequelae type and timing, which may highlight trends, impactfulness, and progressions. With these visual clues, healthcare providers can better justify/advocate need, select the best combination of therapies, monitor progress, identify factors that may aggravate TBI sequelae and impede improvement, and recognize potential warning signs of degenerative conditions in order to intervene.

### **Justifying and advocating need**

A major obstacle to accessing rehabilitative care is justifying need. Typically, physicians are required to prescribe rehabilitation for it to be financially covered (in the U. S.) by other entities. When sequelae have not yet reached an obvious and debilitating stage, as often the case with early mTBI and rmTBI, many physicians may be reluctant to consider it, especially if insurance companies are unwilling to provide coverage [23] and a large portion of primary mTBI injuries resolve. Additionally, the affected individual may also not perceive the need for rehabilitation since it can be time consuming and costly (if funded personally and/or work time is lost), feel pointless since initially there is no serious detriment, and create frustration when comfort zones and personal limitations are challenged. Anosognosia, a psychological need for normalcy, and unknown cumulative injuries may also be hindrances. These issues may be better addressed with observable measures that demonstrate the gamut of rmTBI's influence on the individual. The focus then becomes the beneficial use of rehabilitation therapies as a means to improve current sequelae and attempt to stop, reverse, or slow future potential sequelae progression. TBI graphing may make subtle sequelae trends more understandable through visualization and encourage the physician, sufferer, and associated caregivers into action.

### **Focusing therapies**

Since each TBI sufferer's sequelae profile and expressions may be unique, customizing rehabilitation has been shown to lead to "better outcomes in executive functioning, emotional wellbeing, and community integration" [72]. In 2021, a meta-analysis also indicated that persistent symptoms may be reduced with "specialized interdisciplinary rehabilitation" [73]. The complexity of potential rmTBI sequelae maturation and progression, variability of treatment options and methodologies, and the special needs, abilities,

limitations, and goals of each patient can make the development of individualized rehabilitation plans extremely challenging. The visual clues from TBI graphing and reverse chronology may assist in interdisciplinary rehabilitation planning by showing trends and patterns that may not have been apparent through standard assessments. Periodic and rigorous testing, evaluation, and statistical analysis are important components of formulating and modifying such plans [72] and with additional potential knowledge from these methods, results may help make plans more focused and comprehensive.

### **Monitoring progress**

Monitoring and evaluating patient progress are essential components of rehabilitation since it is the goal of therapy to foster positive change. Various means (e.g., assessments, observations, performative/diagnostic tests) are used to help identify markers that demonstrate the effects of such measures. This gives the provider the ability to alter and evolve care to fit the changing needs of the patient and/or stop treatments that may be ineffective or causing additional harm. Some results from rehabilitation treatment may be obviously observable, but others may take time to develop and only be seen through a change in sequelae degeneration/progression. If the use of rehabilitation is to be expanded for more mTBI and rmTBI cases, it may be necessary to monitor more subtle sequelae over long periods of time and the visual clues from TBI graphing and reverse chronology may add a beneficial layer of analysis.

### **Identifying TBI aggravators**

The nature/location/timing of injury, individual's physiological responses, and medical interventions are not the only factors that impact sequelae onset, severity, and progression. Drugs, heat, blood loss, bodily injury, stress, illness, diet, and more can aggravate brain functioning and regenerative mechanisms, potentially worsening an outcome. Maintaining observance and time chronicling these factors may reveal their influence on sequelae and create an opening for intervention and/or rehabilitation analysis.

### **Identifying degenerative processes**

Failing to identify progressive and neurodegenerative conditions may impact

rehabilitation efforts and result in implementing inappropriate strategies that could further injure rmTBI sufferers. If degenerative conditions exist within the brain, changes somewhere in the functioning spectrum should eventually surface and become observable (visibly and/or diagnostically), but if the changes are subtle, slow, and intermittent, it may be difficult to recognize that the brain is in a declining state until serious and harmful symptoms surface. It also may be challenging to understand the mechanisms causing the decline. Reverse chronicling and TBI graphing may have an advantage by making subtle and slow changes visible to uncover trends (i.e., which sequelae are continuing/worsening, what new sequelae are surfacing, etc.) earlier and provide clues to understand causes.

## Recognizing potential warning signs

From a TBI perspective (with insight derived from these tools), there were numerous warning signs of CW's general decline. The signs went unrecognized, however, due to the lack of knowledge of TBI at that time and inability to take all of his life events collectively into consideration and observe trends. Even with today's knowledge, research, and technological advancements, identifying TBI and its influences can be challenging since there can be a complex set of factors affecting functioning. By identifying trends through visual graphing and reverse chronology, and identifying possible aggravators, an opportunity is created to view causes and effects more easily for the purpose of intervention.

## Discussion

The intention of utilizing rehabilitation therapies for TBI is to optimize the brain and body's own healing/adaptive properties by triggering natural processes to maximize recovery. This includes action at all levels (from molecular/cellular to external stimuli) and with ever-expanding tools and methodologies. Traditionally, the focus of rehabilitation has been restorative, compensatory, and adaptive adjustment [72] mainly for moderate-to-severe cases. An important element of achieving this is working toward preventing, detecting, and managing complications (and possible secondary injuries) with months of regular and specific monitoring immediately following a TBI [74]. Ideally, rehabilitation may have the potential to expand its

use as a preventive/management tool among the majority of mTBI/rmTBI cases with earlier and comprehensive inclusion. Research efforts have been focusing on the efficacy of "reducing inflammation, oxidative stress, axonal injury, and associated neurodegenerative-like pathology" with evolving therapies for rmTBI patients [27], but more opportunities to prevent or lessen sequelae development in rmTBI cases may exist in the future with earlier rehabilitation treatment research and implementation [75,76]. Research suggests that applying appropriate rehabilitation methods as soon as practicable (instead of waiting until sequelae are burdensome/deleterious) should be considered and further investigated. It has been shown that early use of rehabilitation modes that increase heart rate, but do not worsen symptoms, may improve recovery of mTBI [77]. There is also evidence that "rehabilitation interventions can have direct regenerative effects, such as guiding stem cell differentiation, mobilizing stem cells into circulation, and enhancing the secretion of regenerative factors" [72]. Identifying when and what measures would provide the best outcome has been a goal of research for quite some time, especially involving athletes of certain sports.

A deeper understanding of the complex cellular mechanisms of TBI (from initial injury through all stages of regeneration/degeneration processes [27]) is needed to make meaningful diagnostic assessments and provide dynamic care management in order to maximize recovery [21]. Collective knowledge of TBI should be widespread in the general public so it is common to recognize potential injurious situations and sequelae patterns, analyze and identify predictors of sequelae creation and progression, continue surveillance for functioning deficits, and proactively seek intervention and rehabilitation [29].

There must also be robust rehabilitation resources available to accommodate an expansion of treatment to include rmTBI. Even for some moderate-to-severe TBI patients, there are many barriers to rehabilitation access. Research-based development and use of at-home rehabilitation practices (professionally supervised and/or self-directed when applicable) may be a future option to help meet the need for some mTBI and rmTBI sufferers.

Within the past 60 years since the tragedy of CW in Austin, much has been learned about TBI. Repercussions from TBIs, even mTBIs and rmTBIs, are much more far-reaching than previously known and can impact literally almost all areas of functioning. CW's violent ending propagated a wave

of trauma that rippled through generations. It changed many aspects of law enforcement, media, and society within the U. S and even impacted some countries abroad. Using TBI graphing (with reverse chronology) to examine his complex injury history may offer insight that can be used for numerous applications and analyses. For rehabilitators, much may be learned from the results.

In spite of experiencing so many potential brain injuries during his lifetime, CW's ambition and intelligence (measured 127 IQ at last test) caused him to adapt (often inappropriately) and push through his functional changes. Even though he was unaware of what was causing the growing dysfunction, he knew it existed and continuously attempted to "fix himself." Months before the tragedy, CW grew concerned that he was losing his ability to control his aggression and evolving violent thoughts and sought help from the psychiatric staff at the university. The unique essence of his situation went unrecognized. After one visit, CW never returned and noted that he had hoped to stop the problems himself. When an undiagnosed rmTBI sufferer finally reaches a point of seeking help for worsening sequelae, that individual may have been gradually self-adapting for years with potentially counterproductive and harmful tactics that have become so ingrained that they pose additional diagnostic and implementation challenges for rehabilitators (and other healthcare providers).

The results of his reverse chronology also helped identify behavior and actions that may have negatively impacted rehabilitation efforts. For example, the long-term and excessive use of amphetamine was a primary operational aid so he could "stay awake" and reach personal goals. That strategy worked for several years, but had the potential to mask impairments, impact neuroplasticity, lead to neurotoxicity, and produce "adverse psychological effects, including stimulant-induced psychosis" [78]. Regular aspirin use for headaches increased the possibility of intracranial bleeding during additional TBI events [79]. Participation in martial arts and high impact sports made him vulnerable to even more TBIs.

During CW's last several months, witnesses reported that he rarely slept and would stay awake for days at a time. Sleep deprivation had the potential to hinder healing (and foster degenerative processes) and his excessive school, work, and social schedules may have resulted in overexertion and overstimulation (for his condition). Numerous blood donations prior to the tragedy (he was a universal donor) and excessive exposure to high

temperatures strained an already deteriorating condition.

Furthermore, CW's situation is a reminder that bodily symptoms of rmTBI may be more observable clues initially than cognitive, emotional, and/or behavioral deficits in some sufferers. His higher intelligence disguised cognitive impairments and he purposefully and forcefully tried to stay in control of his emotions/behavior, but numerous physical sequelae that have been tied to TBI through research were still present, including: chronic headaches; dizziness; reliance on drugs for functioning; chronic conditions related to immunosuppression; potential subtle liver/kidney dysfunction; changes in eyesight; long-term abdominal and digestive issues; SWD; and abnormal skull thinness. (In addition to being a possible indicator of prolonged inflammation from rmTBI, the abnormal skull thinness suggests a greater susceptibility to TBIs [80].)

CW's TBI graphing results also support the notion that "we must begin to treat children of abuse as brain injury survivors" [81]. AHT is particularly devastating due to its often repetitive and compounding nature that can impact all stages of development (as well as be fatal). Evidence shows that AHT substantially increases the risks of long-term disability, behavioral/learning disorders, developmental delay, motor deficits, visual impairment, and communication deficits [82]. Broadened research of adverse childhood experiences (ACEs), which consists of a wide array of traumatic events that endanger the well-being of a child age 17 or under, also shows that violent behavior manifests in adulthood more often for those with a higher number of ACEs [83]. It is also associated with a higher risk of untimely death [84]. ACEs include serious accidents, physical and/or emotional abuse, witnessing domestic violence, victimization of bullying, and more. Using hindsight as an opportunity to prevent similar future events, using TBI graphing/reverse chronology to monitor those prone to rmTBIs (e.g., military personnel, certain athletes, inmates, domestic violence victims and perpetrators, child abuse victims, etc.) should be considered to open more doors for observation, rehabilitation, and violence prevention.

For many mTBI/rmTBI sufferers (as seen in CW's situation), much of the physical evidence for TBI can only be acquired invasively, through costly diagnostics, and/or only after death through autopsy, dissection, and investigation. The need for ways to identify it more easily and cost-effectively prior to tragedy is evident. For now, TBI graphing and

reverse chronology can be applied to individual cases, but it has the potential to serve as a valuable research tool in the future by identifying trends among different cases. As more graphs are constructed, it may help to uncover previously unknown relationships that can assist with the development/implementation of new rehabilitation treatments.

## **Conclusion**

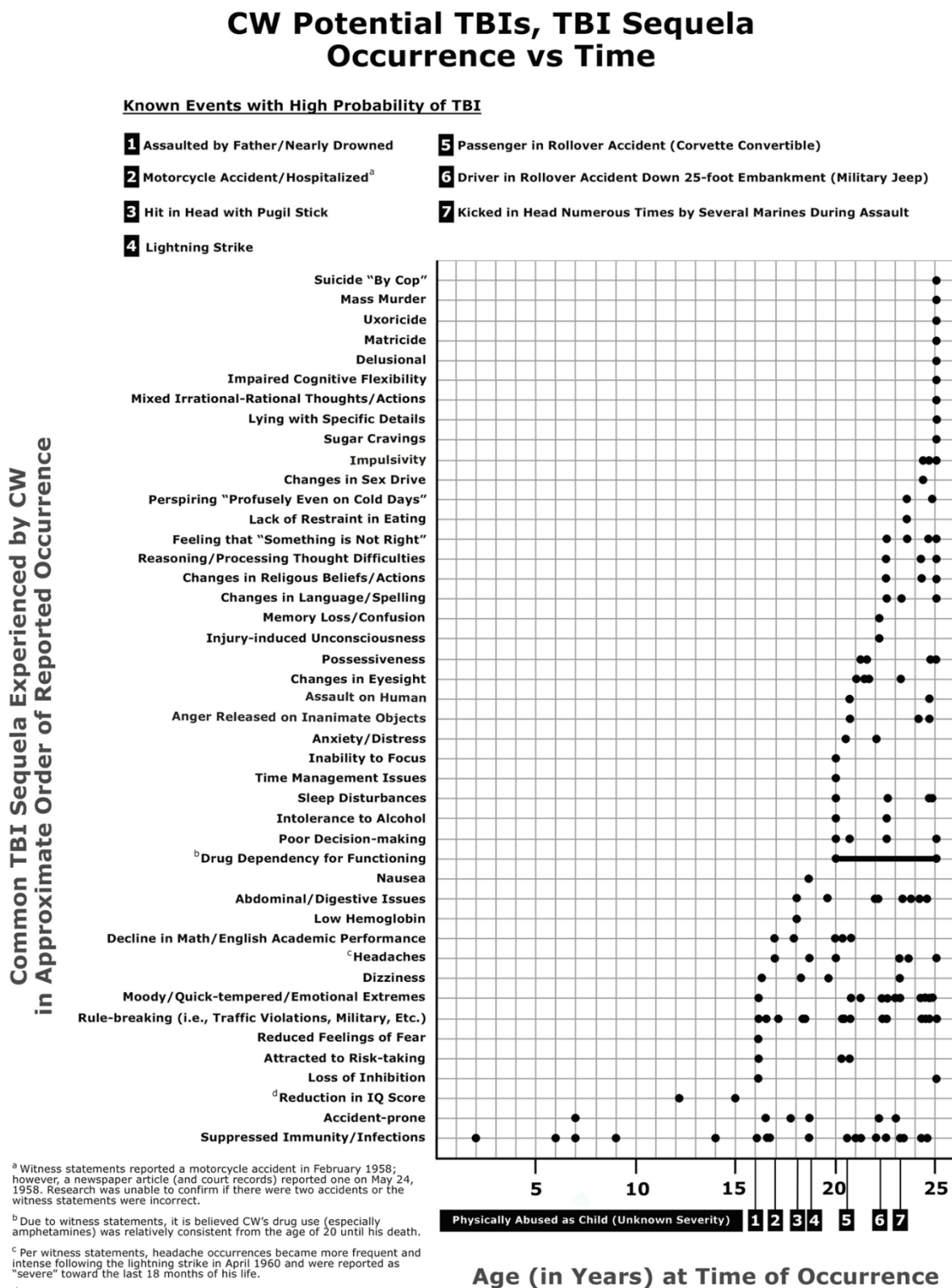
Utilizing data points collected and arranged in reverse chronological order, CW's TBI graph results demonstrate time relationships between potential injuries, sequelae start, and sequelae progression. Expanded research of data points, within the reverse chronology, also identified numerous known TBI aggravators at the time of the tragedy. With clues

made more visible, these tools may provide a means to better justify/advocate the need for rehabilitation, plan more appropriate rehabilitation strategies and timing, monitor the effectiveness of rehabilitation measures, modify treatments, analyze factors impacting rehabilitation and sequelae, identify degenerative processes, and intervene to prevent possible tragic outcomes.

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**Figure 1. TBI graphing results for CW.** CW's TBI graph demonstrates a time relationship between suspected TBI events, sequelae start, and sequelae progression [30].



(Created by K. A. Strube)

**Table 1. Known potential TBI events of CW.** CW experienced potential TBI events throughout his entire life [30].

| <b>Date</b>                 | <b>Incident</b>                                                                                 | <b>Immediate Physical Sequelae</b>                                                                                                   |
|-----------------------------|-------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------|
| <b>October 8, 1964</b>      | Assaulted by several Marines with kicks to the head (case of mistaken identity)                 | Headache<br>Dizziness                                                                                                                |
| <b>September 10, 1963</b>   | Rollover accident down 25-foot embankment in military jeep                                      | 'Groggy,' followed by unconsciousness for several hours (witnessed, not recorded in military documents)                              |
| <b>January 22, 1962</b>     | Involved in rollover accident in Corvette convertible                                           | Stitches to knee injury                                                                                                              |
| <b>April 27, 1960</b>       | Struck by lightning (traveled through line) while using outdoor telephone, thrown against truck | Numb ear<br>Nausea<br>Worsening headaches                                                                                            |
| <b>July/August 1959</b>     | Hard head hit with pugil stick (story shared by CW)                                             |                                                                                                                                      |
| <b>May 1958<sup>a</sup></b> | Involved in motorcycle accident                                                                 | Hospitalized<br>Headaches begin (chronic)                                                                                            |
| <b>June 1957</b>            | Assaulted by father                                                                             | Reportedly nearly drowned<br>Drainage incision for infection on left side of face (~1 month later)<br>Appendectomy (~3 months later) |
| <b>1941-1957</b>            | Physical and emotional abuse by father (admitted, confirmed)                                    |                                                                                                                                      |
| <b>Early 1941</b>           | Mother assaulted by husband while CW was in womb                                                | Mother hospitalized with skull fracture [31]                                                                                         |

<sup>a</sup>Research found data identifying two possible motorcycle accidents; however, only one could be verified with documentation.

## References

1. McKee AC, Cantu RC, Nowinski CJ, Hedley-Whyte ET, Gavett BE, Budson AE, et al. Chronic traumatic encephalopathy in athletes: Progressive tauopathy following repetitive head injury. *J Neuropathol Exp Neurol*. 2009 Jul;68(7):709-35. doi:10.1097/NEN.0b013e3181a9d503.
2. Stern RA, Daneshvar DH, Baugh CM, Seichepine DR, Montenigro PH, Riley DO, et al. Clinical presentation of chronic traumatic encephalopathy. *Neurology*. 2013 Sep 24;81(13):1122-29. doi:10.1212/WNL.0b013e3182a55f7f.
3. Chiasseu M, Fesharaki-Zadeh A, Saito T, Saido TC, Strittmatter SM. Gene-environment interaction promotes Alzheimer's risk as revealed by synergy of repeated mild traumatic brain injury and mouse App knock-in. *Neurobiol Dis*. 2020 Nov;145:105059. doi:10.1016/j.nbd.2020.105059.

4. Pszczółowska M, Walczak K, Miśków W, Antosz K, Batko J, Kurpas D, et al. Chronic traumatic encephalopathy as the course of Alzheimer's disease. *Int J Mol Sci.* 2024 Apr 24;25(9):4639. doi:10.3390/ijms25094639.
5. Smith NIJ, Hogarth L, Corrigan JD, Williams WH. Cumulative effects of mild traumatic brain injury in the general population: Persistent post-concussion symptoms and post-traumatic stress. *Brain Inj.* 2026 Apr;40(6):458-67. doi:10.1080/02699052.2026.2625366.
6. Institute of Medicine (US) Committee on Gulf War and Health. Brain injury in veterans and long-term health outcomes. *Gulf War and health: long-term consequences of traumatic brain injury*, 7th ed. Washington (DC): National Academies Press; 2009. Available from: <https://pubmed.ncbi.nlm.nih.gov/25009909/>
7. Tajik M, Noseworthy MD. A review of molecular and genetic factors for determining mild traumatic brain injury severity and recovery. *Brain Disord.* 2022 Oct;8(16):100058. doi:10.1016/j.dscb.2022.100058.
8. Zeiler FA, McFadyen C, Newcombe VFJ, Synnot A, Donoghue EL, Ripatti S, et al. Genetic influences on patient-oriented outcomes in traumatic brain injury: A living systematic review of non-apolipoprotein E single-nucleotide polymorphisms. *J Neurotrauma.* 2021 Apr 15;38(8):1107-23. doi:10.1089/neu.2017.5583.
9. Schilling KM, Kodama L, Thomas KR, Chanfreau-Coffinier C, VA Million Veteran Program, Merritt VC. Traumatic brain injury, environmental exposures, and subjective cognition in post-9/11 veterans enrolled in the VA Million Veteran Program. *J Neurol Sci.* 2025 Sep 15;476:123625. doi:10.1016/j.jns.2025.123625.
10. Rushworth N, Macdonald A, Brandenburg M, Lonsdale M, Gabbe B, Goldsmith S. The prevalence of acquired brain injury among victims and perpetrators of family violence. *New South Wales: Brain Injury Australia*; 2018. p. 8.
11. Theadom A, Meehan L, McCallum S, Pacheco G. Mild traumatic brain injury increases engagement in criminal behaviour 10 years later: A case-control study. *Front Psychiatry.* 2023 May 2;14:1154707. doi:10.3389/fpsy.2023.1154707.
12. Williams WH, Chitsabesan P, Fazel S, McMillan T, Hughes N, Parsonage M, et al. Traumatic brain injury: A potential cause of violent crime? *Lancet Psychiatry.* 2018 Oct;5(10):836-44. doi:10.1016/S2215-0366(18)30062-2.
13. Dailey NS, Smith R, Bajaj S, Alkozei A, Gottschlich MK, Raikes AC, et al. Elevated aggression and reduced white matter integrity in mild traumatic brain injury: A DTI study. *Front Behav Neurosci.* 2018 Jun 27;12:118. doi:10.3389/fnbeh.2018.00118.
14. Mosti C, Coccaro EF. Mild traumatic brain injury and aggression, impulsivity, and history of other- and self-directed aggression. *J Neuropsychiatry Clin Neurosci.* 2018 Summer;30(3):220-7. doi:10.1176/appi.neuropsych.17070141.
15. Nolan A, Hennessy E, Krukowski K, Guglielmetti C, Chaumeil MM, Sohal VS, et al. Repeated mild head injury leads to wide-ranging deficits in higher-order cognitive functions associated with the prefrontal cortex. *J Neurotrauma.* 2018 Oct 15;35(20):2425-34. doi:10.1089/neu.2018.5731.
16. Vynorius KC, Paquin AM, Seichepine DR. Lifetime multiple mild traumatic brain injuries are associated with cognitive and mood symptoms in young healthy college students. *Front Neurol.* 2016 Oct 31;7:188. doi:10.3389/fneur.2016.00188.
17. Mavroudis I, Ciobica A, Bejenariu AC, Dobrin RP, Apostu M, Dobrin I, et al. Cognitive impairment following mild traumatic brain injury (mTBI): A review. *Medicina (Kaunas).* 2024 Feb 24;60(3):380. doi:10.3390/medicina60030380.
18. Miller SC, Baktash SH, Webb TS, Whitehead CR, Maynard C, Wells TS, et al. Risk for addiction-related disorders following mild traumatic brain injury in a large cohort of active-duty U. S. airmen. *Am J Psychiatry.* 2013 Apr;170(4):383-90. doi:10.1176/appi.ajp.2012.12010126.
19. Schneider BS, Arciniegas DB, Harenski C, Clarke GJB, Kiehl KA, Koenigs M. The prevalence, characteristics, and psychiatric correlates of traumatic brain injury in incarcerated individuals: An examination of two independent samples. *Brain Inj.* 2021 Dec 6;35(14):1690-701. doi:10.1080/02699052.2021.2013534.
20. Williams WH, Mewse AJ, Tonks J, Mills S, Burgess CNW, Cordan G. Traumatic brain injury in a prison population: Prevalence and risk for re-offending. *Brain Inj.* 2010;24(10):1184-8. doi:10.3109/02699052.2010.495697.

21. Silverberg ND, Iaccarino MA, Panenka WJ, Iverson GL, McCulloch KL, Dams-O'Connor K, et al. Management of concussion and mild traumatic brain injury: A synthesis of practice guidelines. *Arch Phys Med Rehabil*. 2020 Feb;101(2):382-93. doi:10.1016/j.apmr.2019.10.179.
22. Dornonville de la Cour FL, Rasmussen MA, Foged EM, Jensen LS, Schow T. Vocational rehabilitation in mild traumatic brain injury: Supporting return to work and daily life functioning. *Front Neurol*. 2019 Feb 21;10:103. doi:10.3389/fneur.2019.00103.
23. Frye CA, Hardy A, Lifrak M. Cognitive rehabilitation improves performance of individuals with mild to moderate traumatic brain injury: A review of comprehensive neuropsychological services as a model approach. *Int J Brain Disord Treat*. 2018;4:025. doi:10.23937/2469-5866/1410025.
24. Madhok DY, Yue JK, Sun X, Suen CG, Coss NA, Jain S, et al. Clinical predictors of 3- and 6-month outcome for mild traumatic brain injury patients with a negative head CT scan in the emergency department: A TRACK-TBI pilot study. *Brain Sci*. 2020 May 1;10(5):269. doi:10.3390/brainsci10050269.
25. Nelson LD, Temkin NR, Dikmen S, Barber J, Giacino JT, Yuh E, et al. Recovery after mild traumatic brain injury in patients presenting to U. S. Level I trauma centers: A transforming research and clinical knowledge in traumatic brain injury (TRACK-TBI) study. *JAMA Neurol*. 2019 Sep 1;76(9):1049-59. doi:10.1001/jamaneurol.2019.1313.
26. Lennon MJ, Brooker H, Creese B, Thayanandan T, Rigney G, Aarsland D, et al. Lifetime traumatic brain injury and cognitive domain deficits in late life: The PROTECT-TBI cohort study. *J Neurotrauma*. 2023 Jul;40(13-14):1423-35. doi:10.1089/neu.2022.0360.
27. Fehily B, Fitzgerald M. Repeated mild traumatic brain injury: Potential mechanisms of damage. *Cell Transplant*. 2017 Jul;26(7):1131-55. doi:10.1177/0963689717714092.
28. Ahmed M, Haq I, Hurley R, Taber K, Rowland J, Yoash-Gantz R, et al. Neural correlates of cognitive anosognosia in veterans with mild traumatic brain injury (mTBI). *Neurology*. 2014 Apr 8;82(10):112-1.008. doi:10.1212/WNL.82.10\_supplement.112-1.008.
29. National Academies of Sciences, Engineering, and Medicine; Health and Medicine Division; Board on Health Care Services; Board on Health Sciences Policy; Committee on Accelerating Progress in Traumatic Brain Injury Research and Care. Gaps, challenges, and opportunities. In: Matney C, Bowman K, Berwick D, editors. *Traumatic brain injury: A roadmap for accelerating progress*. Washington (DC): National Academies Press; 2022. p. 7. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK580090/>
30. Strube KA. Traumatic brain injury graphing: A case study of Charles Whitman. *J Forensic Sci*. 2025 Jul;70(4):1635-44. doi:10.1111/1556-4029.70071.
31. Akers M, Akers N, Friedman R. *Tower sniper: The terror of America's first active shooter on campus*. Houston (TX): John M. Harby Publishing Co.; 2016.
32. Goldstein LE, Fisher AM, Tagge CA, Zhang XL, Velisek L, Sullivan JA, et al. Chronic traumatic encephalopathy in blast-exposed military veterans and a blast neurotrauma mouse model. *Sci Transl Med*. 2012 May 16;4(134):134ra60. doi:10.1126/scitranslmed.3003716.
33. McCay J, Cumley RW. Report to the governor, medical aspects, Charles J. Whitman catastrophe. Houston (TX): The University of Texas M. D. Anderson Hospital and Tumor Institute; 1966.
34. Toumi Z, Chan A, Hadfield MB, Hulton NR. Systematic review of blunt abdominal trauma as a cause of acute appendicitis. *Ann R Coll Surg Engl*. 2010 Sep;92(6):477-82. doi:10.1308/003588410X12664192075936.
35. Minckler TM. General medical background information for the Whitman case requested by Governor John B. Connally. Austin (TX): Ira Iscoe Papers, University of Texas at Austin Dolph Briscoe Center for American History; 1966.
36. Van Genderen MNG, Belgers V, Niers JM, Douw L, Röttgering JG, Gorter M, et al. Tumor location is associated with mood dysfunction in patients with diffuse glioma. *Nat Mental Health*. 2024 Jul;2(7):853-64. doi:10.1038/s44220-024-00275-5.
37. Lupton A, Abu-Suwa H, Bolton GC, Golden C. The implications of brain tumors on aggressive behavior and suicidality: A review. *Aggress Violent Behav*. 2020;54:101416. doi:10.1016/j.avb.2020.101416.
38. Tyagi V, Theobald J, Barger J, Bustoros M, Bayin NS, Modrek AS, et al. Traumatic brain injury and subsequent glioblastoma development: Review of the literature and case reports. *Surg Neurol Int*. 2016 Aug 26;7:78. doi:10.4103/2152-7806.189296.

39. Tse MTL, Cantor A, Floresco SB. Repeated amphetamine exposure disrupts dopaminergic modulation of amygdala-prefrontal circuitry and cognitive/emotional functioning. *J Neurosci*. 2011 Aug 3;31(31):11282-94. doi:10.1523/JNEUROSCI.1810-11.2011.
40. Sakurai A, Atkins CM, Alonso OF, Bramlett HM, Dietrich WD. Mild hyperthermia worsens the neuropathological damage associated with mild traumatic brain injury in rats. *J Neurotrauma*. 2012 Jan 20;29(2):313-21. doi:10.1089/neu.2011.2152.
41. Walter EJ, Carraretto M. The neurological and cognitive consequences of hyperthermia. *Crit Care*. 2016 Jul 14;20(1):199. doi:10.1186/s13054-016-1376-4.
42. Bain AR, Morrison SA, Ainslie PN. Cerebral oxygenation and hyperthermia. *Front Physiol*. 2014 Mar 4;5:92. doi:10.3389/fphys.2014.00092.
43. Yamaguchi T, Shimizu K, Kokubu Y, Nishijima M, Takeda S, Ogura H, et al. Effect of heat stress on blood-brain barrier integrity in iPS cell-derived microvascular endothelial cell models. *PLoS One*. 2019 Sep 4;14(9):e0222113. doi:10.1371/journal.pone.0222113.
44. Chodobski A, Zink BJ, Szmydynger-Chodobska J. Blood-brain barrier pathophysiology in traumatic brain injury. *Transl Stroke Res*. 2011 Dec;2(4):492-516. doi:10.1007/s12975-011-0125-x.
45. Litofsky NS, Martin S, Diaz J, Ge B, Petroski G, Miller DC, et al. The negative impact of anemia in outcome from traumatic brain injury. *World Neurosurg*. 2016 Jun;90:82-90. doi:10.1016/j.wneu.2016.02.076.
46. Esch T, Stefano GB, Fricchione GL, Benson H. The role of stress in neurodegenerative diseases and mental disorders. *Neuro Endocrinol Lett*. 2002 Jun;23(3):199-208.
47. Brand J, McDonald SJ, Gawryluk JR, Christie BR, Shultz SR. Stress and traumatic brain injury: An inherent bi-directional relationship with temporal and synergistic complexities. *Neurosci Biobehav Rev*. 2023 Aug;151:105242. doi:10.1016/j.neubiorev.2023.105242.
48. Kalmbach DA, Conroy DA, Falk H, Rao V, Roy D, Peters ME, et al. Poor sleep is linked to impeded recovery from traumatic brain injury. *Sleep*. 2018 Oct 1;41(10):zsy147. doi:10.1093/sleep/zsy147.
49. Green TRF, Ortiz JB, Wonnacott S, Williams RJ, Rowe RK. The bidirectional relationship between sleep and inflammation links traumatic brain injury and Alzheimer's Disease. *Front Neurosci*. 2020 Aug 25;14:894. doi:10.3389/fnins.2020.00894.
50. Waters F, Chiu V, Atkinson A, Blom JD. Severe sleep deprivation causes hallucinations and a gradual progression toward psychosis with increasing time awake. *Front Psychiatry*. 2018 Jul;9:303. doi:10.3389/fpsy.2018.00303.
51. Kempf J, Werth E, Kaiser PR, Bassetti CL, Baumann CR. Sleep-wake disturbances 3 years after traumatic brain injury. *J Neurol Neurosurg Psychiatry*. 2010 Dec;81(12):1402-5. doi:10.1136/jnnp.2009.201913.
52. Krause AJ, Simon EB, Mander BA, Greer SM, Saletin JM, Goldstein-Piekarski AN, et al. The sleep-deprived human brain. *Nat Rev Neurosci*. 2017 Jul;18(7):404-18. doi:10.1038/nrn.2017.55.
53. Peng Z, Dai C, Ba Y, Zhang L, Shao Y, Tian J. Effect of sleep deprivation on the working memory-related N2-P3 components of the event-related potential waveform. *Front Neurosci*. 2020 May 19;14:469. doi:10.3389/fnins.2020.00469.
54. Vandekerckhove M, Wang YL. Emotion, emotion regulation and sleep: An intimate relationship. *AIMS Neurosci*. 2017 Dec 1;5(1):1-17. doi:10.3934/Neuroscience.2018.1.1.
55. Khan MA, Al-Jahdali H. The consequences of sleep deprivation on cognitive performance. *Neurosciences (Riyadh)*. 2023 Apr;28(2):91-9. doi:10.17712/nsj.2023.2.20220108.
56. Emrani S, Koutures A, Tripodis Y, Uretsky M, Abdolmohammadi B, Nowinski C, et al. Characterizing white matter and vascular pathologies in brain donors exposed to repetitive head impacts. *Acta Neuropathol*. 2025 Mar 6;149(1):24. doi:10.1007/s00401-025-02860-z.
57. Alosco ML, Stein TD, Tripodis Y, Chua AS, Kowall NW, Huber BR, et al. Association of white matter rarefaction, arteriolosclerosis, and tau with dementia in chronic traumatic encephalopathy. *JAMA Neurol*. 2019 Nov 1;76(11):1298-308. doi:10.1001/jamaneurol.2019.2244.
58. Braun M, Vaibhav K, Saad NM, Fatima S, Vender JR, Baban B, et al. White matter damage after traumatic brain injury: A role for damage associated molecular patterns. *Biochim Biophys Acta Mol Basis Dis*. 2017 Oct;1863(10 Pt B):2614-26. doi:10.1016/j.bbadis.2017.05.020.
59. Vo DT, Phan CC, Le HGN, Vo TP, Mai UTT, Le HK, et al. Diffuse axonal injury: A case report and MRI findings. *Radiol Case Rep*. 2021 Nov 1;17(1):91-4. doi:10.1016/j.radcr.2021.10.006.

60. Bayly PV, Cohen TS, Leister EP, Ajo D, Leuthardt E, Genin GM. Deformation of the human brain induced by mild acceleration. *J Neurotrauma*. 2005 Aug;22(8):845-56. doi:10.1089/neu.2005.22.845.
61. Marsland AL, Gianaros PJ, Kuan DC, Sheu LK, Krajina K, Manuck SB. Brain morphology links systemic inflammation to cognitive function in midlife adults. *Brain Behav Immun*. 2015 Aug;48:195-204. doi:10.1016/j.bbi.2015.03.015.
62. Adeloje A, Kattan KR, Silverman FN. Thickness of the normal skull in the American blacks and whites. *Am J Phys Anthropol*. 1975 Jul;43(1):23-30. doi:10.1002/ajpa.1330430105.
63. Akira M, Yuichi T, Tomotaka U, Takaaki K, Kenichi M, Chimi M. The outcome of neurorehabilitation efficacy and management of traumatic brain injury. *Front Hum Neurosci*. 2022 Jun 22;16:870190. doi:10.3389/fnhum.2022.870190.
64. Rodney T, Osier N, Gill J. Pro- and anti-inflammatory biomarkers and traumatic brain injury outcomes: A review. *Cytokine*. 2018 Oct;110:248-56. doi:10.1016/j.cyto.2018.01.012.
65. Al-Rubaie A. Traumatic brain injury and dementia: Mechanisms, risk stratification, and clinical management. *J Clin Neurol*. 2025 Jul;21(4):265-76. doi:10.3988/jcn.2025.0079.
66. Demer LL, Tintut Y. Inflammatory, metabolic, and genetic mechanisms of vascular calcification. *Arterioscler Thromb Vasc Biol*. 2014 Apr;34(4):715-23. doi:10.1161/ATVBAHA.113.302070.
67. Ineichen BV, Okar SV, Proulx ST, Engelhardt B, Lassmann H, Reich DS. Perivascular spaces and their role in neuroinflammation. *Neuron*. 2022 Nov 2;110(21):3566-81. doi:10.1016/j.neuron.2022.10.024.
68. Scott G, Ramlackhansingh AF, Edison P, Hellyer P, Cole J, Veronese M, et al. Amyloid pathology and axonal injury after brain trauma. *Neurology*. 2016 Mar 1;86(9):821-8. doi:10.1212/WNL.0000000000002413.
69. Garcia-Bonilla M, Castaneyra-Ruiz L, Zwick S, Talcott M, Otun A, Isaacs AM, et al. Acquired hydrocephalus is associated with neuroinflammation, progenitor loss, and cellular changes in the subventricular zone and periventricular white matter. *Fluid Barriers CNS*. 2022 Feb 22;19(1):17. doi:10.1186/s12987-022-00313-3.
70. Villapol S. Consequences of hepatic damage after traumatic brain injury: Current outlook and potential therapeutic targets. *Neural Regen Res*. 2016 Feb;11(2):226-7. doi:10.4103/1673-5374.177720.
71. De Vlieger G, Meyfroidt G. Kidney dysfunction after traumatic brain injury: Pathophysiology and general management. *Neurocrit Care*. 2023 Apr;38(2):504-16. doi:10.1007/s12028-022-01630-z.
72. Shen Y, Jiang L, Lai J, Hu J, Liang F, Zhang X, et al. A comprehensive review of rehabilitation approaches for traumatic brain injury: Efficacy and outcomes. *Front Neurol*. 2025 Jun 13;16:1608645. doi:10.3389/fneur.2025.1608645.
73. Möller MC, Lexell J, Wilbe Ramsay K. Effectiveness of specialized rehabilitation after mild traumatic brain injury: A systematic review and meta-analysis. *J Rehabil Med*. 2021 Feb 5;53(2):jrm00149. doi:10.2340/16501977-2791.
74. Marklund N, Bellander BM, Godbolt AK, Levin H, McCrory P, Thelin EP. Treatments and rehabilitation in the acute and chronic state of traumatic brain injury. *J Intern Med*. 2019 Jun;285(6):608-23. doi:10.1111/joim.12900.
75. Mychasiuk R, Hehar H, Ma I, Candy S, Esser MJ. Reducing the time interval between concussion and voluntary exercise restores motor impairment, short-term memory, and alterations to gene expression. *Eur J Neurosci*. 2016 Oct;44(7):2407-17. doi:10.1111/ejn.13360.
76. Leddy JJ, Haider MN, Ellis MJ, Mannix R, Darling SR, Freitas MS, et al. Early subthreshold aerobic exercise for sport-related concussion: A randomized clinical trial. *JAMA Pediatr*. 2019 Apr 1;173(4):319-25. doi:10.1001/jamapediatrics.2018.4397.
77. Coman B, Powell D, Das J, Graham L, Mason R, Harrison M, et al. Active rehabilitation following acute mild traumatic brain injury: A systematic review. *Physiother Can*. 2024 Aug;76(3):295-306. doi:10.3138/ptc-2022-0050.
78. Berman SM, Kuczenski R, McCracken JT, London ED. Potential adverse effects of amphetamine treatment on brain and behavior: A review. *Mol Psychiatry*. 2008 Aug 12;14(2):123-42. doi:10.1038/mp.2008.90.
79. Karamian A, Seifi A, Karamian A, Lucke-Wold B. Incidence of intracranial bleeding in mild traumatic brain injury patients taking oral anticoagulants: A systematic review and meta-analysis. *J Neurol*. 2024 Jul;271(7):3849-68. doi:10.1007/s00415-024-12424-y.

80. Ruan J, Prasad P. The effects of skull thickness variations on human head dynamic impact responses. *Stapp Car Crash J.* 2001 Nov;45:395-414. doi:10.4271/2001-22-0018.
81. Beitchman JA, Dakil S, Stokes M. Repetitive mild traumatic brain injuries in children of abuse. *Ann Child Neurol Soc.* 2023 May 19;1(2):90-5. doi:10.1002/cns3.20024.
82. Nuño M, Ugiliweneza B, Zepeda V, Anderson JE, Coulter K, Magana JN, et al. Long-term impact of abusive head trauma in young children. *Child Abuse Negl.* 2018 Nov;85:39-46. doi:10.1016/j.chiabu.2018.08.011.
83. Jones MS, Hoffmann JP. Adverse childhood experiences and homicide. In: Burgason KA, DeLisi M, editors. *Routledge Handbook of Homicide Studies.* New York: Routledge; 2024. p. 349-68.
84. Brown DW, Anda RF, Tiemeier H, Felitti VJ, Edwards VJ, Croft JB, et al. Adverse childhood experiences and the risk of premature mortality. *Am J Prev Med.* 2009 Nov;37(5):389-96. doi:10.1016/j.amepre.2009.06.021.