

NEUROLOGICAL SOFT SIGNS: A SILENT NEUROPSYCHOLOGICAL COMPONENT IN TRAUMATIC BRAIN INJURIES AND CHRONIC TRAUMATIC ENCEPHALOPATHY

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Abstract

The increasing cases of neurological soft signs (NSS) in contact sports have been the center of public debate in the recent past, with a growing number of people discouraged to engage in these sports. Several health challenges such as deteriorations in motor coordination, sensory integration, head trauma, and the sequencing of complex motor acts are some of the dangers of contact sports. A qualitative research method was used to understand the individual participants' views on contact sports through semi-structured interviews. Snowball sampling was used to ensure only informed participants who would give reliable information were enlisted for the study. As a result, 10 participants who met the eligibility criteria were recruited. Interpretive phenomenological analysis was used to analyze the athletes' personal experiences after engaging in contact sports. Their overall responses were coded into Specific Themes, and those were grouped into Higher Order Themes. The majority of the participants acknowledged that contact sports had adverse and long-term health effects on their lives. Participants mentioned experiencing symptoms of traumatic brain injuries (TBI), chronic traumatic encephalopathy (CTE), and other health risks. This study on neurological soft signs in contact sports with former professional athletes confirmed multiple NSS cases amongst many people engaged in these sports. The study should influence future practices in diagnosing and treating neurological soft signs and whether there should be limitations and changes to the sport.

Keywords: Neurological Soft Signs; Traumatic Brain Injuries; Chronic Traumatic Encephalopathy

Abbreviations: NSS: Neurological soft signs; TBI: Traumatic brain injury; CTE: Chronic traumatic encephalopathy

Introduction

Neurological Soft Signs in Contact Sports

Recently, sports have sparked philosophical and moral debates as to whether parents should let their children get involved in sports, as well as which sport is the safest for their children. Despite the widespread campaigns on sports safety, the greatest concern is physical trauma that may lead to long-term complications, especially head trauma that may then lead to permanent focal or generalized neurological damage. More than 3.5 million children under the age of fourteen get injured annually while indulging in sports activities. The injuries range from bruises to head injuries causing death. Of note is that sport-related Traumatic Brain Injury (TBI) accounts for more than 21 percent of all traumatic head injuries [1].

Few sports exist that mitigate the risk for concussive or sub-concussive hits to the head. Aversive hits to the head do not require violent delivery nor frequent occurrences in order to cause damage [2]. Such injuries may be the result of a hit or kick in the head or with a baseball bat, falling off of a bicycle, or an inadvertent elbow to the nose while playing basketball. These instances are all considered less flagrant than a boxer or UFC fighter being beaten to a bloody pulp or a football player repeatedly using their heads like a battering ram or a hard check in hockey. Low energy trauma to the head can result in complex sequelae of conditions, even after full healing to the initial insult seems to have occurred [3].

A hit to the head can result in a diagnosis of a TBI. TBI is generally delineated as acute or chronic [4]. Acute brain injury in sports-related trauma may lead to concussion, sub-concussion, hemorrhage, or other structural brain damages. The chronic consequence of TBI is Chronic Traumatic Encephalopathy (CTE), a neurodegenerative condition in which progressive clinical symptoms often begin several years after retiring from the sport with abnormal Tau protein accumulation as the histological hallmark. Tau proteins are more abundant in the central nervous system, and they serve to stabilize microtubules - upon injury, they become hyperphosphorylated and have been implicated in dementia as a part of CTE [5].

According to the study of the American Association of Neurological Surgeons (AANS) on sports-related head injuries treated in hospital emergency service settings in the United States in 2009, it is rare for an initial sports injury to result in a fatality. However, they state that subsequent sport related TBI contributes to a significantly higher number of fatalities, as opposed to the initial sports injury. Approximately 21% of the cases of TBI experienced in children and adolescents in America are the result of sport and physical recreational activities.

The top 20 sports and recreational activities with the highest risk of head injuries requiring hospital emergency care or evaluation range from Cycling, Football, and Skateboarding with the highest risk of head injury to lower risk of head injury associated with Basketball, Soccer, and Lacrosse. High-risk sports like Cycling and Football emphasize that a helmet or headgear should always be worn. Proper fitting and safety standard helmets approved by regulatory bodies such as the American Society for Testing and Materials (ASTM) are necessary for the helmet and headgear to provide maximum protection against sports-related head injuries. However, it should be noted that even with lower head injury risk sports, children 14 and below are still susceptible to brain injury and concussions [5].

Sports injuries may lead to numerous long-term neurological complications that may partly or wholly affect the quality of life of a patient. One of the most prevalent conditions is Neurological Soft Signs (NSS). Neurological Soft Signs refer to subtle abnormalities in sensory-perceptual, motor, or other central nervous system functions. Bachmann S, et al. [5], describe NSS as characteristic deficits in sensory integration, motor coordination, and sequencing of complex motor acts, and are mostly seen in patients with schizophrenia [6]. Toro P & Schröder J [7], note that these abnormalities are nonspecific indicators of impairment and are not associated with focal brain dysfunction or any specific disease process. As a result, they may not cause functional impairment [8].

Toro P & Schröder J [7], state that examples of NSS include clumsiness, motor coordination, motor overflow, difficulty with motor sequencing or rapid successive movements, stereognosis or graphesthesia, right-left confusion, and extinction in response to double simultaneous stimulation [8]. Dazzan P & Murray RM [9], showed that the neuroanatomical basis of NSS remains poorly understood, and it has yet to be confirmed whether the symptoms result from specific or diffuse brain abnormalities. Neurological Soft Signs are minor abnormalities such as poor motor coordination, sensory-perceptual difficulties, and difficulties in the sequencing of complex motor tasks [10]. Although NSS

has been present in excess in psychosis, these soft signs of neurological malfunction are also present in apparently healthy individuals, with prevalence rates reported varying between 0 to 50% [11-14].

In another study, the researchers sought to determine the relationship between NSS with PTSD and mild brain injury that was measured using the Behavioral Dyscontrol Scale [15]. The study was comprised of combat veterans who had fought during military operations such as Operation Iraqi Freedom. They found that NSS is closely associated with PTSD due to neurological issues such as reduction in brain activation and cerebral blood flowing to the thalamus compared to healthy control groups. The combat veterans diagnosed with PTSD were found to exhibit signs of hyperactivity in their prefrontal cortex as well as the cerebellum [15]. The research was also used in the evaluation of war veteran subjects who exhibited mild brain injury as a result of issues such as shock waves emanating from blasts. It was found that they exhibited similar signs as those who had PTSD, thus demonstrating the consistent effects of NSS on the cerebellum and prefrontal cortex. It has been found that blast injuries are becoming an increasingly important cause of TBI in civilian and military populations [16].

Extensive research has been conducted examining TBI, and more interest has been expressed regarding the phenomenon of Chronic Traumatic Encephalopathy (CTE). Kulbe JR & Hall ED [17], describe in-depth TBI injury mechanisms in relation to sports and how brain damage is associated with CTE. The findings of the review indicate the need to identify improved neuroprotective strategies for prophylactically attenuating the development of CTE or reducing its progression in affected patients [18].

A different study was conducted by Ng & Lee [79], that focuses on the pathophysiology of TBI and its corresponding underlying molecular mechanisms [20]. Further, researchers provide an update on some of the novel therapeutic targets and agents that can be used in dealing with the condition. Despite numerous studies on TBI, there is limited research on NSS. According to Gunasekaran V, et al. [21], there are limited studies that focus on NSS, with a handful attempting to explore the presence of subtle neurological signs among patients [22]. There appears to be less research and interest focused on NSS as those symptoms appear to be less concerning. Thus, an emerging gap in the literature exists that requires nuanced attention, research, and gathering of information in order to aid in the comprehensive understanding of the complex trajectories of NSS. Notably, less extensive research, focus, and application exists that explores NSS, compared to the abundant knowledge, research, and studies conducted on TBIs.

Statement of the Problem

Head injuries are common in the sports that ordinary people play as well as when played by professional athletes. Recently, much attention has been focused on brain injuries suffered by professional athletes, especially from professional football but also such sports like hockey, collegiate football, and boxing [22]. A major concern has focused on a condition called Chronic Traumatic Encephalopathy (CTE) [23]. This is a condition that appears to manifest years after the initial head injuries occurred. However, it appears that many people with sports-related head injuries exhibit signs of possible brain injuries years before CTE manifests and would be diagnosed. These individuals appear to be exhibiting symptoms of soft neurological signs (NSS). In many instances, these signs may be the precursor to subsequent CTE, but their conditions are not being recognized nor diagnosed.

It is difficult to establish the scope of incidence of CTE and NSS. However, it is estimated that children are the most susceptible to NSS injuries while engaging in sports activities, with more than 3.5 million children under the age of 14 years getting sports injuries each year. Many such injuries consequently include serious traumatic head injuries such as TBI, as it is not just arms, torsos, and legs that children injure. In fact, according to Stanford Children's Health, diagnosed TBI injuries account for more than 21% of all head injuries suffered by children [1]. Although studies are yet to establish the correlation between CTE and NSS, symptoms experienced by adults that are associated with NSS such as difficulties in complex motor tasks' sequencing, sensory-perceptual difficulties, and poor motor coordination, can be perceived as minor abnormalities when the head injury initially occurs. Unfortunately, CTE symptoms, such as problems with thinking, mood, and behavior, occur some years after the initial head trauma(s) [7,24,25].

Further, most CTE cases that have been documented occur largely in specific sports (for instance, soccer, rugby, ice hockey, professional wrestling, American football, and boxing: [26,27]. with indications that NSS symptoms had been present at the time of initial injury. Even though population rates for CTE are unclear, the rates of the disorder are estimated to be about 30 percent of all the individuals who have experienced multiple head injuries [24,25]. There does not appear to be data for people who only suffered a one-lifetime head injury.

There do not appear to be any studies conducted to establish whether NSS symptoms are a precursor to the development of CTE, especially concerning repeated head injuries during sports activities. In this regard, there is a need for research to bridge the gap. This study will be a vital

contribution to the need to determine whether NSS symptoms are often missed when athletes have head injuries. If evidence can be provided that supports the existence of such a problem, then an early diagnosis of NSS could be used to research further to ascertain whether early diagnosis can be utilized as a preventive intervention to curb the development of CTE at a later age. This will be specifically important and valuable to children and adolescents engaging in sports given that their development of mild, NSS symptoms are usually ignored, hence leading to severe problems such as vulnerability to future TBI and even CTE [25]. Whereas children often do not receive the highest level of diagnostic procedures, professional athletes have access to such resources. If their NSS symptoms are being missed, this is a very serious problem beyond that suffered by the athletes.

The best approach to establishing such a relationship would be intensive, individual interviews (such as is provided by the methodology of Interpretive Phenomenological Analysis), as such interviews can tease out subtle NSS that can be missed in less intensive, usual clinical interviews. The sample was selected through snowball non-probabilistic sampling, and the study participants will be individuals who have had head injuries while playing professional sports and have shown signs of TBI [27]. These connections were identified by conducting IPA interviews that provide more information on whether NSS was missed. Furthermore, former professional athletes are a select group, especially former football players, as head injuries affect a small portion of the population, and also, they are an excellent convenience sample in that they are likely to have experienced NSS symptoms [9].

TBI and CTE

TBI results from a brain tissue injury from an external force and may occur without any physical manifestation of trauma, such as in the case of a whiplash injury. The effects range from mild neurologic symptoms to mood disorders and physical disability. According to Selassie AW, et al. [28], every year, approximately 1.7 million American citizens experience some level of TBI, where almost 275,000 individuals are hospitalized, and over 52,000 results in death [29]. They noted that in cases of TBI such as a concussion, these types of injuries can result in chronic impairment and disabilities with long-term neuropsychological implications. Further, over 40% of the people who have endured a TBI usually developed some sort of functional deficits for an extensive period, some surpassing one full year.

Selassie AW, et al. [28], explained that TBI related to sports, together with recreational activities, occurs less often but typically has similar results. Based on the research conducted on sports related TBI, the estimates on the cases of TBI vary. However, Haring et al. asserted that there is agreement among researchers that there is an increasing trend in sports related TBI cases over the past 25 years [30]. Artman Lk & McMahon BT [3], explained that many individuals with TBI injuries have vast limitations as a result of residual symptoms related to the condition [31]. Some of these include sensory deficits, loss of gross and fine motor activity, and physiological and cognitive impairments that may lead to a suboptimal level of efficiency in their former careers. In addition, many authors have termed cognitive and emotional deficits as the most permanent and self-limiting consequences of TBIs. Furthermore, Chronic TBI may complicate Chronic Traumatic Encephalopathy (CTE) outcomes as well as chronic post-concussion syndrome and chronic neurocognitive impairment [32].

According to McKee et al. [2], the limitations associated with CTE can be graded into four stages as per the progression of the disorder: i.e., Stage I (very mild), Stage II (mild), Stage III (moderate), and Stage IV (severe) [22]. In stage I, there is no change in brain weight, but there is mild ventricular enlargement, and it presents with headaches, loss of attention, and the ability to concentrate. It may be asymptomatic in a few, with most of the patients presenting with loss of short-term memory, elevated aggression, and depression, with a few being diagnosed with Post Traumatic Stress Disorder, losing their executive function, and explosivity in their emotions. Stage II presents with gliosis and atrophy of the mammillary bodies with well-delineated tau protein foci in the cerebral cortex, which can only be observed and confirmed in an autopsy post-mortem. The patient presents with depression, unstable moods, and short-term memory deficits. Stage III presents with mild cerebral atrophy and ventricular dilation with a similar presentation with milder forms in addition to aberrant thought processes, impulsivity, and suicidal tendencies. Most of the patients in this group can be termed to be cognitively impaired. Stage IV presents with marked and widespread atrophy, thus a significantly reduced brain weight. In addition, the clinical manifestations include memory loss, attention deficits, visuospatial difficulty, elevated aggression, gait anomalies, impulsivity, depression, paranoia, and Parkinsonism. This degree of impairment, therefore, augments the importance of occupational-based and psychological therapy to this group of patients to enable them to continue performing their normal functions.

In a study by Mez J et al. [33], a convenience sample of 202 deceased patients was used. CTE was diagnosed in 177 football players, including 110 of 111 former National Football League players [34]. However, the authors point out that the causes of the CTE may have been prior to the exposure to sports. The study found that the patients had cognitive, behavioral, and neurological manifestations similar to the four stages of CTE [34].

The increasing trend of cerebral trauma has been linked to an enhanced awareness of the effect of TBI on individual athlete's memory and behaviors, development of stricter regulations on ensuring that sports injuries are reported, as well as improved awareness of the importance of good health-seeking behavior before participating in contact sports activities. Sezgin Caglar A, et al. [35], state that cases of TBI have been reported more frequently in some sports than others, with football accounting for almost 38.1% of the cases of TBI [36]. They further state that in football, TBI has been noted to occur between the ages of 12 to 24 when youth typically participate in middle, high school, and college football. Other cases of TBI are caused by individuals falling from scooters or rollerblading, respectively.

Ling H, Hardy J & Zetterberg H [37], point out that the issues of TBI and CTE in individuals who play contact sports have gained awareness in the media, medical fields, as well as in science [38]. TBI is typically categorized as acute or chronic, where an acute brain injury in the sport can cause a concussion or sub-concussion, as well as other structural damage to the brain. They suggested that some of the acute TBI that occur during competition include skull fracture, rupture of the vertebral artery, as well as an epidural hematoma. They found that the subdural hematoma is one of the major causes of deaths in sport related TBI's and has a higher occurrence rate among boxers. An estimate of 10 deaths is seen annually in boxing due to higher rates of boxers being knocked unconscious. Additionally, they noted that there have been approximately 133 catastrophic brain injuries since 1982 that have been registered in American football and that these are non-professional athletes, with 90% being the athletes in high school and about 8% being college football players.

Much attention has been drawn to the long-term issues that affect athletes who have more damaging exposure to their heads. Some of these effects or impacts are both concussive and sub-concussive impacts. Much attention on this issue has been provided after the publication of autopsy cases of athletes, as well as the increase in the mortality rate of professional athletes that are a result of neurodegenerative diseases. According to Gardner A, Iverson GL, & McCror P [39], the understanding of CTE has been enhanced or redefined from its previous definition that had resembled the delineation of symptoms for Alzheimer's disease [40]. CTE had been recognized and understood as a disorder that affected professional boxers ("punch drunk"), or as another condition that is experienced by military personnel from wartime explosions, but is now recognized with athletes, as well as having some features that are related to psychiatric disorders and some dementia patients.

Gardner A, et al. [39], stated that CTE is currently defined as chronic cognitive as well as neuropsychiatric symptoms of the type of chronic neurodegeneration that usually follow an episode of a severe TBI [40]. They examined 158 case studies of players of contact sports that have been conducted for CTE and recognized an advancement of the version of CTE. Gardner A, et al. [39], explained that there is the difference between the original and modern version of CTE as it relates to the aspect of the age of onset of the neurological deficits, clinical features, progression as well as pathological findings that indicate that these might be very critical conditions [40]. Stein TD, et al. [41], as well as Yue JK, et al. [42], suggested that trauma and exposure to head injury through contact sports comprise some of the risk factors that are associated with CTE [24,39]. Presently, conflicting opinions exist as to whether CTE can occur as a result of a single head injury or if diagnosis of CTE requires recurrent hits to the head. Furthermore, football players who have played for more than five seasons have a higher likelihood of dying from CTE as they are more exposed to the trauma that is known to be a trigger factor for the disease. The trauma mainly results from the rigorous training and game contact.

According to Leddy JJ, Baker JG & Willer B [43], a concussion is a type of mild TBI that usually causes severe symptoms and impacts the brain functioning of the person who received the concussion [39]. The effect of the concussion can either be short term where it lasts for an hour or days, or it can turn into a long-term issue. They suggested that a concussion takes place when an individual suffers a traumatic blow to the head that makes the head, together with the brain, move, causing the brain to bounce as well as twist around in the skull. Such a twist causes stretching, as well as damaging, of the most delicate brain cells and structures. As a result, there is both a physical and chemical change in an individual's brain that affects its functionality.

Leddy JJ, Baker JG & Willer B [43], state that concussions are common in children and that the incidents between 2001 and 2009 rose to 57%, according to the report by the Centers for Disease Control and Prevention (CDC) [39]. Most commonly, children who experience such cases of concussion and TBI do so as a result of a fall. Additionally, they suggested that an estimated 300,000 cases of TBIs take place every year and are related to sports, as noted in the Journal of Athletic Training. In addition, they also noted that there are higher rates of female athletes having concussions compared to males. Some of the sports that report a high level of concussion are Football, Ice hockey, Lacrosse, basketball as well as wrestling. According to Leddy JJ, Baker JG & Willer B [43], people who experience or suffer a concussion often have a hard life and usually get confused and have a lot of headaches, making daily life quite difficult [39]. Others live life with the loss of memories and are unable to remember any event, while others may also lose balance and stability.

Leddy JJ, Baker JG & Zetterberg H [37], suggested that concussion is a major and common acute TBI that is related to sports. It is frequently referred to as mild TBI, and according to them, it is a kind of pathophysiological process that has a negative effect on the brain, and that is usually induced when a biomechanical force causes a blow to the head and thus leads to an impulsive force on the head [39]. McCrory P, et al. [44], state that a concussion may fail to cause a substantial structural injury, hence cannot be detected by means of conventional neuroimaging [45]. In the case where an athlete indicates any concussive clinical symptoms, evaluations and neuropsychiatric testing should be conducted. Leddy JJ, et al. [37], recommended that the athlete should rest for around 24-48 hours immediately after a head trauma as this is an important time period for recovery in the acute symptomatic phase [38]. Giza CC, et al. [46], suggest that many cases of concussion usually get resolved in about 7 to 10 days, although children and some adolescents can experience a longer duration [46].

In summary, researchers note the increase in research focusing on the concept of TBI with reference to sports. In addition, studies provide information on symptoms related to TBI. Studies equally recommend strategies for managing the condition to avoid adverse outcomes on patients.

Biomechanics

Understanding TBI, concussions, and CTE from a biomechanical perspective is imperative due to the rapidly changing biomechanical environment during the head impact and varying levels of impact on the brain. In a study by Meaney DF & Smith DH [47], the multiple complex variations in mechanical response to head strikes are among the factors that create confusion regarding the mechanical etiology of concussions [48]. Consequently, almost every traumatic brain injury or concussion is unique, which complicates diagnosis and treatment monitoring. They further suggested that the two main causal forces that are associated with TBI include inertial and contact forces, whereby both usually occur in the process of impact loading. However, in impulsive head motions, only the inertial loading occurs. Kleiven S [49], supported the earlier finding by Meaney DF & Smith DH [47], noting that oblique impact is the most common fall accident situation that is increasingly associated with the development of head injury due to both increased sensitivity and rotational motion of the human brain [50]. Furthermore, he argued that the bulk modulus of the brain tissue is significantly larger than the shear modulus. Consequently, every impact to the head is likely to deform in shear. The phenomenon is linked to the associated biomechanics whereby a significant portion of the strain from the impact to the brain is given to rotational loading while linear kinematics is given a small level of sensitivity [50]. Thus, understanding the biomechanics of TBI, CTE, and concussions facilitates identification of the primary injuries, how they occur, and their level of impact on the brain. In a related study, variations in biomechanics traumatic injuries to the brain are notable from the different outcomes of different actions leading to the injuries [51]. The study argues that head rotational acceleration may confer concussive tolerance, which also affects the associated clinical outcomes.

According to Post A, et al. [52], the biomechanics of traumatic brain injury varies depending on whether the injuries arise from falls from height or slips. In this study, computer simulations of 9 traumatic injuries from falls indicated that most of the injuries were to the occipital region, especially from falls slipping backward falls [21]. Biomechanically, these falls were associated with low rotational acceleration values from the simulation. Additionally, the study findings suggested that a positive direct correlation exists between the fall height with not only the extent of damage in the occipital region but also the duration of loss of consciousness with extended periods of unconsciousness being reported as a result of high fall heights. An earlier study by Goldsmith W & Plunkett J [48], focusing on children and infants indicated that a proper mechanical understanding of traumatic brain injury might facilitate proper head injury preventive measures among the vulnerable demographics, including athletes and children [53]. The study highlights the need for cooperation between physicists and medical researchers to further advance these developments. Similarly, they support the argument noting that biomechanical engineers should reconstruct complex injury scenarios as this can facilitate an understanding of associated clinical diseases such as hemorrhagic retinopathy, cerebral edema, and subdural hematoma based on the resulting impact on the brain. However, these advances are slower due to reliance on in-vitro models, which are low-evidence studies. Furthermore, up to date, there are very few correlations of clinical brain injury outcomes and the biomechanical metrics. Such parameters are important, especially in the development of protective environments and devices.

Physics

Apart from abrasive forces, G force also plays a role in traumatic brain injuries, concussions, and CTE. In accidents, falls, or collisions during sporting activities, the rapid acceleration, and deceleration of the skull may cause traumatic injury to the brain as it hits against the skull, thereby damaging the tissues [53]. Therefore, this suggests the role of G-force, physics, and Newton's laws in describing fall-related injuries and resulting injuries to the brain. According to Evans V [32], although the role of G-force in traumatic brain injury is still under examination in

research, it is a potential leading of brain injuries both at a chemical and structural level. The concept of G-force and its implications in motor vehicle-related brain injuries is further examined by Weaver C, et al. [54], who conducted an analysis of 374 crashes. The findings indicated for drivers in motor vehicle crashes with an impact of > or = 50 G, there is a 16% chance of developing traumatic brain injuries [55]. On the other hand, the chances of developing a head injury in crashes whose impact was <50 G was estimated to be 1.6%. Understanding the impact of G forces in brain injury development is integral to the development of effective protective measures, especially in motorsport.

Apart from motorsport, the role of G force in the development of traumatic brain injuries, concussions, and CTE has been examined in other outdoor activities. In a related study, Smith DH & Meaney DF [56], examined the impact of G force resulting from roller coasters on the development of traumatic brain injuries [57]. The study argues that although high G forces are well tolerated in many activities, there is a need to regulate the levels of G forces in outdoor activities mostly involving children. Nevertheless, mathematic models of head rotational acceleration in the study did not find most activities as yielding levels of G force high enough to cause traumatic brain injury. However, in their study, Bailes JE, et al. [8], argue that G force resulting from repeated movements and mild brain injuries can culminate into significant axonal injury, neuroinflammation, and blood-brain barrier permeability in the absence of behavioral changes. Additionally, Bailes JE, et al. [8], suggest that CTE and related behavioral changes can result from similar but subconscious level impacts due to significant neurological alterations [58]. The study suggested the need to further examine the concept of "sub concussion" in the emerging research as a potential outcome of G force and repeated mild subconscious injuries to the brain.

In their study, Clark JE & Sirois E [23], explored the role of hydration within the cerebral cortex in concussions as well as the impact of long-term concussions in athletes resulting in the development of CTE. The study argues that altered hydration in the cerebral cortex is a risk factor for concussion and long-term injuries to the brain resulting from contact forces to the head. Although the exact biochemical action involved is not well understood Clark & Sirois [23], argue that as the hydration in the cerebral cortex increases, there is a general increase in the likelihood for disruption of neurofilament proteins as well as exacerbated inflammatory responses after the head trauma [59]. Furthermore, the study posits that increased hydration within the cerebral cortex can also increase dysregulation of the membrane dynamics of the neurons [59]. The findings are consistent with an argument by Alwis DS, et al. [1], indicating that the cerebral cortex has lamina-specific neuronal response alterations reflecting the local circuit changes. Consequently, long-term directional changes in integrative sensory cortical layers are induced by traumatic brain injury [60]. Although hydration is not discussed as the only cortical property affecting resulting injuries, it plays a role in determining the type of sensory processes that are affected a brain injury. Related changes are reported by Tucker LB, et al. [61], in relation to the resulting disruption of multiple neural pathways following a brain injury [62]. According to Keep RF, et al. [63], hydration within the cerebral cortex (brain edema) is associated with poor outcomes following a hemorrhagic and ischemic stroke, and therefore, a concussion or TBI may contribute to a similar outcome [64]. Therefore, understanding the role of the brain tissue fluid is imperative in terms of assessing incident rates and extend of the damage to the brain following a traumatic brain injury or concussion, especially in sporting activities. Additionally, the studies bring to perspective the aspects of brain tissue fluid differences among athletes based on demographics, as well as its combination with inflammation factors, on the development of the long-term physiological and behavioral changes following repetitive head trauma in tracking NSS.

Pathophysiology

Pathophysiology of TBI, concussions and CTE focuses on the extent to neuronal tissue damage resulting from a fall. According to Ng SY & Lee A [19], the two main categories of neuronal tissues damage include primary injuries that are a direct result of mechanical force, especially in the initial fall incident, and secondary injury, which is extended cellular and tissue damage [20]. The study further indicates that in the primary injury, either diffuse or focal injuries may result but co-existent of both types of injuries has been reported. However, over 70% of TBI cases are diffuse axonal injuries (DAI). On the other hand, secondary contusions can develop as a result of secondary impact when the brain strikes the skull, as is the case in rapid acceleration and deceleration injuries. While assessing the implications that mild TBI has on alteration of the signaling pathway Laskowski R, et al. [65], argued that men and women report the impact to the brain differently, whereby women often report noise sensitivity and drowsiness while men complain of amnesia and cognitive deficits [66]. In addition, somatic symptoms commonly reported across genders include fatigue, headache, lethargy, and altered sleep patterns. Repeated mild traumatic injuries to the brain may predispose to CTE with neurobehavioral symptoms correlating with severity, type, and duration. However, Prins M, et al. [67], reported that each case of TBI is usually unique, with patients displaying varying degrees and patterns of injures as well as recovery patterns [68]. Consequently, this complicates the treatment development efforts, as noted by Tra LV [69], with management mostly focusing on supportive care, including correction of cerebral ischemia, monitoring of intracranial pressure, manipulation of serum osmolarity, and lowering the intracranial pressure

[63]. Current research is geared towards finding a cure and effective neuroprotective interventions, including statins, hyperosmolar therapies, and hypothermia.

According to McKee AC, et al. [70], chronic traumatic encephalopathy is primarily an outcome of progressive neurological deterioration [71]. The study further suggests that the pathophysiological process of the development of CTE may depend on the type of injury to the brain, with the three main scenarios commonly associated with the condition being blast injuries, athletics, and other forms of neurotrauma. CTE is a neurodegenerative disorder, and therefore, symptoms range from memory loss, mood and behavioral changes, to dementia and cognitive impairment. Neurophysiological changes reported in CTE include deposition of hyperphosphorylated tau protein. The deposition is usually in the form of clusters around the small cortical blood vessels. A comparative study by DeKosky ST, et al. [29], supports the findings of Gandy S, et al. [40], on the correlation between CTE and repetitive TBI. In addition to agitation, depression, and psychosis, DeKosky ST, et al. [29], reported parkinsonism as a symptom of CTE [72]. According to Saulle & Greenwald [73], in addition to sustaining multiple head injuries as an athlete, military service, or even an older adult, there are known biological risk factors, including the presence of ApoE4 or ApoE3 allele, which bring into focus the possibility of genetic predisposition [74]. Although the exact pathophysiological processes within which the CTE occurs remain largely unknown, Saulle M & Greenwald BD and Iverson GL, et al. [60,73], argue that it may be as a result of the ongoing immunologic and metabolic cascade known as immunoexcitotoxicity [49,74]. Currently, the most compelling strategies that are being applied to combat CTE are prevention and education with an emphasis on athletes and physicians [17]. As highlighted in the reviewed studies, an emerging research gap includes assessment of the genetic risk factors for CTE given the implications the condition has on psychological and physical health as well as professional sports participation.

TBI and CTE Neuroanatomy

Research has extensively examined the correlation between TBI and CTE as well as the neuroanatomy of the affected parts of the brain. According to Lucke-Wold BP, et al. [75], there is a potential link between repeated head trauma and the risk of developing CTE, especially in sports-related concussions among athletes and combat military personnel [76]. Furthermore, they examined the injury-induced pathways noting the actual mechanism by which acute TBI leads to the development of CTE. Anatomically, the study indicates that as a progressive disease, CTE is distinguished by tau neurofibrillary tangles (NFTs). However, the condition may be described based on transactive response DNA binding protein the 43 oligomers. Both occurrences are characterized by the presence of a predilection for subcortical and perivascular areas near microglia and astrocytes. Nonetheless, CTE is only diagnosed postmortem by identifying NFTs neuropathologically. In a comparative study, Turner RC, et al. [104], used model rats to evaluate for tau hyperphosphorylation using the western blot following exposure to head injury [62]. The findings are consistent with those of Lucke-Wold BP, et al. [75], whereby exposure of the rats to repeated blasts resulted in a significant increase in AT8 in the contralateral hippocampus after one month, which provides important insights on both the tau and behavioral changes that occur in human subjects exposed to repeated head injuries or traumatic injury. Additionally, animal studies are pivotal to the early diagnosis of CTE by evaluating and tracking neuroanatomical changes. As noted by Ling H, Hardy J & Zetterberg H [37], traumatic brain injuries may vary with children and adolescents more susceptible to concussions compared to adults as their brains are still developing [38]. Unlike Turner RC, et al. [77], and Lucke-Wold BP, et al. [75], Ling H, et al. [37], argue that both the development of NFTs and tau hyperphosphorylation following TBI and CTE remain speculative. However, CTE-tau pathology is a key i

According to Galgano M, et al. [36], the neuropathology of the brain as a result of CTE can be examined based on the specific part affected which are associated with the patients' behavioral changes [65]. In most patients, psychiatric disturbances lead to suicidal behavior in addition to clinical manifestations ranging from dysarthric speech, difficulty with attention, tremors, memory deficits, and incoordination. In assessing the neuropathology of TBI and CTE, McKee and Daneshvar [78], these behavioral and clinical changes are transient in relation to intracranial lesions [16]. The duration of post-traumatic amnesia (PTA) or loss of consciousness (LOC), as well as the Glasgow Coma Scale (GCS), are useful in quantifying the severity of TBI. Given the susceptibility of axons to acceleration-deceleration injuries in the white matter, severe traumatic injury to the brain often produce a compete transection, a phenomenon commonly referred to as primary axotomy. Additionally, severe injuries are associated with glia and disruption of the blood vessels. On the other hand, in traumatic brain injuries that are less severe, focal pathologic neuro anomalies to axons contribute to delayed axotomy. Traumatic shearing forces, as a result of repeated injuries to the brain or TBI, may increase the membrane's permeability. Consequently, this leads to mitochondrial swelling, calcium influx, and overall axonal swelling [43]. Furthermore, Bigler argued that the ventricle to brain ratio (VBR) is an important indicator of brain integrity following traumatic brain injury, whereby in adults, the normal VBR is approximately 1.5. However, following a severe injury to the brain, the brain volume reduces significantly, leading to an increased ventricular volume as a compensatory outcome, as noted by Castellani [16,37]. Last, Goldstein et al. suggest that

despite the strides in examining the neuro pathologies of concussions, CTE, and TBI, the underpinning mechanisms, as well as the relationships between the disorders, remain poorly understood [37]. Therefore, further advances in research in this area are important to cross-linking the disorders and facilitate early diagnosis.

Neurobiology

The biological underpinning of concussions has remained a center of interest in brain injury studies and associated long-term implications. According to Giza CC & Hovda DA [45], following an injury to the brain, there is a significant increase in energy demands as well as a short period of metabolic crisis due to the release of ionic flux and glutamate [75]. The study further suggests that it is now possible to link these initial neurobiological brain changes to the known characteristics of concussions, including cognitive impairment among some patients, migraines, and increased vulnerability or similar repeat injuries. A comparative study by Howell and Southard supports the arguments of noting that complex, overlapping, and disruptive events usually occur within the brain following a concussion, which may temporarily or permanently affect behavior. In addition to the metabolic crisis reported by Giza CC & Hovda DA [45], Howell as well as Southard state that concussions usually lead to ionic shifts and extensive damage to the neuronal architecture. Additional cellular changes that may occur immediately following a concussion or after a given period include a significant increase in the concentration of inflammatory chemicals, disruption in the cerebral flow of blood, and neuronal crisis. These changes are consistent with the neurobiological alterations reported by Blennow K, et al. [13], noting that the neuropathological changes following concussions are similar to other neurodegenerative conditions such as CTE and dementia [79]. In a related study, Pearce AJ [80], argued that there is a need to assess the neurobiological and neurophysiological changes that occur in both acute and chronic concussions to accurately predict long-term physiological and behavioral implications. Such an approach to research is also critical to differentiating the psychological impact of concussions from other neurodegenerative conditions [44].

According to Barkhoudarian G et al. [10], mild traumatic brain injury or concussions induce cellular changes, including the process of glucose metabolism, axonal injury, cellular structure damage, and mitochondrial dysfunction [81]. Musumeci G, et al. [82], support these findings noting that a wide range of molecular alternation, including energy deficits, mitochondrial dysfunctions, as well as changes in gene and protein expressions that occur after a concussion [83]. Furthermore, the study suggests that these cellular changes often last longer than the clinical symptoms. Through MRS and MRI procedures, it is possible to develop to validate physiological biomarkers associated with injury in order to develop an accurate prognosis of the condition. To provide an in-depth understanding of the neurobiological foundation of concussions delivered with increasing time intervals, Tavazzi B, et al. [84], used a sample of laboratory rats to assess for both nitrative and oxidative stresses [78]. The study indicates that variation in which the concussions were delivered affected the chemical changes in the brain. In general, the study reported that repeated mild traumatic brain injury (concussions) led to a reduction in oxidized glutathione ratio while increasing malondialdehyde. Other brain chemical components that reduced as a result of concussions included nitrate, nitrite, and ascorbic acid. A related study by McDonald et al. examining male soccer players found that about 6-13 days after a concussion, serum NfL (sNfL) increases significantly from the baseline [70]. Additionally, the study notes that in male soccer players, tau and glial fibrillary acidic protein (GFAP) increase between 2 and 13 days of suffering a concussion. Nevertheless, the study did not find any differences in biomarkers from female soccer players. Biomarkers get researchers closer to information regarding the severity of brain trauma; however, in most cases, this must be in conjunction with neuroimaging after significant trauma. Lack of clear information leaves room for NSS to be considered as an efficient screening tool as a first step to triaging.

Neuroimaging

Due to the challenges associated with early diagnosis of TBI and concussions, more research is needed on proper strategies for detection. Gardner A, et al. [39], examine the efficacy of proton magnetic resonance spectroscopy (MRS) in diagnosing sports-related injuries [40]. The review found a significant number of studies reporting MRS abnormalities that were consistent with neurochemistry and, therefore, the study supports the utilization of MRS as a diagnostic tool for concussions or traumatic brain injuries associated with sporting activity. In addition to the identification of neuropathology, they argued that MRS is also an important tool for monitoring recovery in adult athletes. A comparative study by Eierud C, et al. [30], examines the role and efficacy of neuroimaging after mild traumatic brain injury noting that in such conditions, the values of anisotropy are usually depressed, unlike in acute mTBI where the values are elevated [85]. These inconsistencies complicate neuroimaging both in diagnosing TBI and treatment monitoring. Narayana et al. support the argument of Eierud C, et al. [30], noting that as they are, current neuroimaging techniques for mild TBI have low specificity and sensitivity [33]. Larger longitudinal and cross-sectional studies are needed to definitively explore the efficacy and usability of neuroimaging as an mTBI diagnostic tool.

According to Jindal et al., concussions and mild traumatic brain injuries often result in brain abnormalities that are transient and not easy to detect [86]. Similar to Narayana S, et al. [87] and Eierud C, et al. [30], Jindal G, et al. [62], argue that neuroimaging such abnormalities are complex as the mechanisms of injury often lead to specific radiological and clinical presentations. The argument is consistent with the suggestions provided by Bigler ED [12,88], on strategies that can be used to enhance neuroimaging sports related TBI and concussion. The study suggests that in addition to traditional neuroimaging techniques that focus on assessment of the white matter, advanced techniques in MRI that incorporate multiple metrics such as the size of the thickness, shape, and volume should be explored. Similarly, Keightley ML, et al. [71], supported the utilization of dynamic neuroimaging techniques, including the use of MRS, as described by Gardner A, et al. [39]. The study further suggests the need to examine both brain structures metabolism changes post-injury to improve the accuracy of diagnosis.

A larger body of research on neuroimaging concussions and traumatic brain injury is devoted to young athletes. According to Tremblay S, et al. [89], one of the reasons behind this phenomenon is the known correlation between sports concussions and adding including the potential for undiagnosed injuries among young athletes contributing to severe neurological conditions in later years [90]. In one such study, Guenette JP, et al. [51], argued that in this demographic group, advanced neuroimaging techniques are essential not only because of the increased risk impact of brain injuries but also due to the need to identify biomarkers for subtle abnormalities after the concussion [87]. Additionally, Guenette JP, et al. [51], and Odle TG [91], posit that the biomarkers are also imperative to the provision of crucial clinical information related to the appropriate time interval for returning back to sports after a period of rehabilitation following a brain injury. A related study by Churchill et al. reinforces the essence of neuroimaging techniques with a high level of sensitivity and specificity in the identification of biomarkers, especially in accurately diagnosing TBI in athletes with a history of concussion [92]. Therefore, neuroimaging techniques that take into consideration the aspects of aging and staging of concussions are recommended. However, there is a lack of research regarding brain imaging specific to NSS. Upcoming sections will focus on neurological soft signs.

Neurological Soft Signs

According to Mittal VA, et al. [88], the term neurological soft sign (NSS) was initially developed in the 1940's as a description of non-diagnostic abnormalities when carrying out neurological examinations on kids with psychosis [91]. NSS has a close relation to the core pathophysiology of psychosis. Salvador-Cruz J, et al. [93], suggest that NSS is difficult to locate in the human nervous system and is only detectable by clinical neurological exams when neurological injury characteristics are absent [94]. They explain that NSS typically includes some neurological deficits such as motor coordination, perceptive sensory integration, as well as clumsiness; it is important to assess and detect such signs at the early stage, as they can go unnoticed and cause severe results later in life if left unchecked. According to Salvador-Cruz J, et al. [93], and Mittal VA, et al. [88], NSS are associated with some uncertain vulnerability factors that are mainly related to various psychological as well as psychiatric variables. For example, Schizophrenia, Attention Deficit Hyperactivity Disorder, as well as Obsessive-Compulsive Disorder have been associated with NSS.

Although NSS tends to be non-localized and non-specific, it is possible to associate it with underlying issues. Neurological Soft Signs have been closely linked to underlying problems such as tissue breakdown and brain activation problems, especially in the thalamus and cerebellum [95]. As such, the main efferent white matter tracts that link the thalamus and cerebellum could be playing a significant role in helping identify the presence of NSS as a vulnerability marker for subsequent problems such as lasting mild brain injury [80]. NSS indicates the presence of mental and cognitive disorders due to its ability to disrupt motor and stimuli coordination, which marks the onset of cognitive problems [6].

According to Gurvits TV, et al. [53], NSS is more prevalent in combat veterans suffering from Post-Traumatic Stress Disorder (PTSD) compared to patients with other psychiatric disorders [52]. In a study involving 27 Vietnam War veterans who suffered from PTSD and 15 Vietnam War veterans who did not have PTSD, comparisons between these two groups were made [15]. The results showed that the combat veterans who had PTSD were more vulnerable to NSS compared to the veterans who did not have PTSD. The combat veterans suffering from PTSD also reported having had significant enuresis and cognitive problems. These statistics show that NSS are closely associated with PTSD in combat veterans, yet that does not imply specificity in such patients. Thus, differentiating psychiatric conditions from neurological disorders can be complex, with both being associated with NSS-type symptoms.

In conclusion, sports provide entertainment and joy, but at times can result in serious issues that affect the long-term functioning and lives of people who suffer traumatic sport-related injury. Despite the notion that sports contribute to physical fitness, some sports such as football, hockey, as well as boxing are dangerous as the participants can be exposed to significant head injuries in the process of playing. TBI, CTE, and concussion, among other conditions, can worsen in severity and not only affect individuals' careers but can also affect their health in a long-

lasting and negative manner. Additional studies have been conducted on TBI and CTE, but less has been done on the issue of Neurological Soft Signs. Despite the benefits of participating in sports, people should be more aware of the dangers that are associated with various sports and the resulting impacts that may impair their daily lives.

Neuroanatomy of Neurological Soft Signs

As previously discussed, neurological soft signs (NSS) refer to neurological abnormalities that contribute to deficits in motor coordination and sensory integration, among others. These deficits are a direct cause of NSS's impact on specific parts of the brain, which is still an evolving area of neuroscience. In a study by Dazzan P, et al. [27], comprising of 43 healthy individuals to examine the correlation between brain structure and NSS, a notable reduction in the anterior cingulate gyrus, inferior frontal gyrus as well as middle and superior temporal gyrus was reported [67]. Similarly, findings by Fountoulakis et al. [34], from a sample of 122 patients diagnosed with schizophrenia and 122 normal controls supported the argument that neurological soft is a constituent risk factor for schizophrenia [96]. NSS has also been used as a predictor of ultra-high risk (UHR) of psychosis. A comparative study by Wang Y et al. [97], examining 56 antipsychotic-naïve ultra-high risks of psychosis individuals and 35 healthy controls also indicated that compared to the control group, the UHR group had significantly higher NSS scores [98]. However, the study also noted that total NSS was not associated with grey matter volume. Nevertheless, Wang Y et al. [97], argued that the UHR group had greater deficits in terms of sensory integration in the right middle gyrus, the left cerebellum, and the right insula [98]. The findings by Wang Y, et al. [97], and Fountoulakis et al. [34], suggest that structural changes in the brain associated with reduced grey matter volume may be an indicator for developing specific neurological abnormalities. In this study, cortical grey matter reductions both in the temporal and frontal were important integrative signs. Therefore, they may present the neuropathological basis for normal brain development as well as the predictors of psychotic conditions in future.

In their study Chrobak AA, et al. [20], examined the relationship between NSS and implicit motor learning in patients diagnosed with bipolar disorder and schizophrenic patients [98]. Compared to the patient groups, the control group comprising of healthy individuals were characterized by a small number of errors. On the other hand, bipolar and schizophrenic patients demonstrated higher rates of NSS and cerebral soft signs (CSS). These findings are supported by the argument by Rathod et al., indicating that compared to healthy controls, the prevalence of NSS in schizophrenic or psychotic patients are over 50% [99]. Furthermore, the study notes that a significantly high number of psychotic patients are resistant to treatment as a result of the neuroanatomical and neurophysiological basis of schizophrenic conditions that complicate the processes of developing effective pharmacological agents. However, unlike Dazzan P, et al. [27], Rathod B, et al. [98], did not find any positive correlation between the total NSS total scores and cortical thickness in the middle frontal cortex, left paracentral lobule, and the right inferior temporal cortex. On the contrary, a meta-analytical study by Bachmann S, et al. [5], supported the findings indicating that NSS decreased parallel to remission of psychopathological symptoms. Additionally, Herold CJ, et al. [57], found that in addition to the increased NSS scores in schizophrenic subjects, the scores were also strongly correlated with a notable reduction in grey matter in the right gyrus, left thalamus, decline, and the left para hippocampal gyrus [100].

The reviewed studies suggest that as opposed to considering NSS as a state marker or a trait, it should be actively included in patient management in order to improve patients' outcomes in brain injuries.

The Present Study

This study serves to identify and update the literature on Neurological Soft Signs (NSS) and their effects on former professional athletes. There is a paucity of research on this topic as it pertains to athletes. Much of the research on the relationship of NSS to subsequent brain impairments was conducted with severely mentally ill patients, and after 2012 with some athletes, as the general population and the professional community was becoming more familiar with Chronic Traumatic Encephalopathy and interest in that and antecedent conditions increased. However, CTE and TBI's and undetected precursors are currently troubling issues. The present qualitative study is an attempt to update the research on NSS, the manifestation of that symptomatology, and possible treatment implications for working with head trauma victims. Although many of the symptoms experienced by these individuals are related to dementia or CTE, their traumatic experiences are often persistent and prolonged, and therefore may require more detailed, tailored treatment approaches, especially earlier in the process. The small existing literature on this population does not touch on evidence-based treatments for working with this population. And therefore, it is the hope of the researcher to provide the professional community with guidance regarding diagnosis and treatment, especially when NSS are present.

This qualitative study aimed to interview 10-12 former professional athletes, with at least one year of experience in their sport who self-identify as having had a significant head injury while playing. They were asked to complete an approximate 60-minute semi-structured interview. The interview consisted of questions pertaining to neurological symptoms from head trauma while playing professional sports, especially regarding what are commonly termed NSS. It is the belief of the researcher that by having used intensive interviews with the athletes that a more personal narrative is provided, thus creating a better understanding of the current impact of Neurological Soft Signs, and the impact that it has on athletes.

Methods

Participants

Participants were principally recruited through the assistance of both current and former professional athletes. Social professional networking sites such as LinkedIn were utilized. In addition, social networking sites were asked to grant permission to post on their site and granted permission to do so (See Appendix A for request to post the study form). A recruitment message was sent to acquaintances who referred people to the study (potential participants who the researcher did not know personally), which placed the researcher in contact with the participants. Through virtual snowball sampling, the participants were likely to know other athletes who shared the characteristics that made them eligible for study inclusion. However, a snowball sampling approach was implemented as needed in order to meet the requisite amount of athlete's eligible to take part in the proposed study. The participants in the proposed study consisted of former professional athletes who have retired. The researcher included 10 participants, which meets the sampling needs of qualitatively exploring the themes of an Interpretive Phenomenological Analysis [101]. It was projected that once the saturation point of 10 participants was reached, it was unlikely that additional collection of data would add to the themes being explored. Participants who responded to the researcher's post were contacted by the researcher by phone or email to set up a meeting to conduct the interview. They were also informed that the interview would be transcribed by a professional transcriber who signed a confidentiality agreement. Participants included in the study were limited to only former professional athletes who played their sport for at least one year and reported showing symptoms suggesting that NSS may have been present at the time of their head injury.

Measures

Demographic Questions: Demographic data regarding each of the former professional athlete's ethnicity, age, gender, number of years of playing their sport, and number of years that they played professionally were gathered (Appendix A).

Semi-Structured Interview Questions: After review of the literature on Neurological Soft Signs, Traumatic Brain Injuries, and Chronic Traumatic Encephalopathy treatment practices as well as a semi-structured interview instrument was constructed by the researcher as the primary method of data collection for the study (Appendix B). The final semi-structured interview consisted of 10 questions aimed at understanding the state of participants' soft neurological signs and problems and ascertained how they are functioning. The following content areas were covered: common practices of each person and challenges in diagnostic evaluations they have experienced. The questions were open-ended, which allowed for the possibility of idiosyncratic and spontaneous responses to be provided by each participant. Moreover, such an open-ended interview format will enable the researcher to provide a relevant guide to practicing clinicians to help patients elaborate where appropriate and most informative during their assessments and treatment of athletes.

Procedures

Upon meeting the criteria for participation in the proposed study, the researcher then discussed the nature of the project with each participant, verbally reviewed their rights as a participant, and provided an informed consent form. In circumstances in which the subject agreed to participate in the study, the researcher then worked with the participant to set a meeting time for the interviews.

The participants who met eligibility criteria were asked to voluntarily participate in a semi-structured interview for approximately one hour. Participants were informed that the interview would be audio recorded and transcribed for further analysis. Participants were informed that all information is kept confidential. Any identifying information was separated from their responses and excluded during transcription to ensure their confidentiality and maintain the data's integrity. A professional transcriber transcribed all of the interviews. A second reader also reviewed the transcriptions, and he also signed a confidentiality agreement. The second reader was used to calculate inter-rater reliability.

Upon the completion of the semi-structured interview, a verbal and written debriefing statement was provided with the contact

information of the researcher and any necessary wellness referrals. Participants were offered compensation in the form of a \$10 donation paid by the researcher to any charitable organization of the participant's choice. Last, participants requesting a summary of the results and recommendations following the completion of this research project were provided with this information by the investigator. All transcriptions are housed on a password- protected computer and all information was de-identified to protect the confidentiality of the respondents. In addition, the data will be stored for a total of five years in a securely locked bin and then shredded or destroyed.

Results

Introduction

The primary purpose of this study was to qualitatively investigate Neurological Soft Signs as they arise in the field of play in individual athletes. Neurological Soft Signs are associated with Traumatic Brain Injuries (TBI), Chronic Traumatic Encephalopathy (CTE), and other detrimental health risks and have been subject of various outcome studies aiming to identify the most effective treatment form. There is a current gap in the literature regarding whether NSS symptoms are a precursor to the development of CTE, especially concerning repeated head injuries during sports activities. This study provides further evidence supporting the existence of NSS, so that an early diagnosis of NSS can be examined as a potential preventive intervention to curb the development of CTE at a later age.

The purpose of this Chapter is to present the findings that arose from the analysis of the transcripts gathered from the individual interviews. The first section in the Chapter provides demographic information on the study participants. Next, the procedure for data collection is presented. A description of the data analysis conducted for the study then follows.

Demographics

The sample population included 10 participants who participated in a semi-structured interview. These participants were individuals who have had head injuries while playing professional sports and have shown signs of TBI. Most participants (n-8) were African American. The average age of participants was 41.3 years. Most participants (n=7) were married. Most participants (n=9) had children, with an average of two children per participant. Table 1 displays the participant characteristics. Participants were also asked about their experiences on a professional team. Most participants (n=8) played for the NFL. The average years of play was 8.1 years. All participants had concussions, with an average of four concussions. Table 2 displays professional experience characteristics.

Table 1: Participant Demographic Characteristics.

Participant	Age	Marital Status	Ethnicity	# of Children
1	38	single	mixed race (African American/Black/White)	0
2	46	married	African American/Black	3
3	33	married	African American/Black	2
4	39	single	African American/Black	1
5	51	married	African American/Black	3
6	45	married	African American/Black	2
7	37	single	African American/Black	1
8	43	married	African American/Black	4
9	44	married	Caucasian/White	3
10	37	married	Pacific Islander	3

Table 2: Professional Experience.

Participant	Professional Organization	Years of Play	Number of Concussions	Duration of Symptoms	Years since Last Head Injury
1	NBA	5	3	1 week	8
2	NFL	11	3	a few days	18

3	NFL	3	3	a couple days	7
4	NFL	5	3	a few days	9
5	NFL	10	6 or 7	hour or so	22
6	NFL	10	2	a couple days	19
7	NFL	2	3	a few days	17
8	NHL	10	3 or 4	a couple days	7
9	NFL	12	6	a couple days	19
10	NFL	13	2	a few hours	10

Specific themes

Following the recommended procedures for IPA, the researcher read and reread transcripts to identify possible themes. This step resulted in the identification of 46 specific themes. Next, the researcher reviewed these 46 themes for similarities to group these possible specific themes into a final, consolidated list of specific themes. Additionally, the researcher completed an inter-rater agreement process by having an independent reviewer who, like the researcher, is an advanced graduate student on internship in a doctoral, clinical psychology program. The independent reviewer also read the transcripts to identify specific themes, which produced an inter-rater reliability of 98%. After this process, repetitive themes were consolidated and agreed upon resulting in a final list of 35 themes. These themes were defined and given names that represented their content. The number of participants who contributed information to a theme was also noted. Table 3 displays the specific themes that were found in this study.

Table 3: Specific themes found in transcripts and number of participants endorsing each theme.

#	Theme	Number of Participants
1	Experiences with being hit	10
2	Worst experience being hit	10
3	Symptoms experienced during hit	9
4	Experience with being hit during practice	10
5	Symptoms experienced during play	10
6	Headaches experienced during play	8
7	Forgetfulness experienced during play	8
8	Blurred vision experienced during play	5
9	Trouble focusing experienced during play	3
10	No symptoms experienced during play	1
11	Clumsiness experienced during play	1
12	Symptoms experienced before sports	6
13	Headaches before sports	3
14	Blurred vision before sports	1
15	Experience with hiding a head injury	9
16	No experience with hiding a head injury	3
17	Previous experience hiding a head injury	6
18	Medical treatment	10
19	No prior hospitalization for head injury	6
20	Experience with hospitalization for head injury	2

21	Diagnostic testing following head injury	10
22	Flashlight testing	1
23	Concussion protocol	3
24	Basic tests	3
25	Imaging tests	5
26	Cognitive testing	1
27	Life after leaving professional sports	10
28	Decision to allow children to play	10
29	Would allow children to play contact sports	10
30	Other benefits of contact sports	7
31	Prerequisites for children to play	2
32	Worry about children playing contact sports	3
33	Headaches after playing sports	4
34	Blurry vision after playing sports	2
35	Effect of symptoms on life after sports	10

Higher Order Themes

After the list of specific themes was finalized, these themes were sorted into higher order themes based on similarities in content. Table 4 presents the finalized list of the five Higher Order Themes that arose from this iterative, qualitative analysis: Higher Order Theme 1: Head injury during professional play and practice, Higher Order Theme 2: Symptoms during play, Higher Order Theme 3: Experiences with symptoms before sports and after assessments of head injury, Higher Order Theme 4: Assessment of head injury, and Higher Order Theme 5: Influence of head injury in life functioning. The Specific Themes that make up the Higher Order Themes are listed under the Higher Order Theme that subsumes the Specific Theme.

Table 4: Higher Order Themes with the Specific Themes that Comprise Each Higher Order Theme with the number of participants who expressed that Specific Theme.

Higher Order Theme 1: Head Injury During Professional Play and Practice	Higher Order Theme 2: Symptoms During Play	Higher Order Theme 3: Experiences with Symptoms Before Sports and After Head Injury	Higher Order Theme 4: Assessment	Higher Order Theme 5: Influence of Head Injury
Specific Theme 1:	Specific Theme 5:	Specific Theme 12:	Specific Theme 18:	Specific Theme 27:
Experiences with being hit	Symptoms experienced during play	Symptoms experienced before sports	Medical treatment	Life after leaving profes- sional sports
N = 10	N=10	N=6	N=10	N=10
Specific Theme 2:	Specific Theme 6:	Specific Theme 13:	Specific Theme 19:	Specific Theme 28:
Worst experience being hit	Headaches experienced during play	Headaches before sports	No prior hospitalization for head injury	Decision to allow chil- dren to play
N = 10	N=8	N=3	N=6	N=10
Specific Theme 3:	Specific Theme 7:	Specific Theme 14:	Specific Theme 20:	Specific Theme 29:
Symptoms experienced during hit	Forgetfulness experienced during play	Blurred vision before sports	Experience with hospitalization for head injury	Would allow children to play contact sports
N=9	N=8	N=1	N=2	N=10

Specific Theme 4:	Specific Theme 8:	Specific Theme 15:	Specific Theme 21:	Specific Theme 30:
Experience with being hit during practice	Blurred vision experienced during play	Experience with hiding an injury	Diagnostic testing following head injury	Other benefits of contact sports
N=10	N=5	N=9	N=10	N=7
	Specific Theme 9:	Specific Theme 16:	Specific Theme 22:	Specific Theme 31:
	Trouble focusing experi- enced during play	No experience with hiding a head injury	Flashlight testing	Prerequisites for children to play
	N=3	N=3	N=1	N=2
	Specific Theme 10:	Specific Theme 17:	Specific Theme 23:	Specific Theme 32:
	No symptoms experienced during play	Previous experience hiding a head injury	Concussion protocol	Worry about children playing contact sports
	N=1	N=6	N=3	N=3
	Specific Theme 11:		Specific Theme 24:	Specific Theme 33:
	Clumsiness experienced during play		Basic tests	Headaches after playing sports
	N=1		N=3	N=4
			Specific Theme 25:	Specific Theme 34:
			Imaging tests	Blurry vision after play- ing sports
			N=5	N=2
			Specific Theme 26:	Specific Theme 35:
			Cognitive testing	Effect of symptoms on life after sports
			N=1	N=10

It can be seen from Table 4 that the Higher Order Themes tell a story of the athlete receiving a significant head injury (Higher Order Theme 1: Head Injury During Professional Play and Practice) that resulted often in immediate experiences (symptoms) of a brain injury (Higher Order Theme 2: Symptoms During Play). Higher Order Theme 3: Experiences with Symptoms Before Sports and After Head Injury indicates that the athletes continued to experience symptoms of a brain injury, and yet the athletes chose to deal with the symptoms at that time. At some point, there was assessment of the possible brain injury (Higher Order Theme 4), which brings us to the athlete's current condition and the lasting effects of the brain injury on the athlete's life (Higher Order Theme 5: Influence of Head Injury)

Higher Order Theme 1: Head Injury During Professional Play and Practice: relates to participants' descriptions of enduring a head injury during professional play and practice. Participants described their experiences of being the recipient of a big hit or the initiator of a big hit (Specific Theme #1: Experiences with being hit). They also indicated symptoms they experienced during head injuries (Specific Theme #5: Symptoms experienced during play) and whether they were incident symptoms or had been previously experienced prior to their most recent head injuries.

Higher Order Theme 2: Symptoms During Play: conveyed participants' depictions of the variety of symptoms they experienced as a result of being hit or initiating a big hit. Participants shared the symptoms they experienced during play, such as headaches (Specific Theme #6: Headaches experienced during play). They also indicated the extent to which they experienced other symptoms, such as forgetfulness (Specific Theme #7: Forgetfulness experienced during play), blurred vision (Specific Theme #8), trouble focusing (Specific Theme #9: Trouble focusing experienced during play), or clumsiness (Specific Theme #11: Clumsiness experienced during play).

Higher Order Theme 3: Experiences with Symptoms Before Sports and After Head Injury: includes experiences with symptoms before sports and after head injury. This Higher Order Theme encompassed participants' experiences with symptoms before they began playing

professionally (Specific Theme #12: Symptoms experienced before sports) and also included information about participants' hiding a head injury while playing (Specific Theme #15: Experience with hiding a head injury).

Higher Order Theme 4: Assessment of head injury: demonstrated the variety of techniques that were employed following a head injury to assess the injury to the brain. Participants identified different types of diagnostic testing they had received following a head injury (Specific Theme #10: No symptoms experienced during play). They also shared whether or not they had ever gone to the emergency room or had been hospitalized following a head injury (Specific Theme #20: Experience with hospitalization for head injury).

Higher Order Theme 5: Influence of Head Injury: included information about how participants have been affected by their head injuries. Participants conveyed the changes that happened after they retired from pro sports (Specific Theme #27: Life after leaving professional sports). They also shared any lingering symptoms they had related to concussions (Specific Theme #33: Headaches after playing sports and Specific Theme #34: Blurry vision after playing sports). In addition, participants noted the effect that these symptoms have had on their daily lives (Specific Theme #35: Effect of symptoms on life after sports).

Universal Specific Themes

There were 10 Specific Themes that were expressed by every athlete. These Specific Themes that were expressed by every athlete are termed: Universal Specific Themes. Thus, despite a sample size of only 10 athletes, there were some experiences that all the athletes had in common. In that sense, these experiences were universally, without exception, reported by every participant. Those 10 Universal Specific Themes reflect, in a simplified form, and tell the story that the Higher Order Themes reveal in an overall sense, and that the entire list of Specific Themes tells in a detailed form. These Universal Specific Themes are presented in Table 5 in the order of the Higher Order Themes in which they are subsumed.

Table 5: Universal Specific Themes (Specific Themes Expressed by Every Participant) and the Higher Order Themes in Which They Occurred.

Higher Order Theme 1: Head Injury During Professional Play and Practice	Higher Order Theme 2: Symptoms During Play	Higher Order Theme 3: Experiences with symptoms before sports and after head injury	Higher Order Theme 4: Assessment	Higher Order Theme 5: Influence of Head Injury
Specific Theme 1:	Specific Theme 5:	Specific Theme 15:	Specific Theme 18:	Specific Theme 27:
Experiences with being hit	Symptoms experienced during play	Experience with hiding an injury	Medical treatment	Life after leaving professional sports
Specific Theme 2:			Specific Theme 21:	Specific Theme 28:
Worst experience being hit			Diagnostic testing following head injury	Decision to allow children to play
Specific Theme 4:				Specific Theme 29:
Experience with being hit during practice				Would allow children to play contact sports
				Specific Theme 35:
				Effect of symptoms on life after sports

Specific Statements by Participants Illustrating the Specific Themes and Higher Order Themes

Tables 6-10 provide greater details regarding the specific statements that the athletes said that give concrete meaning to the Higher Order Themes. For each Higher Order Theme, specific quotes are provided to give insight into the experience being expressed by the athlete that the Higher Order Theme captures.

Table 6: Example quotes of Specific Themes and the number of participants who expressed that Specific Theme in parentheses for Higher Order Theme 1.

Higher Theme 1: Head Injury During Professional Play and Practice	Quote
Specific Theme 1: Experiences with being hit (10)	"I wouldn't know because the tight end kind of ear holed me, is what it's called. So, in the helmets, you have an ear hole in the helmet, and he hit me spam, smack, right in that ear hole, it felt like. And I just remember ringing in my ear and I had to come off the game and I think that was the worst time ever. But for the most part, thank God, I've been good." (P10)
Specific Theme 2: Worst experience being hit (10)	"My worst experience is I was fighting off of a pick, or screen, and the centre came down and I didn't see him. I was able to move enough out of the way, but it was a moving screen. And as soon as I turned, I mean, you're talking about someone who's 7 foot tall with 270, 280. As soon as I turn, boom, I run into him, and I take the brunt of the hit. And I think that was the worst because I didn't see it coming. And I ran into him and he had his, his body set for a pick." (P1)
Specific Theme 3: Symptoms experienced during hit (9)	"Getting up and trying to walk back and then falling back down. I don't even remember that you fall back down. Looking at practice and you're like, "Oh damn. I can't believe that's me." You know?" (P4)
Specific Theme 4: Experience with being hit during practice (10)	"We didn't do a lot of hitting In the NFL you don't really you got to pay these guys so they can't really risk hurting them too much at practice. In college we would do more, but I was playing corners, so I didn't typically get too much into that stuff." (P5)

Table 7: Example quotes that express Higher Order Theme 2.

Higher Theme 2: Symptoms During Play	Quotes
Specific Theme 5:	"Headaches where I have to take a Tylenol, but nothing major. No blurred
Symptoms experienced during play (10)	vision. No clumsiness. I do have problem focusing. Trouble focusing." (P1)
Specific Theme 6:	"If I sat here and thought about it, yeah, I'm sure I had a headache or two,
Headaches experienced during play (8)	growing up and during sports, maybe." (P6)
Specific Theme 7:	"Yeah, I'm absent minded sometimes, but I used to pride myself in not having
Forgetfulness experienced during play (8)	to write things down." (P7)
Specific Theme 8:	"Yeah Cometimes when I get stressed if I get angreen as " (DE)
Blurred vision experienced during play (5)	"Yeah. Sometimes when I get stressed, if I get angry or so." (P5)
Specific Theme 9:	"I de have trouble with forming It taken me a little hit to get form?" (DO)
Trouble focusing experienced during play (3)	"I do have trouble with focusing It takes me a little bit to get focus." (P8)
Specific Theme 10:	"I don't have any headaches, no blurred vision, no clumsiness, no trouble fo-
No symptoms experienced during play (1)	cusing, and no forgetfulness. I don't have anything like that, thankfully." (P3)
Specific Theme 11:	"You know what I mean? I feel like my balance, my equilibrium is not quite
Clumsiness experienced during play (1)	what it's supposed to be. You know what I'm saying? It's off a little bit." (P7)

Table 8: Example quotes that express Higher Order Theme 3.

Higher Theme 3:	
Experiences with symptoms before sports and after head injury	Quote
Specific Theme 12:	"I was a kid when I started playing. I can't remember. I can't remember having any
Symptoms experienced before sports (6)	headaches. I don't think I had any headaches. I've been playing for so long." (P2)
Specific Theme 13:	"And I think headaches were very rare as a child. If any, you know what I mean? To
Headaches before sports (3)	really speak of." (P7)
Specific Theme 14:	"Before sports I remember having headaches but I needed glasses. So I remember
Blurred vision before sports (1)	having a lot of headaches before I got my glasses, and then after I got my glasses then things were better." (P9)
Specific Theme 15:	"I played in the toughest NFL Division. We were known for hitting and hitting hard, so
Experience with hiding an injury (9)	you always covered up injuries and just kept playing. I'm not going to get into specifics, just know it happens." (P10)
Specific Theme 16:	"Not really. Never had to hide anything. I don't reallyNo, I never had to hide anything.
No experience with hiding a head injury (3)	Thankfully." (P1)
Specific Theme 17:	"Those are the times I told you those, those times I didn't come out the game, I didn't let
Previous experience with hiding a head injury (6)	nobody know nothing. I didn't feel like it was any of their business." (P5)

Table 9: Example quotes that express the Higher Order Theme 4.

Higher Theme 4: Assessment	Quote
Specific Theme 18: Medical treatment (10)	"Your basic tests. You have your CTs and your MRIs. I remember having that done in college, around one of my first concussions. I remember it happening twice in the Pros. But you do go through different testing throughout the year." (P2)
Specific Theme 19: No prior hospitalization for head injury (6)	"No. No. Nothing that bad. I've seen it happen, but nothing that bad that I've been involved in." (P3)
Specific Theme 20: Experience with hospitalization for head injury (2)	"I did go to the emergency one time when I woke up, it's like when your foot might fall asleep and my shit wasn't waking back up and it was bad. I'm like low key limping. And so I did go to the hospital about it that one time, I think that was the initial time. But it's happened a couple times since then too. Maybe three times since then. And they never really know." (P7)
Specific Theme 21: Diagnostic testing following head injury (10)	"I mean, you get a CT scan and an MRI, so those things are done. They do the kind of, the follow my finger test while you're on the field, or at least, that's what we did at, when I was playing. So wasn't much." (P3)
Specific Theme 22: Flashlight testing (1)	"I remember having the flashlight in your eye and going to the doctor each time it happens and then the doctor would tell me rest and don't have a TV on or loud noises. And coach would give me some time off. But I was always able to still hoop. So it wasn't nothing. No big deal." (P1)
Specific Theme 23: Concussion protocol (3)	"You have to go through Later in my career, especially they developed the concussion protocol, so you couldn't come back unless you were cleared." (P10)
Specific Theme 24: Basic tests (3)	"So you get tests done actually before the season, you get tests done during the season and after the season I think they call it like your baseline test and, a mid-season and then after as far as concussions and the way that your brain works, I don't know the words for them, but how your brain works." (P8)

Specific Theme 25:	"I remember actually, after college, that was a big thing for players to come back and have MRIs and CTs done because they were really getting into concussion studies and stuff like that. That
Imaging tests (5)	was a big thing. I've had MRIs and CT scans after playing. I've had them too because I want to give back as much study that can be done. I've had things done." (P6)
Specific Theme 26:	"I had a couple cognitive studies done one with the Cleveland clinic in Vegas when it wasn't
Cognitive testing (1)	Vegas, I'm not sure if it's there anymore." (P5)

Table 10: Specific themes and example quotes in Higher Order Theme 5.

Higher Theme 5:	Quote	
Influence of Head Injury		
Specific Theme 27:	"Life after professional sports has been good to me. I've been able to spend more time with my family." (P2)	
Life after leaving professional sports (10)		
Specific Theme 28:	"You know, injuries happen and they can happen anywhere. Anything you do. I know some people who didn't play any sports and tore, I tore my ACL, but this person I'm	
Decision to allow children to play (10)	thinking about, she tore ACL, she didn't play any sports. She actually just slipped. So injuries can happen anywhere." (P1)	
Specific Theme 29:	"Yeah. Contact sports allowed me to have a great college education at one of the most	
Would allow children to play contact sports (10)	storied universities, as far as football." (P3)	
Specific Theme 30:	"It provides discipline and work ethic. So, for kids to learn that And it's a great way for	
Other benefits of contact sports (7)	them to stay in shape and remain in shape." (P2)	
Specific Theme 31:	"if they can play and they are big like me." (P4)	
Prerequisites for children to play (2)		
Specific Theme 32: Worry about children playing contact sports (3)	"Do I think about some of the, do I want anyone to get hurt? No. Do I think about repercussions? Sure. I just think some people you have to do your best to just make sure that people are prepared. That means properly conditioned, lifting weights, running, making sure you know the plays, and you're studying them, and you're studying your playbook. And that's how you avoid serious injury." (P9)	
Specific Theme 33:	"I do get them now. They don't linger for so long. They might last an hour or two. I'll take some medication. And I'll get some water." (P2)	
Headaches after playing sports (4)		
Specific Theme 34:	"I get blurred vision maybe a couple of times a month maybe. But like I said, I probably	
Blurry vision after playing sports (2)	should wear my glasses more, but not really." (P6)	
Specific Theme 35:	"Network 14 kink and time it wishts but not as faithful " (D4)	
Effect of symptoms on life after sports (10)	"Not really. I think over time it might, but not as of right now." (P4)	

Discussion

This study focused on neurological soft signs (NSS), which are subtle neuro-abnormalities that are observable through conducting clinical examinations. The qualitative nature of this study design enabled the participants to tell their "story" of how their head injury has affected their lives. Many people engaging in sports sustain injuries that lead to the impairment of sensory and motor functions that also affect primitive reflexes. The focus on NSS marks a shift from Chronic Traumatic Encephalopathy (CTE), which has been extensively studied recently.

NSS can be a precursor to CTE, and therefore the study results can lead to recommendations that patients be diagnosed earlier to avoid further medical complications. Identifying the relationship between these conditions will ensure that NSS are not ignored because present vulnerabilities to Traumatic Brain Injury (TBI) or CTE in the future. Brain impairment requires tailored and detailed treatments before the

patient suffers from the long-term effects of the complications of head injuries [37]. However, appropriate treatment and prophylactic actions are not going to occur if it is not recognized that many cases of brain injury are being missed or ignored.

General Findings

There were several key findings that developed from interviews with participants. First, participants shared their experiences with being hit during games and practices (Higher order theme 1: Head Injury during Professional Play). All participants reported their worst experiences with being hit during a game. While some participants distinctly remembered the events leading up to their concussions, some participants did not have much memory of the event. They instead learned about it from other teammates or realized from watching it on tape. In addition to describing experiences with being hit during games participants also commented on being hit in the head during practices. In contrast, some participants reported never experiencing any big hits during practice. For example, participant 5 mentioned, "We didn't do a lot of hitting... In the NFL you don't really... you got to pay these guys so they can't really risk hurting them too much at practice." These participants expressed the range of exposures to hits during their careers.

Participants also commented on the symptoms they experienced after their head injury (Higher order theme 2: Symptoms During Play). These symptoms included headaches, blurred vision, forgetfulness, and clumsiness. There were eight participants who experienced headaches after a head injury. Some participants described their headaches in more detail. For example, participant 1 noted, "I get headaches. I think like anybody else. They're not major. They're just kind of lingering in the background. I can carry on with my day. A couple of times there might be really bad headaches or at least for me, that's bad."

There were five participants who endorsed experiencing blurred vision. Participant 5 shared the circumstances in which he experienced blurred vision, "Sometimes when I get stressed, if I get angry or so." As a last example, one participant indicated that he experienced clumsiness, "I feel like my balance, my equilibrium is not quite what it's supposed to be. You know what I'm saying? It's off a little bit."

In addition to indicating the symptoms they experienced after head injuries, participants also shared if they had any of these symptoms prior to playing sports (Higher order theme 3: Experiences with Symptoms Before Sports and After Head Injury). Most participants did not endorse any symptoms previously. Only three participants posited that they potentially experienced rare headaches when they were children.

Another key finding was that participants received different types of assessments and different levels of medical attention following their head injuries (Higher order theme 4: Assessments of Head Injury). Most participants had some type of imaging diagnostic test. They also reported on whether they had to be admitted to the hospital following a hit. Only a few participants had indicated that they had been hospitalized or admitted to the hospital for a head injury (n=3). In addition, participants commented on whether they had ever hidden a head injury from their medical team. Many participants (n=9) endorsed the experience of hiding a head injury at some point.

A third key finding was that many of the participants in this study reported a limited influence that head injuries had on their lives post-retirement (Higher order theme 5: Influence of Head Injury). Most participants described being very satisfied with their lives after they stopped playing in a professional sports league. Participant 2 conveyed, "Life after professional sports has been good to me. I've been able to spend more time with my family." In contrast, other participants indicated they were still adjusting to their lives without professional sports. For example, participant 4 indicated he was still adjusting, "Oh, the real world. I mean, adjusting to not the catering, the catering aspect and really doing everything for yourself." All but one participant shared that they did not have many symptoms after their retirement that influenced their daily lives.

In addition to describing their life post-sports, participants also shared if they would allow their children to play contact sports. All participants reported that they would allow their children to play. Participant 10 stated, "I have a family, we love football, we're a football family and my boys or my nephews, anything like that want to play, I will let them play." Although all participants said they would allow their children to play, some did voice concerns about having their children play contact sports. For instance, participate 9 opined, "So yeah you worry, but also in that worry you make sure that they're conditioned, and they're fed well, and they take their, it sounds silly but their multivitamins, and protein supplements, or whatever they need to get their bodies in the right position." Participant 4 also qualified participation with a requirement, "Yes, if they can play and they are big like me."

Interpretation of the Findings

This study explored the research findings from the qualitative interviews that were conducted with 10 former professional athletes who had suffered from a head injury. The findings of this study align with some of the existing literature, while other extend what has previously been researched in this area.

The current study sought to examine if people who had sustained head injuries from sports had experienced difficulties resulting from those head injuries that may well have been missed or ignored at the time. Previous research has identified some effects of injuries, ranging from mild neurologic symptoms to mood disorders and physical disability. Selassie et al. noted that in cases of TBI such as a concussion, these types of injuries can result in chronic impairment and disabilities with long term neuropsychological implications [29]. Further, over 40% of the people who have endured a TBI usually developed some sort of functional deficits for an extensive period of time, some surpassing one full year. Additionally, Artman LK & McMahon BT [3], noted that many individuals with TBI injuries have vast limitations as a result of residual symptoms related to the condition [30]. Some of these include sensory deficits, loss of gross and fine motor activity, and physiological and cognitive impairments that may lead to a suboptimal level of efficiency in their former careers. In addition, many authors have termed cognitive and emotional deficits as the most permanent and self-limiting consequences of TBIs.

In the current study, participants shared the extent to which they experienced symptoms after they stopped playing sports. Only one person described suffering from headaches that occasionally interfered with daily life. For the most part, other participants shared symptoms they experienced more immediately following a head injury, namely headache, blurred vision, forgetfulness, and clumsiness. However, these participants did not reveal that symptoms persisted after their retirement. Therefore, findings from the current study provide a different longitudinal experience for individuals suffering from head injury. Despite not endorsing symptoms of lasting brain insult, clinically the participants may in fact meet the criteria for stage I of CTE as they are self-reporting symptoms that match, such as, headaches, loss of attention/concentration and executive function issues [22]. However, at all times, it must be remembered that this study interviewed individuals who self-selected to participate in this study. The fact that these athletes were generally well functioning, despite clearly identifying various functional problems suggests that this study touched the tip of an iceberg. In addition, these participants have been out of their professional sport for 1 to 22 years, thus, as uncomfortable as it is to point out, some of their problems may have yet to emerge.

Another finding from the current study was that participants received varying medical treatment following their injuries. All participants reported receiving similar diagnostic testing, yet only one participant reported completing cognitive testing. Previous literature had demonstrated the importance of including testing beyond traditional imaging to determine the extent of damage after a head injury. McCrory P, et al. [44], state that a concussion may fail to cause a substantial structural injury, hence cannot be detected by means of conventional neuroimaging. In the case where an athlete indicates any concussive clinical symptoms, evaluations and neuropsychiatric testing should be conducted [45]. Furthermore, Gardner A, et al. [39], state that CTE is currently defined as chronic cognitive as well as neuropsychiatric symptoms of the type of chronic neurodegeneration that usually follows an episode of a severe TBI [40]. This previous work highlights the importance of cognitive testing for both evaluating initial effect of head trauma and monitoring changes over time. The current study reveals the lack of consistent neuropsychological testing of athletes with suspected head injury.

Clinical Implications

Hiding Head Injuries

The results from this study have several implications. One implication of the study was the finding that many participants disclosed hiding a head injury from their medical team. Previous literature had demonstrated the importance of identifying, monitoring, and potentially intervening following a head injury. In the case where an athlete indicates any concussive clinical symptoms, evaluations and neuropsychiatric testing should be conducted. Ling H, et al. [37], recommended that the athlete should rest for around 24-48 hours immediately after a head trauma as this is an important time period for recovery in the acute symptomatic phase [30]. Giza CC, et al. [46], suggest that many cases of concussion usually get resolved in about 7 to 10 days, although children and some adolescents can experience a longer duration of symptoms [46]. The current study reveals the prevalence of hiding potential head injuries in a small sample. Participants shared that due to the consequences of missing practice or the atmosphere of the team, they would not disclose a head injury. This belief and practice would be important to address in order to reduce the impact these injuries can have on the lives of athletes.

Psychoeducation

Common phrases participants heard while playing professional sports were that athletes are "spare tires" or "cattle," meaning they could be easily replaced in their sport usually by someone younger and cheaper. This phrasing is part of the culture that causes athletes to participate despite injury. Moreover, the comradery of many sports includes a paramilitary element praising toughness and the "warrior spirit." Many times, athletes forsake their wellbeing to ensure their livelihoods, or for the prospect of generational wealth and prosperity. While some may know the risks of injury that comes with their sports, they often are not taught the dire consequences. Athletes should be advised on the studies

which have revealed that undisclosed TBI may result in neuronal excitotoxicity. Irreversible damage of neuronal membrane due to oxidative stress caused by concussions is attributed to secondary injuries causing dysfunction and cell death [66]. Overall, professional athletes while willing participants need to be routinely educated on the severity of the health risks of their sport.

Traumatic Brain Injuries sustained by athletes are not only a social issue, but a public health issue. Likewise, Jordan describes brain injuries as "substantial public health concern" and emphasizes that when not detected or mismanaged there are greater consequences. In order to prevent athletes from suffering the lasting and life changing symptoms of NSS it will take a significant change [102]. Indeed, coaches, players and medical personnel have a responsibility of ensuring that they identify and disclose brain injuries among their athletes for the necessary recuperative measures to be undertaken in time, which requires psychoeducation about how to recognize NSS. According to the Centers for Disease Control and Prevention (CDC), the rising rates of traumatic brain injuries can be curbed when these conditions are identified and disclosed early. Disclosing brain injuries can help in establishing whether the current diagnostic and treatment techniques can effectively manage these conditions at a particular stage of the injury [93]. Taking such measures will help to reduce the long-term effects of concussions, often resulting in death. Therefore, identifying and educating participants about brain injuries help in providing the most appropriate medical advice and treatment so athletes may recover and resume their normal activities.

Furthermore, there should more awareness regarding the level of severity of brain trauma regardless of how the impact may appear. It may be common to think that there needs to be a large collision or what some would call a "boom." A study by Blennow et al. [14], revealed that CTE is caused by both sub concussive and concussive brain insults which are repeated [4]. Therefore, identifying and disclosing brain injuries among athletes help in avoiding repetitive concussions that eventually cause CTE. Also, early identification and diagnosis of brain injuries help in providing accurate data regarding the causes, types and frequency of brain injuries. Psychoeducation and early detection can create a pathway to collect data which may assist in knowing the ages when players are more prone to brain injuries and whether some athletes could be genetically more susceptible to intense concussions.

Symptoms

A further implication of this study is the demonstration of varied effects that a head injury can have. Participants commented on the types of symptoms they experienced after their injuries. The most commonly reported symptoms were headaches and blurred vision. These symptoms fit into McKee's Stage I. In stage I, there is no change in brain weight, but there is mild ventricular enlargement, and it presents with headaches, loss of attention and the ability to concentrate. Altogether, these athletes can suffer from dysexecutive functioning, behavioral changes, and mood dysregulation. Although, to an untrained individual, such as family members, loved ones, or coaches, these athletes may present as asymptomatic, and NSS symptoms may have gone unnoticed due to lack of psychoeducation on the topic. Also, symptoms may have been masked by the athlete, thought to be minor, or explained as a part of the adjustment to life without the sport if they retire. However, most athletes endorse what appears to be Stage I of CTE, including short-term memory loss, elevated aggression, and depression, with a few being diagnosed with Post Traumatic Stress Disorder, losing their executive function and explosivity in their emotions [22]. In addition, many of the participants met the qualifications for a phrase the researcher has described as "functionally debilitated": when athletes appear to be functional in daily life due to their tactical awareness, muscle memory, grittiness, and for some, their resources. These protective factors appear to be innate to an athlete, are often nurtured while playing sports, and may have been necessary to reach the professional level of competition.

Only a few participants endorsed experiencing any symptoms that fit into McKee's Stage II. Stage II presents with gliosis and atrophy of the mammillary bodies with well delineated tau protein foci in the cerebral cortex, which can only be observed and confirmed, in an autopsy, post-mortem. The patient presents with depression, unstable moods and short-term memory deficits. Stage III presents with mild cerebral atrophy and ventricular dilation with a similar presentation with milder forms in addition to aberrant thought processes, impulsivity and suicidal tendencies. Most of the patients in this group can be termed to be cognitively impaired. Participants in the current study expressed occasional forgetfulness. The current study demonstrates that participants who suffered from NSS did go on to experience minor symptoms of CTE later, which a few in the McKee's Stage I [8]. This finding has implications for the diagnosis of CTE and the usage of NSS when considering diagnostic criteria. The research results indicated that all athletes involved were negatively impacted by head trauma in at least the areas of cognition, memory, attention, executive functioning, and emotional control. Furthermore, most of the participants have communicated poor insight of the severity of symptoms from brain trauma. Identifying NSS may help catch debilitating symptoms of CTE early enough to help these individuals and refer them to a neurorehabilitation program, despite their lack of reporting using clinical terminology, which may lead to a better quality of life.

Assessment of NSS

Another major clinical implication that this study motivated is the need for clinical psychologists to be alert to the assessment of NSS. The participants in the current study reported a variety of assessments they received. However, these assessments were generally imaging or basic testing. Instead of limiting athletes to reporting on symptoms that have been previously expected in these types of injuries, offering more open-ended questions could help to better understand the extent of the impact of a head injury. The questions in this study were open-ended, which allowed for the possibility of idiosyncratic and spontaneous responses to be provided by each participant. If this format were used by clinicians, it would help patients elaborate where appropriate and relevant.

My clinical recommendation is that when working with athletes who have head injuries, one should first use Appendix E: Semi-Structured Interview Schedule. Following this, a clinician should use some combination of the following neuropsychological assessments at minimum twice a year to go along with their annual physical exam, with increasing intervals based on clinical judgment:

- Clinical Interview
- The Neuropsychological Assessment Battery (NAB)
- Hopkins Verbal Learning Test-Revised (HVLT-R; Form 1)
- Brief Visuospatial Memory Test-Revised (BVMT-R; Form 1)
- Symbol Digit Modalities Test (SDMT; Form A):
- Standard and Memory Versions
- Color Trails Tests 1 and 2 (CTT1, CTT2; Form A)
- Pennsylvania State University Cancellation Test (PSU; Form A)
- Controlled Oral Word Association Test (COWAT; FAS)
- Brief Symptoms Inventory-18 (BSI-18) OR Geriatric Depression Scale (GDS)
- Referral from clinician for a computerized tomography (CT) scan/ Magnetic resonance imaging (MRI)

When choosing a test battery from the list above, a clinician should be mindful to assess for all areas of NSS: motor coordination, sensory integration, complex motor tasks, spatial orientation, and primitive reflexes. Additionally, clinicians should monitor for changes in a patient's anxiety and mood profiles; therefore, the test battery should include an assessment to measure emotional or psychological distress. The benefit of the following assessments tools is that they are brief and provide both narrative and robust clinical data. This population requires assessments tools that are easily accessible and accommodate for inattentiveness. A clinician should spend time on the clinical interview to create a space of trust and gain an understanding of the player's vernacular and use of their lexicon, aside from clinical jargon. Expanding the clinical data available is essential to understand best the impact an athlete's trauma has on their cognition and functioning; in contrast, the current data collected rarely helps the athlete while living. Athletes with brain trauma should be regarded as whole people with families and loved ones who depend on them; they deserve the help of clinicians while living. Therefore, routine assessments should be considered the norm by healthcare providers.

In addition to the need for changes in the diagnosis of head injury, there is also a need for further treatment following a head injury. It is critical that professional athletes seek therapy following a head injury, even when NSS are the only symptoms present.

Treatment

Once identified, Neurological Soft Signs can inform the way clinicians move forward with treatment recommendations. Cognitive neurorehabilitation (CR) offered by a Clinical Psychologist and an allied health team may prove to be an essential therapeutic approach in integrating rehabilitation-oriented cases, especially for patients with acquired brain injuries. Fortunately, cognitive rehabilitation can usually be applied in many different settings and modalities such as inpatient, outpatient, as well as individual, family, and group sessions. Likewise, CR can be beneficial at all stages post-injury, and in conjunction with neuroplasticity, can give hope to patients who may be suffering from reduced ability to process, mood disturbances, abnormal behaviors and many other symptoms that result from long-standing past head trauma [73].

Clinical psychologists provide cognitive data that includes emotions and behaviors of patients who have suffered a certain injury or illness

that affected their cognitive abilities. Oberholzer M & Muri RM [92], agree that rehabilitation is a critical part of recovery as it enables patients to improve after an injury. Neuropsychologists are important in the recovery process and should be included in a rehabilitation setting for patients with brain-related disorders as they have diverse training regarding emotional, behavioral, and cognitive difficulties. Objective cognitive data is preferred in neurorehabilitation that is aimed at magnifying the level of functioning of a patient out of their usual environment [91].

Identifying neurological soft signs is clinically important because it can be a diagnostic tool for clinicians. Jöhr J, et al. [64], contend that neuropsychology aids in distinguishing between clinical phenotypes of neurological disorders mental disorders and evaluating their functions for diagnostic criteria and procedures. Hence, neuropsychology is clinically important as it has an impact on the rehabilitation of patients with brain disorders. According to Jöhr J, Et al. [64], neurological diseases can be a result of a certain deficit or unwholesomeness of a certain burden in the community. Some of the neurological diseases that are likely to affect young adults include cerebral palsy and trauma [35]. Neurorehabilitation of the brain is not similar as the extent of the brain damage determines the kind of care to be accorded by neuropsychologists as advised by clinicians. Damage to the brain is often caused by ischemic strokes and is determined by hypertensive hemorrhagic damage that can be localized anywhere in the brain.

Traumatic Brain Injury (TBI) has long-term effects; hence it is a public health concern. Pundlik J et al. [96], claim that the clinical assertion of this complexity varies as different clinicians have different opinions [56]. Besides, individuals with mild TBI can resume their daily functioning quickly and mostly within a year of rehabilitation. However, a few individuals with mild TBI suffer progressive cognitive, emotional, physical, and behavioral symptoms. The clinical implication of TBI follows an initial sequence of coma, disturbed consciousness, and then post-traumatic agitation, and finally, the recovery of body functions occurs [103]. These specific phases of rehabilitation are significant in enhancing the recovery of patients who suffer from brain injuries. Besides, physicians and neuropsychologists follow a specific treatment protocol with a positive clinical implication; for example, brain injury education followed by supportive counseling for the cultivation of treatment team consultations, collaborations, planning, and team feedback about post-injury recovery [56]. Neuropsychologists stress teamwork during rehabilitation as it enables them to understand the level of brain injury and the expected approach to such cases. Neuropsychologists are also aware of the medical complications that may come up such as posttraumatic hydrocephalus, posttraumatic neuroendocrine dysfunctions, and paroxysmal sympathetic hyperactivity [103]. In addition, mild TBI causes cognitive disruptions such as poor emotional control, irritability, and lack of initiative.

There are different ways in which patients can improve with neurorehabilitation. Physicians play a significant role in cognitive rehabilitation. They prepare a detailed history, carry out physical exams, prescribe medication, review high-risk medications, and oversee medication compliance for patients. Often physicians assess and refer patients to neuropsychologists. They interact and work with neuropsychologists to realize the best health outcomes in patients with cognitive, emotional, and behavioral disorders [104].

Khan et al. contend that neurorehabilitation can focus on different areas that will enhance recoveries, such as physiotherapy, speech (language) therapy, occupational therapy, and vision therapy [41]. Physiotherapy enables the body to restore the normal functioning of body muscles by restoring function and movement, especially when a person has suffered an injury. This approach employs a holistic approach that protects the patient from future injury. In addition, it enables the patient to remain stable as different body parts are interconnected with the brain's functioning. Also, speech (language) therapy is essential in neurorehabilitation as it enables the patients to regain their lost sense of functioning, such as movement of the lips and jaws. This technique is specifically essential to children with cerebral palsy. It enables the children to make better-understood speech or words and reduces the difficulty experienced with cerebral palsy.

Occupational therapy also aids patients in the recovery process during rehabilitation. According to Roemmich RT & Bastian AJ [100], occupational therapists' control and manage challenging behaviors and cognitive problems that affect the daily lives of people through acquitting them with special learning sessions [105]. The skills utilized by occupational therapists include problem-solving, attention, and processing speed to facilitate understanding of educative concepts. Vision therapy can also reduce symptoms in patients with neurological diseases. In this case, the idea utilized is that vision is a basic and primary sense that coordinates almost all body functions such as communication, information gathering, and orientation. Therefore, vision is extensively networked with the brain as it integrates the crucial body functions. Consequently, attention process training that requires visual involvement is essential as it will aid in reducing the symptoms of neurological diseases during neurorehabilitation [106,107].

To sum up, neurorehabilitation focuses on improving the functioning of the patient by providing them robust treatment options which promotes neuroplasticity and may be more stimulating than their home environment. This kind of rehabilitation covers all neurological

diseases, including TBI, and facilitates psychological adaptation to disability by encouraging social integration. Also, neurorehabilitation follows a specific process and is divided into interconnected phases for effective results in post-injury recovery. Neuropsychologists work as a team to ascertain various complexities that may be linked to cognitive adaptiveness [108-110].

Limitations

One limitation of this study was the sample size of 10 participants. A larger sample would enable the depiction of the characteristics of NSS not only on the population utilized but would enable greater generalizability. Additionally, this study included only male participants; future studies would benefit greatly from including female athletes. There may be unique experiences female athletes face. Furthermore, their contributions and representation in sports has been remarkable and clinicians should take note of their narrative moving forward. Likewise, the non-probability sampling means that the sample was selected in a snowball fashion, and therefore there are limitations on the degree of representativeness of the entire population.

Another limitation of this study was that the study was not designed to use medical records as another source of information. Therefore, there was a lack of measures to investigate participants whose NSS symptoms may have been missed before TBI symptoms could be detected. Thus, the study relied on the memories of the participants, which, in addition to normal forgetting and distortion, may be impaired due to the head injuries. Despite this limitation in the method of collecting data, the results of the study, will provide a basis for further research so that cognitive and mental disorders affecting athletes with head injuries can be identified and treated at an early stage, as suggested by Bachman and Schroder [6].

Recommendations for Future Studies

The current study was limited to research among a small participant pool using a qualitative methodology. Such an approach reduces the generalizability to the larger population and instead allowed for generalization only to highly similar contexts. For example, the participants in this study were primarily former NFL players. Therefore, the applicability of these findings might be reduced when considering additional populations (e.g. other contact sports). To combat this limitation, future research could expand this study to a larger participant pool. For example, the study could be conducted with a larger sample of participants who had retired from professional sports teams. This could include greater numbers of former NHL and NBA players. It could also include non-contact sports, such as cycling and gymnastics. Moreover, future research could include women's professional sports athletes. In addition, years since retirement has to be a major variable under investigation.

Another recommendation is to use an alternate research design, such as a quantitative methodology to build upon the existing findings in a larger sample. Using surveys or another quantitative methodology, like reviewing medical charts, would allow for data to be collected from a larger population who could be more widely surveyed. This methodology also provides the researcher with a way to evaluate the strength and direction of relationships in the datasets. The resulting data, assuming it is taken from a sufficiently sized population, would be generalizable to the larger population.

Summary and Conclusions

The analysis of these interviews with former professional athletes who had suffered from a head injury revealed multiple Higher Order and Specific Themes that comprised these Higher Order Themes that were related to the overarching research question for this study. The first Higher Order Theme, Head injury during professional play, included participant reports of experiences they had with being hit during games and practices. They described their worst experiences with being hit.

The second Higher Order Theme was Symptoms during play. This Theme included participants depictions of the variety of symptoms they experienced after being hit. Participants indicated whether the experienced headaches, forgetfulness, blurred vision, trouble focusing, or clumsiness.

The third Higher Order Theme, Experiences with symptoms before sports and after head injury, conveyed the participants experiences with various head injury symptoms. They indicated which symptoms they experience before they began playing professionally. This Higher Order Theme also included information about participants' hiding a head injury while playing.

The fourth Higher Order Theme, Assessment, included participant portrayals of the attention they received after a head injury. Participants shared the types of diagnostic testing they received after their injury. They also reported on whether they had to be admitted to the hospital

following a hit. Further, participants commented on whether they had ever hidden a head injury from their medical team.

The fifth and final Higher Order Theme, Influence of head injury, covers participants beliefs about the effect that a head injury has had on their lives after retiring from a professional sports league. Some participants described having great lives they were very satisfied with after retirement. In contrast, other participants indicated they were still adjusting to their lives without professional sports. Additionally, participants disclosed whether they would let their children play in a contact sport. Finally, participants noted any persisting symptoms they had form their head injury and indicated if these symptoms interfered with their lives.

In addition to the future research directions this study motivates, the findings of this qualitative study have implications for clinical practice. This study aimed to examine the effect of experiencing neurological soft signs on outcomes throughout life for former professional athletes. Better understanding this topic can inform the diagnosis and treatment of these injuries in the future. The findings of this study demonstrate a slight connection between neurological soft signs and later functional impairment. However, this association was not observed among all participants. Another important revelation was regarding diagnostic testing. Only one participant in this study received cognitive testing following a suspected head injury. Furthermore, many participants reported hiding their injuries in order to avoid missing practice or game time. These gaps in care need to be addressed to reduce the potential short-term and long-term effects of head injuries. This study has several clinical implications, including the need for more open-ended questions in assessments of patient's post-head injury. Given the qualitative nature of the study, the researcher recommends that a larger, quantitative research study be conducted based on the current findings in order to expand the generalizability of the study.

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Conflict of Interest

None of the authors have a conflict of interest.

References

- 1. Alwis DS, Yan EB, Morganti-Kossmann MC, Rajan R (2012) Sensory cortex underpinnings of traumatic brain injury deficits. PLoS One 7(12): e52169.
- 2. Ann C McKee, Brandon E Gavett, Robert A Stern, Christopher J Nowinski, Robert C Cantu, et al. (201) TDP-43 Proteinopathy and Motor Neuron Disease in Chronic Traumatic Encephalopathy. J Neuropathol Exp Neurol 69(9): 918-929.
- 3. Artman LK, McMahon BT (2013) Functional limitations in TBI and their relationship to job maintenance following work re-entry. Journal of Vocational Rehabilitation 39(1): 13-21.
- 4. Asken BM, Sullan MJ, DeKosky ST, Jaffee MS, Bauer RM (2017) Research gaps and controversies in chronic traumatic encephalopathy: a review. JAMA Neurol 74(10): 1255-1262.
- 5. Bachmann S, Degen C, Geider FJ, Schroder J (2014) Neurological soft signs in the clinical course of schizophrenia: results of a meta-analysis. Front Psychiatry 5: 185.
- 6. Bachmann S, Schroder J (2018) Neurological Soft Signs in Schizophrenia: An Update on the State-versus Trait-Perspective. Front Psychiatry 8: 272.
- 7. Toro P, Schroder J (2018) Neurological Soft Signs in Neuropsychiatric Conditions. Front Psychiatry 9: 736.
- 8. Bailes JE, Petraglia AL, Omalu BI, Nauman E, Talavage T (2013) Role of subconcussion in repetitive mild traumatic brain injury. J Neurosurg 119(5): 1235-1245.
- 9. Dazzan P, Murray RM (2002) Neurological soft signs in first-episode psychosis: a systematic review. Br J Psychiatry Suppl 43: s50-s57.
- 10. Barkhoudarian G, Hovda DA, Giza CC (2011) The molecular pathophysiology of concussive brain injury. Clin Sports Med 30(1): 33-48.
- 11. Bigler ED (2013) Traumatic brain injury, neuroimaging, and neurodegeneration. Front Hum Neurosci 7: 395.
- 12. Bigler ED (2018) Structural neuroimaging in sport-related concussion. Int J Psychophysiol 132(Pt A): 105-123.
- 13. Blennow K, Brody DL, Kochanek PM, Levin H, McKee A, et al. (2016) Traumatic brain injuries. Nat Rev Dis Primers 2: 16084.
- 14. Blennow K, Hardy J, Zetterberg H (2012) The neuropathology and neurobiology of traumatic brain injury. Neuron 76(5): 886-899.
- 15. Castellani RJ (2015) Chronic traumatic encephalopathy: A paradigm in search of evidence? Laboratory Investigation 95(6): 576-584.

- 16. Castellani R, Schmidt C (2018) Brain injury biomechanics and abusive head trauma. Journal of Forensic Science and Medicine 4(2): 91.
- 17. Kulbe JR, Hall ED (2017) Chronic traumatic encephalopathy-integration of canonical traumatic brain injury secondary injury mechanisms with tau pathology. Prog Neurobiology 158: 15-44.
- 18. Cherry JD, Tripodis Y, Alvarez VE, Huber B, Kiernan PT, et al. (2016) Microglial neuroinflammation contributes to tau accumulation in chronic traumatic encephalopathy. Acta Neuropathol Commun 4(1): 112.
- 19. Ng SY, Lee A (2019) Traumatic Brain Injuries: Pathophysiology and Potential Therapeutic Targets. Front Cell Neurosci 13: 528.
- 20. Chrobak AA, Siuda-Krzywicka K, Sołtys Z, Siwek GP, Bohaterewicz B, et al. (2021) Relationship between neurological and cerebellar soft signs, and implicit motor learning in schizophrenia and bipolar disorder. Progress in Neuro-Psychopharmacology and Biological Psychiatry 111: 110137.
- 21. Gunasekaran V, Venkatesh VM, Asokan TV (2016) A Study of Soft Neurological Signs and Its Correlates in Drug-Naive Patients with First Episode Psychosis. Indian J Psychol Med 38(5): 408-413.
- 22. Churchill NW, Hutchison MG, Graham SJ, Schweizer TA (2021) Acute and chronic effects of multiple concussions on midline brain structures. Neurology 97(12): e1170-e1181.
- 23. Clark JE, Sirois E (2020) The possible role of hydration in concussions and long-term symptoms of concussion for athletes. A review of the evidence. Journal of Concussion 4: 205970022093940.
- 24. Cloots RJH, Gervaise HMT, van Dommelen JAW, & Geers MGD (2008) Biomechanics of traumatic brain injury: Influences of the morphologic heterogeneities of the cerebral cortex. Ann Biomed Eng 36(7): 1203-1215.
- Coronado VG, Haileyesus T, Cheng TA, Bell JM, Haarbauer-Krupa J, et al. (2015) Trends in sports-and recreation-related traumatic brain injuries treated in US emergency departments: The National Electronic Injury Surveillance System-All Injury Program (NEISS-AIP) 2001-2012. J Head Trauma Rehabil 30(3): 185-197.
- 26. Cox SM, Ludwig AM (1979) Neurological soft signs and psychopathology: incidence in diagnostic groups. Can J Psychiatry 24(7): 668-673.
- 27. Dazzan P, Morgan KD, Chitnis X, Suckling J, Morgan C, et al. (2006) The structural brain correlates of neurological soft signs in healthy individuals. Cerebral Cortex 16(8): 1225-1231.
- 28. Selassie AW, Wilson DA, Pickelsimer EE, Voronca DC, Williams NR, et al. (2013) Incidence of sport-related traumatic brain injury and risk factors of severity: a population-based epidemiologic study. Ann Epidemiol 23(12): 750-756.
- 29. DeKosky ST, Blennow K, Ikonomovic MD, Gandy S (2013) Acute and chronic traumatic encephalopathies: Pathogenesis and biomarkers. Nat Rev Neurol 9(4): 192-200.
- 30. Eierud C, Craddock RC, Fletcher S, Aulakh M, King-Casas B, et al. (2014) Neuroimaging after mild traumatic brain injury: Review and meta-analysis. NeuroImage Clin 4: 283-294.
- 31. Etikan I, Bala K (2017) Sampling and sampling methods. Biometrics & Biostatistics International Journal 5(6): 00149.
- 32. Evans V (2020) Newton's Laws, G-forces and the impact on the brain. Australasian Journal of Neuroscience 30(1): 24-29.
- 33. Mez J, Daneshvar DH, Kiernan PT, Abdolmohammadi B, Alvarez VE, et al. (2017) Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football. JAMA 318(4): 360-370.
- 34. Fountoulakis KN, Panagiotidis P, Tegos T, Kimiskidis V, Nimatoudis I (2021) Paternal age and specific neurological soft signs as reliable and valid neurobiological markers for the diagnosis of patients with schizophrenia. Eur Arch Psychiatry Clin Neurosci 272(6): 1087-1096.
- 35. Sezgin Caglar A, Tanriverdi F, Karaca Z, Unluhizarci K, Kelestimur F (2019) Sports-related repetitive traumatic brain injury: a novel cause of pituitary dysfunction. J Neurotrauma 36(8): 1195-1202.
- 36. Galgano M, Toshkezi G, Qiu X, Russell T, Chin L, et al. (2017) Traumatic brain injury: Current Treatment Strategies and Future Endeavors. Cell Transplant 26(7): 1118-1130.
- 37. Ling H, Hardy J, Zetterberg H (2015) Neurological consequences of traumatic brain injuries in sports. Mol Cell Neurosci 66(Pt B): 114-122.
- 38. Galindo L, Pastoriza F, Berge D, Mane A, Picado M, et al. (2016) Association between Neurological Soft Signs, Temperament and Character in Patients with Schizophrenia and Non-psychotic Relatives. Peer J 4: e1651.
- 39. Gardner A, Iverson GL, McCrory P (2014) Chronic traumatic encephalopathy in sport: a systematic review. Br J Sports Med 48(2): 84-90.
- 40. Gandy S, Ikonomovic MD, Mitsis E, Elder G, Ahlers ST, et al. (2014) Chronic traumatic encephalopathy: Clinical-biomarker correlations and current concepts in pathogenesis. Mol Neurodegener 9: 37.
- 41. Stein TD, Alvarez VE, McKee AC (2014) Chronic traumatic encephalopathy: a spectrum of neuropathological changes following repetitive brain trauma in athletes and military personnel. Alzheimer's Res Ther 6(1): 4.

- 42. Yue JK, Winkler EA, Burke JF, Chan AK, Dhall SS, et al. (2016) Pediatric sports-related traumatic brain injury in United States trauma centers. Neurosurg Focus 40(4): E3.
- 43. Leddy JJ, Baker JG, Willer B (2016) Active rehabilitation of concussion and post-concussion syndrome. Phys Med Rehabil Clin N Am 27(2): 437-454.
- 44. McCrory P, Feddermann-Demont N, Dvořák J, Cassidy JD, McIntosh A, et al. (2017) What is the definition of sports-related concussion: a systematic review? Br J Sports Med 51(11): 877-887.
- 45. Giza CC, Hovda DA (2014) The new neurometabolic cascade of concussion. Neurosurgery 75 Suppl 4(0 4): S24-S33.
- 46. Giza CC, Prins ML, Hovda DA (2017) It's not all fun and games: sports, concussions, and neuroscience. Neuron 94(6): 1051-1055.
- 47. Meaney DF, Smith DH (2011) Biomechanics of concussion. Clin Sports Med 30(1): 19-31.
- 48. Goldsmith W, Plunkett J (2004) A biomechanical analysis of the causes of traumatic brain injury in infants and children. Am J Forensic Med Pathol 25(2): 89-100.
- 49. Kleiven S (2013) Why most traumatic brain injuries are not caused by linear acceleration but skull fractures are. Front Bioeng Biotechnol 1: 15.
- 50. Tagge CA, Fisher AM, Minaeva O, Gaudreau-Balderrama A, Moncaster JA, et al. (2018) Concussion, microvascular injury, and early tauopathy in young athletes after impact head injury and an impact concussion mouse model. Alzheimer's & Dementia 141(2): 422-458.
- 51. Guenette JP, Shenton ME, Koerte IK (2018) Imaging of concussion in young athletes. Neuroimaging Clin N Am 28(1): 43-53.
- 52. Post A, Taylor K, Hoshizaki TB, Brien S, Cusimano MD, et al. (2017) A biomechanical analysis of traumatic brain injury for slips and falls from height. Trauma 21(1): 27-34.
- 53. Gurvits TV, Gilbertson MW, Lasko NB, Tarhan AS, Simeon D, et al. (2019) Neurologic soft signs in chronic posttraumatic stress disorder. Arch Gen Psychiatry 57(2): 181-186.
- 54. Weaver C, Sloan BK, Brizendine EJ, Bock H (2006) An analysis of maximum vehicle g forces and brain injury in motorsports crashes. Med Sci Sports Exerc 38(2): 246-249.
- 55. Haring RS, Canner JK, Asemota AO, George BP, Selvarajah S, et al. (2015) Trends in incidence and severity of sports-related traumatic brain injury (TBI) in the emergency department, 2006–2011. Brain Inj 29(7-8): 989-992.
- 56. Smith DH, Meaney DF (2002) Roller coasters, g forces, and brain trauma: On the wrong track? J Neurotrauma 19(10): 1117-1120.
- 57. Herold CJ, Essig M, Schröder J (2020) Neurological soft signs (NSS) and brain morphology in patients with chronic schizophrenia and healthy controls. PLoS One 15(4): e0231669.
- 58. Hertzig ME, Birch HG (1968) Neurologic organization in psychiatrically disturbed adolescents: A comparative consideration of sex differences. Arch Gen Psychiatry 19(5): 528-537.
- 59. Howell DR, Southard J (2021) The molecular pathophysiology of concussion. Clin Sports Med 40(1): 39-51.
- 60. Iverson GL, Luoto TM, Karhunen PJ, Castellani RJ (2019) Mild chronic traumatic encephalopathy neuropathology in people with no known participation in contact sports or history of repetitive neurotrauma. J Neuropathol Exp Neurol 78(7): 615-625.
- 61. Tucker LB, Velosky AG, McCabe JT (2018) Applications of the Morris water maze in translational traumatic brain injury research. Neurosci Biobehav Rev 88: 187-200
- 62. Jindal G, Gadhia RR, Dubey P (2021) Neuroimaging in sports-related concussion. Clin Sports Med 40(1): 111-121.
- 63. Keep RF, Hua Y, Xi G (2012) Brain water content: A misunderstood measurement? Transl Stroke Res 3(2): 263-265.
- 64. Jöhr J, Halimi F, Pasquier J, Pincherle A, Schiff N, et al. (2020) Recovery in cognitive motor dissociation after severe brain injury: a cohort study. PLoS One 15(2): e0228474.
- 65. Laskowski R, Creed J, Raghupathi R, Kobeissy F (2015) Pathophysiology of mild TBI: Implications for altered signaling pathways. In: Brain Neurotrauma: Molecular, Neuropsychological, and Rehabilitation Aspects. CRC Press/Taylor & Francis.
- 66. Jordan BD (2013) The clinical spectrum of sport-related traumatic brain injury. Nat Rev Neurol 9(4): 222-230.
- 67. Prins M, Greco T, Alexander D, Giza CC (2013) The pathophysiology of traumatic brain injury at a glance. Dis Model Mech 6(6): 1307-1315.
- 68. Jordan BD (2014) Chronic traumatic encephalopathy and other long-term sequelae. Continuum (Minneap Minn) 20(6 Sports Neurology): 1588-1604.
- 69. Tran LV (2014) Understanding the pathophysiology of traumatic brain injury and the mechanisms of action of neuroprotective interventions. J Trauma Nurs 21(1): 30-35.

- 70. McKee AC, Stein TD, Kiernan PT, Alvarez VE (2015) The neuropathology of chronic traumatic encephalopathy. Brain Pathol 25(3): 350-364.
- 71. Keightley ML, Sinopoli K, Wells G, Chen JK, Taha T, et al. (2013) Neuroimaging dual task performance in youth after sports-related concussion: An fMRI study. British Journal of Sports Medicine 47(5): e152.
- 72. Kennard MA (1960) Value of equivocal signs in neurologic diagnosis. Neurology 10: 753-764.
- 73. Saulle M, Greenwald BD (2012) Chronic traumatic encephalopathy: A review. Rehabil Res Pract 2012: 816069.
- 74. Khan F, Amatya B, Gales MP, Gonzenbach R, Kesselring J (2017) Neurorehabilitation: applied neuroplasticity. J Neurol 264(3): 603-615.
- 75. Lucke-Wold BP, Turner RC, Logsdon AF, Bailes JE, Huber JD, et al. (2014) Linking traumatic brain injury to chronic traumatic encephalopathy: Identification of potential mechanisms leading to neurofibrillary tangle development. J Neurotrauma 31(13): 1129-1138.
- 76. Kong L, Lui SSY, Wang Y, Hung KSY, Ho KKH, et al. (2021) Structural network alterations and their association with neurological soft signs in schizophrenia: Evidence from clinical patients and unaffected siblings. Schizophr Res 248: 345-352.
- 77. Turner RC, Lucke-Wold BP, Logsdon AF, Robson MJ, Dashnaw ML, et al. (2015) The quest to model chronic traumatic encephalopathy: A multiple model and injury paradigm experience. Front Neurol 6: 222.
- 78. Mckee AC, Daneshvar DH (2015) The neuropathology of traumatic brain injury. Handb Clin Neurol 127: 45-66.
- 79. Martínez-Pernía D (2020) Experiential neurorehabilitation: A neurological therapy based on the enactive paradigm. Front Psychol 11: 924.
- 80. Pearce AJ (2019) Transcranial magnetic stimulation: A tool for quantifying neurophysiological changes in the brain following concussion injury in sports. OBM Neurobiology, 3(3): 1-1.
- 81. McDonald SJ, O'Brien WT, Symons GF, Chen Z, Bain J, et al. (2021) Prolonged elevation of serum neurofilament light after concussion in male Australian football players. Biomark Res 9(1): 4.
- 82. Musumeci G, Ravalli S, Amorini AM, Lazzarino G (2019) Concussion in sports. J Funct Morphol Kinesiol 4(2): 37.
- 83. McKee AC, Mez J, Abdolmohammadi B (2017) Chronic traumatic encephalopathy in football players—Reply. JAMA 318(23): 2353.
- 84. Vagnozzi R, Tavazzi B, Signoretti S, Amorini AM, Finocchiaro A, et al. (2007) Temporal window of metabolic brain vulnerability to concussions. Neurosurgery 61(2): 379-389.
- 85. Messinis L, Kosmidis MH, Nasios G, Dardiotis E, Tsaousides T (2019) Cognitive Neurorehabilitation in Acquired Neurological Brain Injury. Behav Neurol 2019: 8241951.
- 86. Mez J, Solomon TM, Daneshvar DH, Murphy L, Kiernan PT, et al. (2015) Assessing clinicopathological correlation in chronic traumatic encephalopathy: rationale and methods for the UNITE study. Alzheimers Res Ther 7(1): 62.
- 87. Narayana S, Charles C, Collins K, Tsao JW, Stanfill AG, et al. (2019) Neuroimaging and neuropsychological studies in sports-related concussions in adolescents: Current state and future directions. Front Neurol 10: 538.
- 88. Mittal VA, Dean DJ, Bernard JA, Orr JM, Pelletier-Baldelli A, et al. (2013) Neurological soft signs predict abnormal cerebellar-thalamic tract development and negative symptoms in adolescents at high risk for psychosis: a longitudinal perspective. Schizophr Bull 40(6): 1204-1215.
- 89. Tremblay S, De Beaumont L, Henry LC, Boulanger Y, Evans AC, et al. (2012) Sports concussions and aging: A neuroimaging investigation. Cereb Cortex 23(5): 1159-1166.
- 90. Naravana PA (2017) White Matter Changes in Patients with Mild Traumatic Brain Injury: MRI Perspective. Concussion 2(2): CNC35.
- 91. Odle TG (2017) Neuroimaging of sports concussions. Radiol Technol 88(6): 621CT-642CT.
- 92. Oberholzer M, Müri RM (2019) Neurorehabilitation of traumatic brain injury (TBI): a clinical review. Med Sci (basel) 7(3): 47.
- 93. Salvador-Cruz J, Tovar-Vital DS, Segura-Villa A, Ledesma-Amaya L, García-Anacleto A, et al. (2019) Neurological Soft Signs and cognitive processes in Mexican schoolchildren aged 6 to 11 years. Acta Colombiana de Psicología 22(2): 28-52.
- 94. Parekh B Berger SE (2022) The Parekh-Berger Hierarchical Interpretive Phenomenological Analysis Model. Nov Res Sci 10(5): 1-6.
- 95. Pierre K, Dyson K, Dagra A, Williams E, Porche K, et al. (2021) Chronic traumatic encephalopathy: Update on current clinical diagnosis and management. Biomedicines 9(4): 415.
- 96. Pundlik J, Perna R, Arenivas A (2020) Mild TBI in interdisciplinary neurorehabilitation: Treatment challenges and insights. NeuroRehabilitation 46(2): 227-241.
- 97. Wang Y, Braam E, Wanaam C, Nelson B (2021) Investigation of structural brain correlates of neurological soft signs in individuals at ultra-high risk for psychosis. Eur Arch Psychiatry Clin Neurosci 271(8): 1475-1485.

- 98. Rathod B, Kaur A, Basavanagowda DM, Mohan D, Mishra N, et al. (2020) Neurological soft signs and brain abnormalities in schizophrenia: A literature review. Cureus 12(10): e11050.
- 99. Rochford JM, Detre T, Tucker GJ, Harrow M (1970) Neuropsychological impairments in functional psychiatric diseases. Arch Gen Psychiatry 22(2): 114-119.
- 100. Roemmich RT, Bastian AJ (2018) Closing the loop: from motor neuroscience to neurorehabilitation. Annu Rev Neurosci 41: 415-429.
- 101. Rothman DJ (2019) An Investigation of Neurological Soft Signs as a Discriminating Factor between Veterans with Post-Traumatic Stress Disorder, mild Traumatic Brain Injury and Co-occurring Post-Traumatic Stress Disorder and mild Traumatic Injury. Virginia Commonwealth University.
- 102. Sahler CS, Greenwald BD (2012) Traumatic brain injury in sports: a review. Rehabil Res Pract 2012: 659652.
- 103. Smith JA, Shinebourne P (2012) Interpretative phenomenological analysis. In: Cooper H, Camic PM, Long DL, Panter AT, Rindskopf D, Sher KJ (Eds.). APA handbook of research methods in psychology. Volume 2 Research designs: Quantitative, qualitative, neuropsychological, and biological. American Psychological Association. pp. 73-82.
- 104. Stanford Childrens Health (2019) Sports Injury Statistics.
- 105. Stemper BD, Pintar FA (2014) Biomechanics of Concussion. Prog Neurol Surg 28: 14-27.
- 106. Stewart W, McNamara PH, Lawlor B, Hutchinson S, Farrell M (2015) Chronic traumatic encephalopathy: a potential late and under recognized consequence of rugby union? QJM 109(1): 11-15.
- 107. Vos PE, Alekseenko Y, Battistin L, Ehler E, Gerstenbrand F, et al. (2012) Mild traumatic brain injury. Eur J Neurol 19(2): 191-198.
- 108. van Heugten C, Wolters Gregório G, Wade D (2012) Evidence-based cognitive rehabilitation after acquired brain injury: a systematic review of content of treatment. Neuropsychol Rehabil 22(5): 653-673.
- 109. Willig C (2001) Qualitative research design. Introducing qualitative research in psychology: Adventures in theory and method. Buckingham: Open University Press.
- 110. Maroon JC, Winkelman R, Bost J, Amos A, Mathyssek C, et al. (2015) Chronic traumatic encephalopathy in contact sports: a systematic review of all reported pathological cases. PLoS One 10(2): e0117338.