FRAMES DEMENTIA HAVE BEEN FOUND TRAUMATIC BRAIN INJURY (TBI AND THE EFFECTS OF OCCUPATIONAL pdf

1: Early TBI Tied to Parkinson's But Not Alzheimer's | Medpage Today

Traumatic brain injury (TBI) is a worldwide public health problem. Over the last several decades, improvements in acute care have resulted in higher survival rates. Unfortunately, the majority of survivors of moderate and severe TBI have chronic neurobehavioral sequelae, including cognitive deficits, changes in personality and increased rates of psychiatric illness.

Research into traumatic brain injury TBI and more specifically, concussion, has greatly increased over the past years. Much of this attention has focused on the world of competitive sports, as doctors, certified athletic trainers, and others have become more cognizant of the effects of these injuries beyond the playing field. That said, experts admit there is still so much unknown about concussion and the details surrounding susceptibility, recovery and the lasting effects of the injury. Great amounts of time, money, and research go into furthering the knowledge so future generations may reap the benefits of our increased knowledge. For now, we know that concussions can have impacts that last anywhere from a few hours to indefinitely. With that in mind, here is a look at a few of the longer-lasting implications of concussion. For some people, this means several weeks; for others, symptoms can persist for a year or even longer. Headaches, by far the most common symptom of post-concussion syndrome, typically appear within the first seven days of the injury. In fact, the likelihood of developing post-concussion syndrome seems to be totally unrelated to the seriousness of the initial injury. Some experts believe the condition is a result of a disturbance in the brain caused by the impact that caused the injury, while others think post-concussion symptoms denote a psychological component. They reason that the most common symptoms, such as headache, dizziness, and sleep disturbancesâ€”are similar to the effects of depression, post-traumatic stress disorder and other psychological diagnoses. As with all other aspects of concussion, further research is needed for more conclusive observations, but current knowledge does indicate some degree of connection between the development of post-concussion syndrome and certain psychological factors. This is by no means conclusive, nor does a lack of these factors preclude a person from developing the condition. The simplest answer, as with most effects relating to concussion, is that memory loss is possible, though not necessarily likely, in the long term. While it is unlikely that a singular episode would cause amnesia over a period of years, the risk of certain side effects does increase with repeated trauma. A considerable amount of the researchâ€”and probably the majority of published researchâ€”concerning long-term effects of trauma related to memory loss focuses on athletes in contact sports, specifically boxers and football players. While there is some emerging evidence that some retired athletes who competed at the highest levels of these sports show cognitive impairment disproportionate to their age, the repeated trauma factor plays a considerable role as well. Chronic Traumatic Encephalopathy CTE is a degenerative brain disease typically found in people with a history of repeated brain trauma. However, recent years have seen an uptick in research of the condition, yielding a more scientific description of the condition. CTE symptoms, generally speaking, begin to develop years after an individual sustains repetitive, mild TBI again, whether diagnosed as concussions or otherwise. A buildup of tau protein in the brain forms and begins to spread, compromising brain cells in specific regions of the brain. The most commonly observed symptoms include impulse control issues, increased aggression, paranoia, and depression. Even without further head trauma, symptoms can progress with age in some patients, while others may stabilize after a period of worsening. Memory loss, confusion, and poor judgment can eventually emerge, ending with progressive dementia in some patients.

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2: Long-Term Effects of Concussion | Nursing

There is controversy regarding the causal link between a single traumatic brain injury (TBI) and the risk of developing dementia. Several studies and meta-analyses have not found an association between TBI and risk of dementia.

Abstract Mild traumatic brain injury mTBI includes concussion, subconcussion, and most exposures to explosive blast from improvised explosive devices. It is also recognized that some mTBIs have persistent, and sometimes progressive, long-term debilitating effects. In addition, repetitive mTBIs can provoke the development of a tauopathy, chronic traumatic encephalopathy. We found early changes of chronic traumatic encephalopathy in four young veterans of the Iraq and Afghanistan conflict who were exposed to explosive blast and in another young veteran who was repetitively concussed. Four of the five veterans with early-stage chronic traumatic encephalopathy were also diagnosed with posttraumatic stress disorder. Advanced chronic traumatic encephalopathy has been found in veterans who experienced repetitive neurotrauma while in service and in others who were accomplished athletes. Clinically, chronic traumatic encephalopathy is associated with behavioral changes, executive dysfunction, memory loss, and cognitive impairments that begin insidiously and progress slowly over decades. Pathologically, chronic traumatic encephalopathy produces atrophy of the frontal and temporal lobes, thalamus, and hypothalamus; septal abnormalities; and abnormal deposits of hyperphosphorylated tau as neurofibrillary tangles and disordered neurites throughout the brain. The incidence and prevalence of chronic traumatic encephalopathy and the genetic risk factors critical to its development are currently unknown. Chronic traumatic encephalopathy has clinical and pathological features that overlap with postconcussion syndrome and posttraumatic stress disorder, suggesting that the three disorders might share some biological underpinnings. Estimates of the prevalence of mTBI among returning service members range from Despite their frequency, the acute and long-term effects of mTBI have been a relatively unexplored area of medical inquiry until very recently. However, there is accumulating evidence that some individuals develop persistent cognitive and behavioral changes after mild neurotrauma. The first large-scale evidence of military-related mTBI occurred in World War I â€” in association with the frequent use of high explosives in trench warfare. At the time, it was unclear whether shell shock was a maladaptive, psychiatric condition related to the stresses of combat or whether the condition was caused by physical injury to the brain. Despite the lack of any pathological studies on the brains of individuals diagnosed with shell shock, wartime committees entreated with the responsibility to inquire into the entity declared the disorder to have psychiatric origins [7]. Martland described unsteadiness of gait, mental confusion, and slowing of muscular movements occasionally combined with hesitancy in speech, tremors of the hands, and nodding of the head. Later, Winterstein summarized the psychiatric manifestations of approximately 50 professional boxers and noted impairment of intelligence, mental dullness, difficulty concentrating, paranoia, and garrulousness [9]. Johnson later added memory loss, dementia, rage reactions, and morbid jealousy to the clinical syndrome [10]. In , Corsellis, Bruton, and Freeman-Browne detailed the neuropathological findings found in the brains of 15 retired boxers and correlated the pathological findings with retrospective clinical symptoms [13]. The authors noted gross neuropatho-logical changes of cerebral atrophy, enlargement of the lateral and third ventricles, thinning of the corpus callosum, cavum septum pellucidum with fenestrations, and cerebellar scarring. The authors speculated that pathology in the limbic structures such as the hippocampus, medial temporal lobe, and fornix was responsible for impairments in learning and memory, that pathology in the substantia nigra accounted for the Parkinsonian features, and that pathology in the septal cortex for the abnormal rage reactions. In this review, we summarize the acute and long-term effects of mTBI that have been reported in veterans and describe the experience over the past 6 years at the VA Boston Brain Bank with military service-related mTBI. Understanding the effects of military-related mTBI presents additional challenges not encountered in studies of neurotrauma associated with play of sports. Unlike the relatively stereotyped features of brain trauma that occur during athletic participation, in which injury is dependent on

the rules of engagement specific to the sport, military-related mTBI is acquired in widely heterogeneous ways, including athletics, recreational activities, physical training practices e. Injury from explosive blasts varies depending on the strength of the explosive; whether the injury occurs in an open field, in, or near buildings; or in a motor vehicle. Concussion, subconcussion A concussion is a mTBI induced by an impulsive force transmitted to the head resulting from a direct or indirect impact to the head, face, neck, or elsewhere [21 , 22]. In the combat setting, most concussions occur as a result of blunt trauma associated with blasts from explosive devices, such as hitting the head against the interior of a vehicle [23]. Signs and symptoms of concussion and PCS include irritability, sleep disturbance, forgetfulness, anxiety, headaches, poor concentration, pain, and psychological distress [24]. Neuropsychological testing in PCS may reveal persistent, yet subtle, cognitive deficits, often in the executive domain [24]. Concussion and subconcussion are produced by acceleration and deceleration forces on the brain that may be linear or rotational [27]. Rapid acceleration, deceleration, or rotational forces cause the brain to elongate and deform, stretching individual cells and blood vessels and altering membrane permeability. Although all cell compartments are affected by the injury, axons are especially vulnerable to shear injury given their relatively long length and high membrane-to-cytoplasma ratio. In addition to axonal damage, the integrity of the microvasculature is compromised, with disruption of the blood-brain barrier and focal cortical hypoperfusion [28]. Pathological studies of concussion and PCS demonstrate microscopic evidence of multifocal traumatic axonal injury TAI [18 , 31] that is best visualized by amyloid precursor protein APP immunohistochemistry [32], often located around small blood vessels in the corpus callosum, fornix, subcortical U-fibers, and cerebellum [18]. In general, the severity of the multifocal axonal injury is parallel to the severity of the TBI. However, APP immunohistochemical identification of traumatic axonal swellings may significantly underestimate the overall magnitude of the axonal damage [33]. TAI affects multiple axonal populations, including large caliber myelinated and non-neuropathologically detected fine-caliber myelinated and unmyelinated fibers. There is evidence that fine-fiber unmyelinated axons are disproportionately vulnerable to traumatic injury and, as such, may be significant contributors to the morbidity associated with mTBI. There are currently no reliable neuro-pathological markers for unmyelinated fine-fiber damage; accordingly, it is likely that the total axonal damage associated with TAI is considerably more than what is detectable with conventional neuropathological methods [33]. In addition, concomitant with the break-down of the axon and myelin sheath, axon terminals also undergo neurodegenerative change and deafferentation. This axonal degeneration and deafferentation likely contribute significantly to the morbidity associated with TAI. Furthermore, deafferentation sets the stage for subsequent neuroplastic changes that may be adaptive or maladaptive. These neuroplastic responses to mild neuro-trauma have not been well studied in the human brain [34 , 35]. After concussion, there is pathological evidence of blood-brain barrier damage with microhemorrhage, astrocytosis, and perivascular clusters of activated microglia [18]. In addition, some cases of recent concussion and PCS show isolated focal perivascular accumulations of hyperphosphorylated tau p-tau as neurofibrillary tangles NFTs and neurites as well as hemosiderin-laden macrophages. The finding of focal p-tau abnormalities in the brains of individuals with PCS in close proximity to focal axonal injury, microhemorrhage, astrocytosis, and perivascular microgliosis suggests that the development of p-tau pathology may be mechanistically linked to axonal injury, breach of the blood-brain barrier, and neuroinflammation [18]. Whether these acute pathologies are reversible and resolve over time is unknown. Because most patients with mTBI recover fully, it suggests that intrinsic mechanisms are generally able to repair low-level injury [33]. However, repetitive TAI superimposed on continuing, perhaps broadening, breach of the blood-brain barrier; disruption of sodium channels; ionic dysregulation; and metabolic irregularities might trigger a self-perpetuating, progressive neurodegenerative cascade in some individuals. Concussion and subconcussive injury are associated with microstructural changes in the white matter and alterations in fiber tract integrity that are detectable with diffusion tensor imaging DTI and susceptibility weighted imaging but are not evident on conventional structural imaging studies such as computed tomography CT scan and magnetic resonance imaging MRI. The

significance of these asymptomatic subconcussive injuries is increasingly recognized in sports such as football, ice hockey, and soccer, in which accumulating evidence demonstrates microstructural changes in the white matter on DTI as well as abnormalities in functional MRI fMRI [26 , 36 â€” 40]. Similar changes recently have been demonstrated in asymptomatic military-related blast injury [37 , 41]. Bazarian and colleagues found a strong association between exposure to blast and reduced first percentile fractional anisotropy FA on DTI that was independent of symptoms of mTBI at the time of blast exposure. Because these asymptomatic mTBIs are difficult to assess, it is likely that DTI and fMRI will become useful tools to guide the prognosis and management of these injuries moving forward. Our experience with repetitive concussive injury includes a year-old male U. Marine veteran with two combat deployments. Similar to many other military personnel, his history was notable for multiple concussions that occurred as a civilian and in combat. His first concussion occurred at age 12 in a bicycle accident with temporary loss-of-consciousness LOC and posttraumatic amnesia PTA. At age 17, he experienced a second concussion without LOC during football practice. At age 25, during his second military deployment, he experienced a third concussion with temporary alteration in mental status but no LOC, after which he was diagnosed with PTSD. Four months later at age 26, he sustained a fourth concussion with temporary LOC and PTA resulting from a motor vehicle-bicycle collision. He subsequently developed persistent anxiety, difficulty concentrating, word-finding difficulties, learning and memory impairment, reduced psychomotor speed, and exacerbation of PTSD symptoms. He died from a self-inflicted gunshot wound 2 years after his last concussion. Neuropathological analysis revealed multiple areas of p-tau immunoreactivity surrounding small blood vessels in the temporal cortex, consistent with the diagnosis of early-stage CTE, although the limited availability of tissue precluded any further histo-logical analysis. The physical effects of the blast depend on many factors, including the characteristics of the improvised explosive device IED ; the relationship of the individual with respect to the source of the blast, including the distance from the blast; whether the exposure occurred in an open environment or in an enclosed space such as a building or vehicle; and whether a solid structure is located between the individual and the device, because the reflection of blast pressure waves off of various surfaces can lead to multiple pressure waves impacting an individual from various directions for a prolonged period [42]. Blast injury is often further complicated by an accompanying concussive mTBI. Blast injury is the result of the rapid transmission of an acoustic wave through the brain tissue and accompanying blast winds [20]. Many animal models of blast-induced injury exist, although the precise biomechanics of blast-related traumatic injury and its neuropathological consequences are the subject of debate [43 , 44]. In addition, simultaneous intracerebral pressure recordings demonstrated that blast waves traversed the brain with minimal change. The blast wavefront transmissions were not associated with thoracovascular or hydrodynamic contributions; indeed, separation of the mouse head from the thorax did not significantly change the blast-induced intracerebral pressure amplitudes. Pathologically, blast injuries produce hemorrhage and edema as blood vessels and brain tissue rapidly contract and expand several times within a fraction of a second after a blast wind, damaging cerebral vasculature [45]. In the acute phases, blast injury may produce large intraparenchymal and subarachnoid hemorrhages [46 â€” 48]. Blast injury is also associated with pseudoaneurysm formation and the development of vasospasm. When severe, vasospasm can lead to cerebral ischemia and clinical deterioration that can be delayed as long as 30 days after initial blast exposure [49 â€” 51]. Lu and colleagues investigated the effects of relatively low-level single and repetitive primary blast injury in nonhuman primates [52]. Ultrastructural analysis and histopathology at 3 days and 1 month postinjury revealed microvascular degeneration and collapse with obliterated capillary lumens, hypertrophic astrocytic end-feet, vacuolated endothelial cytoplasm, and increased perivascular reticuloendothelial cells. Other changes included chromatolysis of cortical neurons, hippocampal pyramidal neurons, and Purkinje cells in the cerebellum; white matter damage; and increased astrocytic aquaporin-4, suggesting cerebral edema. The number of neuropathological analyses of acute blast injury in humans are few. In the instances reported by Mott, there was mild brain edema, severe vascular congestion, variable amounts of extravasation of blood

around small vessels, intraparenchymal and subarachnoid hemorrhage, and generalized chromatolysis of neurons [5 , 6]. Warden and colleagues described the neuroimaging findings in a service member exposed to a series of large explosions. Three months after exposure, the service member still experienced headache, occasional nausea, and eye twitching, but she was found to have a normal neurological examination. Her MRI showed a resolving hematoma in the internal auditory canal and an area of hyperintensity in the cerebellum. Other investigators using DTI have demonstrated lower FA and higher radial diffusivity after blast injury [41 , 54], although the white matter abnormalities found in various studies tend to be heterogeneous and spatially diverse. Using [18 F]-fluorodeoxyglucose positron emission tomography imaging of cerebral glucose metabolism, Peskind and colleagues demonstrated regional brain hypometabolism in veterans with repetitive blast injury [55]. The neuroimaging metrics did not differ between participants with versus those without PTSD [56]. A year-old male U. He was diagnosed with combat-related PTSD. At age 27, he was honorably discharged from the Marines. At age 28, during an early-morning incident in which he allegedly fired on police and other civilians, he was shot and killed. At autopsy, his brain weighed g and was remarkable for slight thinning of the posterior body of the corpus callosum and discoloration of frontal tracts in the cerebral peduncle. Microscopically, there was evidence of severe axonal loss with widespread axonal swellings and axon retraction bulbs, myelinopathy, astrocytosis, and foci of dystrophic calcification in the cerebral subcortical white matter, internal capsule, and cerebellar white matter. Myelinated fiber loss was particularly prominent in the frontal lobes and frontal tracts of the cerebral peduncle.

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3: Mild Traumatic Brain Injury and Related Mental Health Issues

Unfortunately, brain imaging does not yet allow us to identify early who are going to have dementia. However, this is the future goal. Supporting caregivers Significant increase in caregivers to patients with dementia have been made. Unfortunately, caregivers can be at an increased risk for social isolation and depression.

The study, published Tuesday in the medical journal JAMA, examined 34, deaths by suicide over 35 years from a Danish national registry and people who had medical contact for traumatic brain injury TBI. Researchers not only found an increased risk of suicide between traumatic brain injury sufferers and the general population, they also found that the risk of suicide was even higher for people who had experienced severe traumatic brain injury and who had numerous medical visits or longer hospital stays related to the condition. Patients who had longer hospital stays following traumatic brain injuries were at highest risk for suicide in the first six months after being discharged from the hospital, the study found. The study did have limitations: But the study is an interesting look into the possible psychiatric effects of traumatic brain injuries. Traumatic brain injuries have made headlines from the battlefield to the football field. But what are these injuries, how common are they and what other research is needed to understand them? What is a traumatic brain injury and how common is it? According to the Centers for Disease Control , traumatic brain injuries can be defined as a type of head injury that disrupts the normal function of the brain. This can be caused by a bump, blow, jolt to the head, or penetrating injury. Everyone is at risk of experiencing a traumatic brain injury, as these most commonly result from motor vehicle accidents, sports injuries, and falls. Other signs and symptoms include headaches, fatigue, and sleep disturbances, according to the CDC. Traumatic brain injury linked to increased dementia risk: Report While the physical impact of a traumatic brain injury is immediate and can be quite devastating, the long-term health consequences are only starting to come to light through research. Every year in the U. Recently, there has been increased attention to the long-term effects of repeated brain injury, especially as it relates to sports injuries and later development of chronic traumatic encephalopathy CTE. More research is still needed to better understand the long-term effects of CTE, including psychological disturbances. How might traumatic brain injuries put someone at an increased risk of suicide? While the exact mechanism remains unclear, there are some theories. The key might be in preventing the injuries from occurring in the first place, Madsen said. Moving forward, Madsen said she also hopes to improve the treatment of traumatic brain injuries in order to minimize the long-term consequences that could lead to suicidal behavior. She said she is also interested in examining how traumatic brain injury might be associated with social consequences, such as employment status in the years following the injury. All but one of former pro football players had brain injury CTE in study Tayim said there are many ways medical professionals can help support patients. For example, arranging for counseling services in addition to speech, physical and occupational therapies," Tayim said. In addition to the resources listed above, a National Concussion Surveillance System has been developed by the CDC to improve prevention, care, and recovery efforts for those who have experienced a traumatic brain injury.

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4: Treatment of Traumatic Brain Injury (TBI)

Diller L. Pushing the frames of reference in traumatic brain injury rehabilitation. Arch Phys Med Rehabil ; Cognitive rehabilitation is an empirically based field driven by multiple sources of activities and knowledge bases.

With the devastating statistics of morbidity and mortality, swift medical attention is required to provide the best possible care in the hope of preventing long-term damaging effects. Various measures are employed in the hospital and ambulatory setting for individuals with TBI that address physical or cognitive sequelae of the traumatic event. Neuropsychiatric care in TBI patients includes the management of increased intracranial pressure, seizure occurrence, cognitive disturbances, and psychiatric disorders. Traumatic brain injury TBI is a major cause of disability, death, and high healthcare costs in the United States that affects not only the injured persons but also their families and society. TBI can be caused by an external force to the head that disrupts the normal function of the brain. This is useful for promoting prevention, predicting outcomes, focusing on wrap-around services needed, and developing targeted TBI-related interventions and best practices. As previously stated, TBI can be classified as mild, moderate, or severe. This section will focus on moderate-to-severe TBI, which usually requires hospitalization. Acute stabilization, preventing secondary injury, and restoring neuronal function are the goal for medical and pharmacologic interventions in an attempt to improve outcomes in this patient population. Laboratory tests like arterial blood gas, urine drug screen, blood alcohol concentration, and serum electrolytes are necessary to exclude other causes of neurologic dysfunction. Using the GCS and testing the reaction of the pupils to light should be a part of the initial examination. Some acute neurologic symptoms to monitor for include seizures, posttraumatic amnesia, dizziness, moderate-to-severe headache, limb weakness, and paresthesia. Intracranial hypertension and seizure occurrence are common acute neuropsychiatric effects that need to be monitored for, addressed, and managed. Although not the focus of this article, blood pressure control, venous thromboembolism prophylaxis, stress ulcer prophylaxis, glycemic control, possible mechanical ventilation, infection control, and nutrition support are all necessary management protocols to decrease morbidity and mortality in TBI, as with all other critical illnesses. The following will discuss management of intracranial hypertension and seizure occurrence. This is a critical condition and the most common cause of death in patients with severe TBI. There are different thresholds for what is considered abnormal, thus requiring treatment. ICP thresholds of 15, 20, and 25 mmHg have been used in adults and 20 mmHg in pediatric patients. Sedation and analgesia are common first-line therapy for intracranial hypertension. It is usually unknown whether pain is the causative factor because of lack of communication due to brain injury. Midazolam is the preferred benzodiazepine because of its shorter half-life compared to lorazepam, easier titration, and status as the most studied drug in TBI. One study found that dexmedetomidine was safe and not associated with significant changes in intracranial hemodynamics. Fentanyl and remifentanil are also used because of their shorter duration of action as a continuous infusion. Since intermittent bolus has been associated with likely increasing ICP, continuous infusion should be used whenever possible for the above mentioned opioids. These medications are recommended because they provide analgesia, mild sedation, and depression of airway reflexes, which is necessary for intubated and mechanically ventilated patients. Neuromuscular blockers NMBs have also been used concomitantly for patients who are receiving maximum tolerated doses of propofol or who are refractory to all other agents for ICP control. Although NMBs may not have any direct effect on lowering of ICP, they may be beneficial for facilitating mechanical ventilation, minimizing muscle activity and spasms, and improving respiratory compliance. Mannitol is recommended for patients with signs of transtentorial herniation or progressive neurologic deterioration not attributable to extracranial causes. In order to maintain euvolemia, adequate fluid replacement with isotonic saline solution should be used along with osmotic diuresis. Commonly used doses are 0. Side effects to therapy are intravascular dehydration, hypotension, prerenal azotemia, and hyperkalemia. Sometimes mannitol may cause a rebound effect and lead to increased ICP. Its use is contraindicated in

patients with TBI and renal failure because of the risk of pulmonary edema and heart failure. This alternative therapy causes osmotic dehydration and viscosity-related cerebral vasoconstriction. The beneficial effects of HSS in TBI include expansion of intravascular volume, extraction of water from the intracellular space, decrease in the ICP, controlled cerebral edema, and an increase in cardiac contractility. Hyperosmolarity effects such as renal failure and pulmonary edema are not associated with HSS. Therefore, HSS may be a good alternative or in some cases first-line hyperosmolar therapy. Moderate hypothermia may be used in refractory, uncontrolled ICP. Although outcomes of temperature modulation in patients with TBI are controversial, temperature should be controlled and fever aggressively treated in this population. Barbiturates have been proven effective therapies for refractory or second-line intracranial hypertension. They reduce cerebral metabolism and cerebral blood flow and lower ICP. Pentobarbital is recommended for the induction of barbiturate coma. It is recommended in hemodynamically stable, severe TBI patients who are refractory to maximum medical and surgical ICP-lowering therapy. Common adverse effects of barbiturates include hypotension and immunosuppression. Prophylactic use of barbiturates is not recommended. Elevating the head of the bed at a degree angle in hemodynamically stable patients and CSF drainage in patients with ventriculostomy can be recommended. Both methods have been shown to decrease ICP. Decompressive therapy is a surgical procedure to control ICP in which part of the skull is removed to allow a swelling brain room to expand without being squeezed. Although decompressive therapy may be life-saving, evidence regarding its overall effects on outcomes is contradictory. Early-onset seizures occur within 7 days of injury, and late-onset seizures occur 7 days after injury. Prophylactic therapy with antiepileptic drugs AEDs is not recommended for preventing late posttraumatic seizures. However, it is recommended for prophylactic therapy to prevent early posttraumatic seizure in TBI patients who are at high risk for seizures. High-risk patients are defined as having the following: Phenytoin is the drug of choice for the prophylaxis of early posttraumatic seizures. Both valproate and phenobarbital have been studied and have demonstrated little benefit in posttraumatic seizures in high-risk TBI patients. Patients receiving AED prophylaxis should be monitored for potential side effects. Preventive therapy beyond the first 7 days of injury is not recommended. These measures may address physical or cognitive sequelae of the traumatic event. Physical therapy and other rehabilitation measures are often employed to address the physical complications. Individuals can sometimes develop gait and balance disorders that can be resolved through physical therapy. Depending on the cause of the injury and extent of physical injury, situations where limb function, speech, and other bodily functions such as bladder and bowel function, swallowing, breathing, hormonal regulation, motor control, and blood pressure regulation are affected may require physical therapy, acute medical interventions, and other rehabilitation measures. The cognitive decline seen in posttraumatic injury may improve within 6 to 12 months, but may mimic some of the symptoms of various dementias. Attentional skills may be compromised and can easily be mistaken for dementia, so the age of the individual is an important factor to determine the route of evaluation and treatment course. Screening and monitoring for depression, anxiety, and sleep impairment are essential in TBI. The prevalence of depression, anxiety, substance abuse, and other psychiatric disorders varies tremendously and is confounded by the lack of screening, treatment, and follow up. Posttraumatic stress disorder can also occur in TBI, so clinicians should screen accordingly and include as assessment of sleep quality. Cognitive behavioral therapies are also highly effective as monotherapy or as adjuncts to pharmacotherapy. When considering long-term pharmacotherapy for seizure management, attention should be paid to the risk of recurrent seizures and seizure type when deciding to initiate AEDs and choosing a drug. Therefore, it is important to treat only the target symptoms that the patient is experiencing. Before starting pharmacotherapy, always assess for spontaneous resolution of symptoms. Slow titration is very important in this population because of increased sensitivity to cognitive effects of medications. If there is not a beneficial response to medication, then it is not necessary to continue drug therapy. Limiting the number of medications and using the minimal effective doses when possible are also necessary to minimize exposure to side effects that can impede cognitive recovery. Clinicians should develop a treatment plan that allows for

maximal quality of life for the individual. Pharmacists can improve medication outcomes by ensuring that guidelines for TBI are consistently being followed in patient care pathways e. Pharmacists can also ensure that any drugs that may worsen outcomes or increase the likelihood of adverse effects particular to this patient population are avoided i. Adding a clinical pharmacist to a multidisciplinary team will allow for more therapeutic drug monitoring, medication reconciliation, drug interaction screening, and cost-saving initiatives, which can have a positive effect on this highly vulnerable patient population. Traumatic brain injury in the United States: Accessed July 21, What is traumatic brain injury TBI? Incidence of traumatic brain injury across the full disease spectrum: Comparability of national estimates for traumatic brain injury-related medical encounters. J Head Trauma Rehabil. Medical care costs associated with traumatic brain injury over the full spectrum of disease: Recommendations for diagnosing a mild traumatic brain injury: Utilization and cost of health services in individuals with traumatic brain injury. Glob J Health Sci. Management of acute traumatic brain injury. American College of Clinical Pharmacy; Accessed September 11, Romner B, Grande P. Intracranial pressure monitoring in traumatic brain injury. J Neurol Neurosurg Psychiatry. Management of raised intracranial pressure in children with traumatic brain injury. Critical care management of severe traumatic brain injury in adults. Effects of dexmedetomidine on intracranial hemodynamics in severe head injured patients. Clinical practice guidelines for sustained neuromuscular blockade in the adult critically ill patient. Decom-pressive craniectomy in diffuse traumatic brain injury. N Engl J Med.

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5: Glossary of Brain Injury Terms | BrainLine

Specifically, out of 76, veterans diagnosed with a mild TBI developed Parkinson's (%), as did out of 72, with a history of moderate to severe TBI (%). Subsequent analysis â€" after adjusting for sex, age, race, education, and overall health â€" found that traumatic brain injury of any kind increased the risk of Parkinson's by 71% in this group of veterans.

Glossary Introduction Traumatic brain injury TBI is the leading cause of death and disability in children and young adults in the United States. TBI is also a major concern for elderly individuals, with a high rate of death and hospitalization due to falls among people age 75 and older. These figures are likely an underestimate of the true number of TBIs as they exclude people who did not seek medical attention at the emergency room. Although approximately 75 percent of brain injuries are considered mild not life-threatening , as many as 5. Not every TBI is alike. Each injury is unique and can cause changes that affect a person for a short period of time, or sometimes permanently. However, persistent symptoms do occur for some people and may last for weeks or months. Over the past few decades preventive measures, such as seatbelts and helmets, and better critical care have substantially increased survival from severe TBI. Recently, research has expanded from a singular focus on severe TBI to a greater awareness about potential long-term consequences and the need to find better ways to diagnose, treat, and prevent all forms of TBI. Many questions remain unanswered regarding the impact of TBIs, the best treatments, and the most effective methods for promoting recovery of brain function. This publication outlines what is known about TBI, as well as directions for future research. A TBI occurs when physical, external forces impact the brain either from a penetrating object or a bump, blow, or jolt to the head. Not all blows or jolts to the head result in a TBI. For the ones that do, TBIs can range from mild a brief change in mental status or consciousness to severe an extended period of unconsciousness or amnesia after the injury. There are two broad types of head injuries: With this injury, the object enters the brain tissue. Causes include falls, motor vehicle crashes, sports injuries, or being struck by an object. Blast injury due to explosions is a focus of intense study but how it causes brain injury is not fully known. Some accidents such as explosions, natural disasters, or other extreme events can cause both penetrating and non-penetrating TBI in the same person. The type of injury is another determinant of the effect on the brain. These secondary brain injuries are the result of reactive processes that occur after the initial head trauma. There are a variety of immediate effects on the brain, including various types of bleeding and tearing forces that injure nerve fibers and cause inflammation, metabolic changes, and brain swelling. White matter is composed of bundles of axons projections of nerve cells that carry electrical impulses. Like the wires in a computer, axons connect various areas of the brain to one another. This damage commonly occurs in auto accidents, falls, or sports injuries. It usually results from rotational forces twisting or sudden deceleration. It also leads to the release of brain chemicals that can cause further damage. These injuries can cause temporary or permanent damage to the brain, and recovery can be prolonged. Concussionâ€" a type of mild TBI that may be considered a temporary injury to the brain but could take minutes to several months to heal. Concussion can be caused by a number of things including a bump, blow, or jolt to the head, sports injury or fall, motor vehicle accident, weapons blast, or a rapid acceleration or deceleration of the brain within the skull such as the person having been violently shaken. Hematomas can develop when major blood vessels in the head become damaged, causing severe bleeding in and around the brain. The meninges are the protective membranes surrounding the brain, which consist of three layers: These can occur with a delay of minutes to hours after a skull fracture damages an artery under the skull, and are particularly dangerous. Their effects vary depending on their size and extent to which they compress the brain. They are very common in the elderly after a fall. Contusions can occur directly under the impact site i. They can appear after a delay of hours to a day. Generally they occur when the head abruptly decelerates, which causes the brain to bounce back and forth within the skull such as in a high-speed car crash. They are a result of blunt force trauma and can cause damage to the underlying areas of the skull such as the membranes, blood vessels, and brain. One main benefit

of helmets is to prevent skull fracture. For this reason doctors suggest watching a person for changes for 24 hours after a concussion. HPCs occur when an initial contusion from the primary injury continues to bleed and expand over time. This creates a new or larger lesion â€" an area of tissue that has been damaged through injury or disease. This increased exposure to blood, which is toxic to brain cells, leads to swelling and further brain cell loss. The blood-brain barrier preserves the separation between the brain fluid and the very small capillaries that bring the brain nutrients and oxygen through the blood. Once disrupted, blood, plasma proteins, and other foreign substances leak into the space between neurons in the brain and trigger a chain reaction that causes the brain to swell. It also causes multiple biological systems to go into overdrive, including inflammatory responses which can be harmful to the body if they continue for an extended period of time. It also permits the release of neurotransmitters, chemicals used by brain cells to communicate, which can damage or kill nerve cells when depleted or over-expressed. Poor blood flow to the brain can also cause secondary damage. When the brain sustains a powerful blow, swelling occurs just as it would in other parts of the body. When the intracranial pressure becomes too high it prevents blood from flowing to the brain, which deprives it of the oxygen it needs to function. This can permanently damage brain function. Additional information about TBI and its causes can be found on the U. From to alone, falls caused more than half 55 percent of TBIs among children aged 14 and younger. Among Americans age 65 and older, falls accounted for more than two-thirds 81 percent of all reported TBIs. The second and third most common causes of TBI are unintentional blunt trauma accidents that involved being struck by or against an object , followed closely by motor vehicle accidents. Blunt trauma is especially common in children younger than 15 years old, causing nearly a quarter of all TBIs. Assaults account for an additional 10 percent of TBIs, and include abuse-related TBIs, such as head injuries that result from shaken baby syndrome. Unintentional blunt trauma includes sports-related injuries, which are also a major cause of TBI. Overall, bicycling, football, playground activities, basketball, and soccer result in the most TBI-related emergency room visits. The cause of these injuries does vary slightly by gender. According to the CDC, among children age 10 to 19, boys are most often injured while playing football or bicycling. Among girls, TBI occur most often while playing soccer or basketball or while bicycling. TBIs caused by blast trauma from roadside bombs became a common injury to service members in recent military conflicts. From to more than , military service personnel sustained TBIs, though these injuries were not all conflict related. The majority of these TBIs were classified as mild head injuries and due to similar causes as those that occur in civilians. Adults age 65 and older are at greatest risk for being hospitalized and dying from a TBI, most likely from a fall. TBI-related deaths in children aged 4 years and younger are most likely the result of assault. In young adults aged 15 to 24 years, motor vehicle accidents are the most likely cause. In every age group, serious TBI rates are higher for men than for women. Men are more likely to be hospitalized and are nearly three times more likely to die from a TBI than women. These problems may emerge days later. Headache, dizziness, confusion, and fatigue tend to start immediately after an injury, but resolve over time. Emotional symptoms such as frustration and irritability tend to develop later on during the recovery period. Many of the signs and symptoms can be easily missed as people may appear healthy even though they act or feel different. Many of the symptoms overlap with other conditions, such as depression or sleep disorders. If any of the following symptoms appear suddenly or worsen over time following a TBI, especially within the first 24 hours after the injury, people should see a medical professional on an emergency basis.. People should seek immediate medical attention if they experience any of the following symptoms: A child with a TBI may display the following signs or symptoms: CTE occurs in those with extraordinary exposure to multiple blows to the head and as a delayed consequence after many years. Studies of retired boxers have shown that repeated blows to the head can cause a number of issues, including memory problems, tremors, and lack of coordination and dementia. Recent studies have demonstrated rare cases of CTE in other sports with repetitive mild head impacts e. Studies assessing patterns among large populations of people with TBI indicate that moderate or severe TBI in early or mid-life may be associated with increased risk of dementia later in life. Effects on consciousness A TBI can cause problems with arousal, consciousness,

awareness, alertness, and responsiveness. Generally, there are four abnormal states that can result from a severe TBI: Coma generally lasts a few days or weeks after which an individual may regain consciousness, die, or move into a vegetative state. However, they can have periods of unresponsive alertness and may groan, move, or show reflex responses. Although the majority of TBIs are mild they can still have serious health implications. Of greatest concern are injuries that can quickly grow worse. All TBIs require immediate assessment by a professional who has experience evaluating head injuries. A neurological exam will assess motor and sensory skills and the functioning of one or more cranial nerves. It will also test hearing and speech, coordination and balance, mental status, and changes in mood or behavior, among other abilities. Screening tools for coaches and athletic trainers can identify the most concerning concussions for medical evaluation. The ACE is also used to track symptom recovery over time. It also takes into account risk factors including concussion, headache, and psychiatric history that can impact how long it takes to recover from a TBI. When necessary, medical providers will use brain scans to evaluate the extent of the primary brain injuries and determine if surgery will be needed to help repair any damage to the brain. Computed tomography CT is the most common imaging technology used to assess people with suspected moderate to severe TBI. CT scans create a series of cross-sectional x-ray images of the skull and brain and can show fractures, hemorrhage, hematomas, hydrocephalus, contusions, and brain tissue swelling. CT scans are often used to assess the damage of a TBI in emergency room settings.

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6: Suicide risk nearly double for traumatic brain injury sufferers: Study - ABC News

Cognitive Reserve and Early Dementia. The term "cognitive reserve" has been around for approximately twenty years. Only more recently, however, has it become a concept that can be useful to the traumatic brain injury litigator.

L lability â€” state of having notable shifts in emotional state e. A condition resulting from interruption of motor pathways in the ventral pons, usually by infarction. This disconnection of the motor cells in the lower brain stem and spinal cord from controlling signals issued by the brain leaves the patient completely paralyzed and mute, but able to receive and understand sensory stimuli; communication may be possible by code using blinking, or movements of the jaw or eyes, which can be spared. Requires storage and retrieval of information which exceeds the limit of short term memory. Used todetect subtle changes in brain tissue. This includes remembering what you do; remembering what others say to you; and remembering what you see or read. The regulation is carried out by operation of the nervous system. MRI â€” see Magnetic Resonance Imaging muscle tone â€” Used in clinical practice to describe the resistance of a muscle to being stretched. When the peripheral nerve to a muscle is severed, the muscle becomes flaccid limp. When nerve fibers in the brain or spinal cord are damaged, the balance between facilitation and inhibition of muscle tone is disturbed. The tone of some muscles may become increased and they resist being stretched--a condition called hypertonicity or spasticity. This tube allows for direct "tube feeding" to maintain the nutritional status of the patient or removal of stomach acids. Often works closely with schools and employers as well as with family members of the injured person. NG tube â€” see Nasogastric Tube non-fluent aphasia - a condition in which patients have trouble recalling words and speaking in complete sentences. See also vision after head injury. Damage to this lobe can cause visual deficits. The term occupation, as used in occupational therapy, refers to any activity engaged in for evaluating, specifying and treating problems interfering with functional performance. This includes knowing the day, date, month and year; knowing things about yourself; knowing where you are and how to get around; and knowing what happened to you; along with the ability to use this information appropriately in a functional setting. P paraplegia â€” paralysis of the legs from the waist down. PCS â€” see post-concussion syndrome penetrating head injury - a brain injury in which an object pierces the skull and enters the brain tissue. Perseverations may be verbal or motoric. A physician who specializes in physical medicine and rehabilitation. Some physiatrists are experts in neurologic rehabilitation, trained to diagnose and treat disabling conditions. The physiatrist examines the patient to assure that medical issues are addressed; provides appropriate medical information to the patient, family members and members of the treatment team. The physical therapist also evaluates the potential for functional movement, such as ability to move in the bed, transfers and walking and then proceeds to establish an individualized treatment program to help the patient achieve functional independence. May also be called Anterograde Amnesia. Facilities typically provide a full spectrum of clinical therapies, as well as life-skills training in a residential setting. Individuals with deficits in this skill may become "immobilized" when faced with a problem. By being unable to think of possible solutions, they may respond by doing nothing. Having a natural inclination or tendency to something. Combination of kinesthesia and position sense. Psychologists use tests to identify personality and cognitive functioning. This information is shared with team members to assure consistency in approaches. Also called Vocational Counselor. Nurses assist the patient and family in acquiring new information, developing skills, achieving competence and exhibiting behaviors that contribute to the attainment of a healthy state. Minor symptoms include irritability, lethargy, tremors, or vomiting; major symptoms include seizures, coma, stupor, or death. See also Diffuse Axonal Injury. A surgically-placed tube running from the ventricles which deposits fluid into either the abdominal cavity, heart or large veins of the neck. Characteristics may include increase in deep tendon reflexes, resistance to passive stretch, clasp knife phenomenon, and clonus. Staff is present at night and other times when the consumer is present. Staff is available as needed and at planned intervals to offer assistance and support but not to provide supervision.

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7: Practical Neurology - Understanding The Cumulative Effects of Concussion (May)

Research into traumatic brain injury (TBI) and more specifically, concussion, has greatly increased over the past years. Much of this attention has focused on the world of competitive sports, as doctors, certified athletic trainers, and others have become more cognizant of the effects of these injuries beyond the playing field.

Chronic Traumatic Encephalopathy CTE is a progressive neurodegenerative syndrome believed to be caused by single, episodic, or repetitive head trauma or via the transfer of angular and rotational forces to the brain. However, a recent review paper concluded that it is not possible with any certainty to determine the causality or risk factors for CTE. In addition, there is insufficient evidence at this time to classify CTE as a clinical syndrome. Once thought to be exclusive to boxers, the neuropathological changes associated with CTE have been found in former and current professional football, hockey, and soccer players, professional wrestlers, as well as military personnel who have suffered blast injuries and individuals with repetitive physical abuse. Historically CTE has undergone an evolution with over 28 synonymic terms. Some would go on to develop a progressive neurological syndrome leading to mental or physical helplessness. The patients also complained of persistent headaches, dizziness and unsteady gate. There are more than 28 synonymic terms for CTE. In Millspaugh, in describing effects in Navy boxers, coined the term dementia pugilistica. The term CTE first appeared in the literature in the mid s and in Corsellis, Bruton, and Freeman-Browne described three stages of clinical deterioration in CTE, which have yet to be validated. Bennet Omalu6,7 after examining the brain of a former NFL player, Mike Webster, initially found what appeared to be a normal brain; however, microscopic examination demonstrated Tau deposition. He subsequently diagnosed Mr. Webster as having CTE. This was the first documented case in an American football player. He presented his findings to the chair of the Mild Traumatic Brain Injury Committee of the NFL, who rejected the findings both privately and later in a published paper. They found pathological changes almost identical to those described above and correlated the findings with memory loss, behavioral and personality changes, along with parkinsonism, speech and gait abnormalities. These changes can include mild neuronal loss, isomorphic astrogliosis, increased perivascular neuropil histiocytes, reduced pigmentation of the substantia nigra and locus coeruleus with pigmented laden histiocytes in Virchow-Robin spaces. Subcortical and brainstem structures may show mild neuronal loss as well. They can also be found in a perivascular distribution and with clusters around small intracortical blood vessels and a unique regional involvement of subcortical and brainstem structures. It is suspected that traumatic and mild traumatic brain injury induces the activation of kinases which hyper-phosphorylate tau the specific cascade is unknown. Likely a combination of the above occur and future research is needed and underway. Clinically, Stern and colleagues have suggested that the neuropathological changes i. Cognitive deficits were reported in all but two subjects who were asymptomatic at time of death. Two distinct clinical presentations were found: In addition, there were significantly more APOE e4 homozygotes than expected in the general population one to three percent with two of 11 of the cognition group testing positive when compared to one of 22 for the behavioral group. It is hypothesized that the APOE 34 isoform may have direct neurotoxic effects resulting in mitochondrial dysfunction and cytoskeletal changes, resulting in an increased risk of neurodegeneration. The authors asked the question: The authors identified issues with the observational nature of the investigations, potential for bias, methodological issues and confounding factors as reasons for the poor rating. A major focus of CTE research is in the development of biomarkers for the early detection of the disorder. Tau can be measured in the CSF and our group is currently looking at this biomarker in retired NFL players, however less invasive studies are preferred. Retired alumni had significantly greater levels of tau pathology when compared to healthy controls in the amygdala and all subcortical ROIs including caudate, putamen, thalamus, subthalamus, midbrain and cerebellar white matter. This ligand binds to both tau and NFTs. Two case reports of retired NFL players have been published. Interestingly, the distribution of the tauopathy mimicked that of progressive supranuclear palsy PSP , however the patient did not have any signs

or symptoms of a movement disorder. He carried a diagnosis of frontotemporal dementia and had also had a progressive cognitive decline as well as personality changes. Focal [18 F]-Florbetapir retention was noted at the site of impact suggesting focal amyloid aggregation. Three of the athletes with CTE also developed signs and symptoms of progressive motor neuron. In these three cases, there were abundant TDPpositive inclusions and neurites in the spinal cord in addition to tau neurofibrillary changes, motor neuron loss, and corticospinal tract degeneration. The results suggest that TDP proteinopathy seen in CTE can extend into the spinal cord and is associated with motor neuron disease. Studies have shown otherwise healthy former athletes demonstrate abnormalities in the primary motor cortex i. They found that a history of head trauma the results in concussion with a loss of consciousness has a statistically significant association with the risk of developing PD. A Canadian epidemiological study Harris et al. When looking at the subpopulation of head trauma, twenty-seven 18 percent sustained head trauma from athletic activity and related it to the cause of their PD. De Beaumont et al. This reduction correlated with motor sequence learning. Although there are several possible mechanisms as to how head trauma could result in PD, the most likely mechanism is via an inflammatory response. There are numerous case reports our group included showing progressive neuro-cognitive deficits in retired athletes with a history of concussion. AD has a long prodromal phase prior to presentation, therefore mTBI and TBI early in life would not impact the individual until decades later. Furthermore, studies using transgenic mice have shown that amyloid beta may also spread from the initial TBI site to more distant brain regions,43 which is similar to what is hypothesized with tau in CTE. This protein is highly neurotoxic and predisposed to aggregation, which in turn can result in cell death. In addition to beta amyloid deposition, neuro-inflammation and micro glial activation likely play a role in the development of AD in patients with a history of TBI. Animal models have shown an association between TBI and microglial activation and the development of anti-inflammatory cytokines the later of which may be neuroprotective due to their ability to clear beta amyloid. However, activated microglia and pro-inflammatory cytokines can persist for many years after the initial traumatic event where they can have detrimental effects on brain parenchyma. It is now quite evident that multiple concussions, sub-concussive hits and perhaps even a single concussion can place an athlete at increased risk for the early development of neurodegenerative disorders. Could this signal an end to American football? It is unlikely given the strength of the NFL, which continues to report revenues in the billions. It may, however, influence younger athletes to choose other sports such as baseball, basketball and even golf. This trend has actually already begun with a decrease in the number of children playing Pop Warner football over the past few years, and even at the professional level with the retirement of NFL and NHL players in their prime for fear of the effects of head injury. In addition, over the next few years retired NFL alumni are going to begin to be screened for all of the neurodegenerative discussed above. If preliminary results hold true we are about to see a wave of individuals with significant neurological issues. Major questions remain including: What factors influence the development of neurodegenerative disorders i. Is CTE a separate clinical entity? Or are the neuropathological findings a precursor to AD? The later may be the case as new yet to be published evidence from the Mayo Clinic suggests Tau may be a major player in the development of AD. Clearly more research is needed including longitudinal studies looking at cause and effect, neuropsychological and neuropsychiatric symptoms, early diagnosis i. Expect neurologists to be at the forefront of this research. The spectrum of disease in chronic traumatic encephalopathy. Brain , 43â€”64 2 Corsellis J. Observations on the pathology of insidious dementia following head injury. The aftermath of boxing. Chronic traumatic encephalopathy in a national football league player: Neurosurgery 59, â€” discussion: Chronic traumatic encephalopathy in a National Football League player. Neurosurgery 57, â€” discussion: Chronic traumatic encephalopathy in athletes: Anthony Herber Brain damage in boxers: Clinical presentation of chronic traumatic encephalopathy. The clinical presentation of chronic traumatic encephalopathy. Chronic Traumatic Encephalopathy and Suicide: BioMed Research International, Long-term consequences of repetitive brain trauma: Emerging histomorphologic phenotypes of chronic traumatic encephalopathy in American athletes. Neurosurgery 69, â€” discussion: TDP proteinopathy and motor neuron

disease in chronic traumatic encephalopathy. Br J Sorts Med ; Chronic traumatic encephalopathy and other neurodegenerative proteinopathies. Frontiers in Human Neuroscience. White matter integrity and cognition in chronic traumatic brain injury: Repetitive mild traumatic brain injury augments tau pathology and glial activation in aged hTau mice. J Neuropathol Exp Neurol. Head injury and amyotrophic lateral sclerosis. American journal of epidemiology ; Perceptual and motor skills ; Severely increased risk of amyotrophic lateral sclerosis among Italian professional football players. Proportionate mortality of Italian soccer players: European journal of epidemiology ; Neurodegenerative changes after mild traumatic brain injury. The long-term consequences of microglial activation following acute traumatic brain injury.

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8: Managing Neuropsychiatric Symptoms in Traumatic Brain Injury

ABSTRACT: Traumatic brain injury (TBI) is a major cause of disability and death in the United States. With the devastating statistics of morbidity and mortality, swift medical attention is required to provide the best possible care in the hope of preventing long-term damaging effects.

Print Mild Traumatic Brain Injury and Related Mental Health Issues During the last decade, medical science has elucidated a myriad of expressions of mild traumatic brain injury mTBI , and shown that in some cases, there is the presence of cellular and ultrastructural alterations. Most instances of concussions are self-limited, and resolve within the first week. Concussion symptoms usually consist of the following: Headache, memory loss, dizziness, balance abnormalities, sleep disturbance, visual abnormalities, poor school performance, and others. Emerging research is now suggestive that head impacts may commonly occur during contact sports in which symptoms may not develop and there are no outward or visible signs of neurological dysfunction. These impacts are thus not recognized at the clinical level. This evidence includes biophysics data, advanced neuro-imaging findings, and forensic analyses of the brains of former football players who did not have a diagnosis of concussion during their playing career, as well as in laboratory animals subjected to mild traumatic forces. If true, subconcussion is a previously unrecognized phenomenon that needs to be further explored and at the same time, contemporaneously appreciated for its ability to cause important future detrimental neurological effects. The potential relationship between trauma to the brain and long-term neurodegeneration was implied until dementia pugilistica DP was defined in by Harrison Martland, who was the medical examiner in Essex County, New Jersey. He identified changes in the brains of former boxers, consisting of both gross and microscopic alterations. Geddes, MD, now retired but formerly of the University of London, and colleagues discovered that abnormal accumulations of the major brain structural protein tau could be found in humans as a consequence of repetitive head injury. More than 70 years after the discovery of DP, the neuropathologist Bennet Omalu described the first cases of Chronic Traumatic Encephalopathy CTE in modern athletes, primarily in former professional football players. During the last decade, the symptom complex and clinical course of CTE has emerged. However, since most cases were discovered due to their symptomatic condition and occupational exposure, the case series are highly selective and the incidence and prevalence are unknown. Small, MD, has demonstrated that it is now possible to diagnose CTE-like changes in living subjects using a type of positron emission tomography PET scanning. This shows characteristic uptake of the tracer in a typical distribution including not only cortical and subcortical areas of the brain, but particularly the amygdala and thalamus. The symptoms associated with CTE are described in four categories: Cognition, behavior, mood, and occasionally motor. Behavioral changes include amplified aggression, increased impulsiveness, impaired judgment, and risk-taking acts. More than half of CTE sufferers commit suicide. Occasionally, violent behavior, homicide, and explosive outbursts may occur. There have been numerous positive changes in recent years involving contact sports, particularly football, which have resulted in greater safety for all participants. These include limiting contact in practice, eliminating head-to-head hits in youth practice drills, rules changes to penalize and prohibit egregious cranial hits, and improvements in helmet design. I hope in the near future to eliminate the specter of CTE in contact sport athletes. In the meantime, the possibility of repetitive head trauma causing long-term effects including neurodegeneration should be understood.

9: Military-related traumatic brain injury and neurodegeneration

In addition to CTE, patients who suffer repetitive mild traumatic and traumatic brain injury are at risk for other neurodegenerative disorders A growing body of research supports the hypothesis that professional football players are at an increased risk of neurodegeneration, however cause and effect have yet to be established.

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Conceptualising Palestinian institutions: structure, agency, and transition Waving through a window sheet music A whos who of Maltese background persons in Australia and New Zealand Game of thrones espaÃ±ol Object-oriented software metrics I Can Tell the Truth (Doing the Right Thing) Maine j courtney sullivan Computer Performance Evaluation 92 Revolt of Silken Thomas Hopping and Related Phenomena Five Imagining modernity: symbolic terrains of housing Researches Respecting the Book of Sindibad Water and wastewater engineering fair geyer Fishes and invertebrates Civilizational confinement Poems from the Heart and the Darker Side of Me IDreams Fashion Magazine 2013 Internet information services iis manager Iso 20000 1 Flash photography. The Substitute (An Avon Flare Book) Speech of the Right Hon. W. E. Gladstone, M.P. for the University of Oxford Reversal complexity The Filson Club and its activities, 1884-1922 The Greek Koine and the logic of a standard language Stephen Colvin Portraits for Classroom Bulletin Boards Nassau County, Ny Pocket Map Strength and How to Obtain It The hog; his origin and varieties, management with a view to profit, and treatment under disease An honest preface and other essays A season for unicorns I Am Not Going to Read Any Words Today! Shining star intro book Nokia lumia 630 manual Sbi dd form Part III. On utopia and America: post-constitutional America; Ameritopia. A practical guide to tax practice and procedure Eprg model in international business Dangerous Waters (Strange Matter, No. 22) Eastern Exposition of the Gospel of Jesus According to St. John Being an Interpretation Thereof