Neurotrauma & Critical Care NEWS

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AANS/CNS Section on Neurotrauma & Critical Care

Editor: Martina Stippler, MD, FAANS

In This Issue...

Chair's Message

Dear all,

I am honored to serve as your new chair, a job which began at the end of the last executive committee meeting of the AANS/CNS Section on Neurotrauma and Critical Care on May 2, 2016, in Chicago. In my first Chair's Message, I would like to communicate my background, current passions and vision for the field of neurotrauma in general and the section, specifically.

I am currently a neurosurgeon in the Michigan Head and Spine Institute and serve as professor of neurosurgery at the Oakland University William Beaumont School of Medicine. I still take trauma calls every month, as I have for the past 27 years. But as Dr. Evil put it, "the details of my life are really quite inconsequential…" I have sent a brief bio to be posted on our website, along with a head shot, suitable for framing or target practice. I am currently very excited to be the sole neurosurgeon/scientist onthe research team thatdiscovered that a relatively low dose external radiation (2x5Gy treatments) reduces A beta plaque and improves cognition in transgenic Alzheimer's Diseasemice (AD) (1). Based on our preclinical work, I am co-PI on an FDA-approved phase one clinical trial, now crawling through our local IRB. I believe at some point, given the association between AD and TBI, our work may provide some interesting avenues for collaboration.

We find ourselves at an interesting moment in neurotrauma (recall the Chinese curse: may you live in interesting times). Much has been accomplished in basic and clinical brain, spine and critical care at the bench and bedside. Death rates from severe TBI have decreased by about 25 to 30 percentin the past 30 years or so. Yet, being on call the last weekend, again I am frustrated that we are not effectively preventing injury and really have no "magic bullet" to save neurotrauma victims from death and disability. The answers will come, but not without time, effort and lots of money.As your chair, I am committed to advocating for the resources we need to continue to advance the field.

Some may be disappointed at the 4th Edition of the Guidelines for the Management of Severe TBI when they are published. I reviewed the first edition and remember thinking it was one of the greatest steps forward in patient care. But even then, the limits of guidelines were foreseen: they are evidence based. No new evidence; no new guidelines. We must all strive to put guidelines in context and develop useful patient management based on them, whilenot becomingslaves to them.

During the course of my tenure, I hope we can accomplish the following: promote the use of neurotrauma guidelines to improve the care of neurotrauma victims in the U.S. and around the world; tap the expertise of military and non-military neurosurgical masters to provide educational programs for students, residents and practicing neurosurgeons; and continue the close ties with ThinkFirst in promoting neurotrauma prevention through advocacy, research and education.

I attended the National Neurotrauma Society (NNS) meeting in June 2016. It was a great meeting. Kudos to Uzma Samandani, MD, PhD, FAANS, and Jamie Ullman, MD, FAANS, for theirliaison work. I am committed to strengthening the association between our organizations and said so at their awards ceremony. I was pleased to present the neurosurgery resident's awards, thanks to an educational grant from Medtronic. The brain injury award went to David Darrow, MD, from the University of Minnesota, and the Spinal Cord Injury award went to April Herrity, MD, of the University of Louisville. Only through close collaboration with basic neurotraumatologists

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will we find new treatments for our patients.

One of the encouraging trends showcased at the NNS is the use of mega data sets to help explore the nervous system and improve the relevance of clinical trials. Check out the Allen Brain Research Institute'sbrain atlas website (http://www.brain-map.org/). The scientists there have done a fantastic job of organizing and displaying human and mouse brain structure, gene expression studies and even electrophysiology. There are also many online tutorials to get you started, and it is free.

The TRACK-TBI program is another mega data project which seeks to improve clinical trials by collecting large amounts of data on individual patients to be used to define comparable groups of injured patients. These data sets can then be mined for parameters which correlate with outcome (i.e. the Apo E4 allele) or to compare treatment versus control groups in clinical trials. Geoff Manley, MD, PhD, FAANS, a key person in the National Institute of Neurological Disorders and Stroke (NINDS)-funded TRACK-TBI program, stressed the need for multi-institutional collaboration for the ongoing success of the project. Go to https://tracktbi.ucsf.edu/ to learn more about it.

I have nothing but admiration for the work Dr. Ullman and her committee chairs have done during her tenure as chair. I also believe she has put together a dream team for committees and liaisons. I have asked that the chairs maintain their current positions on our Executive Committee, at least until we meet in SanDiego.

I have submitted some of the Executive Committee members to be considered to serve as either AANS International Ambassadors or representatives to the Society of Neurosurgeons (SNS) "portal programs." We are working on a position statement regarding statemotorcycle helmet laws headed up by Dr. Ullman. The officers are also reviewing spine guidelines by Michael Fehling, MD, PhD, FAANS,and I will keep you posted on this.

If you have new ideas or suggestions, please send them to me at danielm@mhsi.us. If you have problems or complaints, I would like to hear them as well;just be sure to suggest a solution.

Finally, to promote the field of neurotrauma, I propose to start a "Neurotrauma Tweet" to be sent out under that name. I ask that you all consider forming Twitter Teams at your institutions, come up with 140 characters or a pic, and send them tome. The goal is to inform and entertain. I would also like to start a "Tips and Tricks" column for this newsletter. If you have any helpful techniques you find useful when operating on trauma patients, send me a paragraph or two or even a drawing, and we will put it in the newsletter.

You will permit the old man a military metaphor from time to time. The problems of neurotrauma can be compared to attacking and overcoming a fortified hill. Who do you want to lead:the guy who says it is going to be murder and we are all probably going to die or the guy who says it isgoing to be a tough fight, but I will lead, and when we get to the top, the drinks are on me? It is a great honor to be your chair!

Best,

Daniel B. Michael, MD, PhD, FACS, FAANS
Chairman, AANS/CNS Section on Neurotrauma and Critical Care 2016-2018

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NREF
Neurosurgery Research & Education Foundation

Dear Supporters of Trauma Section:

The Neurosurgery Research and Education Foundation (NREF) plays a very valuable role in both the education and the research of neurosurgery.

NREF is a not-for-profit, 501 (c)(3) organization created in 1980 by the American Association of Neurological Surgeons (AANS) to support research and educational efforts in neurosurgery. The NREF is dedicated to providing education to neurosurgeons at all stages of their careers, as well as funding research into new and existing neurosurgical treatments, in order to identify links between best practices and improved outcomes in patient care.

Through voluntary public donations, corporate support and donations from allied groups, the NREF supports endeavors that truly impact lives. Donations can be designated to specific areas of education or research.

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Joanne M. Bonaminio
Director of Foundation Services

Cerebrospinal Fluid Leak Management of Basilar Skull Fractures and Craniomaxillofacial Trauma

Quoc-Bao D. Nguyen & Jason H. Huang, MD, FAANS, Department of Neurosurgery, Baylor Scott & White, Department of Surgery, Texas A&M HSC College of Medicine, Temple, Texas

Overview

Basilar (or basal) skull fractures are fractures through the cranial vault. A majority of these fractures are secondary to motor vehicle accidents or other high-velocity impact forces, such as gunshot wounds (1). Incidence of basilar skull fractures after trauma range from 3.5 to 24 percent depending on whether the diagnosis was established clinically and/or radiographically. These fractures are often present with facial trauma and can be associated with parenchymal contusions, hematoma formation and dural lacerations. Craniofacial injuries and basilar skull fractures are commonly seen in males. Approximately 75 percent of traumatic craniofacial injuries are seen in males at an average age of 35. Cerebrospinal fluid (CSF) leaks, also known as CSF fistulas, may form if the space between meningeal tear and outside environment is continuous. CSF leaks are frequently due to trauma, accounting for 80 percent of CSF leaks, while the rest are iatrogenic or spontaneous (2). CSF leak occurrence after head injury with any fracture is relatively rare (approximately 3 percent); however, basilar skull fractures increases the cidence to 12 to 20 percent.

Anatomy

Location of basal skull fractures can be categorized into three fossas: anterior, middle and posterior cranial fossa. Herein, we specifically discuss fractures of the anterior cranial fossa (or frontobasal), which includes frontal bone, ethmoid bone, cribiform plate and sphenoid bone. The anterior skull base is divided into medial and lateral portions: medially, the rhinobase (nose and paranasal sinuses) and laterally, the orbits and the lesser wings of the sphenoid. The smaller and farther the fracture from midline, the lower the rate of infection with CSF leakage. Radiographically, these anterior fractures have been divided into four types: cribiform plate, frontoethmoidal, lateral frontal and complex fractures (3). Facial fractures involve the cranimaxillofacial bony anatomy, which include the mandible, maxilla, zygoma, facial sinuses, frontal bone, orbit, cribriform plate and sphenoid bones.

For completion, the middle cranial fossa includes the greater wing of the sphenoid, sphenoid sinus and clivus. In particular, this middle region contains several important structures such as the internal carotid arteries, cavernous sinus and pituitary gland. Severe fractures may produce sufficient shearing force to

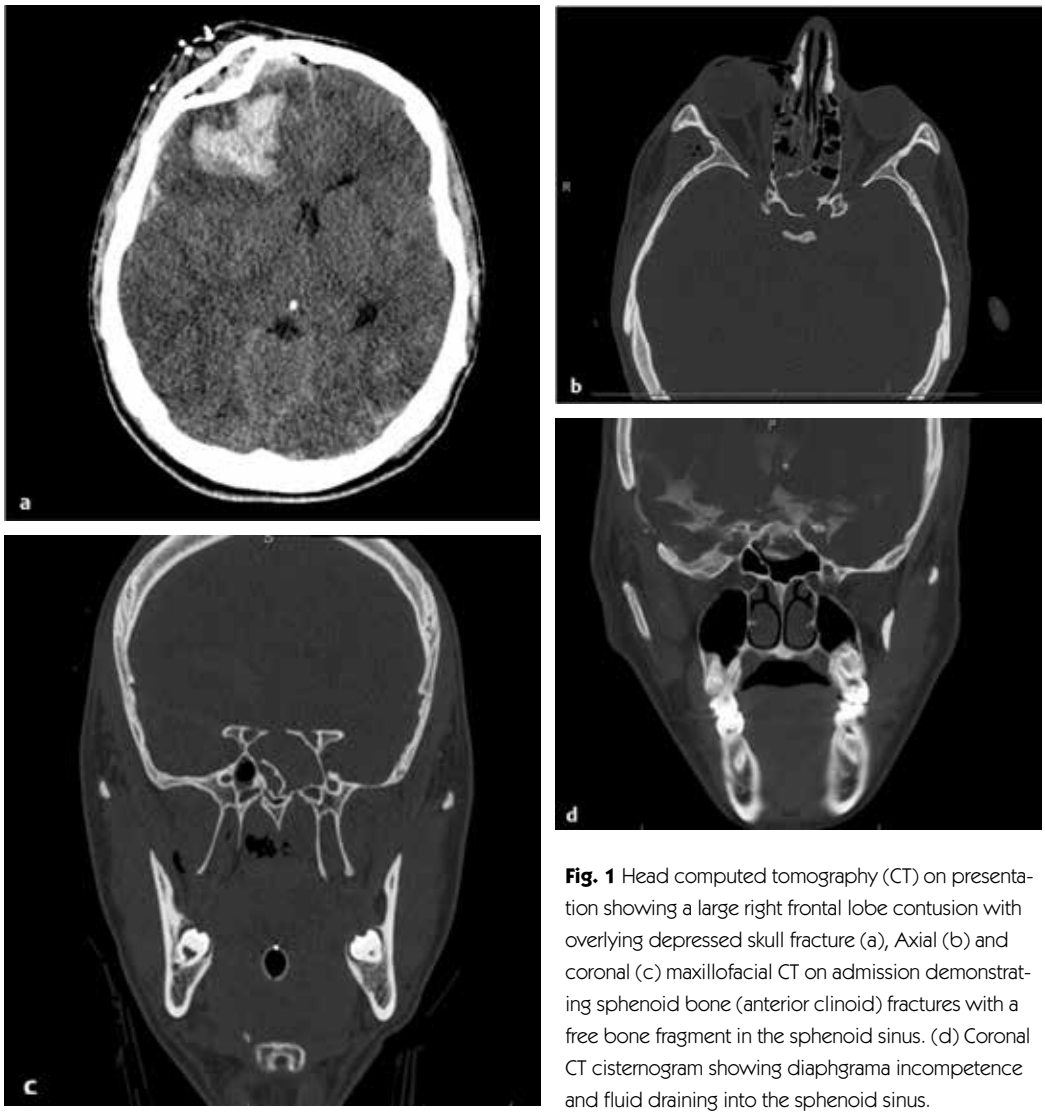

Fig. 1 Head computed tomography (CT) on presentation showing a large right frontal lobe contusion with overlying depressed skull fracture (a), Axial (b) and coronal (c) maxillofacial CT on admission demonstrating sphenoid bone (anterior clinoid) fractures with a free bone fragment in the sphenoid sinus. (d) Coronal CT cisternogram showing diaphgrama incompetence and fluid draining into the sphenoid sinus.

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injure the pituitary gland. Lastly, the posterior cranial fossa includes the petrous temporal bone and occiput.

Clinical Presentation

Classic clinical signs supporting the clinical diagnosis of basilar skull fractures includes periorbital (raccoon eyes) or retromastoid (Battle's sign) ecchymoses, epistaxis, hemotympanum, CSF rhinorrhea or otorrhea and cranial nerve palsies. It is important to note that these signs often appear several hours post-injury. The clinical diagnosis of CSF leakage is typically fairly obvious if clear fluid emanates from the nasal passage; however, confirmatory tests should be done for fluid verification. Facial trauma often coexists with injury to the cervical spine, cranial nerves and internal carotid artery. In a study of 4,786 patients with craniofacial injuries, approximately 10 percent had a concomitant cervical spine fracture with a 2 percent dislocation rate (1). The study reported that the upper face was associated with injuries to the mid to lower cervical spine and cranium, while mandible and midface injuries were associated with fractures of the upper cervical spine and basilar skull. Accompanying anosmia resulted from shearing of CN I, the most commonly injured CN in frontobasal fractures, but other CN dysfunction can occur in 5 percent, with variable recovery. Carotid artery injury occurs in approximately 2 percent in the setting of skull base fracture (5).

Confirmatory Tests and Imaging

The first step in evaluating a patient for suspected CSF leak is to determine if the rhinorrhea or otorrhea contains CSF. Typically, CSF (not contaminated with pus or blood) is clear and described as salty. A quick clinical test to suspect a CSF leak if the CSF mixed with blood is the "halo (or ring) sign", where the clear fluid is dripped on a piece of gauze; if the drainage is CSF, a ring will form around a bloody spot. However, the most sensitive and specific test is ß2-transferrin. ß2-transferrin is highly specific for CSF and is not found in serum, paranasal sinuses, tears or saliva. However, ß2-transferrin is not readily detectable in CSF of newborns and patients with liver disease. In addition, there is a 2 to 4 percent false-negative rate secondary to unusual genetic variants of transferrin. Non-contrast CT cisternography with thin coronal cuts detect the precise location of a leak in more than 90 percent of cases (6). An intrathecal injection of water-soluble contrast can be used for negative plain CT and/or intermittent, refractory or complex leaks. Thin-slice axial bone CT with multiplanar reconstruction is the most sensitive test to detect basilar fractures, with findings of noncoticated, noninterdigitating linear lucencies in the skull base bone. Frontobasal fractures typically run longitudinally parallel to the cribiform plate. Pneumocephalus may be present on plain film or CT and is diagnostic for basilar skull fractures in the absence of an open cranial fracture. Magnetic resonance imaging (MRI) is the preferred method if there is suspicion for meningitis or cerebral parenchymal injury. CT angiography (CTA) or MR angiography (MRA) should be used if there is suspected carotid or vertebral artery dissection or carotid-cavernous fistula.

Treatment Algorithm for CSF leak

A treatment algorithm for managing traumatic CSF leaks is proposed below. The majority of traumatic CSF leaks resolve spontaneously and therefore all non-emergent care begins with conservative management (7).

1. Immediate operative repair with elevation of fracture if significant hematoma or mass effect
2. If simple fracture and leak, bed rest and observation ± prophylactic antibiotics
3. Lumbar drain for three to seven days
4. ± CT myelogram
5. Craniotomy for direct dural repair ± periosteal, temporalis or dural substitute grafting or endoscopic or transfacial repair with use of a gasket seal
6. Ventriculoperitoneal shunt or alternate implanted CSF diversion

Conservative Management and Lumbar Drain

Most CSF leaks after trauma spontaneously resolve in up to 40 percent in three days and 70 percent by one week with simple observation and bed rest (8,9). Fractures of the temporal bone have a significantly higher spontaneous resolution rate than anterior skull base defects (2). The main goal of conservative management is to lower the intracranial pressure. Non-invasive measures, such as acetazolamide and avoidance of straining with stool softeners, are attempted first. If the leak remains persistent, lumbar drain should be considered.

The lumbar drain diverts CSF and reduces hydrostatic pressure through the dural tear, which permits closure of the dural defect by the patient's own inflammatory response. The success rate of CSF diversion at seven days is 70 to 90 percent (7). Typically, the drain output is 10 mL/h, but the output should never exceed 500 mL/d because this exceeds the daily rate of production (approximately 450mL/d). Patients often have a low-pressure headache, which can be treated by decreasing the rate of drainage or lowering the head of the bed. In the rare circumstance of overdraining, cranial nerve and brainstem dysfunction can occur secondary to transtentorial herniation. If this occurs, immediately stop drainage and place patient flat or in slight Trendelenberg with 100 percent oxygen. In addition, CT or plain film should also be obtained to rule out tension pneumocephalus. For this complication, it is advised that all patients with lumbar drains should receive frequent nursing observation and continuous noninvasive monitoring. These drains have a relatively low morbidity rate, with 15 percent nerve root irritation, 5 percent infection rate and one percent persistent lumbar leaks necessitating a blood patch (10).

Although 10 to 25 percent can develop meningitis, a recent Cochrane review of routine prophylactic antibiotics for basilar skull fractures, including 168 studies and five randomized trials, found that overall, there was no difference in the rate of meningitis versus control (11). Nevertheless, the routine use of prophylactic antibiotics still remains controversial. There is evidence that CSF leakage that persists for more than seven days has a significantly higher

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rate of meningitis (2). For this reason, conservative therapy should never be continued for more than seven days unless the patient is too unstable for surgical repair.

Surgical Repair

Indications for operative intervention for CSF leaks after skull base fracture depend on whether delayed, persistent/recurrent or complicated by recurrent meningitis. During the acute phase (from presentation up to five days), surgery is generally performed for hematoma formation or brain contusions (8). The onset of CSF leaks can be delayed up to 30 days and rarely recur late after skull fracture. However, recurrent CSF leaks have been reported as late as nine to 30 years after injury. Delayed diagnosis carries high risk of meningitis, with up to 16 percent of patients with occult CSF leaks presenting with meningitis (17,18). Patients with skull base fractures should therefore be followed accordingly to prevent a missed diagnosis of an occult leak.

Typically, operative repairs occur after failure of conservative management, such as bed rest or lumbar drain diversion. Surgical treatment of frontobasal skull fractures is often a multidisciplinary effort with surgical subspecialists in ear, nose and throat, orbitomaxillofacial surgery and plastic surgery. The exact type and location of surgery depend on the fracture pattern and goal. The most common location of dural tears is at the cribriform plate of the ethmoid bone, posterior frontal sinus or planum sphenoidale (12). The most effective studied technique used for open operative repair of anterior fossa fractures is a bicoronal craniotomy with an intradural pericranial flap. Intradural approaches is typically the procedure of choice given that dissecting the dura off the fossa results in additional dural tearing and difficulty identifying whether the identified dural tear(s) is the cause or iatrogenic. This approach has a low failure rate of less than 3 percent, but there is increased morbidity from brain retraction (13). Approximately 8 percent of intradural approaches for traumatic frontobasal skull fractures have frontal lobe injury. Surgical repair of CSF fistulas can be augmented with a lumbar drain to prevent dislodgment of the graft, though controversy still remains as to whether this has significant benefits.

Another alternative to repairing CSF fistulas surgically is using extracranial approaches, such as endoscopic endonasal skull base repair (2). Endoscopic endonasal skull base repair is most commonly used for spontaneous or iatrogenic CSF leaks but has also been shown to be effective in traumatic CSF leaks with a success rate of 80 to 90 percent (14). Dural tears large as 1.5 cm were able to be repaired. This type of procedure involves finding the exact location of the leak using preoperative thin-cut CT with or without fluroscein and removing all damaged mucosa, granulation tissue and communized bone remnants (15). Once at least 5 mm of bone is circumferentially freed to create a conductive graft site, a trimmed portion of adipose tissue, muscle or fascia lata is placed in the defect. This is then covered with a ridged buttress, fibrin glue and, ideally, a middle turbinate flap. This gasket seal technique has a 5 percent failure rate for large skull base tumors (16). Patients should be instructed not to blow their nose or use a straw until the graft heals. Saline spray should be used beginning the first day after surgery to promote mucosal healing.

Summary

CSF leakage after head injury is relatively rare but can be present in up to 20 percent of patients with basilar skull fractures. If there is no indication for early surgical intervention (i.e., depressed fracture, hematoma or contusion), the preferred initial treatment of CSF leaks is bed rest and observation. There is no strong evidence to support the use of prophylactic antibiotics. Definitive repair should take place no longer than seven days after onset of the leak because the rate of meningitis increases. Craniotomy for direct dural repair and endonasal endoscopic techniques both have high success rates and should be selected based on leak location, surgeon comfort and available multidisciplinary support. The lumbar drain is a useful bedside tool to augment conservative or operative approaches. Rarely CSF leaks can recur or become chronic and may need ventriculoperitoneal shunting.

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Officer in the Spotlight: Odette Harris, MD, MPH, FAANS

By Martina Stippler

Our officer in the spotlight this time is Odette Harris, MD, MPH, FAANS. She is an associate professor of neurosurgery at the Stanford University School of Medicine. She is also the director of brain injury, associate chief of staff of rehabilitation and director of the Defense Veterans Brain Injury Center.

Her clinical focus in neurosurgery is traumatic brain injury (TBI) and peripheral nerve. She competed her medical education and neurosurgery residency at the Stanford University School of Medicine. She also completed the prestigious Van Wagenen Fellowship at the University Hospital of the West Indies and a peripheral nerve fellowship at the Louisiana State University in New Orleans.

Dr. Harris kindly answered the following questions for us:

1. **What do you think is the biggest unanswered question in TBI?**
   There are many that, unfortunately, remain unanswered. If pressed to select one, I would state that a closer link to prognosis with presentation remains elusive in its nuance.

2. **What are the changes in clinical TBI research?**
   I believe that there has been a shift from a search for the magic bullet, i.e. strict pharmaceutical agent, to epidemiology and meta-analysis, with a focus on specificity in identifying injury and tailoring management.

3. **What TBI question did you set out to answer?**
   My focus has changed over time, as it does for everyone in research and clinical care. Nonetheless, I remain driven by epidemiology, as it is key in the armamentarium. Most recently, I have focused on how subpopulations in the greater TBI cohort are impacted by algorithms that might not be applicable. The mismatch is linked to large data management that lacks a nuanced methodological approach and thus, imposes bias that have potential to translate to management. This work builds on noted historical lapses in foundational epidemiological research.

4. **What advise could you give to other neurosurgeons dealing with TBI patients?**
   In addition to surgical skill and training, I believe the best assets are optimism and persistence. As trauma neurosurgeons, we play a critical role at an existential time in the lives of our patients. This is invaluable at the gloomiest of moments. I also hold that we live in exciting times regarding neurotrauma research and care. The potential for impact held by genomics and neuroimaging are promising in aiding our understanding of injury profiles and in targeting therapeutics.

5. **What is your biggest challenge during your day-to-day work?**
   I need more time!

Head Impacts in Contact Sports and Long-term Brain Degeneration

By Vimal Patel, PhD; Julian Bailes, MD, FAANS

The consequences of mild traumatic brain injury (MTBI) have garnered increasing scientific and public attention following reports of altered mood and behavior, as well as progressive neurological dysfunction in athletes who have been exposed to repetitive concussive and subconcussive impacts. Similar mechanisms of injury may occur in military service members exposed to repetitive blast injuries. This has led to TBI research that, in volume and content over the previous decade, supersedes all previous information (1).

TBI is traditionally thought of as involving both primary and secondary injury phases. The primary injury is represented by the moment of impact, resultant from the translation of kinetic energy and force vectors in either a linear acceleration-deceleration mechanism, through rotational vectors, or a combination of both. Additionally, the motion of the brain within the cerebrospinal fluid space can result in brain contact with the underlying irregular bony surface of the skull base, micro-vacuum phenomena within the cerebral tissue and the mechanical tearing and stretching injury to neurons and their projections can result in both local and remote damage (1).

In most instances, the symptoms of concussion are self-limited, resolving on their own within the first week. In about 10 percent of cases, a longer symptomatic period may occur, causing the post-concussion syndrome (up to 12 weeks post-injury) or the prolonged post-concussion syndrome (12 weeks and greater). Concussion symptoms usually consist of the following: headache, memory loss, dizziness, fatigue, nausea, imbalance, sleep disturbance, visual abnormalities, poor school performance, depression, anxiety and others.

The potential relationship between trauma to the brain and long term degeneration was first discovered and defined in 1928 by Harrison S. Martland, MD, who was the medical examiner in Essex County, N.J. He identified changes in the brains of former boxers, consisting of both gross and microscopic abnormalities, which he called Dementia Pugilistica (DP). It is believed that DP occurs in approximately 20 percent of former professional boxers.

Chronic Traumatic Encephalopathy (CTE) was discovered in the brain of a former Pittsburgh Steelers Hall of Fame American Football player, Mike Webster, and reported by Bennet Omalu, MD, MBA, MPH, in 2005. While his brain grossly appeared to be normal, microscopically it had a buildup of tau, a major structural protein in the brain, within neurofibrillary tangles (NFT) and neuritic threads (NT) (2). Only by these special neurodegenerative staining techniques did he see the microscopic clues to this clinical entity. Through further detailed autopsy studies of CTE cases, the areas of the brain with tau staining in CTE are most commonly the frontal and temporal lobes and a characteristic random and patchy perivascular distribution, in a subcortical location and in the amygdala, thalamus and other deep brain structures (3). A 2016 National Institutes of Health (NIH) expert neuropathological panel established that the pattern of CTE is unlike any other form of brain degenerative disease with the collections of NFT or NT occurring in a unique and distinguishing pattern (3). It is believed that the more superficial and frontal areas of the brain are involved due to the direct contact with sports-related head impacts.

These pathophysiological features are associated with a characteristic behavioral syndrome for CTE, with symptoms in four categories: cognition, behavior, mood and occasionally motor. Behavioral changes include amplified aggression, increased impulsiveness, impaired judgment, and risk taking acts. Most often, a 6- to 12-year latency period following retirement from contact sports is then associated with failure in business, financial and marital relationships, homelessness, drug and/or alcohol abuse, depression, mild cognitive impairment and dementia, and many CTE sufferers commit suicide. Researchers have reported that CTE can be considered in two major clinical categories, with one group whose initial features develop at a younger age involving behavioral and/or mood disturbance and another group whose initial symptoms develop later in life and involve cognitive impairment. The language function is usually normal, but intelligence is often ultimately affected by the numerous effects described above (4).

While the risk of developing CTE has historically been discussed in the context of concussive injury and extensive neurotrauma exposure, emerging evidence indicates that a history of diagnosed or major concussions is not a requirement, but instead, repetitive subconcussive injury may play a prominent role in CTE development (5). This finding is based upon lack of documented concussive injury in numerous individuals diagnosed with CTE, although lack of self-reporting by athletes was common, and concussion without loss of consciousness was not recognized and treated as seriously as it is today. Evidence related to subconcussive injury and possible predilection to neurodegenerative disease includes the documented rates and severity of impacts in football linemen, a position in which retired athletes have been diagnosed with CTE (5). Additional evidence is the demonstration of neuroimaging and cognitive changes in those without a history of documented concussions as well as laboratory evidence indicating cellular and ultrastructural alterations without changes in levels of alertness or behavior. Despite the large number of people exposed to concussive and subconcussive injury through various sports and military service, CTE appears problematic for only a small subset of the population exposed to neurotrauma. Nonetheless, we currently do not know the incidence and prevalence of CTE since there has been no longitudinal study conducted to substantiate estimates of several prominent CTE research groups. The largest review to date, Maroon et. al., surveyed the clinical findings in all 153 CTE cases reported in

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the literature and found that 63 had a history of participation in football with majority of these having played at the professional level (6). The most common age at death of individuals with CTE was the range of 60 to 69, with 72.7 percent dying before the age of 70 (6).

Identification of other variables involved as risk factors for CTE remains in its early stage with speculation that genetics and lifestyle may be implicated. Just as in other forms of neurodegeneration such as Alzheimer's disease, it has been postulated that the role of the ApoE ε4 (ApoE4) allele may be a susceptibility factor for the development of CTE; however, this has yet to be borne out.

Research continues to try to identify who is at greatest risk for getting concussions and ultimately, CTE. More studies are needed to follow former athletes over many years in order to know the true prevalence of CTE. A major focus of CTE research is investigating confirmation of the diagnosis of CTE in living individuals with several promising imaging technologies being evaluated, with F18DDNP PET imaging for tau and amyloid protein labeling being the most advanced (7).

Currently, there are no established treatments for CTE; therefore, reducing the risk for CTE development becomes the primary goal by limiting the exposure to concussive and subconcussive head impacts. There have been numerous positive changes in recent years involving contact sports, particularly football, resulting in greater safety for all participants. These include limiting contact in practice, eliminating head-to-head hits in practice drills, rule changes to penalize and prohibit egregious cranial hits, improvements in helmet design, new technology, such as helmet sensors and efforts to mitigate brain slosh, among others. It is hoped that in the near future CTE will be eliminated in contact sport athletes, but in the meantime, the possibility and implications of repetitive head trauma causing long-term effects, including brain degeneration, should be understood.

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The Brain of A Fighter

Martina Stippler, MD, FAANS; Ayesha Quddusi; Xander Sobecki

A few months ago, the death of Muhammad Ali, one of the greatest athletes of our times, was on everybody's mind.

Ali's remarkable journey was retold from various angles, and he died from complications of Parkinson's disease. Surprisingly, none of the journalist or moderator mentioned his Parkinson's diagnosis in relationship to his boxing career and repeated head trauma.

Is it just semantics? Does every news outlet use Parkinsonism as an umbrella term to describe symptoms of muscle rigidity, involuntary tremors and a stiff gait irrespective of the aethology? Is it ignorance, or is it more than that? Does the layperson know that repeated head trauma causes Parkinson-like symptoms, also called "dementia pugilistica," or "boxer's dementia? (4)" Does the media have any responsibility to educate the public about the differences?

When Ali was 12-years-old, his bike was stolen. He wanted to teach the thief a lesson and was advised to learn how to fight before getting into one. Learn he did, and so his career began. Ali went on to win numerous titles and championships and was commonly referred to by fans as "The Greatest." But, did he know about the battle that lay ahead of him? Nearing his retirement, fans and the media began to notice how Ali's speech would sometimes slur. Eventually, he was diagnosed with Parkinsonism, a set of motor symptoms that result in expressionless masked faces, a characteristic pill-rolling tremor and shuffling gait (1).

In 1996, Ali carried the Olympic torch. The world watched in shock and awe as the once-mighty former champion lit the torch, his other hand jerking with involuntary tremors. Despite this, they could still see Ali's fighting spirit. The disease had not defeated him. He became the world's most famous Parkinson's disease patient, raising awareness about the disease as well as funds to support research.

While lauded to the modern day for unmatched athleticism and talent, it is debated within the boxing community if Ali's singular style of fighting elevated his risks for traumatic damage. Regarded as one of the most agile fighters in history, Ali abandoned traditional blocking techniques in favor of weaving away from punches and encouraging his opponents to overextend their attacks. This unorthodox and highly dangerous tactic was admired by fans for being impossibly difficult to achieve, but Ali's daring approach to defense left him vulnerable to full-force impacts much more devastating than those faced by the traditionally trained boxer.

For years, Ali's fans have speculated whether the Parkinsonism was caused by the numerous blows to his head taken during his boxing career. Ali himself would say that he had spent a long career taking blows to his head, so there was a great possibility something could be wrong (2).

What remains undisputed is that Ali did face mild traumatic brain injury (MTBI) as part of his boxing career for decades, and MTBI can cause symptoms of Parkinson's, Alzheimer's and psychosis, depending on where the damage is done within the brain (2). Ali was unusually young for a diagnosis of Parkinson's disease: the average age for the onset of symptoms is 60, whereas he was 42 when he was diagnosed. In fact, his symptoms had first appeared

when he was even younger. Ali was even mistakenly treated for hypothyroidism at first. Only 10 percent of Parkinson's patients may have onset of symptoms before the age of 40 (3).

The earliest complaints Ali had were that he would forget to wipe food from his mouth because his lips would be numb, and he could not feel anything on his face; this was indicative of damage to the brainstem from the MTBIs due to boxing. Over the years, the steady progression of the disease was more indicative of classic Parkinson's disease (3). Ali's doctor, Stanley Fahn, MD, was initially concerned that the boxer's condition might have been a result of the numerous blows to his head during his boxing career and that the boxer might have pugilistic Parkinsonism or post-traumatic Parkinsonism. However, this relationship was lost in the narrative of Ali's life, dissected and retold countless times after his death. Over the years, the steady progression of the disease was more indicative of classic Parkinson's disease according to Wallace Matthews, MD (3).

In 1928, Harrison S. Martland, MD, was the first person to describe dementia pugilistica (DP) in professional boxers. He observed that the boxers experienced declining mental ability, slowed reflexes and rigidity. This phenomenon was commonly known as "punch drunk" (5). Approximately 20 percent of retired boxers exhibit signs and symptoms of DP. The more matches a boxer takes part in, the more likely is the diagnosis of DP. In general, most boxers have some form of cognitive, motor or behavioral deficit by the end of their careers. The punches delivered in a match can have a speed of at least 10 mi/second on impact with the head. This causes

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rotational acceleration of a brain in addition to the shear force of the blow. TBI also results in the formation of more beta amyloid, which is the primary constituent of Alzheimer's plaques (6). In DP patients, scarring is also seen in extra pyramidal tracts, including substantia nigra and the cerebellum (7).

Along with boxers, athletes who take part in other contact sports such as football are exposed to mild neurotrauma during their careers. They are at risk of developing chronic traumatic encephalopathy (CTE), which leads to neurodegenration. DP is considered a type of CTE (7). In recent years, after recognition of the long term sequel of sports-related head trauma and concussion, work has begun toward minimizing and preventing head injuries in sports (8). However, there are still important stakeholders in professional sports who deny the relationship between repeated minor head trauma and CTE.

I am disappointed at the missed opportunity during Ali's death news coverage when no expert drew attention to exposure of many professional athletes today to repeated head trauma, leaving them injured and damaged at a young age.

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ThinkFirst Injury Prevention Abstract Award

Jamie Ullman, MD, FAANS

The ThinkFirst National Injury Prevention Foundation was established in 1986 by the American Association of Neurological Surgeons (AANS) and the Congress of Neurological Surgeons (CNS) to prevent brain, spinal cord and other traumatic injuries. In honor of their 30th anniversary, an award has been established for the best poster or presentation on brain and spinal cord injury prevention. This year the inaugural award was presented to Chunyan Li, PhD, assistant investigator at Northwell Health's Feinstein Institute for Medical Research (FIMR) at the 84th AANS Annual Scientific Meeting, held April 30–May 4, 2016, in Chicago, for the presentation on brain and spinal cord injury prevention or treatment.

Dr. Li was recognized for her presentation entitled "Transient Elevation of Brain Temperature Could Serve as a Surrogate Marker of Cortical Spreading Depolarization," co-authored by Raj K. Narayan, MD, FAANS, chair of Neurosurgery at North Shore University Hospital. The study findings show a correlation between cortical spreading depolarization and brain temperature.

The award of $1,000 will be given for the best poster or presentation accepted at the 2016 CNS Annual Meeting and the 2017 AANS Annual Scientific Meeting. The topic should focus on an area of brain or spinal cord trauma and the prevention or treatment of the injury. Final determination of awards will be determined by the ThinkFirst Awards Committee.

The ThinkFirst Foundation is a 501(c)(3), nonprofit organization with more than 180 chapters worldwide. Its mission is to prevent brain, spinal cord and other traumatic injury through education, research and advocacy. More than 6,300 injury-prevention presentations are provided to students and families each year in the U.S. alone.

Concussion: A Movie Review

Uzma Samadani, MD, PhD, FAANS, is the Rockswold Kaplan Endowed chair for traumatic brain injury (TBI) research at Hennepin County Medical Center and an associate professor of neurosurgery at the University of Minnesota.

Being neither a habitual movie-goer nor a frequent reviewer (this is in fact my first foray into formal movie review writing), I feel compelled to limit this review of Concussion to a discussion of its scientific merit. Those of you seeking the more traditional and greater amusement of standard fare movie reviews, including speculation as to whether Billy Baldwin would have made a better Julian Bailes, MD, FAANS, than Alec Baldwin, are advised to read elsewhere.

Concussion cost $35 million to produce, $14 million to market and grossed $48 million dollars globally, resulting in a loss for its creators. Despite its name, which suggests the topic of acute neurologic dysfunction, the movie portrays the "discovery" of chronic traumatic encephalopathy (CTE) by Bennett Omalu, MD, MBA, MPH, and his subsequent efforts to publicize the dangers of contact sports, despite efforts by the National Football League (NFL) to intimidate him, his wife and his mentor, Cyril Wecht, JD, MD. Omalu published an editorial in the New York Times a few weeks prior to the movie's release, calling for the ban of contact sports in children under the age of 18 years due to the latent risks of repetitive brain injury (http://www.nytimes.com/2015/12/07/opinion/dont-let-kids-play-football.html?_r=0).

In the movie, Omalu is played by Will Smith. I have never met Smith nor can I claim familiarity with his compendium of work, but I have met the real life Omalu and have heard him speak passionately on the topic of brain injury. He genuinely believes that he is a messiah, come to earth to educate regarding the dangers of contact sports. He is an eloquent speaker who presents anecdotes with the conviction of definitive studies.

To put this movie into context, one has to understand a little about CTE. The disease had a gritty start. In 1928, a Newark, N.J., forensic pathologist, Harrison S. Martland, MD, first described a distinct neurologic entity in the brains of deceased boxers with the term "dementia pugilistica."(http://jama.jamanetwork.com/article.aspx?articleid=260461) By 1957, there had been a handful of case reports and series published in the literature, and the eminent Queens Square neurologist, MacDonald Critchley, MD, coined the term CTE based on his findings of Parkinsonian symptoms in 69 living boxers (1). In 1969, Anthony Herber Roberts, also from Queens Square, published an epidemiologic study of a random sample of 250 boxers (out of 16,781 registered) who fought between 1922 and 1955. CTE was found in 17 percent of them (http://practicalneurology.com/2015/05/understanding-the-cumulative-effects-of-concussion).

In 2005, when Dr. Omalu reported finding CTE in the American football player, Mike Webster (2), the British were simply not amused. Calls for retraction of the paper came not only from the alleged NFL-funded physicians cited in the movie but also from the British who reported that the American CTE bore no resemblance to its original counterpart rigorously described by neuropathologist professor J.A.N. Corsellis. They argued that what Dr. Omalu was seeing was a distinct problem both by clinical symptoms and under the microscope. One might speculate that the paper was published in the journal Neurosurgery (which was, rather ironically, edited at that time by NFL consultant and advisor Michael L. J. Apuzzo, MD, PhD) rather than a pathology journal due to disdain by the pathologists. The neuropsychologist Jim Andrikopoulos, PhD, wrote in an editorial to the British Medical Journal that stated "CTE, as defined in America, is not a neurological entity, but is a culture-specific social phenomenon." (http://www.bmj.com/content/350/bmj.h1381/rr-1)

But, CTE in America was not just one entity. Independently from Dr. Omalu, researchers at Boston University, led by pathologist Ann McKee, MD, went to the media with their own findings, ultimately telling PBS' Frontline that CTE was present in 87 of 91 former professional football players. Unlike Dr. Omalu, who stated that a single concussion can lead to CTE, the Boston group indicated that more than one brain injury had to have occurred. The pathologic findings were different in Omalu-CTE and Boston-CTE and neither resembled British-CTE, which could be diagnosed in the living. Multiple scientific reviews of both forms of the American-CTE argued that it could only be diagnosed in the dead.

With confusion rampant in the pathology literature, the government stepped in, sponsoring a "consensus meeting to define neuropathological criteria for the diagnosis of chronic traumatic encephalopathy" which took place Feb. 25-26, 2015. Twenty-five cases were reviewed by seven pathologists in advance, and a definition with four types was established (3).

In the meantime, investigators at Mayo Clinic reported that one-third of all males participating in contact sports were at risk for CTE. However, CTE had no clinical symptoms and was found during autopsy in people who died in their late 70s (4). Finally, the Queens Square group countered with a paper showing that American-CTE, as defined by consensus criteria, is present in equal incidence in people with neurodegenerative diseases as it is in normal healthy controls (5). In fact, the only things that all the neuropathologists can agree on is that American-CTE causes no symptoms in most of the afflicted, and the vast majority of the American demented have neither a history of brain injury nor findings of CTE.

Most recently, given the information that mislocalized and phosphorylated tau, the *sine quae non* of CTE, is neither causative nor pathognomic for post-traumatic dementia (6), there has been a call to abandon the search for identification of CTE in living people due to its clinical irrelevance and to seek schema for better diagnosis and classification of the manifestations of brain injury in the living (6).

Thus, CTE is a disease that either causes dementia and movement disorders in 17 percent of career boxers or no symptoms at all in 12 percent of the normal population, depending on how one defines it. It was not discovered by Dr. Omalu unless one considers changing the pathologic criteria for a disease to something present

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in the general population without associated symptoms as a discovery, and publication by Dr. Omalu was not opposed by the NFL since Dr. Apuzzo, who worked for the NFL at that time, accepted the paper.

Concussion presents Dr. Omalu's mentor forensic pathologist, Dr. Wecht, as indicted and persecuted due to his role in publicizing the ill effects of football on the brain. In real, life Dr. Wecht was investigated for using public funds and facilities for private research prior to any of his work on brain injury as depicted in the movie. (https://en.wikipedia.org/wiki/Cyril_Wecht)

The reality is that brain injury is absolutely, unquestionably bad but also far more complex than can be easily conveyed by a Hollywood film. It impairs memory manifested as shrunken hippocampi, affects cognitive processing and executive decision-making and can lead people into a death spiral of depression and addiction, among other consequences. It is the No. 1 cause of death and disability in American people under the age of 35. A single concussion is associated with increased suicide risk, per a Canadian epidemiologic study (7).

Epidemiologically, the state of California shows an increased risk for dementia in elderly persons incurring mild TBI after the age of 65 years; however, no such association was noted in younger people.

The greatest question is "does playing middle school and high school contact sports mean constant exposure to subclinical damaging brain injury?" Is the risk of sports greater than the benefit?

The challenge for us, as physicians, is that we currently lack the ability to objectively measure CT-negative brain injury, so we are incapable of definitively answering these question. To further complicate the matter, we also have trouble objectively quantitating dementia. Thus, we are trying to assess the impact of something we cannot detect on an outcome we cannot measure.

Suicide rates among both NCAA(8) and NFL (9) football players are less than age-matched, non-athlete peers. A 50-year longitudinal study of high school football players from Rochester, Minn., demonstrated no increased risk of neurodegenerative diseases as compared to members of the chorus, glee club or band (10). In other words, playing high school football carried the same risk for dementia as playing the flute. Football has a lower death rate than equestrian, bicycling, skiing, snowboarding and skateboarding. Swimming and diving have a higher incidence of spinal cord injury.

A multitude of studies suffering from ascertainment bias and lacking controls raise questions about the safety of sports, but few if any of these have been examined in prospective studies.

Dr. Omalu has called for a ban on football, hockey and soccer because children can achieve the same health benefits from other sports. The reality is that there are kids – perhaps at least half the children currently playing football fall into this category – whose body habitus and personality are well-suited for football but not necessarily for other sports. It is naïve to think that the children currently playing football will instead happily run cross-country in the fall or those playing hockey will pick swimming. There is a subset of children with football talent that will not be interested in switching to other sports.

If football is eliminated, a majority of current players will instead become sedentary which will result in an increase in obesity, diabetes, high blood pressure and cardiovascular disease that might offset the original risk of brain injury from football. A sedentary lifestyle is an independent risk factor for dementia: that alone increases the risk by two-fold whereas NFL players have a greater life expectancy, with reduced cardiovascular risk, than their peers (11).

The American Academy of Pediatrics (AAP) has issued a position statement on tackle football, suggesting that it should not be banned but that measures such as safer tackling technique, limited contact practices and neck strengthening measures are incorporated. (http://pediatrics.aappublications.org/content/early/2015/10/20/peds.2015-3282) The implications of this position statement are that, despite our limited knowledge, and acknowledging the need to make sports safer, the play of sports, including with contact, is still better than inactivity.

Concussion is a compelling and emotional story of a few football players who developed neurologic problems that are likely exacerbated by severe repetitive head trauma. The movie translates the pathology findings of these exceptional NFL players to the youth level and argues that children should not play contact sports. One does not generally expect Hollywood to get science history correct, and, in that respect, the movie delivers by ignoring and revising the story of the complex discovery of CTE, over-emphasizing Dr. Omalu's role in that history and downplaying Dr. Bailes' other research on brain injury. The movie also states that Dr. Bailes is a neurologist and not a neurosurgeon. The disservice of this movie is in creating a rabid fear of adolescent sports participation due to risk for brain injury that has not been scientifically validated, yet could have profound consequences on the physical health and mental well-being of our next generation.

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