According to a 2003 report by the CDC, mild traumatic injury results in roughly 500,000 emergency department visits without hospitalization and almost 200,000 hospitalizations per year. However, it’s believed this number is an underestimate because a number of people with mild traumatic brain injury (TBI) are never seen in an ED. Increasingly, TBI is associated with various neurological sequelae, as listed in Table 1. Among these, is epilepsy.

A population study in seizures after TBI by John Annegers, et al. showed that the standardized incidence ratio was 1.5 after mild injury, 2.9 after moderate injury, and 17 after severe injury. Significant risk factors for later seizures included brain contusion with subdural hematoma, skull fracture, loss of consciousness or amnesia for more than one day, and an age of 65 years or older.

Additional data are available from the Southern Arizona VA registry, which show: of 184 patients with TBI, three percent of mild TBI cases experienced seizures, five percent of moderate TBI cases experienced seizures, and 32 percent of severe TBI cases experienced seizures. Of these, 99 had seizure-like spells, with 13 having confirmed seizures, 22 having questionable seizures (requiring further evaluation for diagnosis), and 64 having other causes.

This article is adapted from a presentation by Dr. Hishaw at the American Academy of Neurology 2012 Annual Meeting, held in New Orleans in April 2012.

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UNDERSTANDING THE INJURY
According to the Department of Defense definition, acute injury results from primary (mechanical) physical disruption.

TABLE 1. NEUROLOGICAL CONDITIONS LINKED TO TBI

- Epilepsy
- Dissociation
- Frontal lobe syndrome
- Migraine
- Flashbacks/nightmares
- Panic attacks
- Vertigo
- Psychosis and Schizophrenia
- Substance abuse
- Malingering versus factitious disorder versus conversion disorder
tion of the brain. Traumatically-induced structural injury and/or physiological disruption of brain function as a result of an external force that is indicated by new onset or worsening of at least one of the following clinical signs, immediately after the event:

1. Any period of loss, or a decreased level, of consciousness
2. Any loss of memory for events immediately before or after the event
3. Any alteration in mental state at the time of the injury (confusion, disorientation, slowed thinking, etc.)
4. Neurological deficits (weakness, loss of balance, change in vision, praxis, paresis/plegia, sensory loss, aphasia, etc.) that may or may not be transient
5. Intracranial lesion

Because of the position of the brain in the cranium, sudden acceleration, deceleration, or torsion injuries can cause the brain to strike the skull, causing injury.

There are two types of injuries here: direct trauma causing contusion, hemorrhages and lacerations (coup/contrecoup injuries), and diffuse axonal injury from stretch injuries to the deep white matter.

HOW IT HAPPENS
Seizures originate from gray matter. This includes the cortex and some smaller, deeper areas. Following this down the simplest path, a contusion leads to blood irritation, which leads to an increased likelihood of seizure (This is more or less a primary injury, however, a secondary injury can occur). Exploring further, at the moment of head injury, neurons discharge, resulting in an extensive release of neurotransmitters, followed by neuronal suppression. A flood of glutamate opens the pathophysiological pathway to secondary cell death associated with excitotoxicity. The released glutamate then binds its receptors and causes an increase in intracellular calcium and sodium. As the sodium enters the cell, the osmolality changes, resulting with the cell taking up water. This then results in cytocellular edema.

Glutamate activity will also result in the release of potassium, thereby depolarizing the cell membrane in a manner that mimics spreading depression, and activating sodium/potassium (ATPase) pumps. This activity puts increased energy demands on a cell and puts the system into a state of energy crisis. It’s important to note that the processes can unfold over weeks to months depending on the severity of the injury.

EEG EVIDENCE
Animal models have shown initial epileptiform activity described variously as high amplitude sharp waves, low amplitude high frequency discharges, or generalized high voltage spiking. This is followed quickly by a period of suppressed cortical activity, often appearing nearly isoelectric. EEG suppression can last from 10 seconds to several minutes. Following this suppression period of generalized slowing, a gradual improvement to a normal baseline is seen in 10 minutes to one hour.

In human models, EEG started as soon as 10 to 15 minutes after the injury were seen and most showed no to little alteration in EEG, though a few did show diffuse slowing. Further, boxers have shown a reduced EEG amplitude and increased regular theta activity within 15 to 30 minutes after a fight, and even more so if they had been knocked out. Other studies have shown that long after major closed or penetrating injuries, EEGs showed a wide variety of dysrhythmias, focal slowing, frontal alpha and epileptiform discharges. Neurologists should be mindful that generalized slowing takes longer to resolve than focal slowing but disappears more completely. One study showed focal slowing persisting more than two years in 22 percent of patients, mostly in those who had developed post-traumatic epilepsy.

CLINICAL IMPLICATIONS
A traumatic brain injury may give rise to seizures and epilepsy, with the likelihood of developing seizures related to the severity of the injury. The evidence from EEG studies suggests that TBI may induce multiple changes, including focal slowing. These EEG finding perhaps become important in an individual who does not have an obvious lesion on MRI. An individual with early and focal slowing on EEG may be an individual who is at higher risk for post-traumatic epilepsy, thus decreasing the threshold for management or at least closer monitoring.

Adapted from a presentation by Dr. Hishaw at the American Academy of Neurology 2012 Annual Meeting in New Orleans.

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