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## Anatomy of the Concussion: More Serious than Meets the Eye?

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# Anatomy of the Concussion: More Serious than Meets the Eye?

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## Abstract

Sports-related concussions have unique features that distinguish them from those mild traumatic brain injuries (MTBI's) caused by other mechanisms. Sports-related MTBI's may result from relatively lower-velocity impacts as compared with high-speed motor vehicle collisions, and are thought to cause different neurophysiologic injury. A survey of youth coaches revealed 42% believed a concussion only occurs when an athlete loses consciousness, and 25% would allow the athlete to return to play despite showing symptoms of concussion. Concussions occur not only in youth sports but also at the collegiate and professional levels. It is believed that, post-concussion, an athlete has diminished reaction time, thus increasing the risk of further injury. MTBI tends to occur again in patients with single MTBI. This review of concussion research aims to study the functional effects of MTBI on the brain. Ultimately, investigations should extend towards establishing increased public awareness of concussions with corresponding standardized care criteria.

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## I. Background

One of the most common neurologic disorders is still one the most puzzling – mild traumatic brain injury (MTBI), which is only surpassed in prevalence by migraine and herpes zoster (1). Traumatic brain injury (TBI) can be clinically rated as mild, moderate, or severe. TBI contributes to a third (30.5 percent) of all injury-related deaths in the United States (2). MTBI is the most common rating, with a recent WHO task force reporting that 70-90 percent of all treated TBI fell into this category (3). The ratio in occurrence of mild TBI to severe is about 22:1, with mild TBI accounting for at least 75 percent of patients who survive after TBI each year (4). Due to most mild TBI-injured patients being asymptomatic, a large proportion will receive no medical attention and remain unreported.

According to the American Association of Neurological Surgeons, a concussion is “a clinical syndrome characterized by immediate and transient alteration in brain function, including alteration of mental status and level of consciousness, resulting from mechanical force or trauma” (5). Another accepted definition is “...a complex pathophysiological process affect the brain, induced by traumatic biomechanical forces” (6). In a general sense, a concussion is an impact to the head that causes the brain to collide violently with neighboring rigid structures comprising the skull, thus disrupting normal brain activity.

Additional features that assist in defining a concussion include the severity and duration of symptoms. The presence and duration of three post-injury signs categorize the severity of MTBI: (1) loss of consciousness (LOC); (2) retrograde amnesia (forgetting information of events occurring prior to injury) and anterograde amnesia (forgetting information of events occurring post-injury); and (3) presence/absence of brain structural

and functional alterations as a result of injury (7). Historically, LOC and amnesia have been considered the primary features of concussion. However, a concussion rarely results in the presentation of LOC and/or prolonged amnesia. LOC occurs in only 8 percent of concussion cases (8). In 1998, an *NIH Consensus Statement* noted that “mild traumatic brain injury is an evolving dynamic process that involves multiple interrelated components exerting primary and secondary effects at the level of individual nerve cells (neuron), the level of connected networks of such neurons (neural networks), and the level of human thoughts or cognition” (9).

The major causes of TBI consist of: falls (35.2 percent); motor vehicle involving traffic crashes (17.3 percent); struck by/against events (16.5 percent); and assaults (10 percent). In the United States, falls continue to be the leading cause of TBI. Over 50 percent of TBIs among children aged 0 to 14 years and 61 percent of TBIs among adults aged 65 years and older are resultant of falls. Motor vehicle traffic accidents, as the second leading cause of TBI, result in the largest percentage of TBI-related deaths at 31.8 percent. Collisions with a moving or stationary object are included in struck by/against events. At 25%, these events were the second leading cause of TBI among children aged 0 to 14 years. Assaults account for 2.9 percent of TBIs in children aged 0 to 14 years and 1 percent in adults in adults aged 65 years and older (2). Of MTBIs, at least 20 percent are sports-related injuries (concussions), of which 30-45 percent receive no medical care (10). Injuries to the brain are the most common cause of death in athletes (11). Due to this, athletes represent a population at great risk of experiencing concussive episodes.

## II. Concussion in Sports

Sports are popular, and people of younger ages also participate. Over the years, sports have become more competitive, which has resulted in more injuries occurring – especially concussions. In the case of concussions in children alone, they have caused “more than 500,000 emergency room visits among kids 8 to 19 from 2001 to 2005 (12)” and half were sports-related concussions. Approximately 30-45 million children participate annually in non-scholastic sports programs (13). With children becoming even more competitive when playing sports as opposed to playing for fun and recreation, the incidence of concussions increases. Children develop habits of playing hard rather than for fun, which will continue into adulthood. This would mean that the chances of a concussion occurring have increased greatly from an early age up through adulthood. However, public concern for concussions has significantly decreased.

Sports-related concussions have unique features that distinguish them from those caused by other mechanisms. Sports-related concussions may result from relatively lower-velocity impacts as compared with high-speed motor vehicle collisions, and are thought to cause different neurophysiologic injury (14). With younger athletes less likely to have medical professionals or athletic trainers knowledgeable about concussions at sporting events lack or delay of concussion identification may be more likely in youth sports. Many parents, coaches, and players do not recognize the signs of a concussion and therefore will continue playing, thus increasing the likelihood of greater injury. A survey of youth coaches revealed 42 percent believed a concussion only occurs when an athlete loses consciousness, and 25 percent would allow the athlete to return to play despite showing

symptoms of concussion (15). Not only is it important for coaches and athletic trainers to better understand the severity of concussions, but it is also important for the players to understand. Concussion assessment is often complicated by the fact that athletes may not be honest about the presence of symptoms because they are afraid they will be held from play or experience peer pressure from players and coaches (16).

Concussions are not just occurrences in children- and youth-aged sports. The National Collegiate Athletic Association (NCAA) Injury Surveillance System reported a concussion rate in football of 4.2 per 1,000 athlete exposures, 7.5 percent of all injuries, or in game situations (i.e. not including practices) about 1.1 concussions per season per team. In men's soccer, concussion accounted for about 7 percent of all game injuries, but 11 percent of game injuries in women's soccer. Concussion rates in men's hockey were 1.9 per 1,000 athlete exposures, or 1 concussion every 35 games for a team. Wrestling concussions were 1 concussion every 38 matches for a team. Men's and women's lacrosse revealed that concussion was 11 percent of all injuries. Women's basketball showed that 8 percent of all game injuries were concussions (17).

It is estimated that 100-120 concussions occur in the NFL per year or about 1 every 2-3 games (18). Australian Rules Football reports approximately five concussions per team each year (19-20), 3.6 percent of all injuries and 4.7 concussions per 1,000 players' hours (20). One theory mentioned is that, as a consequence of a concussion, the athlete might have a diminished ability to take action quickly in response to expected events. This delayed response increases the risk of further injury, a possible explanation for the higher likelihood of concussion once an individual has already experienced one. Players reporting



a history of three or more previous concussions were more than three times more likely to sustain a concussion than players with no history of concussion, and these players experienced a slower recovery compared with those with fewer previous concussions. Of repeat concussions sustained within the same season, 92 percent occurred within 10 days of the initial injury and 75 percent occurred within 7 days of the first injury (21).

In order to maximize players' well-being, it is thereby important to understand what is happening to the human body before and after a concussion to institute prophylactic guidelines to prevent concussions from happening and to establish a high standard of care to better clinical outcomes when they do.

### III. Pre-Concussion Brain

A detailed conception of a healthy human brain is necessary to qualify an unhealthy brain – specifically, a brain that has undergone concussion, such that the definition of concussion is dependent on the definition of a healthy brain. The brain, a member of the central nervous system (CNS), is a vital organ that is the source of a person's behavior, thoughts, feelings, and experiences. It is also responsible for regulating body temperature, blood pressure, heart rate, breathing, regulating the flow of information, and for controlling physical motion. There are four lobes of the brain and the cerebellum, each responsible for different functions. Located at the back of the brain, the cerebellum coordinates balance and muscle coordination. The frontal lobe is at the front of the brain and can be divided into anterior and posterior portions. "Higher cognitive functions" and personality are regulated by the anterior or prefrontal cortex. Consisting of premotor and motor areas, the posterior portion modifies and produces movement. The region in the back of the brain,

which is referred to as the occipital lobe, processes visual information. This includes receiving information as well as recognizing shapes and colors. The parietal lobe is divided into right and left, and is located on top of the brain behind the frontal lobe. The right side is responsible for ascertaining visuo-spatial relationships while the left deals with comprehension of spoken and/or written language. There are two temporal lobes on either side of the brain which allow for the differentiation of smell and sound, sorting new information, and short-term memory. Visual memory is monitored by the right and verbal memory by the left.

In order for the brain to relay signals, it is composed of billions of neurons that gather and transmit electrochemical signals over long distances. A neuron is composed of three basic parts: cell body, axon, and dendrites. Cell bodies contain the necessary components for the cell to survive such as nucleus, endoplasmic reticulum and ribosomes, and mitochondria. Electrochemical messages, or the action potential, are carried along the cell's length by long, cable-like projections called axons. In the brain, the axons are non-myelinated meaning that there is no myelin covering the axons. Myelin usually allows for a faster transmission of nerve impulses. The small projections of the cell, called dendrites, allow for a neuron to communicate with other cells and perceive its environment.

The central nervous system is composed of substantia alba and substantia grisea. Substantia grisea, also known as grey matter, is a tissue located in the brain and spinal cord, and is composed of neuronal cell bodies, neuropil (dendrites and unmyelinated and myelinated axons), glial cells, and capillaries. Unlike substantia alba, substantia grisea contains neural cell bodies. Substantia alba, known also as white matter, is composed

primarily of myelinated axons that connect to the cell bodies located in the grey matter and which carry nerve impulses between neurons. There are three possible tracts within the white matter which allow for information transmission: (1) Projection tracts send action potentials from the cortex to other brain regions, out of the brain to muscles, or into the brain from sense receptors; (2) commissural tracts allow the two brain hemispheres to communicate by carrying information between left and right hemispheres over bridges known as commissures; (3) association tracts transmit information between lobes within the same hemisphere (22).

A neuron is considered “at rest” when it is not sending any signals and the inside is negatively charged relative to the outside. This negative charge is due to the cell membrane being only semi-permeable; the different ions are unable to balance out on both sides. Inside the cell there are large concentrations of potassium ions and negatively charged proteins, with smaller concentrations of chloride and sodium ions. Outside of the cell, large concentrations of both chloride and sodium ions exist while there is a smaller concentration of potassium ions. In order to maintain the net negative charge inside of the cell, a pump is used to move “three sodium ions out of the neuron for every two potassium ions” (23).

When a neuron sends out information along an axon, an action potential occurs creating a depolarizing current which causes an exchange of ions across the neuron membrane. Initially, sodium channels open causing an influx of sodium ions into the cell due to the net negative charge inside as compared to outside. This influx causes the neuron to become depolarized due to the sodium ions having a positive charge. As the current

continues traveling down the axon, the sodium channels will begin to close as the potassium channels open. Potassium channels take an increased amount of time to open and, during this time, the potassium ions will reverse the depolarization (repolarization) by rushing out the open channels.

Since it is a gelatin-like substance that is vulnerable to outside trauma, the brain is both protected and cushioned by the meninges and the cerebrospinal fluid (CSF) along with protection from trauma by the skull. However, the skull does not absorb the impacts of violent forces, which can result in injury. “An abrupt blow to the head, or even a rapid deceleration, can cause the brain to bounce against the inner wall of the skull. There is potential for tearing of blood vessels, pulling of nerve fibers and bruising of the brain substance” (5). The meninges are composed of three layers: the dura mater, arachnoid layer, and the pia mater. On the outermost section, the dura mater is a strong, thick and dense membrane. “Within the cranium the dura adheres closely to the inner surface of the bones forming the skull cavity, serving there as the internal periosteum” (24). Found as the middle layer, the arachnoid is composed of white fibers and a mixture of elastic elements. The innermost layer is the pia and is “pierced by the perivascular cuffs of the entering and emergent blood vessels and probably is perforated in the rhombencephalic roof areas” (24). The cerebrospinal fluid surrounding the brain is of great importance. CSF functions by forming a “liquid mantle covering [...] similar to a soft cushion forming a bed for neural structures” (25). This mantle, along with arachnoidal stripes consisting of “trabeculae stretched between the arachnoidal membrane and pia mater” (25), provides hydrostatic stability and protection to the brain. The equilibration of pressure differences that appear in the cranial cavity is considered to be the most important function of CSF.

“The total cranial volume is about 1700 [milliliters] and consists of 1400 [milliliters] of cerebral tissue, [...] and the remaining 200 [milliliters] are filled with cerebrospinal fluid. The volume of the three compartments is not constant...” (25), but the CSF will retract with increased pressure in either of the other two compartments in order to maintain a balance.

#### IV. Impact Mechanisms

This intricate brain system is perturbed by the impact mechanism, which may lead to concussion. There are three basic mechanisms through which minor head injuries occur in sports which include the following: a stationary hit with a forceful blow (impact or compressive force); a moving head hits a nonmoving object (acceleration or tensile force); and the head is struck parallel to its surface (shearing or rotational force) (26). Using fundamental Newtonian physics, an analysis can be applied to sport concussions depending on an acceleration or deceleration experienced by the player. From this, deduction of what brain forces would likely have occurred is a practical way for determining the potential for significant injury.

Accelerations (or decelerations, which are negative accelerations and proportional to the forces causing them) can be determined from a simple relationship:

$$a = \frac{v^2 - v_0^2}{2s}$$

Or

$$a_g = \frac{v^2 - v_0^2}{2sg}$$

In the formula,  $a$  is acceleration,  $v_0$  is the initial speed of acceleration,  $v$  is the final speed of acceleration, and  $s$  is the distance traveled during acceleration. If the acceleration  $a_g$  is to be expressed as a multiple of the acceleration due to gravity ( $g = 32.2 \text{ ft/sec}^2$ ), then the latter equation is used. If, at final acceleration the player is brought to rest, the value of  $v$  is zero (27). Thus, the formula can be simplified to read:

$$a_g = -\frac{v_0^2}{2sg}$$

For example, a football player running at 10 ft/sec collides with another player and is brought to a stop in a distance of 2 in (or 0.167 ft) the acceleration becomes (27):

$$a_g = -\frac{(10\text{ft/sec})^2}{2(0.167\text{ft})(32.2\text{ft/sec}^2 / g)} = 9.3g$$

If it is the player's head that is stopped in this short distance, the forces acting on any elements of his brain are 9.3 times the weight of that element. The effects that this force will have on the brain depends on several factors, including the direction of the force and whether the brain's element is moved by the force relative to its neighbors, thereby creating shear strain or axonal injury (28). In sports injuries to the brain, evaluations of the values of  $v_0$  and  $s$ , and how this accumulated force is transmitted to the head and brain of the player are problems faced.

Four scenarios of acceleration-deceleration models will be considered. These scenarios will be in relation to the sport football, but could be altered to apply to other impact sports. It can also be noted that no single injury may fall exactly into one category, and the following impacts will only be occurring between two individuals.

Model 1. Both players are initially at rest. It becomes a pushing match, and significant shock to the brain does not typically occur. One of the players may be hurled to the ground hard enough to incur injury, but this is discussed in another case.

Model 2. Player A is in motion and Player B is at rest. The question in this scenario is which player will incur injury. In a 1989 study of college football (29), it was determined that there were more significant injuries sustained by those performing the tackles and blocks than by those being tackled or blocked. This means that the player in motion (Player A in this scenario) would sustain more significant injuries than the stationary player.

(a) Player B plants himself firmly as he expects the collision with Player A, and assume B is considerably heavier than A. Because B was prepared, he/she may not accelerate at all. However, the acceleration of A is brought to zero during impact. If A impacts B with either his/her head or shoulder, A's head (and brain) will also be brought to a quick stop and making the value of  $s$  small. This will cause the deceleration of A to be large with significant brain acceleration. If, while A is in motion, B tackles A below the waistline, the distance ( $s$ ) taken to stop A will be larger. This will allow for no significant injury to arise. However, it should be noted that this type of tackle could increase the head's acceleration and thereby increasing the possibility of severe injury.

(b) An alternative is Player B unaware of the impact of A and unable to brace him/herself. This will cause B to be knocked hard into the direction of A's speed,  $v_0$ . If upon impact A holds onto B and the two players have approximately the same weights, they will end up with a final speed half of A's  $v_0$ . However, if A pushes B forward but is able to stop him/herself from moving forward as well, A's  $v_0$  could be fully transferred to B. If B is hit

below the waistline, as is the case for legal impacts in football, his/her head will lag behind the speed of the rest of the body until his/her neck is bent at an extreme angle. Once in this extreme angle, the head will “snap” into the new speed over a very short distance. This results in a large acceleration over a small  $s$ . These circumstances will cause A to most likely suffer no trauma, but B will likely be severely injured.

Model 3. Upon impact, both players A and B are in motion with exact speeds yet opposite directions.

(a) If the impact results with a head-on impact, both players (if equal weight) experience the same decelerations. Significant brain accelerations can be assumed with impacts of head to head or shoulder to shoulder. However, the significance of injury can be decreased if player A low-tackles B in a way that A passes under B. This will cause the stopping distance  $s$  to increase.

(b) A slightly less dangerous impact would be if both players hit at an angle as the players' heads are less likely to collide, but the shoulder-to-shoulder impact may produce head accelerations. Again, advance awareness is important because if a player does not anticipate the collision, said player will likely experience whiplash at an oblique angle of the hit. Due to less flexibility of the neck in a sideways direction and a shorter distance  $s$ , the acceleration and potential brain injury worse.

Model 4. In this model, a single player hits the ground. A player who simple falls will most likely not have any problems. However, running at top speed with his/her feet being tackled or him/her stumbling, he/she may hit the ground with most of the forward running



speed. Two factors that will then be brought into question are which body part first touches the ground and the toughness of the ground. Also, how short of a distance s his/her head is brought to a stop is questioned.

Consider a person running at a speed of 15 ft/sec stumbling and with a stopping distance of 0.2 ft. Using the formula  $a_g = v^2/2sg$ , it can be determined that:

$$a_g = -\frac{(15\text{ft/sec})^2}{2(0.2\text{ft})(32.2\text{ft/sec}^2 / g)} = 17.5g$$

This would be a likely injury case in football and, though the force is not likely to cause permanent neurologic dysfunction after a single event, it is still a considerable force (27).

Low-velocity impacts are impacts to an object that produce appreciable acceleration and an accompanying deformation of the body. When a person experiences an impact with an object, said person's body will move in translation and rotate in conformity with the imposed constraints. The impact forces from a blow to the head will result in a local structural deformation wave that gets propagated into the head's constituents, as well as an angular and linear acceleration of the head and neck. When an impact is not severe enough to cause a depressed skull fracture and a dura mater tear, then any injury to the intracranial contents would be due to the deformation wave propagation into the brain and/or the translational and rotational acceleration of the head. The hyperextension-hyperflexion or "whiplash" motion of the head and neck results in injury that is solely due to induced translational and rotational accelerations (30). Between 1996 and 2001, the highest risk associated with concussion that was reported in National Football League games was from helmet impacts. Other high risks of concussion resulted from contact with

other players' body parts at 21 percent and contact with the ground at 11 percent. Loss of consciousness was not associated with 91 percent of these cases (31).

During an impact, the primary risk factors for concussion are linear and rotational head accelerations, which induce brain tissue strain patterns, and can result from both direct and inertial (i.e. whiplash) head loadings (32). It is thought that a rotational impact will produce rotation of the cerebrum about the brainstem, thus causing shearing and tensile strains. The midbrain and upper brainstem are understood to control alertness and responsiveness. Therefore, rotational mechanisms are believed by some to be more responsible for loss of consciousness than linear impacts. Ommaya and Hirsch estimated that about 50 percent of concussive potential for an unprotected head is derived from rotational forces; they predicted that a minimum rotational acceleration of 1800 rad/sec<sup>2</sup> would be needed to induce concussion (33). It was also shown that while pure translational accelerations could not produce concussion in experiments with subhuman primate subjects, the combination of rotational and translational accelerations could (34).

Supplementing these empirical data are biomechanical finite element models such as the Wayne State University Brain Injury Model (WSUBIM), which models an elastic brain with shear modulus:

$$G(t) = G_{\infty} + (G_0 - G_{\infty})e^{-t\beta}$$

In the WSUBIM model,  $G_0$  and  $G_{\infty}$  are short and long-term moduli, respectively,  $\beta$  is the decay constant, and  $t$  is strain duration. The model indicates that intracranial pressure, skull deformation, and, most notably, shear stress – particularly at peak regions, the corpus

callosum and the brainstem – are much higher due to lateral impact than frontal impact (35). Factors predicting the amount of axonal damage involve an interaction between the magnitude and force as determined by the mass, surface area, velocity, and hardness of the impacting object (26).

When assessing the biomechanical damages obtained through a concussion, several factors must be taken into consideration. These factors include the shape of the skull, its size and geometry, density and mass of neural tissue, thickness of scalp and skull, extent/type and direction of the concussive blow, head—body relationships and mobility of the head and neck (36). Deceleration and rotation are able to define areas of the brain deformation that occur subsequent to an impact, thus leading to concussion signs and symptoms. A *coup* injury is a brain's early response to a linear injury occurring directly adjacent to the impact site. A coup injury produces enough force in the form of an opposite velocity vector to possibly cause the brain to strike the inner skull in the direction of initial travel. A majority of concussion impacts in the NFL occur laterally or obliquely, thus causing the temporal lobe to show the earliest signs of brain deformation or strain. A subsequent response is a slightly delayed *contrecoup* injury occurring on the opposite side of the brain from impact and typically in the temporal lobe opposite the impact site, though any area of the brain may be affected. These injuries and the magnitude of brain tissue alteration are intensified with excessive rotational forces being applied.

## V. Post-Concussion Brain

With any traumatic and disruptive movement that occurs to the brain, the possibility arises for blood vessel tearing, nerve fiber pulling, and brain substance bruising.

Brain swelling is a potential risk that can amplify the severity of a concussion. The brain is confined to a rigid area by the skull bones. When swelling occurs, blood flow limitations result due to the brain and blood vessels being compressed. This inadequate blood flow prevents the necessary oxygen and glucose from reaching the brain, with a stroke or ischemia ensuing. Those suffering from MTBI are extremely vulnerable to changes in cerebral blood flow (CBF), slight increase in intracranial pressure, and apnea. Brain vulnerability may be sustained due to the metabolic dysfunctions, including an increase in glucose need and incomprehensible CBF reduction occurring post-concussion.

Normally CBF is tightly coupled to neuronal activity and glucose metabolism (7), though this coupling can be disrupted due to MTBI. A reduction of CBF up to 50 percent of normal is possible and has been shown to occur in experimentally induced fluid percussion following brain injury (37). This decline will cause the available energy for neuronal activity to be greatly diminished and, during hyperglucolysis (increased glucose use), this energy supply and demand disparity could potentially result in a damaging energy crisis. After the initial spike in glucose usage, an injured brain will enter a period of decreased metabolism that could last seven to ten days and is correlated to cognitive deficits (38). This could lead to a longer and worsening energy crisis, exposing the brain to potential long-term damage. Experimental animal evidence has shown that following the initial stage of hyperglycolysis, the CBF was found to be diminished by twenty-four hours post-injury and remained low for the next five to ten days (38). Neurons become less capable of metabolically responding to peripheral stimulation because of the depression. The effects of MTBI have been shown to alter the brain chemistry and metabolism in studies involving rats. Evidence of oxidative stress has been observed, including a decrease in ascorbate of

16.4 percent in the cortex and 29.7 percent in the cortex and hippocampus compared to an untreated control group; membrane disruption involving a decrease in total level of phosphocholine and glycerophosphocholine of 23.0 percent and 19.0 percent in the cortex and hippocampus, respectively; and neuronal injury demonstrated by N-acetylaspartate decreases of 15.3 percent in the cortex and 9.7 percent in the hippocampus.

The alteration in CBF and fluid and glucose demand render the neurovascular system unable to respond to energy demands that are required to return the neurochemical and ionic environments to normal homeostasis. Further examination of the post-concussed brain on the microscopic level shows that neurochemical changes result in paralyzed, dysfunctional brain cells and create increased injury vulnerability. This neurochemical and metabolic cascade begins within the first hour of insult and continues for up to 10 days post injury (46). Though cells do not become irreversibly destroyed, they exist in a vulnerable state. Beginning the first hour of vulnerability, a neurotransmitter – primarily glutamate – is released from the cell, causing extracellular glutamate concentration to increase. This increase causes a potassium influx up to seven times the normal amount. Normally, an excessive concentration of extracellular potassium is neutralized by surrounding glial cells, allowing the brain to maintain physiological equilibrium of potassium following mild disturbances (7). Initially, a massive excitatory process occurs due to excessive concentration of potassium, followed by an abrupt wave of relative neuronal deactivation (potassium ion outflow). This cycle, known as *spreading depression* is a notion that acute loss of consciousness, memory loss, and cognitive abnormalities are direct manifestations of post-traumatic *spreading depression* (41). This flooding of ions leaves the cell unable to produce an action potential, thus leaving the

sodium/potassium pump dysfunctional, the ATP-dependent sodium—potassium pumps become activated, and increased metabolic stress. The stress to damaged neural tissue maintains low levels of CBF.

Reduction of CBF also results in an influx of calcium ions. When the brain is injured, calcium ion channel blockers (nimodipine and dextromethorphan) mediate this influx by changing the body's ability to increase CBF. These changes create an imbalance in the ratio between the utilization of fuel (glucose) and fuel delivery (41). An imbalance between energy demand and production creates the metabolic dysfunction that causes the brain to enter a state of hypermetabolism (a depressive state) that coincides with a state of lactic acidosis. This state results in a decrease of glucose, oxidative metabolism, and a further decrease in CBF, thus potentially leading to ischemia (26).

Even with a single concussion, the damage to the brain can begin with “limited but significant damage to the neurons” (39) resulting. As the acceleration/deceleration forces increase, the number of cells damaged will increase as well due to the damage progressively occurring in deeper structures. The damage occurs deeper in tissues, and processing systems become more affected thus slowed. Low magnitude brain injury can disrupt high order cognitive functioning (40) Concussions, referred to structurally as “diffuse axonal injuries (DAI),” result in some degree of functional impairment as a result of the damage caused by stretching and twisting of neuronal axons. Functional disruption caused by a concussion can result from diffuse damage to white matter tracts that are more susceptible to the shearing forces of MTBI (40). This type of DAI can disrupt critical

cortical-subcortical pathways that lead to widespread cognitive dysfunction, with lesions primarily in the corpus callosum, internal capsule, and centrum semiovale (41).

The amount of force and direction of head movement are large determinants of the type of neurons injured by acceleration/deceleration forces. Studies have demonstrated that when axons become susceptible to damage, they often change direction. Examples of events that induce directional changes include accommodation of a blood vessel's presence, the need to enter target nuclei, and the occurrence of decussation within the brain parenchyma (39). Damages due to acceleration/deceleration injuries typically affect large myelinated cells that decussate as well as those that transfer information to and from the cortex. This implies that diffuse cellular damage involves communication between different brain regions within a neural system.

Axonal fibers that are stretched via MTBI become swollen, beaded, and varicose, causing the neurons to become dysfunctional even though the neuron is still alive. If these same fibers are stretched to a rupture point due to a repeat concussion, more permanent brain damage is thought to occur (42). These "centripetal" strains occur higher at the surface than at the depth of the brain, decreasing radially toward the center of the brain. Areas of the brain that are especially vulnerable to rotational shear strains include the midbrain stalk, the cerebral hemispheres above, and the cerebellar structures below with the latter two due to mass. Most often, MTBI occurs with Grade I DAI, which is classified with widespread axonal damage in the corpus callosum, white matter of the cerebral hemispheres, and the brain stem. Grade II DAI exhibits focal abnormalities in the corpus callosum and is often associated with small tissue tear hemorrhages in addition to all Grade

I injuries. Grade III DAI shows, in addition to Grade II injuries, additional axonal abnormalities commonly in the rostral brain stem as a result of tissue tear hemorrhages. Immediate changes in membrane structure and damage to the physical structure of the neural cell are a result of the physical stretch at the time of injury. These changes result in the disruption of the capability to maintain ionic gradients resulting in adverse neurochemical changes (26).

Through various studies, it has been noted that the brain appears to tolerate sagittal movements best and then motions in the horizontal plane, whereas the brain is most vulnerable when moved laterally (26). In addition to linear *coup-contrecoup* injury mechanisms, diffuse axonal injury can result in disruption to brain centers responsible for breathing, heart rate, and consciousness; more typically though they result in memory loss, cognitive deficits, balance disturbances, and other somatic symptoms. When a skull experiences an impact, it absorbs part of the blow. This denting is associated with an equal and opposite bulging that could result in a fracture remote from the injury site. Immediately, this bulge fills with displaced cerebrospinal fluid but due to the cranium distorting faster than the brain, a vacuum is created opposite the injury site and local blood vessels may rupture (43). The build-up of CSF in the bulge creates problems by occupying too much of the brain's space because it is unable to retract soon enough. Intraventricular hemorrhage is possible due to the negative pressure created and rapid aspiration of CSF.

An area of the brain especially vulnerable to damage is the prefrontal cortex, and damage leads to long-term cognitive impairments. White matter of the frontal lobe and cellular damage has been demonstrated by DAI, as well as impaired neuronal integrity and



associated cognitive impairment. Reduced NAA/creatine ratio and increased choline/creatine ratio can be observed and are highly correlated to head injury severity (7).

## VI. Conclusion

Understanding a concussion and the effects that can be accrued is vital to the continued health of people, especially those participating in impact sports as the probability of multiple concussions – concussion loading – is great after the initial occurs. Early onset of cognitive decline and dementia can develop over a period of time due to the cumulative effects of multiple concussions. While a single concussion is able to damage the neuronal function via its connections, the chances greatly increase for post-concussion injuries to occur due to decreased cognitive and neurologic abilities. The neurons of the brain are subjected to increased vulnerability during the repair stages after concussion events due to the possibility of a repeat concussion occurring before full recovery is obtained. Systems in the process of recovering when a subsequent injury occurs will most likely lose what recovery had been obtained from the initial injury, as well as sustaining further damage to a new set of neurons (39). Regardless of the type, attribute, or severity of a particular impact, the end result is the same, and that is that the mass of the head has become too large for the body to overcome the acceleration or deceleration forces that have sent it in motion (32).

It is thus that the human head – and thus its owner – is presented with the risk of concussion and its profoundly negative effects in day-to-day living, particularly with

regards to sports. The issue of concussions is not only a matter of neurological biomechanics but also one of public health.

It would be both practical and beneficial to establish benchmarks based on empirical evidence of what minimum forces and accelerations induce brain tissue damage with both short-term and long-term adverse effects. For instance, football helmets could be better designed with respect to what angular accelerations induce concussive shear strain on nervous tissue – the long-term effects of a concussion could then be better prevented through more adequate cushioning or even helmet shape or material change. Laboratory quantification of the range of concussive forces and other biomechanical variables can be translated to positive outcomes beyond the laboratory; that is, benefits go from bench to bedside to the general public. It is in this fashion that comprehensive review of previous concussion research and subsequent directed pursuit of future research and development are advantageous.

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