

Increasing Awareness about Possible Neurological Alterations in Brain Status Secondary to Intimate Violence

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Introduction

Issues of domestic violence were first addressed and identified in the late 1970s (Chescheir, 1996; Healey, Smith & O'Sullivan, 1998; Muelleman et al., 1996). These early studies focused on the characteristics of the abuser, punishment of the perpetrator, the relationship between the perpetrator and the recipient of abuse, and intervention strategies. In the late 1990s, what was termed domestic violence expanded and now is more accurately called intimate violence.

Although the psychodynamics of intimate violence have been researched widely, the types of traumatic injuries, neurochemical changes and/or structural alterations in the brain resulting from intimate violence altercations have not. The purpose of this paper is to identify and explain the possible types and etiologies for traumatic injuries, neurochemical changes and structural alterations that may occur in the brain as a result of intimate violence, most of which are not being diagnosed. Additionally, discussion will focus on the short- and long-term consequences of undiagnosed TBI for the person in an intimate violent relationship, linking together the specific type of intimate violent acts with what may happen to the brain.

Persons with injuries from intimate violence may not be aware of the signs of TBI, putting them at risk for subsequent injury. Additionally, medical personnel addressing issues of persons both with or without apparent injuries from physical violence may not be aware of the signs and symptoms of TBI, resulting in these injuries being undiagnosed and untreated, as well as misdiagnosed. This paper will focus on the effects of violent interactions on brain structures and processes with the hope of increasing awareness of the issue. A general overview of intimate violence will be followed by an explanation of the types of TBIs which may result. It is believed raising awareness will lead to better diagnosis and improved treatment of those who sustain intimate violence.

Intimate Violence

For the purposes of this article, “intimate violence” is defined as a relationship between two people who may or may not: be of the same sex, cohabitate or currently be in a relationship (Alpert, Cohen & Sege, 1997; Barrier, 1998; Loring & Smith, 1994; Moehling, 1988; Muelleman et al., 1996; Novello, 1992; Saltzman & Johnson, 1996). Like domestic violence, intimate violence includes the establishment of abusive control and power over another person through fear, isolation and/or intimidation (Alpert, Cohen & Sege, 1997; Campbell & Soeken, 1999; Healey, Smith & O’Sullivan, 1998; Muelleman et al., 1996; Saltzman & Johnson, 1996). Violent behavior (i.e., the act of engaging in intimate violence) often is thought of as direct “hands-on” infliction of pain but also includes implied threat or actual physical, sexual and emotional abuse, including withholding finances and medical equipment (Campbell & Soeken, 1999; Healey, Smith & O’Sullivan, 1998; Loring & Smith, 1994; McCoy, 1996; Muelleman et al., 1996; Saltzman & Johnson, 1996). Intimate violence takes place across all cultures, socioeconomic statuses, geographic regions and ages, including teenage dating relationships and geriatric populations (Chambliss, 1997; Healey, Smith & O’Sullivan, 1998; Keller, 1996; Melvin & Rhyne, 1998; Muelleman et al., 1996).

Statistics about intimate violence are based on reported acts, with between two to four million women being abused physically by an intimate each year (Campbell & Soeken, 1999; Gaffigan-Bender, 1998; Keller, 1996; Muelleman et al., 1996; Novello, 1992). In fact, intimate violence is the leading cause of serious injury to American women between the ages of 15 and 44 (Gaffigan-Bender, 1998; Grisso et al., 1991; Keller, 1996; Novello, 1992; Melvin & Rhyne, 1998), with approximately one in four women being abused by a partner in her lifetime (Jecker, 1993; Loring & Smith, 1994). In 70% of homes where a woman is beaten, children also are beaten (Muelleman et al., 1996). Because many instances of intimate violence go unreported, the actual numbers may be greater. Although men can be abused and women can be abusers, 95% of recipients of reported intimate violence acts are women (Alpert, Cohen & Sege, 1997; Casardi, Langhinrichsen & Vivian, 1992; Gaffigan-Bender & Narula, 1998; Keller, 1996; Novello, 1992).

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Researchers have indicated up to 40% of women who visit hospital emergency rooms are there for symptoms related to physical abuse; however, depending on the statistics cited, as few as 2.8% to 10% of those women are identified as such (Alpert, Cohen & Sege, 1997; Barrier, 1998; Campbell et al., 1994; Cascardi, Langhinrichsen & Vivian, 1992; Gaffigan-Bender, 1998; Jecker, 1993; Loring & Smith, 1994; Keller, 1996; McFarlane et al., 1991; Muelleman et al., 1996; Novella, 1992; Plichta, Duncan & Plichta, 1996). Women are more likely to be injured severely in intimate violent relationships and present contusions, concussions, lacerations, sensory organ injury, burns, stab wounds, miscarriages and gunshot wounds in emergency departments (Barrier, 1998; Campbell et al., 1994; Cascardi, Langhinrichsen & Vivian, 1992; Gaffigan-Bender & Narula, 1998; Loring & Smith, 1994; McFarlane et al., 1991; Melvin & Rhyne, 1998; Muelleman, Lenaghan & Pakieser, 1996). With the head, face and neck injured most frequently during physical intimate violence, (Barrier, 1998; Campbell et al., 1994; Dym, 1995; Melvin & Rhyne, 1998), these women are more likely to experience black eyes, broken teeth, broken bones, fractures, joint damage, chronic pain, gynecological problems, sexually transmitted diseases, hyperventilation, choking sensations, chest pain, gastrointestinal symptoms, insomnia, fatigue, seizures, headaches, impaired cognition, dizziness and disability (Barrier, 1998; Campbell & Soeken, 1999; Cascardi, Langhinrichsen & Vivian, 1992; Chescheir, 1996; Gaffigan-Bender & Narula, 1998; Gremillion & Kanof, 1996; Loring & Smith, 1994; McFarlane et al., 1991; Melvin & Rhyne, 1998; Plichta, Duncan & Plichta, 1996; Warshaw, 1996). Hickman (as cited in Stancliff, 1997, p. 23) states, "A woman is hit an average of 35 times before she calls the police, and she will leave her abuser five or six times before she leaves for good."

Types Of Brain Injuries Which May Result From Physical Violence

A thorough literature search was unable to reveal research directly examining TBIs as a result of intimate violence, although numerous studies have been conducted on TBIs occurring in sports-related incidents. Falco (1997) reports persons with sports-related TBIs present the same deficits as persons who have sustained head trauma in other circumstances and situations (e.g., sports-related studies will serve as correlative reference for this paper). Death, brain injury and/or permanent damage may result from lethal violence. The brain can be assaulted directly by physical force and neurochemically from the prolonged effects of stress from intimate violence (Campbell et al., 1994).

Of significance to intimate violence and TBI is the examination of the type of blow delivered to the head and the resultant type of TBI incurred, with numerous studies on boxers providing correlative information. Researchers indicate a boxer wearing a six to eight ounce glove can generate an impact force of more than half a ton (Drachman & Newell, 1999; Richards, 1995). Because of this force, gloves are worn during boxing to “soften the blow” (Falco, 1997). Erlanger and colleagues (1999) note contact sports-related TBIs result from lower velocity impacts than those sustained in non-contact sport contexts. This author did not find statistics regarding the force directed by a bare fist or use of an object.

Women who were injured physically in intimate violent relationships report they were pushed, kicked and/or hit with a fist or other object (Pakieser, Lenaghan & Muellman, 1998). The added strength behind the use of the large muscle groups of the body, such as with a kick or a stomp, increases the force delivered to the head. If the person’s head hits a hard surface after being struck or actually is slammed into a hard surface (i.e., the pavement) the impact also is increased. Likewise, use of a solid object (i.e., a bat or tire iron) increases the force greatly, as compared to a punch or swing. Direct force to the head can result in a closed or open head injury, with firearms providing the “ultimate” force resulting in a penetration-type injury.

Closed Head Injuries

A closed head injury results when a force causing injury to the brain impacts the head, and the skull remains intact (Senelick & Ryan, 1998; Swiercinsky, Price & Leaf, 1993). As the brain swells, the intracranial pressure increases and may cause diffuse axonal injury, edema and even death (Senelick & Ryan, 1998; Swiercinsky, Price & Leaf, 1993). Initially, with closed head injury there may be no outward apparent signs of TBI. When reporting injuries—including skull fractures—in the emergency room (ER), studies indicate a disproportionate amount of women are reporting they have fallen (Grisso et al., 1991). These women are too young to be considered in a risk category for falls and may be part of the population with undiagnosed TBIs (Grisso et al., 1991).

Open Head Injuries

An open head injury exists if the skull actually fractures or is displaced by an outside force, leading to an increased opportunity for infection as the brain is exposed to foreign material (Senelick & Ryan, 1998; Swiercinsky, Price, & Leaf, 1993). In addition to being classified as open or closed, TBIs are categorized by the type of injury to the brain (i.e., penetrating injury, focal ischemic lesions, diffuse axonal injury, anoxia) (Drachman & Newell, 1999; Erlanger et al., 1999; Falco, 1997).

Types Of Brain Injury Secondary To Force

Penetrating Injuries

Firearms used in intimate violence are 12 times as likely to cause death than use of any other weapon (National Center for Injury Prevention and Control, 1998). Handguns are the weapons used most often against women in intimate violent relationships (Saltzman & Johnson, 1996). The devastating TBIs caused by bullet wounds result in a 91% firearm-related death rate overall (National Center for Injury Prevention and Control, 1999; Stone et al., 1995). Firearms are the single largest cause of death from TBIs, accounting for approximately 44% (Fontanarosa, 1995; Harrison et al., 1998).

Penetrating injury to the brain occurs from the impact of a missile (i.e., a bullet) that forces hair, skin, bone and missile fragments into the brain (Brumback, 1996). Low velocity missiles can cause a ricochet effect within the cranium, which widens the area of damage (Brumback, 1996). A “through-and-through” injury occurs if the missile enters and exits the cranium (Brumback, 1996). Through-and-through TBIs include the effects of penetration injuries, plus additional shearing, stretching and rupture of brain tissue (Brumback, 1996).

Gunshot wounds may be the most obvious types of lethal violence occurring in intimate violent relationships. Unfortunately, there are many forms of TBIs which are not as recognizable and may go undiagnosed. Therefore, the purpose of this paper is to increase awareness of all TBIs.

Focal Ischemic Lesions

Signs of focal ischemic lesions may not be obvious at first and persons may not be aware of the neurological repercussions. Focal ischemic lesions even can develop a few days after the incident (Lampert & Hardman, 1984). Ischemia (i.e., vascular inefficiency to the neural tissue) can result in cell death (Nolte, 1993; Swiercinsky, Price & Leaf, 1993).

A straight parallel blow to the head results in linear acceleration movement of the skull, causing a gliding movement of the brain (Cantu, 1996; Erlanger et al., 1999; Lampert & Hardman, 1984) and a stretching and contracting of the axons (Erlanger et al., 1999). A very forceful linear acceleration (i.e., when a person is shoved or hit exceptionally hard) causes neck hyperextension, which can result in loss of consciousness secondary to axonal damage in the medullapontine angle and the reticular substance (Lampert & Hardman, 1984).

Although cerebral spinal fluid acts as a shock absorber to alleviate the stress on the moving brain, ischemia, contusions and axonal damage in the brainstem may develop (Erlanger et al., 1999; Lampert & Hardman, 1984). A contusion is a type of focal ischemic lesion occurring when small blood vessels in the brain break and deprive contiguous brain tissue of blood (Swiercinsky, Price & Leaf, 1993). Contusions that occur at the direct site of impact and the opposite side of impact are termed “coup contracoup” lesions (Brumback, 1996). For instance, a coup contracoup lesion may occur if a person is hit in the face hard enough to cause the brain to move and slam against the back of the skull. In this case, the brain would be injured focally in both the front and back areas. Conversely, a type of TBI that causes widespread damage to the brain is called a diffuse axonal injury.

Diffuse Axonal Injury

A person who is hit in the head in another way may sustain a different type of TBI. An “uppercut” is a blow that causes the head to turn with a rotational acceleration (Adams & Burton, 1989), increasing the force of the blow. With each ensuing blow to the head, the force imparted becomes even greater as the neck becomes more relaxed (Cantu, 1996; Erlanger et al., 1999). This phenomenon may be understood better by applying Newton’s law, which states force equals mass times acceleration or acceleration equals force divided by mass (Cantu, 1996). If the neck muscles are contracted in anticipation of a blow, the head can accept greater forces without injury because the mass of the head is the same as the mass of the body (Cantu, 1996). When the neck muscles are subtle, the mass of the head decreases to its own weight, therefore becoming more susceptible to an equivalent force which can deliver an increased acceleration (Cantu, 1996; Lampert & Hardman, 1984).

During rotation acceleration, injury occurs because the static brain delays behind the acceleration and deceleration of the skull, thus causing structures to tear (Lampert & Hardman, 1984). Rotational acceleration creates shearing stress and can produce diffuse axonal injury, intracerebral hemorrhage and subdural hematoma (Cantu, 1996; Erlanger et al., 1999; Falco, 1997; Jordan et al., 1996; Lampert & Hardman, 1984).

A diffuse axonal injury occurs when the brain is subjected to acceleration and deceleration forces, such as when a person is shaken violently. This shaking—similar to what is known as Shaken Baby Syndrome in infants and young children—recently has been termed Shaken Adult Syndrome (Carrigan, 2000). A violent shaking episode can cause extensive tearing of axons, mainly in the subcortical regions, white matter, corpus callosum and brain stem (Cantu, 1996; Erlanger et al., 1999; Falco, 1997; Lampert & Hardman, 1984). The torn axons cause disruption of nerve impulse transmission and neurochemical release that further impairs the brain (Swiercinsky, Price & Leaf, 1993). The neuropathway disturbance may produce temporary or permanent widespread brain damage including intracerebral and extracerebral hemorrhages, edema, coma and even death (Lampert & Hardman, 1984; Lampert & Hardman, 1984, as cited in Erlanger, Kutner, Barth & Barnes, 1999). A person sustaining a diffuse axonal injury may present dizziness, inefficient thinking, memory loss and behavior inhibition (Senelick & Ryan, 1998; Swiercinsky, Price & Leaf, 1993).

Concussion

The types of impact to the head that can result in a concussion—the most common type of mild brain injury—include a direct blow, gunshot wounds and/or force from a whiplash-type injury (i.e., when a person is shaken violently) (Cantu, 1996; Swiercinsky, Price & Leaf, 1993), as well as both closed and open head injuries (Bodel, 1999; Swiercinsky, Price & Leaf, 1993). A concussion results from impact to the head that causes neuronal, neurochemical and/or structural damage with or without brain cell death (Cantu, 1996). Hematomas (i.e., bleeding in the brain) may result in brain tissue destruction and death (Brumback, 1996). In some instances, a blood clot in the brain may transpire secondary to a concussion and can be fatal (Brain Injury Association, 2000).

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Early signs of concussion include confusion, dizziness, vomiting, headache and nausea (Bodel, 1999; Kelly & Savage, 1999; Savage, 1998). Late signs of concussion are persistent headache, poor attention, irritability, ringing in the ears, restlessness, depressed mood, lightheadedness, memory problems, blurry vision, fatigue and anxiety (Bodel, 1999; Kelly & Savage, 1999; Savage, 1998). Behavioral changes signaling concussion include: blank staring, decreased response time when answering questions or following directions, confusion, distractibility, inability to carry out regular activities of daily living, disorientation, ambulation in the wrong direction, slurred speech, impaired production of thought content, extreme range of emotions, impaired memory and loss of consciousness (Kelly & Savage, 1999; Savage, 1998).

A loss of consciousness secondary to concussion frequently follows the injury and may last for seconds or minutes (Savage, 1998). The severity of a concussion may be classified as a Grade 1, 2 or 3, based on symptomology (Kelly & Savage, 1999; Savage, 1998). For sports players, the grades are used as an indication as to when an athlete may return to play (Kelly & Savage, 1999; Savage, 1998).

The Standardized Assessment of Concussion (SAC) Manual was developed for coaches and physicians to assess a player's readiness to return to play after a concussion (McCrea, Kelly & Randolph, 1998). The SAC is the first instrument that quantifiably measures the neurocognitive effects of concussion, specifically the functional areas most likely to be affected by concussion: orientation, immediate memory, concentration and delayed recall memory (McCrea, Kelly & Randolph, 1998).

Post-concussion Syndrome

Post-concussion syndrome may occur following a concussion as a result of altered neurotransmitter function (Cantu, 1996; Swenson, 1997). Post-concussion syndrome is characterized by headache, dizziness and personality changes (Brumback, 1996; Cantu, 1996). A “migraine-like” chronic headache may be constant or throbbing (Brumback, 1996; Cantu, 1996). Dizziness may present secondary to vestibular nerve damage (Brumback, 1996; Cantu, 1996; Swenson, 1997). Additional presentations of post-concussion syndrome may include amnesia, impaired concentration, impaired ability to learn, emotional lability, irritability, fatigue, aggressiveness, depression, anxiety and hyperactivity (Brumback, 1996; Cantu, 1996; Swenson, 1997).

Second Impact Syndrome

Second impact syndrome—also termed recurrent TBI—occurs when a person sustains a second TBI prior to the resolution of symptoms from the first concussion (Cantu, 1996; Kelly & Savage, 1999; Salcido & Costich, 1992). Again, the majority of research about second impact syndrome has focused on athletic injuries. Researchers studying athletes have found that even a blow to the chest may cause acceleration of the head, resulting in a second concussion (Cantu, 1996). Salcido and Costich (1992) indicate personal or family dysfunction as an increased risk factor for second impact syndrome.

Symptoms of second impact syndrome obvious within seconds to minutes of incidence include collapse, rapidly dilating pupils, loss of eye movement, presentation of respiratory failure and evolution to a semicomatose state (Cantu, 1996). The pathophysiology of second impact syndrome (Cantu, 1996) results as a loss of autoregulation of the brain’s blood supply. Cantu (1996) states:

This loss of autoregulation leads to vascular engorgement within the cranium, which in turn markedly increases intracranial pressure and leads to herniation either of the medial surface (uncus) of the temporal lobe or lobes below the tentorium or of the cerebellar tonsils through the foramen magnum. Animal research has shown that vascular engorgement after a mild head injury is difficult if not impossible to control. (p. 293)

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Because death may occur rapidly, initiation of emergency medical treatment is imperative (Cantu, 1996; Swenson, 1997). Cumulative consequences of recurrent brain injury include: dystonia, emotional lability, hallucinations and cognitive impairment (Salcido & Costich, 1992). Additionally, dementia pugilistica can result.

Dementia Pugilistica

Persons who are hit repeatedly in the head—even across a span of years—can experience a cumulative type of TBI. Recurrent brain trauma caused by repeated blows to the head can produce the neurological repercussion termed “dementia pugilistica,” also called chronic traumatic encephalopathy or “punch drunk syndrome” (American Academy of Pediatrics Committee on Sports Medicine and Fitness, 1997; Bouras et al., 1997; Drachman & Newell, 1999; Erlanger et al., 1999; Falco, 1997; Geddes et al., 1999; Jordon et al., 1995; Jordon et al., 1996; Jordon et al., 1997; Kemp et al., 1995; Richards, 1995; Zasler, 2000). Dementia pugilistica is characterized by findings of damage to one or more of the pyramidal, extrapyramidal or cerebellar systems with the presentation of dementia, behavioral change, impaired cognition, ataxia, confusion, psychosis and motor dysfunction (Falco, 1997; Jordan et al., 1997; Roberts 1988; Roberts, Allsop & Bruton, 1990).

Studies on dementia pugilistica have been focused on boxers and supplemented with limited animal studies of recurrent brain injury (Geddes et al., 1999). Researchers performing a single case autopsy examination of a physically abused woman who had presented with dementia, however, found brain pathology identical to that of boxers with dementia pugilistica (Geddes et al., 1999; Roberts et al., 1990). Furthermore, researchers conducting an autopsy examination of an autistic woman who had displayed the self-injuring behavior of head-banging, revealed similar findings as others who had endured repeated head injuries (Geddes et al., 1999; Hof et al., 1991). Collectively, research suggests a similar mechanism of action, subsequent or severe impacts to the head, generates the neuropathological degeneration associated with dementia pugilistica.

The clinical manifestations of dementia pugilistica occur in three stages (Falco, 1997; Richards, 1995), with progression through the three stages and/or plateauing possible at any level (Falco, 1997). Symptoms can appear years after the brain trauma and even late in life (Falco, 1997). Erlanger and colleagues (1999) state mild neurocognitive deficits will develop prior to a diagnosis of dementia pugilistica. Psychotic symptoms and affective disturbances appear in the first stage (Falco, 1997; Richards, 1995; Roberts, Allsop & Bruton, 1990). Memory loss, Parkinsonism, social instability and psychiatric symptoms present in the second stage (Falco, 1997; Richards, 1995; Roberts, Allsop & Bruton, 1990). In the final stage of dementia pugilistica, declining cognitive function with dementia and pyramidal, extrapyramidal and cerebellar disease is typified (Falco, 1997; Richards, 1995; Roberts, Allsop & Bruton, 1990).

Neurologically, the hallmark presentations of dementia pugilistica are damage to the septum pellucidum, cerebral atrophy, enlargement of the ventricles, focal scarring of the cerebellum, reduced cholinergic activity in the basal forebrain, loss of pigmented neurons in the substantia nigra and neurofibrillary tangles in the cortex, particularly in the temporal lobe (Buee et al., 1994; Bouras et al., 1997; Dale et al., 1991; Drachman & Newell, 1999; Falco, 1997; Hof et al., 1992; Jordan et al., 1995; Jordan et al., 1997; Richards, 1995; Roberts, 1988; Roberts, Allsop & Bruton, 1990; Roberts et al., 1990). The neurofibrillary tangles of dementia pugilistica are identical morphologically to those associated with Alzheimer's disease (Bouras et al., 1997; Dale et al., 1991; Lennox et al., 1988; Roberts, 1988; Roberts, Allsop & Bruton, 1990). Contrary to Alzheimer's, the neurofibrillary tangles observed in dementia pugilistica do not or rarely present neuritic senile plaques and are concentrated in the superficial layers of the neocortex (Bouras et al., 1997; Dale et al., 1991; Hof et al., 1991; Richards, 1995; Roberts, 1988; Roberts, Allsop & Bruton, 1990; Scully et al., 1999).

It is evidenced that TBIs produce metabolic changes in the brain (Geddes et al., 1999). Little is known, however, about the initial histological changes that take place in the brain following trauma and prior to the development of dementia pugilistica (Geddes et al., 1999). Recent research indicates groups of neurofibrillary tangles consistently situate on blood vessels in the most injured areas and deep in the sulci of the brain (Geddes et al., 1999; Hof et al., 1991). Researchers believe this may be because the neuronal cytoskeleton abates shortly after ischemic insult or because of direct damage to the vessels (Geddes et al., 1999; Jordan et

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al., 1995). Furthermore, researchers believe that since the blood vessels are sites where axons change directions, they may be more susceptible to shearing stress (Geddes et al., 1999). Cytoskeleton neuronal cell body abnormalities may be induced by cytotoxins released during shearing episodes (Geddes et al., 1999).

Researchers suggest some people have a genetic predisposition which makes them more susceptible to the devastation of TBI (Geddes et al., 1999; Zasler, 2000). Persons with the genetic predisposition can be expected to present a greater severity of impairment (Geddes et al., 1999; Zasler, 2000). Research by Nemetz et al. (1999) concluded that persons with TBI and a predisposition to Alzheimer's developed Alzheimer's disease in a median of 10 years, versus a median of 18 years for persons without a prior TBI (Zasler, 2000). The onset of Alzheimer's disease was shorter also for persons with the predisposition who had experienced a TBI prior to age 65 (Nemetz et al., 1999). The relationship between TBI and Alzheimer's is complex. Although Zasler (2000) addresses the issue on a deeper level, there is a lack of study regarding the chronic implications for persons who have been the recipients of long-term physical intimate violence.

Anoxia

During intimate violence, anoxic brain injury may occur following strangulation, near-drowning, head impact and/or blood loss from open lesions, such as stab wounds. The brain can be deprived of oxygen during intimate violence because of forced restriction by the perpetrator. Anoxia results from an inadequate amount of oxygen in the respiratory system and can result in brain injury, permanent brain damage or even death (Swiercinsky, Price & Leaf, 1993). Ischemia, reflex hypotension and/or artery impingement can develop secondary to a blow to the head (Erlanger et al., 1999).

Strangulation

During a violent altercation, a perpetrator may attempt to control a person by silencing them, often using choking as the mechanism. San Diego Deputy City Attorney Gail B. Strack and Dr. George McClane study cases of strangulation and choking, educating law enforcement officials to increase awareness of these instances (Strack, 2000; Strack & McClane, 1999; Strack & McClane, 2000). Strack (2000) estimates that between 15-50% of sexual assaults involve strangulation.

Strack reports forced restriction of oxygen can be attained by use of the perpetrator's hands or by use of an object (i.e., a rope or cord) (Strack, 2000; Strack & McClane, 1999; Strack & McClane, 2000). Only 11 pounds of pressure applied for 10 seconds to the neck is necessary to force the restriction of oxygen to cause unconsciousness (Strack & McClane, 1999). Death can occur in four to five minutes with application of 30 pounds of pressure (Strack & McClane, 1999).

Physical substantiation after strangulation may not be apparent—although internal injury could have transpired—and internal neck swelling can compromise the airway passage (Strack, 2000; Strack & McClane, 1999; Strack & McClane, 2000). If the person's medical condition is not addressed, reports of death from the compromised airway passage can result in 36 hours (Strack, 2000; Strack & McClane, 1999; Strack & McClane, 2000). Persons without visible injury have died several weeks after strangulation as the result of undiagnosed brain damage (Strack & McClane, 1999).

Strack and McClane (1999, 2000) examined 100 strangulation cases in San Diego, indicating that 62% of the reports about people who were strangled contained no documentation about visible injuries and only three had sought medical treatment. Because the injury was not obvious, 1) medical attention was not sought by the person who had been choked or strangled and 2) medical attention was not recommended by those encountering the person (Strack & McClane, 1999; Strack & McClane, 2000).

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Strack and McClane (1999, 2000) report the following signs of strangulation or choking that should prompt medical attention and care: scratches, bruises, red spots (from capillaries bursting), blood-red eyes, rope or cord burns, neck swelling (subtle to massive), red linear vertical or horizontal marks, complaints of neck pain, difficulty in swallowing or breathing, vomiting blood, coughing and a lost or raspy voice. Furthermore, Strack is concerned that persons who have been strangled can present agitation associated with TBI, which may be misinterpreted by the responding law enforcement officers as hostile or uncooperative (Strack, 2000; Strack & McClane, 1999).

Blood Flow Disregulation

An anoxic injury also can occur if the head is punched repeatedly and the neck buckles, causing impingement of the carotid artery (Erlanger et al., 1999; Lampert & Hardman, 1984). Additionally, direct blows to the carotid artery or large vessels can create thrombosis (i.e., a blood clot which can obstruct blood flow), aneurysm and reflex hypotension (Erlanger et al., 1999; Hof et al., 1992; Lampert & Hardman, 1984). Part of a blood clot (termed an embolus) can break off and travel to the brain causing a cerebral vascular accident or death (Nolte, 1993). An aneurysm is defined as a ballooning of the arterial wall, which can compress surrounding structures or rupture (Nolte, 1993). Reflex hypotension secondary to pressure on the carotid sinus decreases blood flow to the brain, causing fainting and brachycardia (Erlanger et al., 1999; Lampert & Hardman, 1984).

Other Possible Injuries to the Brain Secondary to Intimate Violence

Direct physical contact is not the only way the brain may be injured in intimate violent relationships. Sexual violence, sexually transmitted diseases, emotional stress and sleep deprivation can cause changes in brain chemicals, resulting in a limitation in functional status and onset of mental illness (Campbell, Kub & Rose, 1996; Jacobson et al., 1997; Raskin, 1997; Russouw et al., 1997; Sheline et al., 1999). Research of women with a history of suicide attempts indicates that up to 50% of them have experienced intimate violence (Hartzell, Botek & Goldberg, 1996). As many as 66% of female inpatients with mental illness have been abused (Gaffigan-Bender & Narula, 1998; Hadley, 1992). Psychiatric manifestations can include anxiety, depression, eating disorders, self-mutilation, substance abuse, post-traumatic stress disorder and suicide attempts (Barkan & Gary, 1996; Barrier, 1998; Campbell & Soeken, 1999; Casscardi, Langhinrichsen & Vivian, 1992; Gaffigan-Bender & Narula, 1998; Gremillion & Kanof, 1996; Keller, 1996; Loring & Smith, 1994; McFarlane et al., 1991; Plichta, Duncan & Plichta, 1996).

Sexual Assault

Sexual assault can occur within intimate violence relationships. Although the relationship between sexual assault in intimate violence and traumatic brain injuries is not documented well, there appears to be a link. Smikle et al. (1995) attest that women in their study reported a 75% rate of traumatic injury if they were abused both physically and sexually, as compared to women who only were abused sexually (34%) or only physically abused (23%). Women in the Smikle et al. study (1995) who had been abused both sexually and physically reported 60% of their injuries to the head, face, and neck. Koss (1992) reports that half of rape-related injuries treated in the ER presented contusions or abrasions to the head, although the report does not specify the number of rape-related injuries as a result of intimate violence specifically. Other authors indicate that up to 50% of women who are abused physically in intimate violent relationships also are abused sexually (Acierno, Resnick & Kilpatrick, 1997; Quillan, 1996). Hartzell, Botek and Goldberg (1996) indicate 35% of females who have experienced sexual violence in their study also presented orbital fractures.

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Ocular Injuries

Forces inflicted upon the eyes can result in a variety of ocular injuries, which can occur acutely or over time (Falco, 1997; Richards, 1995). A study of 74 professional boxers by Giovanazzo et al. presented the following findings: 66% had ocular damage, 58% had vision threatening complications and 24% had retinal tears (Giovanazzo et al., 1987, as cited in Falco, 1997, as cited in Richards, 1995). It is estimated that 20%-30% of persons with TBI have visual deficits (Sabel & Kasten, 2000).

Lampert and Hardman (1984) state linear acceleration forces particularly are responsible for retinal detachment. Corneal abrasion, lens displacement, cataract formation and glaucoma also can manifest as a result of eye injuries (Falco, 1997; Smith 1988, as cited in Richards, 1995). Because the eyes are an extension of the brain, it is important to note that visual pathway disruption and impairment of the way the brain and eyes work together can cause diplopia, perception disturbance and thwarted reaction times (Richards, 1995; Scheiman, 1997).

Sexually Transmitted Diseases (STDs)

STDs have been estimated to occur in 3%-30% of persons who have been raped (Chescheir, 1996; Family and Intimate Violence Prevention Team, 1998; Koss, 1992). This is a concern for persons in intimate violent relationships considering some STDs, such as syphilis and AIDS, can spread to the brain (Campbell & Soeken, 1999; Family and Intimate Violence Prevention Team, 1998; Warshaw, 1996) and, as with AIDS, be fatal. Neurosyphilis can produce dementia, personality change, impaired cognition, dysphoric or elevated mood, hallucinations, delusions or delirium (Russouw et al., 1997). AIDS-related dementia presents behavioral change and impaired cognition (Jacobson et al., 1997). Recent research by Brian (University of Iowa College of Medicine, 1999) indicates the brain inflammation associated with HIV-dementia can cause the blood flow in the brain to become maldistributed, leading to ischemia or brain damage, resulting in even more damage. Rothenberg and Paskey (1995) believe women with HIV are more likely to be abused and report high levels of depression and prior physical abuse. Furthermore, research indicates persons with a history of sexual abuse who sustain a mild TBI demonstrate greater deficits in the areas of working memory and executive functioning (Raskin, 1997).

Stress

The experience of intimate violence itself—as well as the witnessing of the act by children and others—can be very stressful. Recent research presents the effects of prolonged stress on the brain, indicating that stress can cause permanent neurochemical and structural changes in the brain (Kotulak, 1997; LeDoux, 1998; Raskin, 1997; Scheutzow & Wiercisiewski, 1999; Van der Kolk, McFarlane & Weisaeth, 1996). Changes in brain neurochemicals and brain structures can lead to impaired cognition and psychiatric disorders (Herman, 1992; Raskin, 1997; Scheutzow & Wiercisiewski, 1999; Van der Kolk, McFarlane & Weisaeth, 1996). Moreover, a TBI can compound the effects of neurochemical changes, brain structure changes, impaired cognition and psychiatric conditions.

Post-traumatic stress disorder (PTSD) can develop as a result of intimate violence (Barkan & Gary, 1996; Gaffigan-Bender & Narula, 1998; Muelleman et al., 1996; Van der Kolk, McFarlane & Weisaeth, 1996), with Raskin (1997) indicating that more pronounced cognitive impairments are presented in persons with both PTSD and TBI. Scheutzow and Wiercisiewski (1999) suggest persons with TBIs resulting in the disruption of serotonergic systems may be more predisposed to panic attacks, which also are associated with PTSD. PTSD, depression and chronic stress can cause neuronal cell death and reduction in brain structure size (LeDoux, 1998; Sheline et al., 1999; Van der Kolk, McFarlane & Weisaeth, 1996).

During chronic stress and depression experienced by persons in intimate violent relationships, glucocorticoid production increases to the point of being neurotoxic, with cells being killed directly or by apoptosis (programmed cell death or “cell suicide” in which a controlled or programmed sequence of events leads to the elimination of cells without releasing harmful substances into the surrounding area) (Sheline et al., 1999). Human and animal research demonstrates a size decrease in the hippocampus (i.e., the structure of the brain responsible for sorting and categorizing incoming information into memories) secondary to the increase in cortisol (Sheline et al., 1999; Van der Kolk, McFarlane & Weisaeth, 1996). The amount of volume loss is in direct correlation with the length of depressive episode and cortisol levels (Sheline et al., 1999; Van der Kolk, McFarlane & Weisaeth, 1996). Because of the neuronal decrease, there are deficits in verbal memory and short-term memory skills (LeDoux, 1998; Raskin, 1997; Sheline et al., 1999; Van der Kolk,

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McFarlane & Weisaeth, 1996). Research on combat veterans with PTSD indicates the hippocampus can shrink up to 26% and verbal memory skills can decrease by up to 40% (Van der Kolk, McFarlane & Weisaeth, 1996). In addition, the risk of ischemia, superoxide radical generators and hypoglycemia is increased because the neurotoxic effects of the glucocorticoid increase neuronal susceptibility (Sheline et al., 1999). Sheline et al., (1999) state “depression-related volume loss does appear to be cumulative, suggesting that immediate recognition and treatment of depressive episodes is important in preventing cumulative damage that occurs with repeated episodes of depression” (p. 5042).

Additionally, the amygdala (i.e., the structure of the brain responsible for assigning emotional meaning to incoming stimuli and making associations with past memories) decreases in size in direct proportion to the hippocampus (Sheline et al., 1999; Van der Kolk, McFarlane & Weisaeth, 1996). For persons who have experienced sexual violence and have PTSD, heightened activity in the right visual cortex induces visual memories of the traumatic experiences (Raskin, 1997; Van der Kolk, McFarlane & Weisaeth, 1996). Of particular significance is the deactivation of the Broca’s area (Van der Kolk, McFarlane & Weisaeth, 1996) which has been suggested as an explanation of why persons with PTSD present emotions in physical states and have difficulties putting feelings into words (Van der Kolk, McFarlane & Weisaeth, 1996).

Sleep Deprivation

Persons in intimate violent relationships can experience interrupted sleep for a variety of reasons (i.e., stress, physical symptomology and/or the abuser may wake them up repeatedly during the night as a means of control). Sleep disturbance and sleep deprivation also are very common following TBI (Clinchot et al., 1998; Drake & Bradshaw, 1999; Tobe et al., 1999). The exact relationship between sleep disruption and TBI is unknown, but may be the result of interrupted neurochemical reactions that take place during sleep (Clinchot et al., 1998; Drummond et al., 2000). It appears reasonable to assume a person with TBI in an intimate violent relationship would have disrupted sleep.

Persons with TBI can experience difficulty falling and remaining asleep (Clinchot et al., 1998; Drake & Bradshaw, 1999; Tobe et al., 1999). Females with TBIs report an increased rate of sleep disruption (Clinchot et al., 1998). Persons with TBIs and sleep impairments are apt to experience

decreased levels of function in activities of daily living (ADLS) and vocation, as well as behavioral problems, cognitive impairment, communicative impairment, depression and anxiety (Clinchot et al., 1998; Drake & Bradshaw, 1999). Sleep deprivation can impair cognitive performance in persons with or without TBI (Drummond et al., 2000). Free recall memory and verbal learning is impaired particularly because of a disruption of neural substrates (Drummond et al., 2000). As cognition improves, sleep patterns appear to improve with recovery (Drake & Bradshaw, 1999).

Discussion

Traditionally, it has been asked why a person in an intimate violent relationship does not leave. The time has come to ask such questions as “has a person’s neurological status been altered by being in an intimate violent relationship,” “how many persons have died as a result of undiagnosed TBIs incurred during intimate violence” and “how many of those deaths could have been prevented?”

This article and literature search presented many types of brain alterations and injuries that may result from intimate violence. Combinations of TBI types and other brain status alterations can coexist at the same time, thus magnifying the manifestations.

Research is lacking regarding the incidence and types of TBIs resulting from intimate violence. Additionally, the obtainment of detailed facts is integral to increase diagnosis and treatment. The short- and long-term consequences of stress, TBI and relationships to other diseases must be regarded to the same degree, if not more, than it has been researched already in sports-related injuries. As mentioned previously, the adaptability of the SAC Manual—developed to assess concussions in athletes—needs to be investigated as a screening tool for persons who may have sustained a TBI from intimate physical violence.

Policy makers also must be aware that persons presenting injuries from intimate violence have been denied health, life and disability insurance coverage (Cohen, De Vos & Newberger, 1997; Gremillion & Kanof, 1996; Hyman, 1996; Warshaw, 1996). Reasons for denial have included terming intimate violence as a pre-existing condition, high-risk condition or as a life choice (Gremillion & Kanof, 1996; Hyman, 1996; Warshaw, 1996). Paying out of pocket for health services can prevent persons in intimate violent relationships from seeking healthcare (Hyman, 1996). Persons with undiagnosed TBIs may self-medicate symptoms with illegal drugs or alcohol, which only magnifies the effects of the TBI and can be fatal. Persons in intimate violent relationships should have the opportunity to achieve maximal levels of functioning- physically, emotionally, sexually, and cognitively.

All personnel likely to encounter persons affected by intimate violence must be educated to increase detection and diagnosis of TBI. Additionally, persons in intimate violent relationships must be advised of possible symptoms and when to seek emergency help. For this to occur there must be an increased public awareness of the incidence of TBI caused by intimate violence.

In our society, if an altercation takes place between two people in public there is a plan of action that follows (Jecker, 1993). Ideally, rescue personnel would be called to take care of the wounded and law enforcement would apprehend the perpetrator. Intimate violence altercations traditionally have been viewed as private affairs in our society, and it has been up to the persons involved—most likely the recipient of the violence—to initiate medical and legal attention (Jecker, 1993). If that individual has cognitive impairments from neurological injury and alterations secondary to TBI from intimate violence, his/her ability to do so efficiently will be decreased. It is known that when a person attempts to leave an intimate violent relationship, the chance of fatal injury increases to 75% (Healey, Smith & O’Sullivan, 1998). Therefore, it is imperative that a person attempting to leave such a situation should make very careful, calculated decisions. It follows that a person with TBI and impaired cognition would be at substantial risk during this life or death time period.

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Conclusion

Impairments resulting from intimate violence must be looked at holistically, as all ADLs and daily roles can be affected. The cumulative consequences of undiagnosed TBI as a result of intimate violence spreads with a ripple effect, from the person's immediate family to throughout society. The loss of potential to family and society is a tragic waste. The time has come for the "privatization" of intimate violence to become public, as violence is recognized as a public health concern. Research into the relationship of TBI to intimate violence is imperative, with a focus on what happens neurologically to the person in an intimate violent relationship. In terms of this research, the crucial question remains, "Has being in an intimate violent relationship altered the person's neurological status?"

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