

# [Is impulsive sexual disorder deviance or addiction psychology essay](https://assignbuster.com/is-impulsive-sexual-disorder-deviance-or-addiction-psychology-essay/)

In the last twenty- five years people of moral character have been deemed as having an addiction to various sexual activities. Example, Jimmy Swaggart: adulterous indiscretions with a prostitute Swaggart was subsequently forced to step down from his pulpit for a year (New York Times). Swaggart was caught again by California police three years later in 1991 with another prostitute (King, Wayne). Ted Haggard: regularly visited male prostitutes who also provided him with methamphetamine eventually he admitting to second (and possible multiple homosexual relationships with male church members (CNN-TV Larry King). Douglass Goodman: an evangelical preacher, jailed for three and a half years for the sexual assault of four members of his congregation in 2004(BBC News)( jesusblogger) Lonnie Latham: 2006, Latham, the senior pastor of South Tulsa Baptist Church was arrested for “ offering to engage in an act of lewdness” with a male undercover police officer (Associated Baptist Press). Earl Paulk: (no relation to John Paulk) was the founder and head pastor of Chapel Hill Harvester Church in Decatur, Georgia from 1960 until the 1990s. A number of women from the congregation came forward during the 1990s claiming that Paulk had sexual relations with them. Some of these claims have subsequently been proven correct. Moreover, Donnie Earl Paulk, the current senior pastor of the church and nephew of Earl Paulk, had a court-ordered DNA test in 2007 which showed that he was Earl’s son, not his nephew, which means that Earl and his sister-in-law had had a sexual relationship which led to Donnie’s birth (J. Lee Grady). Joe Barron: arrested on May 15, 2008 for solicitation of a minor after driving from the Dallas area to Bryan, Texas, in order to allegedly engage in sexual relations with what he thought to be a 13 year-old girl he had met online. The “ girl” turned out to be an undercover law enforcement official (Dallas Morning News).

The previous referenced individuals share a few things in common, ministry background, societal view of having a higher moral fiber, all are considered to have an addiction to a sexual behavior or an impulse control issue in relation to a sexual act. You would have to justify being a man of the cloth would be able to differentiate between bad and good character decisions. We see that same issue with professional athletes, over all bad choices, repetitive sexual deviance issues that afflict many football players, basketball players, hockey players, soccer players. In relation to professional ministers and athletes we see that they share the same behavior issue, the lack of self control. What we see justified in athletes as a reason for their behavior is frontal cortex trauma to the brain. Most individuals after some crisis brought on by their own sexually impulsive behaviors seek recovery in some form of addiction treatment center. Further labeling the individual as a, “ Sex Addict”. Currently the DSM-IV has no diagnosable format for sexual addiction. It is classified as impulse control disorder that is not classified elsewhere in the list of impulse control disorders. Currently the DSM-IV groups’ impulse control disorders in a residual category called, “ Impulse Control Disorders Not Classified Elsewhere,” (DSM) because impulse disturbances also occur in bulimia, mania, substance abuse, and paraphilias, and in borderline and antisocial personality disorders. DSM-IV’s residual category has five specific impulse control disorders: pathological gambling, kleptomania, pyromania, intermittent explosive disorder and trichotillomania (the impulse to pull one’s own hair out).(DSM) Clearly there has been no defined psychopathology guideline to determine the, “ Classical Symptoms” of what some are considering a sexual addiction (not by DSM-IV standards anyways.) Clearly misdiagnosis have taken place, because the lack of standards in the field of psychology there is no direct diagnoses code in order to be able to clearly diagnose anyone with having a sexual addiction, in addition to the lack of research for biological and neurological factors that may be present in clinical patients case studies. Many of these factors may be overlooked as a result of poor psychopathology and the lack of treatment guidelines within a classification system in the current DSM-IV standards.

The biological factors that may be present typically are over looked in a case study such as TBI or better known as Traumatic Brain Injury. The brain is the bodies and minds consciousness regulator. Injure that regulator and it will have devastating effects on one’s mind and body. The link between brain injuries and behavior lies within the correlated damage to the prefrontal cortex with psychopathic behavior and the inability to make morally and socially acceptable decisions; as duplicate by all previous individuals(not say they suffered from a MTBI, just suggesting it may be a possibility). Unfortunately, the prefrontal cortex is the forehead region of the brain and is often the site of most injuries. Researchers at the University of Sweden have found the prefrontal cortex to be precisely the area of the brain that is impaired in murderers, rapists, and other violent criminals that re-offend. At the November 1999 annual meeting of the Society for Neuroscience, Asa Bergvall and her colleagues presented findings on their study of violent offenders. The brains of the violent offenders performed normally in every task except the one, which taps prefrontal function, “ In that,” says Bergvall, “ It was as if they were retarded.” They had an impaired ability to shift their attention in order to view the world in a different way – a function linked to the lateral prefrontal cortex.(Society for Neuroscience) Neurology professor Dr. Antonio Damasio and colleagues at the University Of Iowa College Of Medicine reported on two cases of early brain damage to the prefrontal cortex. As adults, both patients showed an almost total lack of guilt. (Sports Med)University of Southern California psychopathologist Adrian Raine has documented prefrontal damage in people with Antisocial Personality Disorder, which is characterized by irresponsibility and deceitfulness, lack of emotional depth and remorse. His article in the Archives of General Psychiatry, February 1, 2000 stated that, “ The antisocial men actually had 11-14% less brain tissue volume in their prefrontal cortexes, compared to normal males – a deficit of about two teaspoons’ worth.” (Archives of General Psychiatry) The “ amygdala” is a pair of small almond-shaped structures situated between the cerebral cortex and the limbic/emotional center of the brain. When this neural circuit for processing emotional information is damaged, the prefrontal cortex cannot interpret feedback from the limbic system. Uninhibited signals from the amygdala lead to free expression of emotions, and may manifest in violent and aggressive behavior, a common complaint following mild traumatic brain injury. The amygdala is responsible for emotional reactions that have to do with survival, including our ability to learn what is fearful to us. Brain injury consists of primary and secondary events. Primary brain injuries – fractures, bruises, blood clots, lacerations of brain tissue or blood vessels – are more or less complete at the time of impact. But, a secondary cycle of biochemical events is set in motion by the trauma and is the major contributor to long-term deficits associated with brain injury. Like personal tectonic plates, the human braincase is composed of eight unique cranial bones. On either side of your skull, layers of material help protect your brain from normal wear and tear. On the outside are muscle, skin, and hair. On the inside, connective tissue and fibrous membranes do the cushioning. Within your skull, your gelatinous brain floats in a sea of cerebrospinal fluid that bathes and supports this precious organ, while acting as a shock absorber during rapid head movements. Although the outer surface of the skull is smooth, parts of its inner surface are rough and jagged and can cause significant damage in acceleration/deceleration, or “ closed head injuries.” In this type of injury there may be no external damage, but because the head abruptly stops after being in motion, the brain rebounds back and forth against the skull’s interior bony structures. This trauma initiates a cycle of biochemical events responsible for the major long-term deficits associated with brain injury. Unfortunately, the area of the head most vulnerable to injury is also where the most fragile and crucial region of the human brain is located. Behind your forehead lies your prefrontal cortex, the center of your higher-order “ executive functions,” as well as home to your social awareness and moral conscience. Injury to the prefrontal cortex can affect your most human qualities: the ability to process information and solve problems; to concentrate, remember, and learn. Damage here can lead to personality changes that manifest in impulsive and socially inappropriate behavior, depression, violence and social norm violations. The prefrontal cortex is the last to form the deep fissures that give the outer layer of the human brain its characteristic cauliflower-like appearance – and its vast array of higher functions. In the womb, this area is the slowest to develop. After birth, brain cells in the prefrontal cortex form connections more slowly than any other brain area and levels of the key neurotransmitter dopamine rise very gradually. The prefrontal cortex bestows humans with “ executive functions,” such as working memory and multi-tasking. The area above your eyes is sometimes called the “ dashboard” of your brain. Like the dashboard of a car – where bundles of insulated electrical wires connect to the vehicle’s other systems – your prefrontal cortex is integrated with regions deep in your brain by bundles of insulated nerve fibers. Here, a subsystem in the prefrontal cortex (the orbitofrontal region) is supported by sharp-edged bony protrusions of the skull’s interior. Although the protrusions do a good job of protecting the olfactory cranial nerve, they become a highly significant factor in brain injury during acceleration-deceleration forces to the brain. What usually happens in brain injury at the cellular level is a combination of primary and secondary damage known as “ axonal injury.” Axons are the microscopic nerve fibers of neurons, the brain cells that communicate with each other. Axons form the long connecting nerve fibers of the neural networks throughout the brain.

After a closed head injury, the shifting and rotation of the brain inside the skull causes a shearing injury to the brain’s complex circuitry. This axonal shearing can occur in localized areas or throughout the brain. The latter is called “ diffuse axonal shear.” Furthermore, the brain cells particularly important to learning and memory (cholinergic neurons), are apparently more vulnerable to trauma than other neurotransmitter systems. Axonal shear is a microscopic tear along the myelin sheath surrounding the nerve fiber that is often followed by micro swelling and the formation of scar tissue. According to Kit W. Harrison, Ph. D., at the Houston Behavioral Health Associates: “ First, the nerve fiber itself may be damaged and begin to swell. The swelling usually acutely reduces functioning of that cell but some neurocognitive functions may be restored soon after as swelling reduces. The process of scarring, however, follows and can take weeks, months, or even years, to complete. As the axon scars over, fewer and fewer impulses can be carried through the tough scar tissue, and the axon may begin to necrotize (die) and lose connectivity function over time. This accounts for a number of symptoms which could worsen with time,” (Journal of Neuropathology and Experimental Neurology). For the past decade, Drs. Maxwell and Graham at the Institute of Biomedical and Life Sciences, University of Glasgow, Scotland have focused on the effect of brain injury at the level of the axon. They have concluded that “ two different mechanisms of injury may be occurring in non-impact injury to the head. The first is shearing of axons and sealing of fragmented axonal membranes within 60 minutes. A second mechanism occurs in other fibers where perturbation of the axon results in axonal swelling and disconnection at a minimum of 2 hours after injury”(Berl). Researchers at the University of Pennsylvania Medical Center have determined that after brain trauma one of the initial events triggering long-term problems includes a massive flood of electrically-charged calcium atoms that enter axons (Journal of Neurosurgery.)” It appears that that the physical motions of trauma literally tears open proteins that act as gates on the axon membrane,” explains Douglas Smith, MD, an associate professor in the Penn Department of Neurosurgery, “ We have now found that it is the rapid flow of sodium ions through the damaged gates that triggers a subsequent inflow of calcium ions.” By evaluating therapies that block the sodium channels, Smith is convinced that the damage can be slowed down and eventually even stopped(Neuroscience). Hours, even months, can go by after a head injury before progressive damage to the axons becomes so severe that the neurons can no longer function. Myelin is a fatty substance that coats and protects the axons. A myelin sheath insulates these individual axons and is crucial to the speed and accuracy of its electrochemical impulse. If the myelin sheath is structurally damaged, then its electrophysiological properties are disrupted, and the electrochemical impulse will become abnormal and uncoordinated down the length of the axon. Consequently the information being conveyed by these nerve fibers will be scrambled or cut off. Most significantly, “ myelination” of the prefrontal cortex is especially slow – not beginning until the ninth prenatal month, and continuing as late as the mid-twenties. That’s why brain injury at an early age can be the most devastating. A key factor in recovery time is the extent of damage to the white matter, the myelinated neuronal axons that serve as cables linking the different areas of the brain. When they are injured, then vital connections needed to allocate functions elsewhere are lost. “ The involvement of white matter tracts portends slower and reduced recovery,” said Dr. Keith Thulborn, director of MR research at the University of Illinois. “ This may reflect reduced capacity to redistribute workload when the connectivity through white matter is disrupted”(Neurology).

You don’t have to be knocked out in order to sustain a brain injury. Mild traumatic brain injury (MTBI), also known as concussion, is becoming a serious public health problem. Most brain injuries are considered mild and appear to be trivial blows to the head, but it turns out that the consequences are not so mild and often lead to deep and prolonged impairments of the brain. The biggest problem in diagnosing someone with a MTBI is the fact they don’t seek treatment according the CDC statics on brain injury that the number that don’t receive treatment is uncountable (CDC). The patient is typically the primary course for what is perceived as the most serious question: Did you lose consciousness? How reliable of a source is a person with a potential brain injury, to answer such a question certainly not without significant examination of the person’s recollection of events. More significantly the loss of consciousness is not the limitless test for brain injury. Any change in mental state can be significant. Headache, lack of consistency in reported symptomatology, nausea and the need for oxygen could be a sign as well. MTBI are likely the most undiagnosed types of head wound. This is because most people don’t realize that any blow to the head, no matter how minor, can be significant. When a minor head blow occurs it is not uncommon for the person to continue to do what they were doing, or even if they do take a time out they don’t get themselves checked out, they don’t get themselves checked out because all seems normal to them. The problem with head injuries is that the wound is located under the skull; it is not seen as the same thing as a broken bone or external laceration which is more obvious. Traumatic Brain Injury has long been referred as the silent epidemic because many individuals suffer and go undiagnosed. According to the U. S. Centers for Disease Control every year 1. 4 million sustained a TBI in the U. S and of that statistic; 1. 1 million are treated in our emergency rooms and released (CDC). Many of these are likely mild traumatic brain injuries as it is common for severe and even moderate brain injuries to require prolonged hospitalization or additional treatment. Many people go untreated due to the fact they simply do not realize that they have a problem.

Mild traumatic brain injuries are often difficult to assess because many individuals do not feel any different after the initial symptom of a head injury began to resolve. CONCUSSIONS are a form of mild TBI, and many people live their lives normally after suffering a concussion, but realistically this is a low level head injury. Any or all of the above symptoms may appear and the ones that emerge may appear consistently or sporadically. There is no set symptom range sense every mild TBI is different no two injuries are the same. The unique problem with them is that so may go undiagnosed making them difficult to detect. As the CDC indicates many undiagnosed cases of TB occur each year and there is no hard statistic on the number of undiagnosed cases. Treatment is the biggest factor in determining what possible side effects someone may have with a MTBI. With the amount of undiagnosed cases in the U. S, this could explain the reason for such erratic behavior by some individuals. The moral abnormalities, impulsive control issues, erratic acts of violence could very well be an underlying reaction to an unknown head injury. A Scottish study found that 47% of people classified as having mild head injuries were actually disabled to some extent one year later, and that they received little rehabilitation or follow-up care with social workers. (British Medical Journal) Many psychiatric delusions appear to be associated with mild traumatic brain injury. Examples include content-specific personality changes, such as when the patient believes that family members are impostors or identical doubles. An extremely common delusion among domestic abusers and stalkers is pathological jealousy and preoccupation with another person (sounds remarkably familiar to O. J Simpson).

Brain injury causes lesions that appear and change over time in the prefrontal cortex and its pathways to the older regions of the brain. This explains the wide spectrum of complex neurobehavioral complaints following MTBI: compulsive and explosive behavior (as we seen in varying professional athletes, in specific professional football players), sensory anomalies, memory loss – as well as behavioral dis-inhibition, domestic violence, and alcohol intolerance. One of the biggest problems is the cognitive and neurological effects of repetitive head injuries (RHI) (As seen in the professional football player). Almost every concussion causes some damage to the brain. Regardless of severity, a second brain injury can be life-threatening if experienced within hours or days of a first. After one brain injury, you have a three-time greater risk for a second injury and an eight-time greater risk for subsequent injuries (Journal of Neurochemistry). Many people attribute violent, antisocial, aggressive behavior to environmental factors such as childhood abuse, but it may surprise you to learn that there can be physical factors as well. Researchers are finding more and more links between violent behavior and brain damage to certain regions of the brain. While no cure is currently available, these eye-opening studies reinforce the need for protections against head injury. Children who experience early damage in the prefrontal cortex never completely develop social or moral reasoning. As adults, even on an intellectual level, they cannot refer to such behavior because they have little concept of it. In contrast, individuals with adult-acquired damage are usually aware of proper social and moral conduct, but are unable to apply such behaviors.

Neurology professor Dr. Antonio Damasio and colleagues at the University Of Iowa College Of Medicine reported on two cases of early brain damage to the prefrontal cortex. As adults, both patients showed the same two distinctive features: an almost total lack of guilt and an inability to plan for the future – but were normal in almost every other type of mental ability (Nature Neuroscience). The patients had problems with violence and resembled “ psychopathic individuals, who are characterized by high levels of aggression and antisocial behavior performed without guilt or empathy for their victims,” commented Raymond Dolan of Institute of Neurology in London (Nature Neuroscience). Their brains were just not capable of acquiring social and moral knowledge even at a normal level. Empirical data on head injury and human sexuality was mainly limited to small series or few parameters until now. In the last 4 years, substantive studies have documented the high prevalence and common patterns of disturbance to sexual behavior following closed and penetrating head injuries of varying severity. Further light has been shed on the anatomical localization of our polymorphous perversity, and studies of non-traumatic brain injury have illuminated atypical changes in sexual behavior after penetrating head injury. Head injury comprises traumatic damage to the skull and its contents, from penetration or acceleration/deceleration forces. Clinically, it implies evidence of raised intracranial pressure, loss of consciousness, post-traumatic amnesia, neurological signs of impaired brain function, and/or skull fracture.

According to US statistics, for every million of the population, 4000 head injuries occur each year; Adding annually a further , one hundred survivors of serious injury (Garden). Consequential disturbance of sexual functioning is the rule rather than the exception as explained by Blackerby. As 85% of all head injuries’ occur before age 25, there is often arrested development of sexual self-concept (Blackerby). He classifies some altered sexual behaviors following head injury as transitory and normative; most as the sexual content of acting out behaviors, sometimes impulsive; many as dysfunctions, arising from cognitive and physical deficits; and a few as the results of sexual identity confusion. The brain damage itself, social environmental response, and pre-injury social skills and experiences all affect subsequent sexual behavior. Brain trauma may lead to poor judgment, social norm violations, egocentricity or insensitivity to the partner, inability to tolerate delayed gratification, poor memory, distractibility, impaired motor functions and side effects of medications.

Social environmental responses of the patient may include social isolation, depression or anxiety, altered body image and self-concept, social norm, violations and role changes on the part of the spouse or partner. Pre-morbid factors comprise general knowledge concerning sexuality, social skills in interacting with others, experiences with friendships, dating, marriage and sex. Blackerby proposes that sexual drive, sub served by deep structures, is rarely disturbed by non-penetrating head-injury; that it is motivation and initiation which are damaged, by blunt frontal lobe trauma. Sexual arousal may also be reduced by loss of touch sensations, impaired sense of smell, or loss of capacity for visual imagery (Hayden and Hart).

Early, middle and late stages of recovery from more than mild head injury are characterized by changing and overlapping patterns of disturbance of sexual behavior (Blackerby). Initially, there is often exhibitionistic exposure and masturbation. Sexual delusions may be evident. Confabulation extends into the middle stage, which is characterized by inappropriate verbal allusions, joking and approaches. Approaches may be physical, accompanying increased sexual drive. Later “ re-entry” behaviors are more influenced by insensitivity to others, distractibility, poor judgment, memory disturbance, spouse or family role change, depression, social isolation, anxiety, medication effects, altered body image and self-concept. Incomplete control of any seizures, and the medications prescribed, may be accompanied by diminished libido.

Lezak (1978) largely attributes spousal sexual and affectionless frustration to patients’ reduced interpersonal sensitivity and ability to empathies, and the mismatch between desire and performance. Many patients make incessant demands, whether or not those demands can be satisfied. Sexual sharing is often one-sided – taking by the patient, giving by the partner. The patient frequently blames the partner for any sexual dysfunction. The patient and the relationship may be so altered that some wives feel as if they are being “ unfaithful” during sexual relating after head-injury (Hayden and Hart).

Early studies of head-injured populations established the high prevalence of sexual behavior disturbance, and attempted to correlate some of the clinical variables. Bond (1976) assessed the psychosocial outcome of severe head injury, using neurophysical, mental and social scales. In his study of 57 patients, length of post-traumatic amnesia and levels of physical disability or cognitive impairment did not predict the occurrence or severity of sexual disturbance.

Kosteljanetz (1981) studied a sample of 19 males who were unconscious for less than 15 minutes and who had post-concussive symptoms lasting a minimum of 6 months. 10 (53%) reported reduced libido, 8 (42%) erectile dysfunction. In his study, sexual dysfunction was more common in the intellectually impaired patients.

Dreaming was not reduced overall following head injury, but the two-thirds of patients who were initially in coma reported a significant decrease in sexual content (Benyakar). Whether or not there was initial unconsciousness, dreams of threatening content increased. As impaired self-perception is usual following traumatic brain injury, a number of authors have attempted to validate the responses of patients by questioning their sexual partners independently.

A series of 50 adults with a minimum of 24 hours post-traumatic amnesia included 12 married patients (Oddy). Six months after the injury they reported no persistent sexual problems and increases as often as decreases in coital frequency. Six months later still (Oddy and Humphrey), three of 7 spouses felt less affectionate towards their injured mates but still reported no significant change in sexual behavior. The only exception was a patient who had developed “ partial impotence”: his wife felt both partners were experiencing less satisfaction even when sex was technically satisfactory. Rosenbaum and Najenson (1976) interviewed the wives of 10 severely brain-injured and 6 spinal-cord injured soldiers 1 year after the event. Sexual activity was better maintained by the brain-injured, and patients’ distress about changes in sexual functioning was less, but their wives reported the greater mood disturbances. The authors partially attributed these wives’ greater dislike of physical intimacy to the brain-injured men’s “ being more self-oriented and exhibiting more childlike dependency” (including less involvement in childrearing and family finances.) 47% of mothers and wives reported that brain-damaged patients, half of them traumatically head-injured, developed sexual preoccupation or lack of interest (Mauss-Clum and Ryan). In contrast with the Oddy et al. series, patients’ inflexibility (20%), inappropriate public behavior (40%), self-centeredness (43%) and decreased self-control (47%) mitigated against sexual re-adjustment. A quarter of the wives had been verbally abused, one in five threatened with physical violence and criticized by their spouses for providing poor care. A third felt they were married to a stranger, nearly a half that they were “ married but don’t have a husband.” Roughly three-quarters of the wives responded with frustration, irritability, depression and anger. Peters (1990) of Winnipeg found that wives of severely head-injured men reported they received significantly less expression of affection than those of mildly or moderately injured patients.

Kreutzer and Zasler (1989) have attempted to assess more comprehensively and specifically changes in sexual functioning following traumatic brain injury. Their Psychosexual Assessment Questionnaire (PAQ) addresses behavior, affect, and self-esteem and qualitative attributes of relationships. The authors have so far reported its use only in 21 male outpatients. Garden (1990) undertook a similar study of 11 male and 4 female patients and their spouses, using questionnaires adapted from the American Medical Association’s Self-Evaluation of Sexual Behavior and Gratification. Clark (1988), studied 33 patients who had a period of unconsciousness followed by at least 24 hours’ post-traumatic amnesia. They demonstrated a fall in testosterone during the first three days after head injury, apparently due to damage to the hypothalamus. This hypogonadism effect, which correlated positively with severity of injury, persisted at 3-6 months in 5 out of the 21 patients retested. Study of disturbances in sexuality after focal brain injury may give clues about areas of brain involved in normal sexual response. Sabhesan and Natarajan (1989) attempted to correlate evidence of persistent neurological damage with disturbances of sexual functioning still evident in 21 out of 34 East Indian patients a year after head injury. Sexually-inappropriate behavior (purposeful use of lewd language, exhibitionism, sadism and rape) occurring for the first time following the head-injury, was consistently associated with other evidence of frontal lobe damage. In the other three out of eight patients with frontal lobe syndrome (constricted emotional expression, reduced inhibition, impaired foresight, personality change, usually intellectual impairment); there was total loss of libido as part of global motivation.

Patients with continuing sexual disturbances (sexually-inappropriate behavior and dysfunctions) were distinguishable from the recovered controls in having significant prevalence of delusional disorder, depression and other neurotic features. Hypersexual behavior is much less common than hypo sexuality following brain injury. Miller (1986) attempted to correlate the development of hypersexual states with the site of the lesion in 4 patients with non-traumatic brain injury. Two of the patients had basal frontal lesions, whereas a third developed injury to the thalamic and per ventricular regions of the right hemisphere, accompanied by a sexual preoccupation in the context of a manic syndrome. The fourth Miller patient, who had temporal lobe damage, developed interictal hypo sexuality punctuated by hypersexual arousal after seizures. Similar hyper sexuality has been documented following temporal lobotomy for epilepsy (notably Blumer). The Kluver-Bucy Syndrome (visual agnosia, placidity, altered sexual activity, irresistible impulse to touch, hyperorality and altered dietary habits), was first described in rhesus monkeys. Including a striking increase in the amount and diversity of sexual manifestations, it has similarly been described (Isern) after a gunshot wound to the temporal lobe. Temporal lobe structures also appear to mediate sexual preference. The Kluver-Bucy Syndrome in humans, both a traumatic and following head injury is usually associated with aphasia, amnesia, dementia and sometimes seizures. It has involved changes in sexual preference more commonly than hyper sexuality (Lilly). These case-reports echo that of Mitchell (1954) whose patient’s temporal lobe epilepsy was invariably triggered by viewing of a fetish object (safety-pin): not only the epilepsy, but also the fetish itself, was abolished by temporal lobectomy. Miller described four further patients who develope