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TRAUMATIC AMNESIA, REPRESSION, AND HIPPOCAMPUS INJURY DUE TO EMOTIONAL STRESS, CORTICOSTEROIDS AND ENKEPHALINS

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ABSTRACT

The role of stress, arousal, emotional trauma, and corticosteroid and enkephalin secretion on memory and the hippocampus, and the development of traumatic amnesia and repressed memory syndrome are detailed. Animal and human studies are reviewed. Trauma-induced memory deficits appear to be secondary to abnormal neocortical and hippocampal arousal, and corticosteroid and enkephalin secretion which can induce atrophy or seizures within the hippocampus, suppress hippocampal theta activity and long term potentiation, as well as injure hippocampal pyramidal cells. Predisposing factors include individual, age, and sex differences in arousal, and previous emotional trauma or temporal lobe or hippocampal injury. However, as the amygdala processes and stores emotional experiences in memory, patients may also demonstrate trauma related symptoms, including flashbacks as well as shrinking retrograde amnesia.

It is well established that fear, pain, sexual activity, and emotional stress affect neocortical and limbic system

arousal as well as learning and memory. However, although low and intermediate levels of arousal improve learning, as arousal and stress levels dramatically increase, memory deteriorates and fewer details are recalled, including events which occurred immediately prior to or following each high stress episode, such that an inverted-U shaped learning curve is produced. 1,2,3,5,6,7,8,9,10,11,12,13 Under excessive and prolonged conditions of stress, excitation, and arousal, learning and memory may be completely eclipsed, inducing a profound amnesia, 11,14,15 and abnormal activity and injury to the hippocampus. 16,17,18,19,20,21,22,23,24 As is well known, the hippocampus assists in storing words, places, conversations, written material, contextual details, and spatial relationships in long-term memory. 25-36

In part, it appears that high levels of stress and arousal interfere with memory and new learning by inducing abnormal neocortical and hippocampal activity. 11,14-18 Excessive stress and arousal also eliminate hippocampal theta activity and long-term potentiation (LTP). 37-41 LTP represents prolonged neural excitation which is associated with learning and memory. 42-46

In fact, the hippocampus may atrophy due to the repeated stress induced secretion of corticosteroids and enkephalins, 19-24 which attack hippocampal neurons, disrupt LTP, and produce an inverted-U shaped learning curve. 37,47-51

With direct hippocampal activation, subjects may suffer a profound amnesia which may extend backward

in time from minutes to days and even weeks. 14,15 However, as with amnesia due to head injury, patients may subsequently demonstrate some memory recall and a shrinking retrograde amnesia. 11,14,15

As will be detailed below, the deleterious effects of excessive and prolonged stress and arousal on brain functioning, coupled with other predisposing factors, may well explain the flashbacks, disturbances of memory, traumatic amnesia and the trauma-induced repressed memory syndrome which afflicts some victims of sexual abuse, 11,52-56 rape, 56-59 physical assault, 60-63 front line combat, 64-67 and natural disasters and related trauma. 68-70 Indeed, adults who have been severely traumatized or sexually abused as children also display hippocampal atrophy and disorders of memory. 19,20 Hence, this relationship is not merely hypothetical or coincidental as again, it is well known that hippocampal injury disrupts the ability to convert short-term memories into long-term memories, and when injured patients suffer from varying degrees of amnesia. 25-56

TRAUMATIC AMNESIA:

AGE, INDIVIDUAL DIFFERENCES, PREDISPOSING FACTORS

Because the hippocampus may be repeatedly injured by excessive arousal and repeated stress induced enkephalin and corticosteroid secretory episodes, 21-24,71 traumas which are prolonged or repeatedly suffered are often more difficult to remember than a single episode of severe turmoil. 11,70 However, situational and individual differences are important

contributing factors.

For example, children are more at risk for traumatic memory loss than older individuals. 11,72-75 This may be due in part to the immaturity of the hippocampus which remains exceedingly plastic and does not complete its cycle of myelination until well after the first decade. 11,76 Another factor is the prolonged immaturity of the corpus callosum which is limited in its ability to transfer information and memories between the right and left hemisphere until well after the age of five. 11,77,78

As the right hemisphere and right temporal lobe are dominant for the storage of personal and emotionally laden experiences, 11 callosal immaturity prevents the language dominant left hemisphere from gaining access to this data. This also explains why most individuals have difficulty recalling events which occurred prior to age three-and-a-half. 11 Hence, the younger the victim and the more prolonged the trauma, the more pronounced might be the memory loss.

Consider, for example, the nationally publicized trauma of "Baby Jessica" McClure who spent 58 terrifying and painful hours trapped 22 feet deep below the Earth's surface in a narrow hole when she was just 18 months old. Although this incident was internationally televised and subject to intense media attention, several books, and one television movie, and despite the skin grafts and the amputation of one toe, 10-year-old Jessica cannot remember anything of her ordeal. 79

In yet another well publicized incident, 3-year-old Tara

Burke was kidnaped, held captive in a van, and repeatedly sexually assaulted by two men; an ordeal which lasted for 10 months and which was filmed by her tormentors. Despite intense media scrutiny and a subsequent court trial, 18-year-old Tara nevertheless reports that the "memory has been erased" from her mind. "It's like a story that has happened to someone else." 80,81

However, although age and callosal immaturity play a significant role in memory loss for early chilhood experiences, childhood amnesia is not always due to trauma and in this regard, must be distinguished from trauma-induced repression. Rather, although age, sex, predisposing factors and individual differences are contributory, 11 it is the prolonged, terrifying nature of the trauma which explains why disturbances of memory and traumatic amnesia are not limited to children but includes hardened soldiers, 64-67 as well as adult victims of rape. 57-60

For example, Donaldson and Gardner 56 describe a woman who was kidnaped and repeatedly raped over a period of weeks. She suffered profound memory loss and was able to recall only bits a pieces of what occurred until nightmares, flashbacks and therapeutic assistance enabled her to remember.

As with children, 11,53,70 a single instance of terror can also induce memory loss in adults. Fisher, 83 for example, describes a druggist who was terrorized, handcuffed and robbed by two thieves, and subsequently became amnesic and lost all memories of what occurred. In another case, a woman became amnesic for 18 hours after seeing her husband die right

before her eyes. 83

However, in instances of amnesia secondary to a single terrifying event, those afflicted may well have a history of previous emotional trauma, or hippocampal-temporal lobe injury which places them at risk and predisposes them to suffering memory loss. 11, 83 If there are predisposing factors, an emotional shock may induce an amnesia so profound even personal identity may be forgotten.

For example, Schacter and colleagues 84 described a 21-year-old man (PN) who was discovered wondering the streets of Toronto and who had no idea as to his name, address, or any other personal information. Following his hospitalization and the placement of his picture in the newspaper, a cousin came forward, and reported that PN's dearly beloved grandfather had died the week before; a traumatic event which immediately preceded the amnesia, and which PN claimed not to remember. PN may well have been predisposed to become temporarily amnesic if provided sufficient emotional shock, for a CT scan revealed a right temporal lobe injury suggestive of gliosis.

Similarly, Christianson and Nilsson 58 describe a 23-year-old female who was raped and beaten while out jogging. When found by police she had no memory of her identity or that of her relatives, friends, boyfriend, or place of work. All memories of her past life, including all aspects of the rape were forgotten. Once her memory returned, it was determined that she had been repeatedly sexually abused as a child.

Presumably, this childhood sexual trauma predisposed

this young woman to suffering memory loss and/or further neurological injury in response to this latter severe emotional stress. A CT scan or MRI may have in fact revealed a hippocampal injury as has been reported in similar cases.

11 Again, it has been reported that children who were sexually abused, as well as adults suffering from post traumatic stress disorder, display hippocampal injury and atrophy, 19,20 as well as disturbances of memory, including trauma-induced repressed memory syndrome.

Hence, a victim's age, personality, emotional history, rearing environment, predisposing factors, and the nature of the trauma may differentially contribute to the stress response including any disturbances of memory. Indeed, in addition to age and other predisposing factors, there are sex, intellectual, and thus individual differences in regard to the effects of emotionality and arousal on learning and memory. 4,7,10,11,74,75,82

Given the above, it should not be surprising that an intelligent, stable, well-adjusted child, who is fondled on only a few occasions by a "loving" adult who has provided her with toys and gifts and where the abuse is discovered and becomes public, will not be as profoundly affected as a battered child who is secretly and repeatedly raped and tortured, and who is threatened with death and told "you're crazy.... it never happened... someone has been planting false memories."

PUBLIC TRAUMAS & MEMORY LOSS

Not just secret and personal events, but well publicized national traumas can induce varying degrees of memory loss, even when the incident is openly and repeatedly discussed with friends, family and in the media. 11,74,75 Again, however, age, temperament, and individual differences differentially contribute to any subsequent memory loss.

For example, in their study of children's recollections following the Challenger space craft explosion, Warren and Swartwood 74 found that children age 5 were less accurate and more likely to delete features and forget details as compared to older children, and that those who had been the most emotionally upset suffered the greatest degree of memory loss.

Winograd and Killinger 75 in examining memory for the death of President Kennedy and Robert Kennedy, found a steep gradient of forgetting which became more profound for memories formed between the ages of 1-7. Only approximately 50% of those who had been 4.5 years old or older could verbally recall the news and provide at least one verbal detail. Those who were younger than 3 had no verbal recollections regarding context or associated events or information sources, and only a few who were younger than 5 were able to demonstrate detailed verbal knowledge or memories when questioned as adults.

Again, however, even adults demonstrate a significant degree of forgetting. For example, Neisser and Harsch 85 had subjects fill out a questionnaire regarding where they were and how they heard about the Challenger accident and so on. When these subjects were questioned again 32-34 months later, 75% could not

recall filling out the questionnaire. Many of the subjects in fact had forgotten considerable detail regarding the accident. According to Neisser and Harsch, "As far as we can tell, the original memories are just gone."

HIPPOCAMPAL INJURY & MEMORY LOSS

It is thus quite apparent that even highly emotional public events can have negative influences on verbal recall such that even supposedly vivid ("flashbulb") memories tend to become riddled with gaps which may be filled with confabulatory ideas and errors. 11,77,78 Although memory loss in the above instances could be attributed to "normal forgetting," it can also be surmised that the prolonged emotional shock and stress experienced secondary to these national traumas may well have exerted deleterious effects on the functional integrity of the hippocampus--which when injured can result in profound memory loss and amnesia.

Consider, for example, the famous case of H.M. who can recall almost nothing following bilateral hippocampal amputation. 26,27 Every time he discovers his favorite uncle died he suffers the same grief as if he had just been informed for the first time. Dr. Brenda Milner has worked with H.M. for over 25 years and yet she is an utter stranger to him.

Although without memory for new (non-motor) information, HM has adequate intelligence, is painfully aware of his deficit and constantly apologizes for his problem. "Right now, I'm wondering" he once said, "Have I done or said anything amiss? You see, at this moment everything looks clear to me, but what

happened just before? It's like waking from a dream. I just don't remember...Every day is alone in itself, whatever enjoyment I've had, and whatever sorrow I've had...I just don't remember" (p.96). 86

MEMORY LOSS & HIPPOCAMPAL HYPERACTIVATION

There is considerable evidence which indicates that the hippocampus places various short-term memories into long-term storage. Presumably the hippocampus encodes new information during the storage and consolidation (long-term storage) phase, and assists in the gating of afferent streams of information destined for the neocortex by filtering or suppressing irrelevant sense data which may interfere with memory consolidation. 25-36,87-89

In part, it appears that long-term memory consolidation is made possible through hippocampal influences on thalamic and neocortical arousal. 11,14-18,90 The bidirectional neural pathways linking these tissues, and the fact that the medial hippocampus is contiguous with the medial temporal lobe, presumably enables the hippocampus to activate specific neocortical regions, to track information as it is stored in the neocortex, and to form conjunctions between different brain regions which process associated memories.11,27,29,33,87,88,89 This may also be accomplished, in part, through the modulation of arousal and selective attention.

For example, the hippocampus exerts desynchronizing and synchronizing influences on the neocortex and various thalamic nuclei and can augment or decrease

thalamic and neocortical activity. 11,14-18,90-93 Through activational influences it is presumably able to block or enhance information transfer to various neocortical areas where perceptual processing is taking place and where various memories and perceptual experiences are presumably stored.

However, as noted, under high levels of arousal, the neocortex and the hippocampus may become abnormally activated and overwhelmed. 14-18,90 These conditions give rise to an inverse-U shaped learning curve and an amnesia which may extend backward in time from minutes to days and even weeks. 14,15

For example, when the neocortex becomes desynchronized (indicating neocortical arousal), the hippocampus sometimes develops slow wave, synchronous theta activity which correlates with new learning and the stabilization and preservation of those memories presumably destined for the neocortex. 11,16-19,41 However, when both the neocortex and the hippocampus become highly aroused and desynchronized, theta disappears and distractibility, confusion, disorientation, and hyperresponsiveness results 16,90 Distractability and hyperresponsiveness also occurs following hippocampal lesions in rats and other animals. 29,94,95

Similarly, under conditions of overwhelming terror or high levels of arousal, the hippocampus becomes desynchronized, and the subject may seem petrified with fear and will be unable to respond to environmental stimuli, be it a threatening or painful stimulus. 11,16,90 Sensory processing is eclipsed, what

is experienced may be forgotten or stored abnormally and independently of the hippocampus, and those afflicted may suffer a profound hippocampal amnesia. 14,15 Patients may also experience a variety of emotional disturbances, including flashbacks and nightmares, as these aspects of emotional memory and recall are in part mediated by the amygdala. 11,77

HIPPOCAMPAL LTP, THETA, AROUSAL & MEMORY LOSS

It has been demonstrated that learning and memory are associated with synaptic changes in the growth and morphology of dendritic spines, and the development of long-term synaptic potentiation (LTP) and EEG evoked responses and theta activity within the hippocampus. 37,42,43,44,45,46,87,88

For example, not only are correlations between hippocampal LTP, theta activity, and learning found across a variety of tasks, 42-46 but theta and especially LTP may be triggered by very brief periods of excitation, and then, in the case of LTP, continue to build up within the first half hour during memory acquisition, and then persist from hours to days to weeks to months. 42-46,96 Presumably this prolonged activity and the memories it represents may remain vulnerable for an equal period of time and is thus disrupted by stress or severe head injuries such that patients may suffer a retrograde amnesia, which extends backwards in time for hours, days, weeks, or months.

Presumably, this long lasting synaptic activity acts to bind the pre- and post-synaptic surfaces and thus

reflects synaptic activity in those neurons responsible for learning and transferring information from short-term, to long term memory. 11,33,44,45,88,96,97 However, once this information has been consolidated and placed in long-term storage, LTP ceases to be a factor in memory maintenance. That is, through LTP the hippocampus presumably acts to bind various divergent neocortical sites so as to form a circuit of experience and create specific neural networks which supports or maintains specific memories. 11,33,44,45,87,88

Again, however, hippocampal LTP and theta activity are disrupted and suppressed by high levels of arousal, fear, and stress, 18,38,39,40,90, and there is synaptic depression, and the hippocampus may be beset by irregular electrophysiological activity. 41,90

If abnormal activation and excitability is prolonged, the hippocampus may become prone to seizures, 98,99 and non-emotional learning and memory suffer significantly, 14,15 due presumably to the eradication of hippocampal LTP which may have been building for weeks. Again, however, these same patients may subsequently experience some degree of memory recovery; i.e. shrinking retrograde amnesia.

CORTICOSTEROIDS, FEAR, STRESS, LTP & HIPPOCAMPAL ATROPHY

Normally, as part of the "fight or flight" response, and in reaction to fear, pain, and emotional stress, the hypothalamic-pituitary-adrenal (HPA) axis prepare sthe brain and the body for possible catastrophic consequences by secreting large amounts of the amino

peptide, corticotropin-releasing factor and a variety of corticosteroids (which are secreted by the adrenals). These stress hormones potentiate behavioral and autonomic reactions when confronted with or following bodily injury, and thus provide protective as well as activating influence which enables the organism to continue to function and thus escape or fight for their life.

As noted, high corticosteroids levels inhibit the hippocampus, 22,24,100,101 eliminate hippocampal theta, 40 and can block and prevent hippocampal LTP, 37,38,51 thus producing profound memory deficits. 37,47-50,102 Although low levels of corticosteroids can exert mild increases in arousal and enhance learning and memory, as corticosteroid levels increase memory deteriorates and an inverted-U shaped learning curve is produced. 22,37,47 For example, the oral administration of 10 mg of hydrocortisone in rats, 48 or repeated doses of 80 mg of prednisone in humans, significantly disrupt learning and memory, and interferes with recognition memory, 49,50 including the ability to discriminate between relevant and irrelevant stimuli. 22

High levels of corticosteroids also injure hippocampal pyramidal neurons, 71 kill cells in the dentate gyrus and Ammon's horn, 22 and induce hippocampal atrophy 21-23,101 --an affect exacerbated by activation of Type II adrenal steroid receptors which abound within the hippocampus. 22,102 In fact, the overproduction of corticosteroids is directly correlated with hippocampal atrophy and memory loss among individuals with Cushing's syndrome. 103

In part, the deleterious effects of high corticosteroids levels on learning, memory, LTP, theta, and the hippocampus, are due to suppression of membrane receptor proteins which affect excitability and information transmission between neurons. 104,105 Moreover, corticosteroids can detach the cellular receptor from its attached protein; 106 a condition which interferes with messenger RNA protein transcription, 107 and thus the genetics of memory. Incoming messages cannot be acted on, learning cannot take place, injured or damage cells cannot be repaired due to RNA/DNA interference, and in consequence memory dysfunction and hippocampal atrophy results.

OPIATES & HIPPOCAMPAL AMNESIA

In response to physical injury, terror, and severe emotional stress, the amygdala, hypothalamus, brainstem, striatum and related limbic system nuclei secrete opiate-like substances, enkephalins. Like corticosteroids, enkephalins are released as part of the fight or flight response, and insure that an animal or human can continue to do battle, or to successfully run away, although severely injured. Indeed, it is the massive secretion of opiates which may account for the narcotic-like bliss associated with "near death experiences" and the numbing which enables a severely wounded warrior to keep fighting, or a hunted and wounded beast to lie down and calmly allow itself to be eaten alive. 11

Like corticosteroids, enkephalins abolish LTP and theta activity, 39,98,99 disrupt learning and memory, 2,3 and induce hippocampal epileptiform and seizure activity (albeit in the absence of convulsions) which is

accompanied by abnormal, high voltage EEG paroxysmal waves which can last from 15 to 30 minutes. 100,101 Enkephalins can also trigger hyperactivation of hippocampal pyramidal cells, 100,101 --neurons which normally display synaptic growth and dendritic proliferation in response to new learning. 87,88 Enkephalins can also alter the pre- and post-synaptic substrates, 108,109 thereby injuring hippocampal neurons and producing a hippocampal amnesia as well as a state dependent memory loss. 11

CONCLUSIONS

In response to high levels of prolonged arousal, and repetitive trauma, stress, and fear, the brain in general, and the hippocampus in particular, are repeatedly pummeled by neurochemical insults and abnormal neurophysiological activity. LTP and theta activity may be abolished, hippocampal neurons may atrophy or die, and learning and memory may be so disrupted that patients become amnesic. Prolonnged, and repetitive levels of extreme stress and the secretion of enkephalins and corticosteroids exert a deleterious and harmful effect on memory and the brain, the hippocampus in particular.

The evidence reviewed above strongly indicates that the deleterious effects of prolonged and high levels of stress, fear, and arousal on the hippocampus and learning and memory may account for the disturbances of memory, and the trauma-induced repressed memory syndrome which sometimes afflicts victims of sexual abuse, rape, physical injury, combat, and natural disasters. These highly arousing and stressful conditions partly explain not only the memory loss, but

the hippocampal atrophy which has been recently demonstrated among those subjected to combat conditions, or repeated sexual abuse during childhood. 19,20,110,111 Sexual abuse injures the hippocampus.

Unfortunately, although suffering from a profound, albeit circumscribed traumatic amnesia, victims may remain emotionally traumatized as emotion, fear, and trauma, the felt aspects of memory, are processed and stored by the amygdala. 11,112-121 The amygdala and its neural pathways, in fact, display neural plasticity as well as LTP in response to fear and emotional learning. 44,122,123 When activated, the amygdala can trigger forgotten memories, intrusive images, and flashbacks of various highly emotional incidents involving intense fear, terror, or sexual intercourse. 110,111,124,125 Amygdala activation can also induce the remembrance of at least some aspects of those traumas experienced in the absence of hippocampal participation. 11,119

Indeed, although hippocampal functioning may be disrupted under prolonged, highly stressful conditions, the events in question may still be partially consolidated, yet suffer an abnormal fate. 11,33 Coupled with the participation of the amygdala in personal and emotional memory storage, the abnormal storage of these memories may well explain the initial amnesia which is sometimes followed by flashbacks, traumatic dreams, and partial memory recovery (shrinking retrograde amnesia). 126,127,128

Thus trauma-induced amnesia and the repressed memory syndrome appear to be due to the deleterious effects of excessive stress, fear, and arousal on the functional and structural integrity of the hippocampus.

By contrast, symptom formation, flashbacks, and intrusive imagery may be due in part, to the mediation, retention, and recollection of the emotional, traumatic attributes of memory by the amygdala; a nucleus which may also be negatively impacted and thus function abnormally following prolonged or repeated emotional traumas. 11

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