

A Review of the Psychological Consequences of Lightning and Electrical Injury

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Abstract—A hypothesis is proposed for the causation of remote injuries from Electrical and Lightning Injuries (ELI). This theory takes instruction from recent developments in biological psychiatry and in particular the cortisol theory of depression. This is reviewed and its consequences are given.

The theory is developed as follows. Cortisol is a known stress hormone, and is likely to be activated by the trauma of an electrical injury. Intense trauma chronically affects the control mechanisms for cortisol production leading to chronic cortisol level excess. Cortisol damages the areas of the brain involved in the memory, learning, and fluency dysfunctions seen in ELI, and leads to loss of volume of those regions, due to cell death. Cortisol levels affect the production of BDNF – Brain Derived Neurotropic Factor – in a proportion of individuals. BDNF exists in polymorphic forms and BDNF in one form can be severely depleted by stress. The brain loses its plasticity as a result. Depression results, and is of a type consistent with ELI, along with other psychological features of ELI.

It is hoped this theory will lead to experiments to support it, and will guide the use of medication for the injury.

I. INTRODUCTION

It is often not appreciated that the consequences of a lightning injury (LI), as well as an Electrical Injury (EI), contain several elements, both physical and psychological. The injury certainly can show symptoms and signs in the line of current passage, and these can be attributed simply to injury from the current itself. However there are a significant group of symptoms held in common that are psychological in nature and these cannot be attributed to current passage in all but the rarest case. Indeed “expert” opinions on the condition can blindly state that the psychological manifestations are manufactured and represent malingering. The opinion is that if there is no evidence of current traversing the brain, then the injury is not real, or not attributable to an electrical mechanism. In essence for many, the injury is allowable only if confined to the line of the current.

These views fail to recognise the fact that the psychological syndrome is well described [1-10] and is consistent between victims who have had no chance of colluding or manufacturing their symptoms.

Electrical Injuries and Lightning injuries in general are different, but they do share a commonality in the psychological

consequences. Observations applying to one group are broadly applicable to the other when considering psychological injury.

The author regards it as “the holy grail” of electrical and lightning injuries (ELI) to discover the mechanism underlying these symptoms in order to understand and treat them. They are often termed “remote symptoms” in that they arise in areas quite separate from the line of current passage, all the more so in lightning injury. Broadly it is presumed that remote symptoms imply the generation of psychological symptoms presumably from some interference with brain function, when the current passage is peripheral.

It has been suggested that these injuries are the cause of much more suffering, not to mention loss of work, than any other[11]. The suffering includes work related matters, such as loss of ability to perform work, loss of cognitive ability, reliance on workmates, loss of promotion, and eventually unemployment. In the domestic environment, there is inability to support one’s family, loss of income and opportunity, inability to partake in family activities, inability to enjoy usual hobbies, and personality change leading to relationship disturbance and often marital breakdown.

The author would prefer to see the totality of the injury referred to as a “Post Electric Shock Syndrome” and for it to be recognised as such in the compendia.

It is my intent in this paper to document the injury, and provide information for those less familiar with the psychological components. I would also like to document competing theories for their causation.

II. REMOTE INJURIES

The injuries that are seen as the remote group of injuries are set out below[12]. These represent the author’s summary.

Memory Disturbance	71%
Concentration Disturbance	63%
Aggression and Irritation	67%
Wariness and Phobia	58%
Loss of Mental Powers	50%
Social Isolation	38%
Sleep Disorder	38%

and others including confusion, word finding disability, anxiety, depression, and learning disorder.

An important part of evaluating a victim is to submit them to neuropsychological testing. The aim of such testing, in part, is to objectify the dysfunctions that are seen, and if possible, subdivide them more specifically. In the cohort reported by the author[2], this testing was undertaken and demonstrated the existence of

Memory and Learning Deficits	
globally	19%
more specifically	
Visual	35%
Visuospatial	38%
Auditory	62%
Verbal Learning Deficit	54%
Verbal Fluency deficit	46%
Concentration and Attention Deficit	46%/42%
Executive Function Deficit	38%
Reduced Executive Speed	62%

and others including General and Verbal IQ decrease, Dynamic Coordination decrease, Slowed information processing, Deficit in fine motor skills, phobia, and anxiety and depression.

Morse [13] lists short term memory loss, general memory loss, confusion, phobia, personality change, sleep disorders, loss of attention span and concentration, emotionality, temper.

Crews[14] emphasise loss of confidence in vocational activities, distractibility, anxiety, with decreased libido, irritability and anger, and high stress indices.

When neuropsychological testing was performed the comment that these deficits seemed to have an “organic” rather than a “functional” origin was made. The distinction is important and the terminology will be examined below. The strong appearance of auditory and verbal memory and learning deficits, (these are often termed “spatial” deficits) supports an organic origin to the deficits also. Grossman [15] particularly makes this distinction, and notes psychomotor slowing, memory loss and sleep dysfunction, cognitive deficit, fatigue, and sexual dysfunction. Multiple other writers report similar phenomena [1, 4, 7, 8, 16-20]

The enigma raised therefore is how brain injury, remote from the passage of electric current could occur and this paper hypothesizes on this question (see also for example [13]).

The aim of this paper is examine hypotheses for how remote injuries may occur. Such an examination must consider two major aspects:

- a. How a remote injury might occur

- b. How the observed delays in onset of some features may occur.

In considering general mechanisms for remote injury causation, we might include major general groups of theories:

1. A neurohumoral mechanism, where the injury causes peripheral release of some neuroactive substance, which in turn act on the brain, and alternatively within the brain itself.

2. A reverse conduction mechanism. It is known that peripheral activation of pain receptors activates a complex set of interactions at spinal cord level (turning acute pain into a chronic pain syndrome where “Pain is the Disease” rather than its cause), and reverse conduction superiorly via the cord to the brain.

3. Parallel current paths indeed involving the brain however small the degree

4. And possibly others.

We first ask if there is evidence of brain change underlying at least some of these facets.

Andrews [1] has undertaken speculative imaging in these injuries, using SPECT scans. These are interpreted with caution, as many factors may influence them. Nonetheless broad alteration in cerebral function is seen.

Ramati et al., [21], note deficiencies inattention, learning and working memory. They state the nub of the problem in “... cognitive changes occur in EI survivors, even in cases in whom the head was not in direct contact with the ... source”. They quote inconsistent findings in cerebral function including decreased perfusion in mesial temporal cortex and the caudate nucleus of the brain. We will return to these sites later. After a learning paradigm, fMRI was used to examine different areas of brain activity. The areas of the brain most involved with cognitive based eye movement required for working memory and procedural learning, are the dorsolateral prefrontal cortex, anterior cingulate, and dorsal striatum. Prefiguring later comments, these have direct connections with the limbic system of the brain, and an area named the hippocampus. Working memory and procedural learning were evaluated. The electrically injured patients showed disturbance in areas of the brain involved in cognitive processes, and these regions could represent underlying neural deficiencies in working memory and learning deficiency seen in neuropsychological testing. Areas of increased activation were seen with working memory tasks, implying inefficiency and compensation in these brain areas. There were decreases in activation in areas involved in cognitive processing. The dichotomy between increases in some areas, and decreases in others seemed to be a hallmark of electrical injury. The underlying pathological reasons for the differences were left open.

III. TERMINOLOGY AND UNDERLYING CONCEPTS

A. Organic versus Functional Causation

The terms “organic” and “functional” are terms that the reader will often see with regard to causation of psychological disorders. They will be used in this paper also. In one sense

they derive from historical views of theories of mental dysfunction, and with the benefit of current knowledge, are not as clear cut as once thought. The brief discussion here will start from an older viewpoint to make the distinction clearer.

There have been, in the past, two relatively clear-cut circumstances in which mental illness has been thought to occur.

First, an “organic” cause. It is quite obvious that a neurological disease or demonstrable brain disturbance can show emotional consequences. For example, multiple sclerosis, brain tumours, and other changes in brain structure can produce psychological symptoms. Lay people often term such changes as evidence of “brain damage”, in the sense that they are observable when recourse is made to investigations of body structure, e.g CT scans, ultrasound, MRI scans, and so on. These are “organic” in the sense that they result from definite tissue abnormality superficially thought to reside “within the person” – whether at macroscopic or microscopic level.

On the other hand, “functional” illness was identified as a dysfunction for which such an organic causation could not be identified. Sometimes the cause was seen not as residing in the individual themselves, for example, it may well have been seen as a psychological reaction which was more a reaction to circumstances than a malady with identifiable cause. Other examples include psychological disturbance as a result of malfunction of other body chemicals, for example the psychological consequences of thyroid disease.

The distinction nowadays is artificial. Even with those maladies we might have called functional, refinements in measuring and observational techniques do identify biochemical changes which are not observable on structural investigations. So functional illness may be just as organic, with the causative process simply being microscopic.

In another context, Andrews et al., have suggested that the distinction between organic and functional illness is more dependant on our measuring techniques and their resolution than any biological difference[22]. This also brings to mind the distinction between structural and functional investigation modalities. Structural investigations are just that – they demonstrate structure. These include XRays, MRI scans, CT scans, ultrasound, and so on.

Functional investigations aim to demonstrate chemical and biochemical activity, for example nuclear scans and the like. Hints at abnormalities for ELI victims can be demonstrated in these. These include, for example, deriving an image of the distribution of a radioactive natural body chemical and any abnormality in its distribution.

There is however one caveat. If a functional investigation is to demonstrate abnormality, it must “look” in the place where the damage exists. For example, this author would suggest that the major neuro-muscular (physical) abnormality in ELI exists at the level of the motor end plate (the locus where nerve connects to muscle to activate it and fatigue of which can well be seen as the typical muscle fatigue of the ELI victim). Traditional Nerve Conduction Studies are normal, however it

should be remembered that these test the function of the major nerve trunks, and not the endplates.

Similarly the sensory transducers are microscopic fine structures in and around skin and major organs. There is little in the way of investigation to test these.

B. Depression

Depression is a disorder which, in the older view, we could determine as being either organic or functional depending on perceived causation. It covers a multitude of symptoms in addition to the mood disturbance. It includes loss of volition, loss of concentration, loss of memory, loss of enjoyment, and loss of initiative, along with sleep disorder, appetite disturbance, weight loss, disturbance in pain sensation, disturbed sexual ability, among several others. A symptom often overlooked is a testiness and bad-tempered-ness – “bitchiness” – “a short fuse”. Further there are particular types of anxiety often associated with depression, and these include specifically the social anxieties and the phobic anxieties. It can include the learning, memory, and fluency features shown above.

A long established theory of depression maintains that neurotransmitters, which mediate the chemical signaling between nerve cells, are depleted in concentration at the signaling point (the “synapse”). These are mono-amine chemicals, and the aim of treatment is to restore their levels.

Returning to causation, depression can result from “organic” causes – brain tumours, and neuronal disorders and degenerations, to include but a few.

Nonetheless, historically there have been many (probably the majority) of depressions for which no structural disease was evident, and these were considered to be functional. Often they may have been regarded as a reaction to environmental factors. It has been shown that mono-amine deficiency is still exhibited in these cases, making the distinction between organic and functional illness very fuzzy. In the functional case however, it still begs the question of why the monoamine levels have become diminished in the first place.

Of passing interest is the observation that tumours distant from the brain can cause a marked depression, and pancreatic malignancies are strong in this regard. This raises the interesting observation that there must indeed be some communication from periphery to the brain, not completely understood. Hence the old dictum, that if an older patient presents for the first time with a mental illness at a more mature age, they must first be investigated fully for other disease before mental illness.

Recent depression research has implicated other chemicals in the genesis of depression, particularly the stress hormone cortisol and its production pathway and its interaction at neuronal level. It is this observation that gives rise to one hypothesis of causation in this paper.

I make a further observation. In electrical injury there are depressive elements and also elements of PTSD. Yet the syndrome is wider. When expert psychiatric reports are

obtained on the injury for court purposes, one find these diagnosed as entities, along with “adjustment” disorder and the like. A reporter in a particular discipline makes a diagnosis against the criteria currently existing within that discipline, e.g. in psychiatry, the DSM-IV or V. There is in fact more to an electrical injury than the current diagnostic framework allows. Thus diagnoses given are usually “as close as we can get” while remaining true to current criteria. They should not be regarded as final – they imply diagnosis and modes of treatment which are, more often than not, simply symptomatic. There is a great need to expand diagnostic criteria to include a more accurate and complete “post electric shock syndrome”. This writer hopes that future editions of DSM might include this.

C. The HPA Axis

Cortisol is a hormone which controls many body functions. It is a hormone particularly involved in the body stress response. It is produced in the adrenal gland, which is a small gland sitting on the top pole of each kidney. [Figure 1]

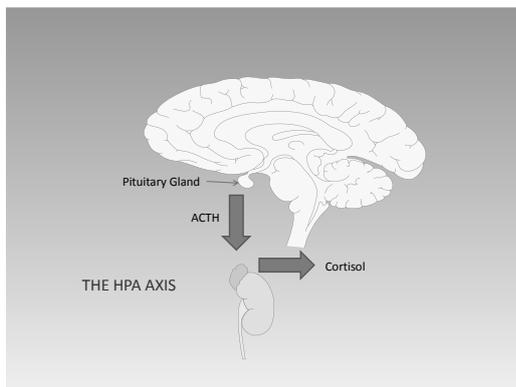


Figure 1. Cortisol is produced by the Adrenal Gland, in reponse of ACTH from the Pituitary Gland. The Pituitary is situated at the base of the brain, and is controlled by other brain centres including the Hypothalamus(Fig 2). The system is referred to as the Hypothalamic-Pituitary-Adrenal (HPA) Axis.

The adrenal production of cortisol is stimulated by a hormone ACTH (adreno-cortico-trophic-hormone) which is released by the pituitary gland at the base of the brain. An increase in ACTH increases the output of cortisol and vice versa.

The pituitary gland produces ACTH on “command” from higher brain centres. The “command” is given by CRH (Cortico-trophin-releasing hormone), which is released from the hypothalamus, an integrative area just above the pituitary gland. “CRH has been localized to the paraventricular nucleus

(PVN) of the hypothalamus, which projects to the median eminence [part of the pituitary] and other hypothalamic and midbrain targets. The CRH gene exists in cellular DNA and therefore there is some genetic element in its expression and production (as is also seen in a family predisposition to depression). Recently, a family of CRH-related peptides, termed the urocortins, has been identified. These peptides probably play a role in integrating multiple aspects of the stress-response, although their functions are largely unknown”.[23]

Signalling for the production of cortisol commences from areas having an influence on the hypothalamus (including the area termed the limbic system, of which one component is the hippocampus, and the hippocampus will figure in later discussion). CRH is released in the hypothalamus, and it acts on the pituitary which produces ACTH, which in turn acts on the adrenal to produce cortisol. Cortisol is thus highly inducible by stress. The level of cortisol is partly controlled by negative feedback to various parts of this axis.

D. The action of BDNF

BDNF (Brain Derived Neurotropic Factor) is an important chemical in maintaining cell growth and connections in the hippocampus especially. It is a protein produced from synthetic processes which transcribe the BDNF gene in cells supporting neurons in the brain. It’s physiology is complex and not yet well understood. [24].

Concurrent levels in normal subjects indicate that BDNF and cortisol adopt the same circadian rhythm, positively correlated[25].

E. The hippocampus hypothalamus and limbic system (Figures 2,3)

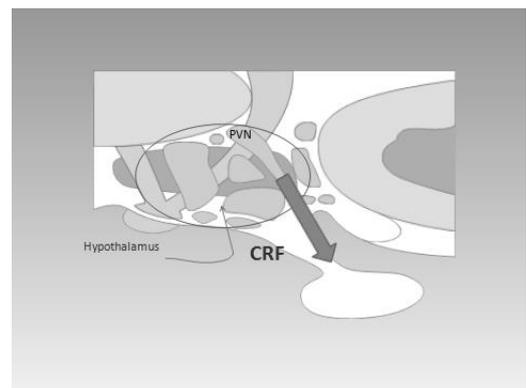


Figure 2. The Pituitary Gland secretes ACTH in response to the secretion of Corticotrophin Releasing Factor (CRF) which is released by the integration of higher signals from the brain (Fig 3).

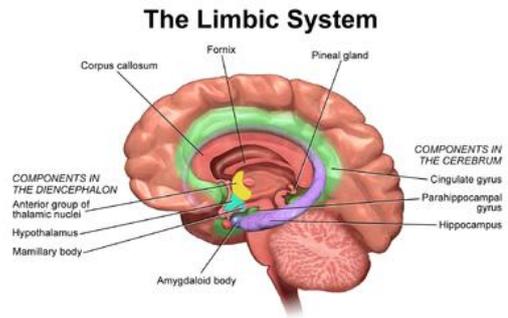
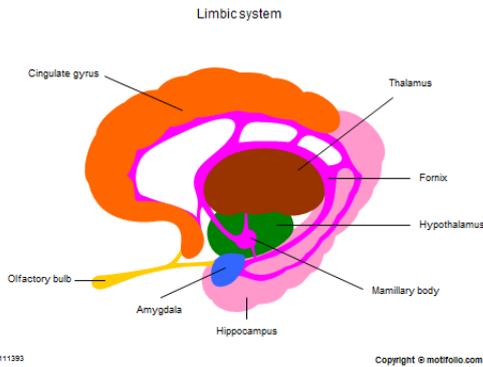


Figure 3. The hippocampus is directly connected to the limbic system, which includes the Amydala, Mamillary Bodies, Olfactory Bulbs and internal structures of the brain. Integrated signals from these structures are used to control CRF secretion.

Although the exact inputs to the hypothalamus are not known, these neurons are largely controlled by serotonin-mediated input from the amygdala and hippocampus of the limbic system.[23] It is interesting to note, again in passing, that serotonin is one of the key mono-amines depleted in depression. The restoration of serotonin levels is the function of one set of anti-depressant medications.

It is important to note that the limbic system is a system of the brain, which includes the hippocampus and the amygdala, and is strongly involved in emotional responses, as well as memory and learning. Damage to these areas will therefore affect these functions.

A good description of limbic system function is found in the following:

Those who research clinical depression have been interested in a particular part of the brain called the limbic system. This is the area of the brain that regulates activities such as emotions, physical and sexual drives, and the stress response. There are various structures of the limbic system that are of particular importance. The hypothalamus is a small structure located at the base of the brain. It is responsible for many basic functions such as body temperature, sleep, appetite, sexual drive, stress reaction, and the regulation of other activities. The hypothalamus also controls the function of the pituitary gland which in turn regulates key hormones. Other structures within the limbic system that are associated with emotional reaction [and also memory and learning] are the amygdala and hippocampus. [26]

A little neuroanatomy may make this clearer. [Figures 4 5 6]

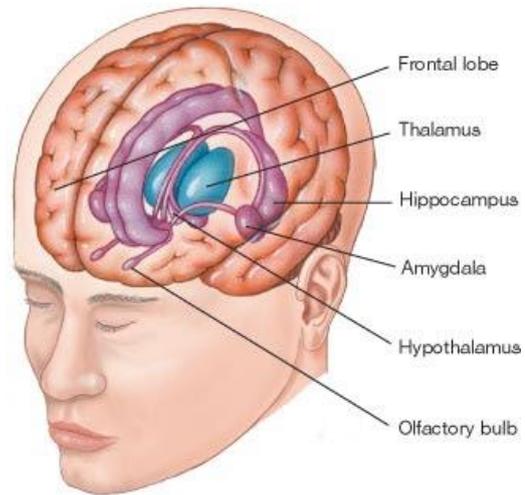


Figure 4. Limbic system.

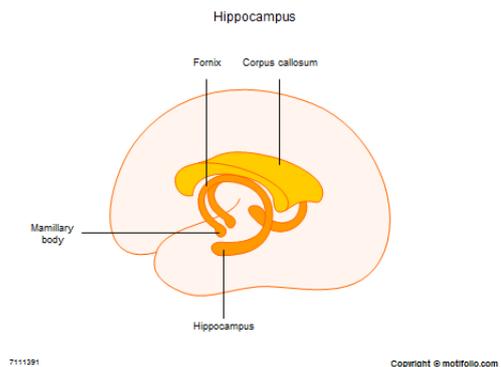


Figure 5. The Hippocampus

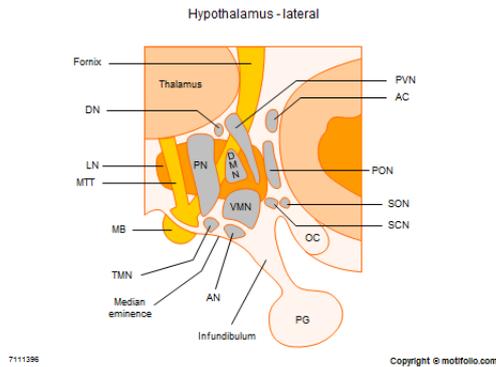


Figure 6. The Hypothalamus and its connection to the pituitary gland via the infundibulum (abbreviations in text).

The limbic system is one of the systems of the brain responsible for particular functions. It is a complex set of brain structures [Fig 4] that lies on both sides of the thalamus, right under the cerebrum. It is not a separate system, but a collection of including the olfactory bulbs, hippocampus, amygdala, anterior thalamic nuclei, fornix, columns of fornix, mammillary body, cingulate gyrus, parahippocampal gyrus, limbic cortex, and limbic midbrain areas.

The limbic system supports a variety of functions, including emotion, behaviour, motivation, long-term memory, and olfaction. It appears to be primarily responsible for emotional life, and it has a great deal to do with the formation of memories. It is a fairly primitive region, and instils these functions in an instinctual way.

The prefrontal cortex is intimately connected to the limbic system, and is where “the primitive brain” meets the “higher brain”. Thus higher control can be exercised on primitive emotions.

The hippocampus may be seen in Figure 5. It is involved in cognition (spatial memory, and learning) and memory. It is very vulnerable to circulatory and toxic insult. It connects closely to the limbic system.

The hypothalamus is situated centrally. It is a well-connected area, integrating the outputs of the limbic system and hypothalamus. Its direct output is twofold – firstly via the autonomic nervous system and secondly via the pituitary gland.

Thus emotional outbursts, stress, insult, emotional responses, can be mediated outwardly via hormonal output and autonomic nervous output.

The consequences of ELI can therefore be appreciated, and foreshadowing later sections, ELI can specifically damage the hippocampus leading to the cerebral dysfunction seen, as well as the ongoing initiation and perpetuation of emotional responses.

F. Depression in ELI

The author proceeds from the point that depression in ELI is organic in origin. That is, there is some mechanism for the

injury to affect the brain to produce the dysfunction seen. The author is swayed by reports from neuropsychologists who opine that the symptoms that they say are similar to those seen in organic injury. Further the author is also swayed by the commonality between some hundreds of victims of ELI that he has seen without any chance of collusion between them.

There will always be a debate however given that there are those who will say that the depression seen in ELI is purely functional and by implication reactive. Indeed they will say that the symptoms claimed above can be seen in any depression whether caused by something deriving from the injury itself, or simply as a reaction to it.

The psychoanalyst might say that the common feature of all depression is loss of some kind. If this is accepted as true, however, a victim of ELI has every reason to appreciate loss keenly, if only from the marked persistent physical symptoms they suffer. In this context loss of function is profound, and there is every reason for depression of a functional kind.

In the end it is of little matter. Even in functional depression chemical changes within the body may be seen, whatever they are ultimately discovered to be.

And in particular, the ongoing physical disturbance will constitute a distress of the highest level, and result in the chronic production of stress hormones like cortisol, which is important in the hypothesis below.

IV. CAUSATION IN REMOTE INJURIES

After the text edit has been completed, the paper is ready for the template. Duplicate the template file by using the Save As command, and use the naming convention prescribed by your conference for the name of your paper. In this newly created file, highlight all of the contents and import your prepared text file. You are now ready to style your paper; use the scroll down window on the left of the MS Word Formatting toolbar.

A. Authors and Affiliations

Two major theories of causation will be mentioned.

a) *Oxidative Radicals, and the hippocampus (Reisner, A.D).*

b) *Hippocampal Theories (Andrews, C)*

I outline these in the following. In this paper I apologise for dwelling perhaps at excessive length on the second, however Dr Reisner’s theories do not preclude my own, and I have attempted to take my own previous theory of cortisol related causation via BDNF, and incorporate Dr Reisner’s postulates.

1) *Oxidative Radicals, and the hippocampus*

Reisner has speculated on causation and also emphasises the hippocampus.

He presented a case of a patient with delayed onset of symptoms[9]. In considering causation, he draws a parallel with memory loss after ECT, and also notes the sensitivity of the hypothalamus to electrical injury. This writer considers a combination of theories may ultimately be explanatory

including that of hippocampal atrophy. He states, and I quote his excellent summary at length:

The hippocampus, an area of the brain associated with memory, is quite sensitive to neurological damage or dysfunction from a variety of sources [1]. Electrical over-stimulation to the hippocampus [2], prolonged seizures [3], unremitting stress [4] and PTSD [5] may all cause damage or dysfunction in the hippocampus and, thus, impair memory. This damage or dysfunction may result from over-stimulation of glutamate (an excitatory amino acid, EAA) receptors in the hippocampus and may be partially mediated by stress-induced glucocorticoid hormones [6]. Glucocorticoids increase the length of time that glutamate receptor channels are open, thus creating vulnerability when over-stimulation occurs [7]. van Zomeren et al. [8] suggest that autonomic dysregulation after lightning injury may account for certain vegetative symptoms. Although they do not specifically mention glucocorticoids, it seems likely that the autonomic nervous system would be partially responsible for increased levels of stress-related hormones after lightning injury. Electroconvulsive therapy may create memory problems when the resulting seizure overstimulates glutamate receptors in the hippocampus [16] and, thus, impairs Long Term Potentiation (a model of learning and memory) [9]. Direct electrical kindling stimulation to the hippocampus has been shown to produce neuronal loss [10], but seizures produced in an animal model of ECT have not produced neuronal loss [11]. Lightning injury may create perfect conditions for memory impairment via delivering electrical over-stimulation to the brain (possibly including glutamate receptors in the hippocampus) along with concomitant glucocorticoid-inducing traumatic stress. The delayed-onset of some cognitive symptoms in lightning injury cases may, thus, be partially mediated by delayed-onset PTSD, a solidly established psychiatric disorder [12].

In addition to having immediate destructive effects, over-stimulation of glutamate (or other EAA) receptors may lead to oxidative stress and subsequent cumulative neuronal damage or loss [13] in the hippocampus [14] and elsewhere in the brain [22]. Oxidative stress produces free radicals which can damage neurons [15]. Cumulative effects of EAA-related oxidative stress may account for apparent delayed effects in some neurodegenerative processes [16]. In fact, independent of the lightning injury literature, oxidative stress related to glutamatergic over-stimulation has been suspected as being involved in Parkinson's disease [17] and amyotrophic lateral sclerosis (ALS) [18], two of the delayed neurodegenerative disorders occasionally seen 'days to months' after lightning injury [19]. Cumulative effects of excitatory amino acid-related oxidative stress should be given consideration as possible contributing factors when neurodegenerative diseases follow lightning injury, as well as in instances of delayed or progressive cognitive dysfunction.

[I have removed Dr Reisner's reference for sake of confusion and warmly recommend reading these from his referenced paper.]

Reisner also discussed theories of causation in a second work [27]. In that exposition, he not only emphasizes the

cortisol and free radical hypersecretion, but also the consequences of glutamate hypersecretion.

2) *Hippocampal theory*

The second theory also focuses on the hippocampus, but approaches its function from a different perspective. Recent insights in depression research motivate this theory.

Strong evidence has been advanced that there is cellular destruction in the hippocampus as a result of depression, remembering incidentally that the hippocampus is strongly responsible for the learning and memory deficits associated with ELI. MRI evidence strongly supports the loss of volume of the hippocampus in depression, in the presence of otherwise normal brain size.

Patients with depression had a statistically significant 19% smaller left hippocampal volume than comparison subjects, without smaller volumes of comparison regions (amygdala, caudate, frontal lobe, and temporal lobe) or whole brain volume.[28]

The loss of hippocampal volume is thought to be a consequence of depression rather than a precursor of it[29]. A meta-analysis of studies confirms the loss[30]. In addition, there is suggestion that the atrophy is proportional to the length of the depression and may not be reversible[29]. It is also known cortisol is cytotoxic and that the atrophy is caused by cortisol toxicity[28, 29, 31]. Truly it may be said that untreated depression causes brain damage.

Stress-induced structural remodelling in the adult hippocampus, involving debranching and shortening of dendrites and suppression of neurogenesis, provides a cellular basis for understanding the impairment of neural plasticity in the human hippocampus in depressive illness. [32] It was particularly noted that production of granule precursor cells was decreased, and this occurred after an initial application of only 7 days of stress.

Plasticity refers to the ability of the brain to generate and repair itself in a plastic rather than fixed way. The link suggested is that BDNF is needed for nerve cell regeneration and repair, i.e plasticity, and plasticity is the key loss in depression. In the normal person, cortisol is accompanied by a rise in BDNF promoting plastic repair, but not in the abnormal, discussed below.

When the consequences of depression were investigated post-depressives scored lower in verbal memory, a neuropsychological measure of hippocampal function, suggesting that the volume loss was related to an aspect of cognitive functioning[31].

This finding of decreased verbal function – memory, fluency and learning in ELI – is just what documentation of the injury indicates, along with a broader cognitive deficit also seen. This deficit is said to be the case in 50% of depressed patients, and of these those with hippocampal atrophy were especially likely to suffer from a subtype of depression with the deficits seen above and proportional to cases where cortisol oversecretion was most marked[29, 31].

The hippocampal atrophy seen is thought to be a result of either inhibition of nerve cell growth or toxic destruction of nerve cells[33]. The loss is also seen in Cushing's Syndrome, where there is an over secretion of cortisol.

Thus excess of cortisol (and this is unquestionably the case in depression resulting from stress) appears to damage neurons in the hippocampus, negatively affecting long-term potentiation of neurons, and showing as memory, cognition, and learning deficits[34].

Further, the implication of cortisol in depression and the loss of hippocampal volume seems undoubted, and this is a "new" proposal in the theory of depression.

The question arises that if cortisol destroys nerve cells, why does the brain's plastic protectivity "rescue" it using a mechanism involving BDNF in the normal person but not in the depressed. (It has also been shown in double-blind studies that anti-cortisol agents can act as antidepressants if the patient is diagnosed as one showing high cortisol [35]).

So the question now can be restated as whether or not stress inhibits neurogenesis of the hippocampus.

Studies show that long term potentiation of hippocampal cells which enhances the memory and learning referred to above, are interfered with by stress, and infusion of BDNF is protective. [36]

BDNF infusion gives significant protection against both brain tissue losses and spatial cognitive impairments. These findings indicate that stress is associated with cognitive deficits, and that BDNF pretreatment is protective against both cellular loss and spatial/memory impairment.[37][38]

While BDNF production parallels cortisol production in the normal person it is interesting that its production is empirically decreased by stress, and indeed stress, in reducing BDNF, deprives the brain of its plastic ability to adapt to stress[24]. That is, it is a key feature of the link between BDNF and depression that there is a lack of parallel relationship between BDNF and cortisol, and a marked negative proportionality (increased cortisol and decreased BDNF) which alters the balance between the two negatively.

But only in some. It is interesting that only some electrically injured patients will show the long term problems, while others simply seem to "brush it off".

In attempting to explain this variability, it is known that there is a genetic polymorphism of BDNF. One form of BDNF seems to be highly correlated with the production of stress related depression with the alternate is protective. The "harmful" form is associated with dysregulation of the HPA axis and the decoupling of cortisol and BDNF thus depriving the brain of its plasticity[39, 40]. The polymorphism has been investigated. There is a gender difference and also an individual difference within sexes of the presence of each polymorphic form. Thus there is a difference in predisposition to depression from the same stress dependent on the polymorphism of BDNF[41].

One writer has proposed a difference in stress response between the amygdala and the hippocampus[42]

V. CONCLUSION - AN HYPOTHESIS FOR ELI

The author collects these disparate findings together to form a hypothesis for the production of the remote symptoms in ELI.

It is entirely reasonable to consider ELI a highly stressful event and thus it is entirely reasonable that it is productive of an intense cortisol response. That response is maintained by ongoing pain and dysfunction over some weeks, remembering that the experimental observations above were generated in animals subject to stress for as little as seven days.

In a short time, excess cortisol has a cytotoxic effect in the hippocampus and this direct cytotoxic effect is highlighted by Reisner. Production of oxidative radicals perpetuating this can also be incorporated, The consequence is further dysregulation of the HPA axis maintaining the state, along with the cytotoxic action on the vulnerable hippocampus.

The further consequence is hippocampal atrophy, remembering that the hippocampus is strongly integrated into the learning and memory deficits associated with electrical injury.

There is a delay in onset of the neuropsychological picture, and this theory only partly accounts for that fact.

One enigmatic feature is that only some people, even if subject to roughly the same stress, will demonstrate the Post Electric Shock Syndrome. This may be accounted for by the observation of the polymorphism of the BDNF gene, and the fact that there is a predisposition to the syndrome in only some who have the dysmorphism.

The disorder, once commenced is self-perpetuating, and experience with the injury indicated that the disability lasts for a long period, and rarely if ever completely resolves. This is commensurate with the view of some that the atrophy and cellular loss is never completely reversible.

These observations lead the author to a firmer basis for experiments to test the hypothesis and for the rational use of antidepressants, and also more novel agents mentioned in the relevant literature. It may be that BDNF assay and/or infusion may be worthwhile avenues, and possibly anti-cortisol agents might also be trialled.

VI. SUPPORT

The author admits that these hypotheses are quite speculative. However there is an interesting avenue of support in study of Kurtulus et al.[43]. In that study the authors subjected rats to electric shock with household electricity. There was found to be a statistically significant loss of neurones in the hippocampus of their rats on day 3 post shock.

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