

# The Origin of Remote Symptoms in Electrical and Lightning Injuries: An Attempt at Explanation and a Hypothesis for Testing

Christopher J. Andrews\*

7 Morningview St., Chapel Hill, QLD A069, Australia

**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.

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

**Keywords:** ELI remote symptoms, ELI psychological hypothesis, Cortisol, BDNF, ELI psychological syndrome, Hippocampus.

## 1. INTRODUCTION

Lightning and Electric Injuries share one feature in common, and this feature is often a stumbling block to those who do not understand the injuries. 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. But there are a significant group of symptoms that are psychological in nature, and these cannot be attributed to current passage in all but the rarest case. Indeed the writer sees many injured people, and reads “expert” opinions on their condition, which state that their 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 that injury is confined to the line of the current only.

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.

The author regards it as “the holy grail” of electrical and lightning injuries (“ELI”) to discover the mechanism underlying these symptoms – often termed “remote symptoms” – in order to understand and treat them. Indeed 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 author would prefer to see the totality of the injury referred to as a “Post Electric Shock Syndrome”.

## 2. REMOTE INJURIES

The injuries that are seen as the remote group of injuries are set out below [12].

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, and anxiety and depression.

\*Address correspondence to this author at the 7 Morning View St., Chapel Hill, QLD A069, Australia; Tel: + 617 3878 5594; Fax: + 61738782842; E-mail: and.c@pobox.com

Often neuropsychological testing gave rise to the comment, that these deficits were more often seen in “organic” rather than “functional” conditions. 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.

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]).

Electrical and lightning injuries produce physical symptoms of Pain, Fatiguability, and Weakness, as well as the remote symptoms above. The incidence of these was incidentally found to be:

Pain	71%
Fatiguability and Weakness	7%/4%
Headache	50%

and others like clumsiness and incoordination, sensory disturbance, and autonomic disturbances. These are disabling for a long period after the injury also.

The aim of this paper is to setup a plausible hypothesis for how remote injuries may occur. It is fully admitted that this hypothesis is speculative and has not been tested. Nonetheless it is not without some potential support.

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

1. A neurohumoural mechanism, where the injury causes peripheral release of some neuroactive substance, which in turn acts on the brain.
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. and perhaps others.

The author wishes to explore the first of these, and suggests that, in fact, we do not necessarily have to search for a hitherto unknown chemical mediator. Insights in other areas, especially in neuropsychiatry and modern views of the causation of depression, may assist in the formulation.

### 3. TERMINOLOGY AND UNDERLYING CONCEPTS

#### 3.1. 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.

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.

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.

In Electrical Injury and Lightning Injury, (ELI), these are usually normal.

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 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.

It is not surprising then that gross investigations in ELI are normal.

### 3.2. Depression

Depression is a disorder which, in the older view, we could determine as being either organic or functional depending on perceived causation.

Depression is not just “feeling blue”. 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 the hypothesis in this paper.

### 3.3. 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.

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 the hippocampus, which is a discrete region of the brain. The “command” is given by CRH (Cortico-trophin-releasing hormone), which is released from the hypothalamus. “CRH has been localized to the paraventricular nucleus (PVN) of the hypothalamus, which projects to the median eminence 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” [14].

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). 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.

### 3.4. 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 [15].

Concurrent levels in normal subjects indicate that BDNF and cortisol adopt the same circadian rhythm, positively correlated [16].

### 3.5. The Hippocampus Hypothalamus and Limbic System

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 [14]. 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 [17].

### 3.6. 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.

## 4. RECENT DEPRESSION RESEARCH

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 [18].

The loss of hippocampal volume is thought to be a consequence of depression rather than a precursor to it [19]. A meta-analysis of studies confirms the loss [20]. In addition, there is suggestion that the atrophy is proportional to the length of the depression and may not be reversible [19]. It is also known cortisol is cytotoxic and that the atrophy is caused by cortisol toxicity [18, 19, 21] (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 [22]. 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 [21].

This finding of decreased verbal function – memory, fluency and learning in LEI – 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 [19, 21].

The hippocampal atrophy seen is thought to be a result of either inhibition of nerve cell growth or toxic destruction of nerve cells [23]. 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 [24].

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 by mechanism of BDNF (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 [25]).

So the question now becomes whether or not stress inhibits neurogenesis of the hippocampus.

Studies show that long term potentiation (LTP) of hippocampal cells which enhances the memory and learning referred to above, are interfered with by stress, and infusion of BDNF is protective [26].

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 [27, 28].

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 [15]. 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.

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 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 [29, 30]. 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 dependant on the polymorphism of BDNF [31].

One writer has proposed a difference in stress response between the amygdala and the hippocampus [32].

#### 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. The consequence is further dysregulation of the HPA axis maintaining the state.

The further consequence is hippocampal atrophy, remembering that the hippocampus is strongly responsible for the learning and memory deficits associated with ELI.

There is a delay in onset of the neuropsychological picture, and this theory 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 in ELI and for the rational

use of antidepressants, and also more novel agents mentioned in the relevant literature.

The author admits that this hypothesis is quite speculative, but draws some support from the study of Kurtulus *et al.* [33]. In that study the authors subjected rats to electric shock with household electricity and found statistically significant loss of neurones in the hippocampus of their rats on day 3 post shock.

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#### CONFLICT OF INTEREST

Declared none.

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