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INVITED REVIEW

Neurological and neuropsychological consequences of electrical and lightning shock: review and theories of causation

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Abstract
Injuries from lightning and electrical injuries involve multiple systems of the body, however neurological symptoms are very widely reported. A disabling neuropsychological syndrome is also noted. This paper presents a comprehensive review of neurological and neuropsychological symptoms. Partial theories of causation for these injuries have been advanced, however, there is no convincing explanation for both delay in onset of symptoms and also the genesis of the neuropsychological syndrome. A theory of causation is proposed which satisfies both these constraints. This theory suggests circulating hormones such as cortisol, together with nitric oxide and oxidant free radicals from glutamatergic hyper-stimulation, act on tissues remote from the injury path including the hippocampus. This theory opens a research path to explore treatment options.

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Introduction
Electrical and lightning injuries (ELI) have multiple consequences. The majority of these are identified as neurological damage. While neurological symptoms may be prominent, some suggest this indicates preferential damage to the nervous system. There is no doubt that the nervous system may be directly damaged in some circumstances, however, it is debated whether this represents primary injury to nervous tissue, as opposed to secondary damage. Nonetheless, neurological symptoms result. These can be viewed as either or both of neurological consequences, and neurophysiological consequences.

Further, a proportion of victims also demonstrate substantial neuropsychological symptoms. It has been argued that these are indeed the most debilitating aspects of the ongoing disability (Cooper, 2001; Cooper and Andrews, 2005). Once again, debate regarding the cause of these problems exists, with one extreme suggesting they represent malingering, others suggesting that if electric current has not traversed the brain then there cannot be psychological injury. Others question whether organic brain changes exist to explain these symptoms, and still others whether they are functional in origin.

Shock from lightning, and shock from electric current, give rise to different types of injury. The major differences are demonstrated in the nature and extent of burns, and in the presence of injuries by some ancillary mechanisms, for example blast, and also in cardiac and respiratory consequences. Other differences exist, however there remain items in common. In particular, the physical neurological symptoms are reported similarly, and the neuropsychological symptoms are held largely in common. This article examines the latter two areas.

Any explanations for the symptoms must address several points:
- The reason for the actual existence of the symptoms, and their localisation;
- The fact that some symptoms seem to involve loci displaced from any overt immediate current passage;
- The fact that there can be substantial delay in onset of symptomatology from the initial insult.

Most authors document the symptoms, and then in attempting to ascribe causation, describe pathological tissue change, without considering how those in turn come about. Tissue change has been observed for some time, although observational techniques have improved. The tissue changes described are relatively "high level" changes, and little has been advanced to explain the deeper causes giving rise to these changes. Causation of neuropsychological changes remains an active subject of research.

This paper provides a review of the described neurological symptoms, both in physical neurology, and also in neuropsychological syndromes. The symptoms and signs of ELI

are reported very much in isolation. Such isolation does not lead to easy identification of one underlying causative mechanism. This paper documents the symptoms and signs as an empirical description of the injury, and from this causation is considered. Present theories of causation are given, with some examination of treatment possibilities, particularly those which might arise from the symptoms and the theories of causation.

Physical Neurological Symptoms

Physical symptoms have been commonly reported, and a classification of these has been advanced. This classification is predicated on the time course of symptoms. Cherington's classification is given in the following using his own examples (Cherington, 1995, 2003):

1. Immediate and transient symptoms (*e.g.*, kearaunoparalysis, lichtenberg figures)
2. Immediate and prolonged symptoms (*e.g.*, hypoxic encephalopathy, intracranial (IC) bleeding, cerebral infarction (especially parieto-occipital), cerebellar syndromes, spinal cord injury (some of which Cherington notes to be permanent), chronic epilepsy, peripheral nerve lesions (*e.g.*, neuropathy, myopathy, neuromuscular junction disorders, polyneuropathy)).
3. Delayed neurological syndromes. Motor neuron disease following days, months, and even years.
4. Secondary symptoms (perhaps due to blast). IC haemorrhage, barotrauma, ruptured tympanic membrane.

We have framed the injuries in an alternate anatomic classification and will discuss them under those headings. The following anatomic structure is advanced.

1. Central syndromes
   a. Cerebral haemorrhage - those consequences following bleeding within the cerebral structures
   b. Cerebral infarction
   c. Central encephalopathy, including hypoxic encephalopathy, that is, alteration in neuronal function due to loss or diminution of oxygen supply
   d. Convulsions
   e. Movement disorders, including Parkinsonism and other disorders of normal movement derived from central causes, whether added movements, modulation of normal movement, attenuation of normal movement, or loss of control of normal movement
2. Cerebellar syndromes
3. Cranial nerve and related disorders
   a. Specific cranial nerve involvement, including speech and articulation disorders
   b. Ocular neurology
   c. Cochleo-vestibular neural disorders
   d. Synaesthesia
4. Autonomic syndromes
   a. Complex regional pain syndromes (CRPS) including Causalgia and reflex sympathetic dystrophy (RSD)
   b. Postural hypotension – and other disturbances of autonomic function
   c. Cerebral salt wasting – this may be endocrinological in origin, however also mediated by neural influence on the hypothalamus and pituitary gland.
5. Spinal cord disorders
   a. Transverse myelopathy
   b. Amyotrophic lateral sclerosis (ALS), and other motor neurone disorders - the loss of function of neural pathways controlling movement, in this case involving specific neurones in the anterior horn of the spinal cord. The generic "motor neurone disorder" includes more specific disorders such as ALS
   c. Spinal origin dystonia
6. Paralyses
   a. Keraunoparalysis
   b. Paraplegia and quadriplegia
7. Peripheral nerve disorders
   a. Specific peripheral nerve involvement
   b. Myopathy
   c. Myoclonus
   d. Weakness syndrome and fatiguability
8. Sensory abnormalities

   We now discuss the reported syndromes under the anatomical classification above.

Central syndromes

Multiple cerebral syndromes have been reported. Cerebral haemorrhage of almost all variations appear (Morgan et al., 1958; Mann et al., 1983; Stanley and Suss, 1985; Wakasugi and Masui, 1986; Thomas et al., 1991; Cherington et al., 1993, 1997; Steinbaum et al., 1994; Ozgun and Castillo, 1995; Ohashi et al., 2001; Knaggs, 2002; Cherington, 2003; Caksen, 2004; Carrera-Izquierdo et al., 2004; Guardiola et al., 2010). Many are accompanied by skull fracture, and reports indicating some chronicity, plus associations with other consequent neurological signs exist. In particular, the basal ganglia seem prone to injury, and basal ganglion symptoms are particularly reported. Cerebral Infarction is only briefly mentioned, and naturally considered to be vascular in origin (Huan-Jui et al., 2010).

As the genesis of many cerebral pathologies is postulated to be vascular, ischemic encephalopathy is expected and encephalopathic syndromes include confusion and amnesia, seizures, hallucinations, dysphasias, and the appearance of diffuse white matter changes, are documented (Paterson and et al., 1944; Poser, 1994; Cherington, 1995; Milton et al., 1996; Courtman et al., 2003; Isao et al., 2005; Kleiter et al., 2007; Bryan, 2009; Kruja et al., 2016). Various long term findings questionably related to ELI, though only isolated cases are presented. These include, for example, glioma, and cerebral atrophy. Bryan (2009) postulates mechanisms – current effects, thermal burns, and mechanical trauma, as well as hypoxia.

Movement disorders are reported (O'Brien, 1995; Morris et al., 1998; Quinn, 2000; Cherington, 2003), and differentiation between these as central syndromes, versus peripheral movement disorders is emphasised. O'Brien (1995) particularly itemises Parkinsonism, choreoathetosis, dystonia,

myoclonus, and cerebral tremor, under the heading of movement disorders. He considers basal ganglion ischemia, and cerebral hypoxia and anoxia to be key mechanisms. Jankovic and Pardo (1986) reports the electrical origin of myoclonus, and also demyelination, in some of the 18 patients in his series, and claims a surprising latency in some of up to 33 years.

Motor neurone disease (MND) is widely reported, and largely as a delayed onset syndrome (Gallagher and Talbert, 1991; Ghosh et al., 1995b; Jafari et al., 2001; Cherington, 2003; Zoccolella et al., 2008). A similarity with ALS is mentioned. Delayed onset and slow progression seem to mark these disorders. Some writers even report years of delay before the onset of many syndromes. The commonality between the generic MND and more specific ALS is shown by the involvement in both cases of the final motor neurones in the movement pathway.

Cerebellar syndromes

There are only a small number of reports of cerebellar syndromes (Suri and Vijayan, 1978; Gilbert, 1994; Cherington, 2003). Cherington itemises four cases, three of which resolved, and one of which was a case of cerebellar atrophy (Cherington et al., 1993). Gilbert (1994) presented a case where previous rapid and convoluted speech became "robotic", and suggested this was cerebellar in origin. Cherington, in a reply, questioned the cerebellar origin of this. Suri identified cerebellar signs in a soldier struck when using a telephone. Scholars (Andrews, 1993, 1995; Andrews and Darveniza, 1994) have paid attention to cranial orifices as portals of entry to the body, and access to the cerebellum would seem consistent with this.

Cranial nerve syndromes

Many cranial nerve lesions have been individually reported as due to ELI, and in particular ocular nerves. Baqain (Baqain et al., 2004) reports multiple cranial nerve effects, especially leading to diplopia as well as abnormal lingual sensation. Facial Palsy, and Bulbar palsy leading to dysphagia have been presented (Richards, 1973; Silbergleit and Trenkner, 1988; Saddler and Thomas, 1990). Articulation disturbances implicate other cranial nerves (Caksen, 2004). Caksen reports this phenomenon in conjunction with thalamic haemorrhage. Speech impediments are also reported (Isao et al., 2005; Desai and Fairclough, 2011).

Ocular pathology is noted in its own right, with mydriasis, anisocoria, Horner's syndrome, failure of accommodation, and long term cataract generation, along with the individual nerve pareses (Lea, 1920; Abt, 1985; Graber et al., 1996). Norman and Younge (1999) reports a case of visual loss responding to high dose methylprednisolone. The unreliability of pupil response in assessing resuscitation response has been highlighted.

Cochleovestibular disorders are reported, including in the animal literature (Poulsen and Knudstrup, 1986; Bedenice et al., 2001; Modayil et al., 2013) with tinnitus, decreased hearing, and dizziness seen as symptoms.

One isolated report of synaesthesia in two patients has been made (Farina et al., 2016).

Autonomic syndromes

Three categories of autonomic dysfunction have taken attention. Firstly RSD and Causalgia, as previously known, have been reported along with several reports of a CRPS (Rosenberg, 1989; Belsole and Smith, 1990; Shantha, 1991; Demun et al., 1993; Hendler, 1994; Kim, 2001; Jost et al., 2005; Grubb and Karabin, 2007).

Interesting cases of electrolyte disturbance and a salt-wasting syndrome have been reported, and the origin of these has been advanced as hypothalamic and physeal (Weeramanthri et al., 1991; Hawkes and Thorpe, 1992; Lim et al., 2001; Ozdemir, 2002; Emet et al., 2010; Orbak and Kara, 2010) though these may also be endocrine in origin.

Persistent hypertension in two cases was thought to be central in origin (Suri and Vijayan, 1978; Weeramanthri et al., 1991).

Spinal cord disorders

It is in this area that much interest has been generated. The possibility of ALS and related demyelination, both immediate and delayed, has been widely presented. The relation between ALS and MND has been stated above. Related paralyses are reported separately below. Causation is intuitively easier to ascribe to the direct action of electric current at cord level, though we suggest that this is an explanation that needs further detailed exposition.

ALS and transverse myelopathy are explicitly reported along with related demyelination syndromes. Delay is highlighted prominently (Butler and Grant, 1977; Christensen et al., 1980; Davidson and Deck, 1988; Koller, 1989; Clouston, 1995; Arévalo, 1999; Deveci, 2001; Bariar, 2002; Cherington, 2003; Ko, 2004; Abhinav et al., 2007; Erkin, 2007; Lakshminarayanan et al., 2009).

Other related syndromes at spinal level have been presented – corticospinal pareses (Johansen, 2008), Anterior horn myelopathy (Fu, 2008), and dystonias specifically lingual and peripheral (Adler and Caviness, 1997; Ondo, 1997). MRI findings have been used to support the diagnoses (Freeman et al., 2004).

Paralyses

For an area providing so many reports, the previous section seems small, however consideration of paralyses is taken as a separate section, and this is largely since one syndrome unique to lightning injury, keraunoparalysis, is of particular interest.

Keraunoparalysis is a transient syndrome, lasting a small number of hours. It is seen in a limb in the line of current passage, and is thought to be due to vascular spasm to the region. The limb becomes cold, blanched, numb, and pulseless, and is confused with a compartment syndrome (ten Duis et al., 1985; Graber et al., 1996; Gouse et al., 2015).

Paraplegia and quadriplegia are well reported (Sharma and Smith, 1978; Varghese, 1986; Gathier, 1988; Kanitkar, 1988;

Hawkes and Thorpe, 1992; Steffen et al., 1992; Clouston, 1995; Breugem et al., 1999; Baqain, 2004; Caksen, 2004; Ko, 2004; Fu, 2008; Gouse et al., 2015). All forms are represented – flaccid, spastic; transient, long-lasting; immediate, delayed; paraplegia, quadriplegia; unilateral, bilateral.

Peripheral nerve disorders

Individual peripheral nerves may be affected, and in combinations. In the arms, various combinations of median, ulnar, and radial nerve involvement are reported (Butler and Grant, 1977; Rosenberg, 1989; Engrav, 1990; Clouston, 1995; Cahill et al., 2014; Patnaik et al., 2015). Similar findings in the legs are reported, and involve sciatic and femoral nerves, and more distant branches (Butler and Grant, 1977; Rosenberg, 1989; Engrav, 1990; Clouston, 1995; Wilbourn, 1995; Cherington, 2003; Fan, 2005; Cahill et al., 2014; Patnaik et al., 2015).

In consequence, a Guillian-Barre like syndrome has also been reported (Girard and Jacquot, 1954; Panagiotis et al., 2008).

A syndrome very commonly seen by one author (CJA) is that of muscular weakness especially in the muscles in the line of current passage. Coupled with this is a loss of stamina, and easy fatiguability. Weakness is commonly reported (Butler and Grant, 1977; Steinbaum et al., 1994; Clouston, 1995; Courtman et al., 2003; Baqain, 2004; Fu, 2008; Bhargava et al., 2014), however the fatiguing seems only to attract passing reference. Most often this is attributed to a myopathy. An alternative is suggested below.

Sensory abnormality

Finally, we turn to sensory abnormality, and the sensation most commonly reported is pain, associated with the muscular weakness. CRPS have already been mentioned.

Mechanisms are conjectured, in the light of histological observations of myelin sheath disintegration, and nerve cell swelling.

One author (CJA) is mindful that Nerve Conduction Studies (NCS) in electrical injuries are often normal. Some advance this in the compensation arena as indicating that no injury has occurred. Yet symptoms still are manifest. It is postulated then that the locus of damage may well be outside that examined in NCS, and may well reside in damage to neuromuscular junctions (NMJ), with this also explaining weakness and especially early fatiguing. Similarly, perhaps the sensory dysfunction may be due to sensory nerve terminal damage.

Overall

The spectrum of symptoms which are reported is indeed wide. Ascription of cause has been attempted with these reports, but generally only at a "high" level describing changes that some have seen. There is a need to go beyond this to a lower level and to determine the underlying forces leading to those changes.

Neuropsychological Symptoms

There is a consensus among scholars working in the field of ELI that individuals can experience neuropsychological impairments subsequent to the injury (Hopewell, 1983; Kelley, 1994; Primeau et al., 1995a; Janus and Barrash, 1996; Crews Jr et al., 1997; van Zomeren et al., 1998; Duff and McCaffrey, 2001; Cherington, 2003; Martin et al., 2003; Morse and Morse, 2004, 2005; Morse et al., 2004; Hendler, 2005; Andrews, 2006; Pliskin et al., 2006; Reisner, 2006; Bailey et al., 2008; Chudasama et al., 2010)

In a review (Andrews, 2006) lists problems with memory (71%), concentration (63%), and loss of mental powers (50%), as being some of the neuropsychological symptoms often seen in cases of ELI. Auditory learning and auditory memory seem to be particularly affected. A review by Duff and McCaffrey (2001) indicates that a wide variety of neuropsychological tests have been used in studies, including the Wechsler Memory Scale, Trails A & B, Finger Tapping, Tactual Performance Test, Halstead-Reitan Neuropsychological Battery, Wechsler Adult Intelligence Scale-Revised, Wechsler Memory Scale-Revised, Wide Range Achievement Test-3, Rey Auditory Verbal Learning Test, Seashore Rhythm Test, Speech Sounds Perception Test, Bender Gestalt Test, and Luria Nebraska Neuropsychological Battery. Although these authors view the quality of studies, and the specificity of impairments noted in the studies, to be variable, researchers have found, following ELI, deficits in memory, visuospatial reasoning, motor deficits, "diffuse mild to moderate neurocognitive dysfunction..." (p. 105), decreased visual reaction times, decreased verbal memory, and overall neuropsychological deficits as measured by Impairment Indexes on both the Halstead-Reitan and Luria Nebraska test batteries. Perhaps just as important as neuropsychological test findings are the observations of the first author (CJA) that many victims of ELI lose the ability to run a business, to keep schedules and financial books, to maintain a diary, and other practical daily tasks. Deficits found on tests can translate into real world disabilities.

The findings of neuropsychological deficits as well as certain physical and psychiatric symptoms in individuals experiencing ELI have led a number of researchers to suggest that there is a specific syndrome associated with electrical and lightning and injury (Primeau et al., 1995a, b; Janus and Barrash, 1996; Andrews, 2006). The symptoms of ELI have been viewed as similar to symptoms seen in concussions (Yarnell, 2005), head injuries (Primeau et al., 1995a, b), and whiplash injuries with chronic pain (Yarnell, 2005). It has also been suggested, though, that chronic pain itself, which is often seen in ELI (71% of cases; (Andrews, 2006)), may contribute to the often seen difficulties with attention and concentration (Hendler, 2005). It is often noted that depression, anxiety, irritability, fatigue, and posttraumatic stress disorder (PTSD) are common problems after ELI (Primeau et al., 1995a, b; van Zomeren et al., 1998; Duff and McCaffrey, 2001; Cherington, 2003). It is generally acknowledged that psychological symptoms and cognitive symptoms may interact, with psychological symptoms contributing to cognitive deficits, and frustration and depression over cognitive deficits leading to worsening psychological

symptoms (Primeau et al., 1995a, b; Janus and Barrash, 1996; Reisner, 2006).

Taking into account the physical, psychiatric, and neuropsychological symptoms often seen together in ELI, the first author (CJA) has formulated a potential diagnostic schema for post electric shock syndrome (PESS) and post lightning shock syndrome (PLSS). It is thought that a diagnosis will foster scientific research and inform the medical community of symptoms to evaluate currently, and of delayed symptoms that may appear in cases of lightning and electrical injury. A group of authors writing from the perspective of Emergency Medicine state that it is known that neurological and neuropsychological consequences of ELI can appear "long after their discharge from the ED [Emergency Department]" thus the need to follow-up with patients "in the same manner as with patients treated with head trauma" (Bailey et al., 2008). Moreover, for those who have suffered these distressing and sometimes debilitating symptoms, a specific diagnosis will provide a sort of validation, an assurance to themselves and others that their suffering and impairments are real and should not be minimized or dismissed.

Pliskin and his colleagues (Pliskin et al., 2006) as well as Reisner (Reisner, 2006, 2014) have independently noted similarities in cognitive/memory dysfunctions in ELI and the cognitive/memory dysfunctions seen after electroconvulsive therapy (ECT). It is important to note that even though ECT is a therapeutic procedure with memory deficits as a side effect, and ELI is a destructive event, there can still be some similarities with regard to the mechanisms which bring about memory impairments. It must be considered that in both situations there is an excitatory input – electrical, psychological or both. In the case of ECT, the excitatory electrical input is augmented by a seizure, and in the case of ELI, the electrical input is much stronger. (How the electric current might reach or effect the brain in cases of ELI where the main path of current does not appear to traverse the brain will be addressed in a subsequent section of this paper.) Pliskin and colleagues (Pliskin et al., 2006) write: "We hypothesize that neuropsychological and neuropsychiatric changes in EI [electrical injury] relate to electrochemical alterations in brain systems. By way of comparison, electroconvulsive therapy (ECT) has been studied extensively and we speculate that ECT may serve as a model for understanding the impact of more extreme field strength exposure suffered by electrical injury patients."

The relationship between glutamatergic hyper-stimulation and the role of cortisol is of great relevance in both ELI and ECT-related cognitive dysfunction. Elevated cortisol frequently accompanies stressful events and is thought to have cytotoxic effects of its own, including in the hippocampus, an area associated with memory (Andrews, 2012). Additionally, in the context of ECT, glucocorticoids (of which cortisol is one) may hold glutamate receptors open for longer periods of time and more frequently, thus perhaps augmenting glutamatergic hyper-stimulation (Chamberlin and Tsai, 1998). Reul and Nutt (2008) also suggest a synergistic effect between cortisol and glutamate. An empirical study found that patients who had higher levels of cortisol prior to ECT experienced more memory deficits than did ECT patients who had lower levels of cortisol prior to ECT (Neylan et al., 2001). It stands to reason that individuals traumatized by lightning and electrical injury would have elevated levels of cortisol, given that elevated cortisol levels are associated with stress. Elevated cortisol levels in combination with increased glutamatergic stimulation from lightning and electric injury may have a particularly negative effect on memory.

In ECT, there are well known memory problems that occur as a side effect, and this occurs in the absence of evidence of gross structural change or damage noted in imaging studies (Reisner, 2003). With some exceptions (Reisner, 2013) imaging studies generally do not detect signs of structural brain damage with lightning and electrical injury (Pliskin et al., 2006; Reisner, 2013). In ECT it is thought that hyper-stimulation of glutamate receptors in the hippocampus may interfere with long-term potentiation (LTP), a model of learning (Chamberlin and Tsai, 1998). If such hyper-stimulation is of the intensity as found in ECT, neuronal cell death does not occur; but if the hyper-stimulation is of sufficient intensity, as occurs in certain animal studies, cell death in the hippocampus can occur (Chamberlin and Tsai, 1998; Reisner, 2003). Provided that electricity reaches the brain in lightning and electrical injury, or that otherwise a massive hyper-stimulation occurs affecting the excitatory neurotransmitter, glutamate, one might anticipate disruption in the hippocampus mediated by glutamatergic hyper-stimulation, and thus subsequent memory problems. This comparison of ELI and ECT may help explain the prevalence of memory problems with electrical and lightning injury.

Theories of Causation

The desire to reach fundamental process descriptions of causation has been stated. Generally, in existing reports, there are attempts to ascribe causation. The central syndromes, including cerebellar syndromes, are often assigned to traumatic events, and also to mechanical effects of current flow. Thermal effects are considered for direct neural injury, autonomic dysfunction, and peripheral injuries. Vascular causes of infarction are postulated, with vascular obliteration due to electrical current. Vascular phenomena are similarly promoted especially in the spinal cord lesions. Direct neural degeneration is postulated in cases of Parkinsonism, ALS, and MND. While these mechanisms are attractive in varying degrees, they do not fully account for the phenomena and particularly for the delay in onset often seen. We postulate later alternative mechanisms which address this issue, and also subsume the causation of neuropsychological syndromes under one causation.

Tissue level changes have been reported, and these changes have been confirmed and documented consistently.

Critchley, as long ago as 1934, documented a number of features. These are high-level features, and we highlight the need to explore prior mechanisms giving rise to these changes:

1. Focal petechial haemorrhages – especially in the ce-

rebrum and medulla, and also in the anterior horn grey matter of the spinal cord. He says these are more common after AC shocks, and also large vascular tears are seen after lightning injury.

2. Chromatolysis – especially in pyramidal cells of the medullary nuclei, in anterior horn cells, and the Purkinje cells of the cerebellum. These changes are said to be patchy and sharply demarcated.

3. "Curious" dilatation of perivascular spaces, which he suggests are due to gas release, being seen largely in legal electrocution.

4. Tortuosity and fragmentation of the axons of peripheral nerves with breakdown of Schwann sheaths. Infiltration of the epineurium is seen with endothelial cells.

5. Spiral-like changes in muscle fibres.

6. In severe injury, the "entire brain and parts of the cord may be swollen softened and even diffluent."

The following classes of causative processes are advanced, either in reports, or included in the present paper for completeness:

1. Common symptomatic endpoint.

It is noted that the long term disability seen in ELI resembles head injury cases (van Zomeren et al., 1998). Indeed, there are several injuries where somewhat similar features may be seen. This includes some symptomatology of traumatic brain injury, viral illness, autoimmune disease, and others.

Some authors suggest that what is seen in ELI is actually a syndrome representing the common expression of several insults. As such, it may be that therapeutic modalities for some will inform others.

2. Direct electrical\ damage

Direct damage due to the passage of current through tissues may account for symptoms in the line of current passage. Mechanisms of purely electrical damage include the electrostatic separation of tissues, and also cell death due to electroporation. The latter refers to the action of an electric field on cell membranes to cause the appearance of pores in the membrane (Lee, 1997). Some advance the use of labelled polaxomers to seal pores and map damage.

It is pertinent to dispel certain myths regarding the conduction of electrical current in tissue (Andrews, 2017). Many reports attempting to explain this offer simplistic and unrealistic explanations of current conduction in tissue. The myths involved include (1) "Electricity" flows in a single straight line (alt. path); (2) Damage is seen only in this direct line, and (3) if current does not identifiably flow in a given tissue then there can be no effect on that tissue.

Regarding (1), all tissues form parallel conduction pathways in the body. Current flows in all of these in inverse proportion to the resistance of the pathway. Coupling (2) with this, no tissue is therefore without influence of current flow, however small it may be. A salutary example is to consider current flow hand to hand. The least resistance in this path is offered by blood, largely across the subclavians. A proportion of the current will flow in this pathway. A potential difference will exist between the roots of the carotids and a (possibly small) fraction of the current will flow in one carotid, across the circle of Willis, and *via* the opposite carotid. Thus, there is a fraction that will indeed enter the brain. Hence it is incorrect that direct flow from hand to hand, for example, cannot affect the brain "as it is not in the pathway". This is very elegantly shown in finite element analyses (Seckler, 2016).

3. Thermal damage

The passage of electric current through a resistance, represented by the tissues through which it passes, generates dissipative heat. This is also in direct proportion to the applied potential, and in inverse proportion to the tissues resistance. Thus, it is incorrect to say that tissues of the highest resistance dissipate the most heat.

In any event, tissue heating disrupts tissues, and denatures proteins in the pathway.

4. Tissue separation - by force or by electrostatic effects

The electrostatic separation of tissues has been mentioned, and may explain observations of Critchley, above.

There are also those that suggest generation of gas phases with heating also separate tissues.

5. Ischemic changes

Possibly the theory which is most often mentioned is that the conduction of current *via* the blood vessels (and this contention is correct to the extent that blood has the lowest resistance, along with cerebrospinal fluid (CSF), and other fluids) leads to vascular spasm and ischaemia in the tissues supplied by the given vessel.

While this accounts for the immediate effects of the current, it is extended in one of two ways to explain the delay in onset of symptoms. First, some postulate chronic ischaemia existing after the original injury, which ultimately slowly and progressively damages the tissue it supplies in time. Second, the existence of a chronic inflammatory response is also advanced to achieve the same ends.

6. Circulating neurohumoural actions

The authors have advanced a further theory and will develop it below. It is suggested that this theory, involving released neurohormones, explains both "action at a distance", and also "delayed onset" symptomatology.

These then are the features that must be explained by a cogent theory – that there is immediate damage, that there is delayed onset of symptoms, and that there are symptoms seen involving areas distant from the perceived current passage, in particular effects giving neuropsychiatric symptoms which must involve cerebral tissue and systems.

The mystery of delayed syndromes and an alternative hypothesis

It is easy to grasp intuitively the notion of severe damage to tissues and systems directly following the extreme physical insult of lightning and electrical current. But delayed psychiatric, cognitive, and neurological damage is more difficult to understand and explain. The phenomena which are most dramatic and difficult to explain are neurological disorders that appear months or over one year after a lightning or elec-

trical injury.

In a previous article, the second author (Reisner, 2013) found evidence in the literature for delayed syndromes that include motor neuron disorder (Cherington, 2003), amyotrophic lateral sclerosis, parkinsonism, focal dystonia and tics (Cherington, 2005). Psychiatric symptoms can occur in delayed fashion, such as personality change, poor concentration, emotional lability, forgetfulness (van Zomeren et al., 1998). Cognitive functioning can deteriorate after a delay (Primeau et al., 1995a, b; Martin et al., 2003). Although some are sceptical regarding these delayed syndromes actually being related to the electrical or lightning injury (Muehlberger et al., 2001), the second author (Reisner, 2013) previously noted that in one study, one of several neuropsychological functions that decreased over time after electrical injury was grip strength, something unlikely to decline because of psychological factors alone. Some cases involved delayed spinal cord atrophy (Farrell and Starr, 1968; Low, 1976).

One group of authors suggests that vascular damage from electrical injury leading to chronic ischaemia may contribute to delayed injury; they also suggest that electrical current may damage DNA (Ghosh et al., 1995a). A particularly interesting theory was put forward by Farrell and Starr (1968). They suggested that electrical current alters protein and/or DNA and that this may adversely affect vascular endothelial cells, leading to decreased blood supply to the central nervous system.

A neurohumoural hypothesis

It is known that that blood vessels carry most current. No evidence is left of the current passage, as blood is a self-sealing tissue. Further, generated heated is carried away by the mechanical flow. We postulate that under influence of the current, circulating substances are released, and are carried in the blood stream to act at a distance. One demonstration of this was given by Park and colleagues (Park et al., 2012). Alterations in arterial function was noted in the arteries directly in the line of current passage. However, alteration in arterial function was also noted in arteries where current had "not" flowed – the opposite side of the body.

The authors postulate the action of released chemicals – notably cortisol and free radicals – particularly on areas of the brain, accounting for neuropsychological symptoms. Processes are set up which develop immediately, and also over time. Evidence has been presented suggesting that cortisol augments hyperstimulation of glutamate receptors (Chamberlin and Tsai, 1998; Reul and Nutt, 2008), which in turn augments release of destructive free radicals (Coyle and Puttfarcken, 1993).

In previous articles, the second author (Reisner, 2013, 2014) noted that Farrell and Starr's hypothesis can be bolstered, and added to, *via* consideration of scientific developments involving glutamatergic stimulation leading to the production of free radicals through oxidative stress, and the free radicals contributing to neurodegenerative diseases (Coyle and Puttfarcken, 1993). One study demonstrated that free radicals are elevated after lightning injury (Bailey et al., 2003). Another study found that glutamatergic stimulation can lead to oxidative stress-related free radicals, and that this can cause apoptosis (cell death) in cerebrovascular endothelial cells (Parfenova et al., 2006). It is noted that glutamatergic neurons exist in both brain and spinal cord (Juurlink and Paterson, 1998). Essentially, if electrical current reaches the spinal cord and the vascular tissue feeding it, this may hyper-stimulate glutamatergic neurons leading to free radical formation, which subsequently gradually breaks down endothelial cells in the blood vessels feeding the spinal cord, ultimately leading to thrombosis and cell death in the spinal cord (Reisner, 2013, 2014).

Nitric oxide is known to be secreted from vascular structures, and is a potent vasoactive compound. It has been implicated both as a neurotransmitter and also as a mediator of glutamate excitotoxicity in neuronal structures (Kader et al., 1993). It was particularly released in ischaemic events in rat neuronal tissue. It is also implicated in neuronal death, and thus in neurodegenerative diseases (Boje et al., 1993). It is also implicated in the inhibition of responses mediated by the N-methyl-D-aspartate (NMDA)-glutamate receptor (Lei et al., 1992). NMDA controls synaptic plasticity and memory. Increasing hypofunction of NMDA receptors is known to be associated with decreases in memory and learning performance (Newcomer et al., 2000).

Thus, release of nitric oxide from blood vessel tissues decrease the response of glutamate-NMDA receptors in enhancing plasticity and memory.

The first author (CJA) notes the role of cortisol which is released in trauma. It is found to be in excess in PTSD and also depression. Further, the toxic action of cortisol on neuronal tissue is noted. The literature on depression has shown the shrinkage of the hippocampus in depression as a consistent feature (Sheline et al., 1996, 1999; Wolkowitz, 1999; Bremner, 2000; Sapolsky, 2001; Videbach, 2004) along with BDNF suppression. Implicating cortisol excess in the decrease in hippocampal volume is a short step, and it is noted that the hippocampus is very accessible from the otic portals of entry in LI, and is essential in various lacking functions seen in ELI. Further experimental evidence is provided by Kurtulus et al. (2008), who note hippocampal volume loss after electrical injury.

The stimulation of glutamate and cortisol are not mutually exclusive, and it is suggested that a complex interaction of electric current, vessel walls, nitric oxide, glutamate and cortisol, have an endpoint action on memory, and learning, and other ELI symptoms, and intimately involving the hippocampus.

The authors of this paper suggest that this theory is the most cogent to date explaining both delay and also remote action. Further research is needed.

Treatment

Definitive treatment strategies have not been considered in detail. Certainly, the use of an antidepressant can be supported, and agents for neuritic pain relief, such as pregabalin and/or congeners.

The discussion above may well encourage the use of nitric oxide antagonists, glutamate antagonists, cortisol antagonists, and antioxidants. These agents may well become the subject of research. Juurlink and Paterson (1998) propose (1) the use of reduced-glutathione levels achieved by the use of cysteine precursors, (2) platelet activating factors, and (3) nutritional support maximising anti-oxidants, including the use of flavonoids.

We suggest that these techniques remain experimental and open fruitful lines of research. Coupled with pharmacological agents, verbal strategies in neuropsychological counselling are indicated.

Conclusion

This paper has presented a review of neurological symptoms of ELI under an anatomical categorisation. Similarly, the spectrum of neuropsychological symptoms has been reported, indicating the severe disability victims of ELI suffer.

A theory of causation of ELI symptoms has been advanced, and this theory suggests that circulating neurohormones explain enigmas like the delay in onset of symptoms, and the appearance of symptoms attributable to body systems distant from the passage of electric current. Fruitful lines of continuing research are suggested.

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