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Neurological sequelae of lightning injury

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Introduction

Man has been exposed to lightning ever since his appearance on earth. This is particularly so when he moves about in open spaces with the risk of being struck by lightning being about 30 times greater in rural areas than in cities[1]. Transient paraplegia following lightning strike was described by Orton in 1848[2] and Charcot in 1889[3]. Arborescent red lines or burns, as carefully described in Dr. Zammit Maempel's case report in this issue of the journal, usually indicate the point of contact of lightning, and are considered to be pathognomonic features[4]. However, the path taken through the body can be deduced only approximately from the clinical sequelae. Table 1, adapted from the 1970 monograph by Panse[4] on electrical lesions of the nervous system, summarises the typical physical consequences of a lightning strike.

Table 1 - The physical consequences of lightning srike

	Entry at the head	Entry elsewhere on the body
Fatal Strokes	• instantaneous cerebral death • scalp and skull burns • lightning figures on skin surface • mechanical tearing of clothes • melting of metal objects close to body	• much rarer
Short term Sequelae	• severe coma lasting up to several days • agitated confusion or psychosis • seizure disorder[5] • other signs as per fatal strokes	• brief loss of consciousness • short-lived sensory motor deficits according to pathway of strike • vasomotor symptoms at affected body parts • other signs as per entry at head
Long term Sequelae	• hypoxic encephalopathy • cerebral vein thrombosis[6] • striatal disorders • hemiparesis and/or dysphasia • psychosis	• spinal amyotrophy • transverse myelopathy • deafness

Table 2 - Transient neurological defects caused by lightning strike
Certain neurological sequelae are typical[4]:

- some degree of clouding of consciousness;
- psychological and psychiatric reactions;
- transient disturbances of motor and sensory functions of single or all limbs;
- autonomic nervous system disorders;
- myoclonic jerking of one or more extremities, as probably occurred in the case described by Dr. Zammit Maempel in this issue of the journal.

Initial loss of consciousness is quite common, and it usually clears up completely within a few seconds or minutes[7]. However, impairment of consciousness lasting several days has also been reported, particularly when the head or neighbouring parts have been struck directly. There are also a few documented cases where the victim repeatedly lapsed into brief unconsciousness[7]. A transient seizure disorder is frequently reported in survivors[5].

The impairment of consciousness may be followed by mental changes comprising disorientation, emotional lability, psychomotor retardation or even an agitated-confusional state which may persist for a week or two. Frank psychosis may develop rarely. Retrograde amnesia has been recorded in a few cases, and usually involves a very brief period just prior to the lightning strike[4].

Immediate 'lightning paralysis' characterised by complete tetraplegia and loss of sensory awareness of the trunk and all four limbs may be considered virtually pathognomonic[3]. Sometimes only the lower limbs, or an arm and a leg are affected, depending on where the lightning had struck[4]. Very often the sensory and motor paralysis is accompanied by marked autonomic and vasomotor disturbances[8,9,10], as described in Dr. Zammit Maempel's case. The affected limbs become pale and cold or cyanotic. The arterial pulses may transiently disappear. The affected limb can rarely become oedematous. In 1955 Panse described a board-like induration of one calf, perhaps as a result of 'electric oedema' or tetanic contracture[11]. In

typical uncomplicated cases of 'lightning paralysis', sensation and motor function usually return after a few minutes and at the latest up to a few days, accompanied by parasthesiae[4].

Cranial nerve involvement has been documented with lightning strikes as far down as the back of the neck[4]. Damage to the auditory apparatus with transient or permanent deafness and tinnitus frequently features in the literature, and rupture of the tympanic membrane has also been reported[4].

About a quarter of lightning victims develop generalised autonomic disturbances[12] including sleep disorders[4], changes in appetite and weight[13], diabetes insipidus[14], disorders of libido[13], menstrual irregularities[4], hyperthyroidism and hyperpyrexia[14].

Table 3 - Permanent neurologial defects caused by lightning strike

The long term neurological sequelae of lightning strikes can be very diverse:

- segmental amyotrophy, or less frequently a syndrome simulating amyotrophic lateral sclerosis (ALS)[15,16] or transverse myelopathy[17,18,19,20];
- striatal disorders[21,22,23], including parkinsonism and choreoathetosis;
- cerebellar ataxia[24];
- hemiparesis, with or without dysphasia[25].

As expected, there may be lasting spinal cord sequelae, depending on the path taken by the lightning strike through the body, usually arm to arm or arm to leg[1]. The first case of spinal amyotrophy was documented by Le Roy de Mericourt in 1860[26]. Later similar reports became more frequent which were characterised by focal or diffuse amyotrophy with or without sensory involvement[4]. There have also been some cases of transverse myelopathy with spastic paraparesis and sphincteric disturbances[4].

It is difficult to establish the time interval between the injury and the onset of amyotrophy since the early stages can be overlooked by both patient and doctor[4]. Often the spinal syndrome develops immediately. However, in some cases there has been a latency period of up to a maximum of 24 months before the onset of muscle wasting[16]. The course of spinal amyotrophy can be initially progressive for a few months or years, and then eventually regress or become static[4]. On the other hand, there can be unrelenting progression leading to death, particularly in those who develop an ALS-like syndrome[16].

The proposed pathogenesis of delayed spinal cord injury includes slow endothelial fibrosis and intimal thrombosis with vascular occlusion or ischaemia of the microvascular circulation[16]. This has been likened to the delayed radiation effects on the spinal cord[27]. Stanley has proposed that delayed injury is secondary to vasospasm with a similar mechanism as that seen with subarachnoid haemorrhage[28]. Farrell and Starr proposed alternative explanations, including injury to cellular DNA[27]. This may be secondary to thermal, mechanical, or vascular insult, with subsequent neuronal cell death[29].

In contrast to the spinal atrophic syndromes less rare but slighter and more transient neurological manifestations have also been reported. These include parasthesiae in certain fingers of one or both hands lasting several weeks, subjective weakness or clumsiness without objective reflex changes or muscle wasting, and transient disorders of micturition and potency[4].

Peripheral nerves lying in an area of a severe electric burn are usually severely, and often irreversibly, damaged[10]. Delayed neuropathic effects include nerve entrapment and nerve pain syndromes[5].

Persistent seizures are surprisingly rare[1]. However, in a small number of surviving patients, after an asymptomatic interval of days to months, there has been an apoplectic onset of hemiplegia with or without aphasia, or a striatal or brainstem syndrome presumably due to thrombotic occlusion of cerebral vessels with infarction of tissue[1].

'Electric cataracts' are fairly typical consequences of lightning strikes to the face or forehead. The usual latency period is at least four to six weeks, and at most 18 to 24 months[11].

Review of Investigation Results

In 1957 Schmidt et al reported important electroencephalographic findings suggesting a high degree of cerebral involvement[30]. In one case there was mainly irregular background activity of 9 - 10 Hz with paroxysmal dysrhythmic discharges at rest which persisted for several months following the injury in the absence of clinically overt epileptic manifestations. In another case, an alpha rhythm of 8 Hz with superimposed high voltage steep spikes of up to 130 microvolts over the frontal regions was recorded in a deeply comatose woman. In patients with severe skull burns, there can be ipsilateral loss of alpha activity in association with high amplitude theta waves as well as epileptic discharges[31].

The most common brain imaging finding that can be attributed to a lightning strike is basal ganglia haemorrhage which can be bilateral[32].

Heavily blood-stained cerebrospinal fluid (CSF) has been reported in some survivors who complained of headache and neck stiffness[33]. Some went on to develop communicating hydrocephalus[34]. The CSF in patients with spinal amyotrophy has only shown a mildly raised protein content[35].

Neuropathological Findings

Lightning that strikes the head is particularly dangerous, proving fatal in 30 percent of cases[9]. Death is due to ventricular fibrillation or to the effects of intense desiccating heat on the brain[1]. Apnoea may also develop as a result of injury to the medullary centres[5].

A detailed description of the neuropathological changes in fatal lightning injury has been given by Critchley[36]. These changes include cerebral oedema, extensive subarachnoid haemorrhages secondary to ruptured arteries, and focal petechial haemorrhages in the brain and spinal cord, especially in the medulla and the anterior horns. Irregular tears and fissures can appear in the brain tissue, and adjacent cortical layers can be separated from one another[37]. There can also be crinkling, granulation and spongy loosening of brain tissue due to the heat. Wide dilatations of the

perivascular spaces measuring 25 to 250 microns in diameter have also been reported[36]. Peripheral nerve examination has revealed fragmentation of axons and changes in the myelin sheaths[36].

When a person is struck by lightning he may be flung violently to the ground. Injuries from mechanical trauma, involving especially the skull and its contents, must be distinguished from the lesions produced by the electrical trauma[38].

Conclusion

Lightning strikes are not rare. Fortunately, the majority survive with no long term sequelae. Any part of the peripheral or central nervous system may be involved. Of crucial importance are the points of contact, and therefore the path of current flow. An accident involving the passage of current between the head and foot or between one arm and the other is likely to damage the brain and spinal cord respectively[5]. The effects may be immediate, which is understandable, but of greater interest are the rare instances of neurological damage that occur many days or months after the accident. The immediate effects are apparently the result of direct heating of the nervous tissue, but the pathogenesis of the delayed effects is still not well understood[1].

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