The current state and possible perspectives are discussed in this communication.

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**1740**
**WFN15-1802**
**Neurology and Special Environments T 5.1**
**The CNS in lightning**

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In developed countries, lightning injury is primarily a neurological, not a burn, injury. It can extend from the nearly harmless simple static discharge similar to walking across a carpet to cardiac arrest with hypoxic bran damage. Between these two extremes, brain injury similar to post-concussion syndrome and nerve injury, with resultant chronic pain, are the main components of lightning injury. The autonomic, and sometimes the endocrine system, can also be affected. Concussive injury from the explosive effect very near a lightning strike or muscle contractions throwing a victim can cause musculoskeletal problems.

Only 3-5% of injuries are from direct strike. The majority are from ground current (40-50%), side flash from another object (20-30%), contact injury (15-25%), or upward streamer (10-15%), all of which deliver only a small portion of lightning's usual energy to the victim. Because of this, thermal injuries occur in less than half of the survivors so that lack of skin markings, burns or scars cannot be used to rule out lightning injury.

Although brain injury with adult attention deficit, memory, executive function, irritability, depression, and other signs have been well described, defining the cellular and tissue level damage has been more difficult due to lack of funding and research trained keraunomedicine practitioners as well as available subjects. Controlled longitudinal studies to determine long term effects have also not been done for similar reasons.

In the US and many other developed countries, lightning injuries and fatalities have been markedly decreased through public education. However, lightning injury continues to be a major concern in lesser developed countries due to lack of lightning-safe buildings and vehicles, poor awareness, and the much higher lightning densities that occur in tropical and subtropical countries.

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**1741**
**WFN15-1817**
**Neurology and Special Environments T 5.1**
**The brain in acute mountain sickness and cerebral edema**

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Ascent to altitudes above 2500 m can cause acute mountain sickness (AMS), which may progress to high altitude cerebral edema (HACE). AMS is characterized by headache, lassitude, anorexia and nausea/vomiting, which are all usually self-limiting with a day of rest and symptomatic treatment. Truncal ataxia and altered consciousness are the cardinal symptoms of HACE, which is often lethal when not treated with oxygen, dexamethasone and descent. Slow ascent, or acetazolamide, or glucocorticosteroids during ascent can attenuate or prevent these illnesses.

The pathophysiology underlying the cerebral symptoms of AMS is unclear. Several mechanisms could activate pain sensitive structures and vegetative centers in the brain. Candidates are brain edema and increased intracranial pressure (ICP), distension of large vessels because of increased cerebral blood flow (CBF) in hypoxia and possibly impaired autoregulation of CBF, restricted venous drainage of the brain, activation of the trigemino-vascular system by hypoxia, or increased permeability of the blood brain barrier (BBB) in hypoxia with release of inflammatory mediators. It is conceivable that several of these factors are involved in the pathophysiology of AMS, since no single factor is strongly associated with the development of AMS.

Increased ICP and vasogenic edema located predominantly in the corpus callosum (CC) are consistent findings in HACE. Hemosiderin depositions in the CC usually detectable by susceptibility weighted MRI after HACE indicate leakage of red blood cells through the BBB and are footprints of HACE. Increased CBF, impaired venous outflow and increased vascular permeability may all contribute to the BBB leak in hypoxia.

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**1742**
**WFN15-1627**
**Neurological Care During Disasters T 23.1**
**Acute phase management for neurological patients in a disaster**

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I summarize what we did at the earthquake on March 11, 2011 and thereafter.

**Acute phase (a week or two weeks after the earthquake)**

We did our roles as a doctor, not specialists. We triaged emergent, serious patients and cared them in our hospital or transferred them to distant, intact hospitals.

**Subacute phase(one month to two months after the earthquake)**

We, as neurologists, treated serious patients with encephalitis, MG crisis and MS relapse, who were not able to be managed in other hospitals.

**Recovery phase to usual neurology (three months to a half year after the earthquake)**

At this phase, we gradually recovered to the usual neurologists. The patients living near our hospital came back to our outpatient clinic.

**Chronic phase, usual neurology (several months after the earthquake)**

We mostly spent our time the same as before the earthquake. At this stage, we should take care of care givers as well as the patients because some care-givers had some mental problems due to the disaster.

**Long term works (one year or more later)**

In most areas in Fukushima, people live just like before earthquake because of the same radiation level as the other places in the world. The radiation makes some of us unable to come back to the original living place, which has produced several health problems due to incorrect lifestyles as follows: the higher blood pressure and hyperlipidemia in the people living in the temporary houses, and the cerebrovascular incidence increment after the disaster.

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**1743**
**WFN15-1660**
**Neurological Care During Disasters T 23.1**
**Establishing a nationwide disaster management system for neurologically ill patients**

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Establishing a nationwide disaster managing system for neurologically ill patients.

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Japan and Chile have a common problem of earthquakes and tsunami, which cause amassive national disaster. Critically ill neurological patients such as with amyotrophiclateral sclerosis on a respirator succumbed to death in the last Eastern Japan Earthquake. Some of them died from tsunami, the others of respirator failuredue to power loss. There have been a number of discussions to reduce such casualties. One is to prepare emergency power source driven by oil. The easiest one is tosupply electric power from cars. Of course, continued supply of gasoline or diesel for cars is needed. Local medical personnels have to retain a list of patients with a respirator at home. Japanese government is now promoting these activities to prepare for the expected Southern Japan earthquake, which shouldcome in 50 years with the likelihood of more than 50%.

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**1744**
**WFN15-1667**
**Neurological Care During Disasters T 23.1**
**Challenges of mental health care in Latin America in the aftermath of disasters**

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Disasters are increasing its frequency in the world and Latin America. Post-traumatic mental disorders are one of the most important consequences of these events, with significant costs for the public health of communities.

During the last decades, the world has designed and organized different models of intervention to prevent and treat post-disaster mental disorders. Latin America - as a region in course of development - has been facing its own local challenges to accomplish this task.

In order to get a glimpse about the key challenges of disaster mental health in Latin America, we surveyed the public health officials in charge of disaster mental health in different countries of the region, asking them the to identify the most important challenges they are facing in their duties. In this keynote, we present the most important results and discuss the implications for future public health actions.

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**1745**
**WFN15-1925**
**Neurological Care During Disasters T 23.1**
**Mental health after the Great East Japan Earthquake**

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We experienced the Great EastJapan Earthquake on March 11, 2011. This disaster caused unprecedented damage, with nearly 20,000 peopledead or missing. Since March 17, our university dispatched medical rescue teams9 times in total, the last three focused on psychiatric problems and mental supportin Fukushima prefecture. The disaster in Fukushima could be recognized asmultifaceted with tsunami and possible radiation exposure caused by the nuclearaccident in addition to the earthquake damage per se. These complex problems causedtremendous distress to vulnerable people including aged persons, individualswith various medical/neurological/psychiatric conditions, and specificallypeople with cognitive impairment. Psychiatric problems which required forintervention included worsening of original psychiatric diseases, acute stressdisorder, post-traumatic stress disorder, adjustment disorder, suicidal attempt/idea,and alcohol related problems. One exceptional and extraordinary situation in Soma district in Fukushima prefecture was the effect of Nakamura Domainoie-sodo (internal trouble), "Soma Incident" which occurred during 1880's. Thefeudal lord Tomotane Soma is considered to have had schizophrenia. As adistorted consequence with stigma, there existed no psychiatric facilities inSoma district even before the earthquake, and citizens with psychiatricproblems in Soma had been forced to visit neighboring Minami-Soma and Futabadistricts. However, the psychiatric facilities in Minami-Soma and Futabadistricts were completely destroyed by the nuclear accident. We helped Departmentof Neuropsychiatry, Fukushima Medical University provide suitable mentalsupport for the survivors. In addition, to create new psychiatric services isan urgent issue including outreach activities and remote medical/psychiatriccare.

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**1746**
**WFN15-1848**
**Neuro-Ophthalmology T 8.1**
**How can Ocular Coherence Tomography (OCT) Inform the Neurologist?**

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In the 1990's a team from New England Eye Center of Tufts University led by James Fujimoto Ph.D., Joel Schuman M.D. and Carmen Puliafito M.D. was one of the groups that developed the technology of Opthical Coherence Tomography for Opthalmology. The flat retina could now be seen side ways as a histological specimen and differente layers could be measured. OCT is an excellent tool for examining the optic nerve head and the retina. The inner retina, ganglion cells and axons are central nervous system equivalent to the spino-thalamic pathway. As such they can be part of degenerative brain diseases. There are numerous papers dealing with thinning of the inner plexiform, retinal ganglion cells and nerve fiber layers in the macular area and the optic nerve area in different nerurological diseases. The Stratus-Time Domain machine has a definition of 15um., while the Cirrus-Spectral Domain has 7um. definition and the Heidelberg-SD has 10um. definition with 40.000 scans/sec. With these machines it is possible to diagnose a subtle papilledema ophthalmoscopically invisible, a small macular hole that explains a visual loss or fluid under the macular from a disc oedema. In neurodegenerative disorders thinning of the retinal nerve fiber layer in MS or ganglion cell-internal plexiform layer thinning in Parkinson's disease are new elements that contribute to the diganosis and can be part of the prognostic factors. The spatial resolution of the different machines have to be taken in account when different results are compared.

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**1747**
**WFN15-1659**
**Neuro-Ophthalmology T 8.1**
**What's new in idiopathic intracranial hypertension?**

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Idiopathic intracranial hypertension (IIH) is a disorder typically affecting young, obese women, producing a syndrome of increased intracranial pressure without identifiable cause.

Despite a large number of hypotheses and publications overthe past decade, the etiology of IIH is still unknown. Numerous studies have emphasized radiologic abnormalities in IIH, including orbital signs of raised intracranial pressure, empty sella, meningoceles and cerebral transverse sinus stenosis. Identification of subgroups of patients at high-risk for irreversible visual loss, such as black patients, men, morbidly