Saturday, 31 May

EFNS/ENS/EFNA Awareness Session: Brain Disorders: The Communication Challenge – Focus on Traumatic Brain Injury

SPS1-1
Case-study: acquired brain injury

N. Steinhoff
Grimmenstein, Austria

In traumatic brain injury a complex and sudden impact leads to many simultaneous lesions of the central nervous system. This creates a sudden change of situation of life for the injured and the family. For communication in life several centres of the brain have to collude in order to make interaction between human or other beings possible. Corresponding to the complex injury of the brain through TBI several barriers arise in communication with the traumatic brain injured, concerning speech understanding and production, alertness, impetus, awareness, language, cultural problems, premorbid and emotional situation or the social environment. The environment and the injured have to adapt quickly to the new situation, which often creates new problems of interaction. But there is also a problem of time on the professional side and a lack of understanding TBI itself that reduces the capacity of perceiving the actual and in any case individual situation. Especially the nursing staff and caregivers are exposed to the sudden changes of moods and aggressive approach to communication. Most of communication problems are due to lack of training how to deal with the individual situation after TBI. This can go so far, that inadequate prognosis might be given to the TBI affected persons. Most of aggression and agitation are avoidable through professional training. The readaptation syndrome after TBI and its single factors are often resulting out of lack of transporting the most important information about TBI to the family and injured at the very beginning after TBI. The several steps of communication and its challenge after TBI shall be presented in this contribution and made visible through an example of a TBI injured person.

Disclosure: I disclose potential or actual conflicts of interest concerning this presentation.
Monday, 2 June

European Basal Ganglia Club

SPS5-1

Aetiology and pathophysiology of focal dystonias
A. Berardelli
Rome, Italy

Primary focal dystonias are disorders characterized by involuntary muscle spasms causing abnormal postures and patterned movements. The various types of focal dystonias have different clinical, epidemiological and etiological features. Focal dystonias, however, share a number of factors. Shared features include genes, molecular and cellular pathways and anatomical circuits. Focal dystonias also share pathophysiological abnormalities. Loss of inhibition has been demonstrated by studies of spinal cord and brainstem reflexes (blink reflexes, reciprocal inhibition and long-latency reflexes). Reduced inhibition is also present at the level of primary motor cortex, as demonstrated by the abnormalities of intracortical inhibition, silent period and surround inhibition. Loss of inhibition contributes to the defect in focusing motor command and causes the abnormalities in cortical plasticity present in dystonia. Whether neurophysiological abnormalities depend on changes at cortical, brainstem and cerebellum is still unclear. Finally a defect in sensory and sensorimotor integration is another important abnormality of dystonia and may be responsible for some of the motor dysfunction. Differences and similarities in focal dystonia can be explained by a recently proposed conceptual model (Jinnah et al 2013).

Disclosure: Nothing to disclose
Tuesday, 3 June

Mediterranean session – Jointly organised by EFNS / ENS and PAUNS (Pan Arab Union of Neurological Societies): Dementia and Alzheimer’s disease

SPS7-1

Epidemiology of dementia – does the Mediterranean diet matter?
R. Gouider
Tunis, Tunisia

Introduction: Dementia is a major public health problem in Mediterranean populations. Its prevalence depends on the interaction of genetic and environmental risk factors including dietary habits. The Mediterranean diet is presented as a cultural and healthy model characterized by a plant-based dietary rich in anti-oxidants and polyunsaturated fatty acids. Its effects on preventing dementia are controversial.

Objective: To conduct a review of the epidemiological data concerning dementia in Mediterranean countries and assessing the influence of the adherence to Mediterranean diet on the risk of cognitive impairment and dementia.

Methods: Articles and reviews relevant to the topic were selected through a PubMed search using the search terms “Mediterranean diet”, “nutrition”, “dementia incidence” and “cognitive decline”. The search results included epidemiological and interventional studies.

Results: Data from population-based studies carried out in Mediterranean countries shows higher incidence rates of dementia in Northern than in Southern countries. Several epidemiological cohort studies found a relationship between Mediterranean diet and lower risk of cognitive decline and dementia. Higher adherence to Mediterranean diet seems to be associated with reduced risk of Mild Cognitive Impairment (MCI) and Alzheimer’s disease (AD). However, there are contrasting findings between studies about the impact of the Mediterranean diet on the risk of progression from MCI to AD.

Conclusion: Environmental risk factors such as dietary habits, interacting with genetic factors, may influence the epidemiology of dementia and contribute to the north-south gradient observed across Mediterranean countries. Indeed, Mediterranean diet appears to have a protective role against cognitive decline and progression to dementia.

Disclosure: Nothing to disclose

SPS7-2

Transcultural approach in dementia
M. El Alaoui Faris
Rabat, Morocco

Objectives: As the elderly population increases faster in non-Western countries than in Western countries, the prevalence of dementia will increase more rapidly in these countries. We want to present the situation of dementia in these countries.

Methods: We studied the publications on dementia in non-Western countries, mostly in Asia, Africa and the Middle East, to know the state of knowledge of dementia in these countries.

Results: Although two thirds of people with dementia live in non-Western countries, few studies concerning these countries. Prevalence of dementia in some of these countries is as high as in Western countries, however, the diagnosis of dementia is often at an advanced stage of the disease and most people living with dementia have not received a formal diagnosis, one study in India suggesting that about 90% of dementia remain unidentified. The delay and the lack of the diagnosis of dementia are probably due to several factors including lack of cognitive tests in native languages and on neuropsychological tests adapted to illiterate people. Furthermore, the perception of dementia in different cultures is decisive in the fact that families bring or not the patient in consultation, we need anthropological studies to understand this situation and to see how to use the resources of various cultures to help patients and their families.

Conclusions: Dementia in non-Western countries remains dangerously neglected by researchers and by policy makers. Clinical and epidemiological studies are likely to provide valuable information on patients living with dementia in these countries.

Disclosure: Nothing to disclose
SPS7-3

Alzheimer's disease from pathophysiology to treatment?

M.N. Rossor

London, United Kingdom

Our understanding of the histopathology of Alzheimer’s disease has expanded enormously over the last 20 years, although drug targets have to date proved disappointing. One of the challenges is that the hallmarks of AD, senile plaques and neurofibrillary tangles, show an association with cognitive impairment but not a close correlation with severity, particularly in the elderly. Nevertheless, the pathophysiology of amyloid deposition, driven by studies of familial Alzheimer’s disease and of tau accumulation, have led to trials targeting these misfolded proteins. So far amyloid immunotherapy trials have failed to meet primary endpoints but there are a number of studies commencing which trial these drugs in early or premanifest cases. Numerous other potential targets that are being explored such as inflammation, and the unfolded protein response. Although current efforts are focused on disease modifying treatments, symptomatic treatments based on neurotransmitter modulation will remain an important approach to treatment, building on the current use of cholinergic enhancement and (cholinesterase inhibitors) and glutamate modulation (memantine). Finally, treatment of comorbidities which can alter synaptic function or indeed amyloid accumulation are important, as are non-pharmacological interventions.

Disclosure: Nothing to disclose