Nature Reviews Neurology **9**, 659 (2013); published online 12 November 2013; doi:10.1038/nrneurol.2013.236; doi:10.1038/nrneurol.2013.237; doi:10.1038/nrneurol.2013.238; doi:10.1038/nrneurol.2013.239

IN BRIEF

SPINAL CORD INJURY

Deep brain stimulation could provide a new approach for restoring motor function after spinal cord injury

Stimulation of the mesencephalic locomotor region might improve limb function after incomplete spinal cord injury (SCI), a recent study suggests. Excitatory deep brain stimulation (DBS) restored hindlimb function to nearnormal levels in rats with 20–30% sparing of reticulospinal fibres after chronic SCI. In many patients with SCI, a small number of such fibres persist after injury. The new findings indicate that DBS could provide a therapeutic strategy for treating gait disturbances in these individuals.

Original article Bachmann, L. B. *et al.* Deep brain stimulation of the midbrain locomotor region improves paretic hindlimb function after spinal cord injury in rats. *Sci. Transl. Med.* doi:10.1126/scitranslmed.3005972

EPILEPSY

Childhood convulsive status epilepticus may lead to progressive hippocampal volume loss

Sequential MRI assessment of 80 children during the first year after convulsive status epilepticus (CSE) has revealed that hippocampal injury not only occurs in the acute phase that immediately follows CSE, but may also progress over time. Contrary to previous assumptions, hippocampal volume loss could result from CSE of any aetiology, and was not limited to children with febrile seizures.

Original article Yoong, M. *et al*. Hippocampal volume loss following childhood convulsive status epilepticus is not limited to prolonged febrile seizures. *Epilepsia* doi:10.1111/epi.12426

ALZHEIMER DISEASE

Amyloid plaques are associated with mitochondrial dysfunction in AD

Amyloid- β (A β) deposition is an ubiquitous finding in Alzheimer disease (AD), but the role of A β in the disease progression is subject to debate. Xie *et al.* found abnormalities in structure and function of mitochondria specifically in the vicinity of A β plaques in a mouse model of AD. The results provide evidence for involvement of A β neurotoxity in the pathological process that impairs mitochondrial function in AD.

Original article Xie, H. *et al.* Mitochondrial alterations near amyloid plaques in an Alzheimer's disease mouse model. *J. Neurosci.* doi:10.1523/JNEUROSCI.1836-13.2013

STROKE

Novel oral anticoagulants for stroke prevention are associated with reduced risk of intracranial haemorrhage

New-generation oral anticoagulants carry a lower risk of intracranial haemorrhage (ICH) than does warfarin, a new systematic review and meta-analysis shows. Chatterjee *et al.* analysed six studies, which had recruited a total of 57,491 patients with atrial fibrillation who received anticoagulants for stroke prevention. All novel anticoagulants, which interact directly with coagulating proteins, were associated with a 50% reduced risk of ICH compared with warfarin-based vitamin K antagonists.

Original article Chatterjee, S. et al. New oral anticoagulants and the risk of intracranial hemorrhage. JAMA Neurol. doi:10.1001/jamaneurol.2013.4021