REM sleep behavior disorder can precede development of neurodegenerative disease

Rapid eye movement (REM) sleep behavior disorder (RBD) is a condition in which patients act out their unpleasant dreams and experience a loss of the normal muscle atonia associated with REM sleep. Evidence indicates that RBD can be the first manifestation of a neurodegenerative disease in some patients, sometimes preceding the neurodegenerative disease by years.

In a recent descriptive study conducted in Spain, researchers retrospectively assessed the records of 44 patients (39 male, 5 female; mean age 74 years) who attended their sleep clinic and had been diagnosed with idiopathic RBD. All patients had received at least 2 years of clinical follow-up involving assessments every 3–12 months by both sleep and neurodegenerative experts, which included neurological examinations, neuropsychological testing and parkinsonism rating scales.

After a mean of 5.1 years of follow-up from the time of diagnosis of idiopathic RBD, 20 patients were found to have developed a neurological disorder: nine patients developed Parkinson's disease, six patients developed dementia with Lewy bodies, four developed mild cognitive impairment, and one developed multiple system atrophy with predominant cerebellar syndrome. The mean duration from the time of the reported onset of RBD to development of the neurological disease was 11.5 years.

The authors conclude that patients diagnosed with idiopathic RBD should be closely monitored for the development of neurodegenerative disease, as early detection and symptomatic treatment might improve their long-term outcome.

Original article Iranzo A *et al.* (2006) Rapid-eye-movement sleep behaviour disorder as an early marker for a neurodegenerative disorder: a descriptive study. *Lancet Neurol* **5:** 572–577

Severe traumatic brain injury leads to cellular immunosuppression

Severe traumatic brain injury (TBI) can lead to immune system impairment, resulting in increased morbidity and mortality. Mazzeo *et al.* have studied the early effect of severe TBI on cell-mediated immunity and examined whether early infusion of the immunosuppressant ciclosporin A—which has been shown to be beneficial in TBI—further affects early cell-mediated immunological function after TBI.

The study included 49 patients with severe TBI and a Glasgow Coma Scale score of ≤8. Thirty-six patients received one or two 24 h infusions of ciclosporin, and 10 patients received placebo; 3 patients were not included in the ciclosporin substudy.

At admission, $CD3^+$, $CD4^+$ and $CD8^+$ T-lymphocyte counts were low in 65%, 70% and 39% of patients, respectively. Injury severity score increased with decreasing T-lymphocyte counts, indicating that patients with the most-severe injuries had the worst immunological impairment. Reduced T-lymphocyte counts were also associated with increased susceptibility to pulmonary infection. Higher injury severity scores were associated with worse neurological outcomes. Absolute T-lymphocyte counts were similar in patients receiving ciclosporin and patients receiving placebo. Histopathological investigation of contused brain tissue from one patient showed large numbers of T cells—mainly $CD8^+$ T cells—infiltrating the traumatized brain parenchyma.

The authors conclude that cellular immunosuppression often follows TBI, and that a reduction in T-lymphocyte count is related to worse neurological outcome and increased risk of pulmonary infection. They state that ciclosporin administration after head injury is safe and does not interfere with the cellular immune response.

Original article Mazzeo AT *et al.* (2006) Severe human traumatic brain injury, but not cyclosporin A treatment, depresses activated T lymphocytes early after injury. *J Neurotrauma* **23:** 962–975

H. pylori eradication improves symptoms of Parkinson's disease

The *Helicobacter pylori* bacterium lives in the mucus layer of the stomach and is a common cause of gastric and duodenal ulcers. Although up to one-third of the world's population might be infected with *H. pylori,* most people are asymptomatic. *H. pylori* might, however, interfere with levodopa activity, and preliminary results have suggested that the response to levodopa of *H. pylori*-infected patients with Parkinson's