Gérard Said

Secretary General and Founder, European Neurological Society (ENS)

Gérard Said is Secretary General of the European Neurological Society (ENS), which he co-founded in 1986, and a Consultant Neurologist in the Fédération des Maladies Neurologiques at the Hôpital de la Salpétrière in Paris. Prior to this, he served as Chief of the Neurology Service at Hôpital Bicetre, a position he held from 1988 to 2007. Previously, he served as an Associate Professor of Neurology at the Medical School of the University Paris-Sud. Professor Said graduated from the Medical School of Paris in 1971 and qualified in neurology in 1974. Professor Said is also Director of the Research Group on Neuromuscular Disorders of the World Federation of Neurology (WFN), and between 1998 and 2000 he was President of the Peripheral Nerve Society (PNS).

The focus of the European Neurological Society (ENS) 2008 meeting confirms the steady interest and progress in the treatment of chronic neurological disorders, which represent a major factor of disability. In the field of multiple sclerosis (MS), a number of new developments have been announced with the wider use of early treatment. The results of clinical trials support the hypothesis that inflammation is necessary for new lesion formation and conditions axon degeneration. The implication is that immunological therapies will best prevent sustained accumulation of disability and disease progression if given early in the course and before the cascade of events leading to axon degeneration is irretrievably established. In clinically isolated syndromes, epidemiological studies and clinical trials have demonstrated that, within two years, approximately 90% of patients can be diagnosed as having MS according to the McDonald criteria. Patients with a high number of lesions or gadolinium-enhancing lesions in brain and spinal cord magnetic resonance imaging (MRI) have a very high risk of experiencing a second attack shortly after the first, with an increased risk of long-term disability. The subsequent question will be which preventative treatment to use: Avonex®, Betaferon® or Copaxone®? The decision belongs to the patient, but the role of the neurologist is crucial to explain to the asymptomatic patient why a certain treatment is advisable. In MS patients who continue to experience disease activity despite treatment with traditional disease-modifying therapies, a more effective therapy becomes available. In this respect, natalizumab (Tysabri®) has reduced relapse rates by up to 68% and the risk of sustained disability progression by up to 54% in phase III clinical studies in patients with relapsing MS.

Alemtuzumab, a CD52-specific monoclonal antibody that targets lymphocytes, markedly suppresses disease activity when administered during the early stages of relapsing–remitting MS (RRMS). Recent studies presented at the ENS meeting show that alemtuzumab is significantly more effective than interferon-β-1a at suppressing relapses and disability in patients with RRMS over three years of follow-up regardless of patient sex, age, race or nationality. Early tests on laquinimod are also very promising.

Several therapeutic trials are under way in hereditary disorders of the nervous system. Friedreich ataxia (FRDA) is an autosomal recessive neurodegenerative disorder caused by a mutation in the FXN gene, which encodes a protein named frataxin. As a result, frataxin is quantitatively reduced but qualitatively normal. Recent studies have demonstrated the efficacy of recombinant human erythropoietin (rhuEPO) in increasing frataxin levels in cultured cells, and also in the lymphocytes of FRDA patients receiving rhuEPO subcutaneously. Mariotti and co-workers have started a monocentric, randomised, double-blind, placebo-controlled, dose-finding study aimed at assessing the efficacy and toxicity of rhuEPO in FRDA patients. Fx-1006A is a novel, selective and potent stabiliser of wild-type and variant transthyretin (TTR) that may confer disease modification and halt disease progression in patients with TTR amyloidosis, a devastating neuropathy common in Portugal, Japan and Sweden and now identified in a number of countries thanks to molecular genetic analysis. An international study of the efficacy of this compound in familial amyloid polyneuropathy is under way. In inflammatory demyelinating polyneuropathies, which often respond to treatment with high-dose intravenous immunoglobulins (IVIgs), the use of the recently introduced 10% liquid IVIg , instead of the 5% concentration IVIg currently used, is associated with substantial reductions in infusion times in 85% of patients and a similar efficacy.

All of these new works confirm the strong interest of researchers and pharmaceutical companies in the treatment of chronic neurological disorders, hope that the rich variety of articles contained within the pages of *European Neurological Review* prove an informative and insightful read. ■

Nineteenth Meeting of the
European Neurological Society

June 20–24, 2009

Milan, Italy

Neurology: Learning, knowledge, progress and the future

Key symposia:

- Management of stroke: from bench to guidelines
- The molecular era of neuromuscular disorders
- From pathophysiology to new treatments in epilepsy
- Parkinson´s disease: advances in diagnosis and treatment
- Critical issues on MS diagnosis and treatment

The congress programme includes interactive case presentations, 23 teaching courses, 16 workshops organised by the ENS subcommittees, practical breakfast sessions in clinical neurophysiology and selected scientific sessions in the form of oral sessions, poster sessions (guided poster walks) and satellite symposia.

Abstract Submission Deadline: February 11, 2009
Early Registration Deadline: April 22, 2009

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