

Stroke remains the dominant topic for neurological research and practice across Europe. In the year to December 2015, over 20% of papers published in the *European Journal of Neurology* were related to stroke [1–48]. A substantial amount of work is focused on the pathophysiology of stroke, including genetic causes and risk factors, atrial fibrillation, arterial dissection and the role of infection in increasing risk in the acute and chronic stage. More attention is also being paid to intracranial small vessel disease including micro haemorrhages, often assumed to be a consequence of ageing. Inevitably the acute treatment of ischaemic stroke remains directed towards early detection and intravenous thrombolysis. Probably the most important limiting factor to the application of this therapy is access to specialized centres for diagnosis and treatment. European countries are relatively far advanced in providing such therapy for their citizens, but inevitably geography and resources continue to play crucial roles in determining outcome across Europe. The uncertainty of effectiveness of intravenous thrombolysis in the 3–4.5+ h time window following onset provides further challenge to the design of community response to stroke.

Multiple sclerosis (MS) is a field of neurology that has undergone tremendous advances within only the last 15 years [49–90]. The advent of immunological therapies has transformed outcome for patients with relapsing-remitting disease, although the effect on chronic progressive disease is less certain. The aetiology and pathogenesis of MS remain unknown. There is increasing evidence for a genetic component to MS risk, and certain lifestyle and environmental factors may influence this or act independently. Vitamin D deficiency appears to be a reproducible factor for increased risk.

The cause and treatment of Parkinson disease (PD) remains the most important component of research into movement disorders [91–117]. Substantial advances have been made in understanding the aetiopathogenesis of PD. Several genetic causes of familial PD have been described and genome-wide association studies have identified additional genes and cell function pathways that influence cause and onset. Probably the most important of all these are mutations in the glucocere-brosidase (GBA) gene. Mutations of this gene are found across Europe, with particularly high prevalence in the Ashkenazi community; overall, it is estimated that 10% of all PD patients carry a GBA mutation. Mutations of the gene also increase the risk for dementia with Lewy bodies. The non-motor aspects of PD have attracted increasing attention particularly over the last 10 years; non-motor features account for the greatest impairment of quality of life in advanced disease. Although some aspects may respond partially to dopaminergic drugs, treatment generally is limited. An interesting application for the identification of non-motor features has come in the detection of the pre-motor prodrome of PD. Abnormalities of olfaction, mood (depression) and the appearance of sleep disturbance in the form of rapid eye movement sleep behaviour disorder may all appear before motor features. Although each is non-specific, it has been proposed that, when used in combination and together with imaging, the specificity for detecting early PD may be increased significantly. Dystonia and other movement disorders are now attracting increasing attention in terms of treatment, particularly with non-medical forms including deep brain stimulation [118–133].

The management of Alzheimer and other dementias represents an increasing challenge to neurologists [134–150]. The aetiology and pathogenesis of Alzheimer disease and other forms of dementia have followed a parallel path to that in PD. Genetics attract substantial attention in the hope that causes of familial dementias and genetic variations that significantly influence risk are likely to reveal biological pathways that may be of relevance to therapeutic intervention. The role of inflammation in Alzheimer disease is an example of this dividend. However, it is recognized that the control of vascular risk factors such as hypertension, diabetes and hypercholesterolaemia, atrial fibrillation and smoking plays a crucially important role in reducing dementia, and improved management of these factors probably accounts for the recent decline in Alzheimer incidence. Other neurodegenerative diseases such as motor neuron disease and the hereditary spastic paraplegias [151–159] have likewise benefited from the revolution of neurogenetics.

The treatment of epilepsy has been able to take advantage of the introduction of a number of new drugs with good seizure control and improved adverse event profile [160–168]. Status epilepsy remains the most important life-threatening dimension of epilepsy.

There is predictably some overlap between neuromuscular disease [169–186] and neuro-inflammation [187–192]. Indeed the role of the immune system in neurological disease, both peripheral and central, is attracting considerable attention. Autoimmunity is associated with neuromuscular junction disease, such as myasthenia gravis and Lambert-Eaton syndrome, and the inflammatory myopathies for which an increasing number and range of antigens have been identified. Antibody-mediated auto-immune encephalitis continues to represent an important diagnostic and management challenge.

Headache is probably the commonest cause of referral to neurologists [193–200]. Migraine continues to present a significant therapeutic challenge, particularly in some of its more refractory forms such as cluster headache. The relationship of migraine to other neurological diseases such as epilepsy and stroke is important.

Advances in neurology occur across a broad front [201–225] including neuro-oncology, trauma, neuro-otology and neurodevelopment. These areas also overlap with other specialist areas within the neuro-sciences. The journal has an advantage in accommodating a wide range of subspecialty neuroscience papers within its pages.

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