



Highlights from this issue

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Ever since Hughlings Jackson's pioneering work, neurologists have tried to localise a clinical presentation—either explicitly or subconsciously—in terms of the level of the nervous system affected. This approach is particularly important when trying to disentangle challenging clinical problems—exemplified by the clinicopathological conference (page 64)—as well as being a key concept for teaching and learning neurology from medical school onwards. Symptoms and signs are synthesised to an anatomical location and then the differential is expanded, based on that localisation.

For example, a presentation with leg weakness, sensory loss, sphincter disturbance and reflex loss comprises a cauda equina syndrome; this prompts urgent assessment and imaging under orthopaedic or neurosurgical teams given the immediate concern of cauda equina compression. But if the scan is negative, what then? Neurologists are often asked to assess such 'scan-negative' patients. Ingrid Hoeritzauer and colleagues bring a practical approach to this clinical presentation (page 6), outlining the other conditions to consider in this situation, and how best to investigate and manage such patients, particularly when all investigations prove negative.

Paraneoplastic processes may manifest at all levels of the nervous system, from brain to muscle, from encephalitis to myopathy. The range of antibodies involved and our understanding of their pathogenesis have increased dramatically over the last few decades, characterising several clinical phenotypes and their linked antibodies. Sarosh Irani and his team discuss the best approach to the clinical assessment, investigation and treatment of patients with these important conditions (page 19).

It is often challenging to synthesise syndromes that involve more than one level of the nervous system. A slowly progressive bilateral vestibular syndrome is an unusual presentation, and even more so when the ataxia is compounded by a cerebellar syndrome,

a sensory neuropathy (including impaired position sense) and autonomic involvement (including postural hypotension). However, this combination is very characteristic for cerebellar ataxia, neuropathy, vestibular areflexia syndrome (CANVAS), and a long-standing dry cough provides a further clue. Andrea Cortese and colleagues (page 14) provide a clear description of this disorder—the most common recessive cause of ataxia—which their team have recently genetically characterised.

CANVAS may in time appear as readily recognisable clinically as Huntington's disease, a genetic condition also caused by a pathogenic repeat expansion. Thomas Stoker and colleagues provide an up-to-date review of the diagnosis and management of Huntington's disease (page 32). One newer issue is deciding at what point someone with a known mutation, determined from predictive testing, has developed the condition. Frustratingly, recent trials of disease-modifying treatments have ceased but the team describe the range and application of currently available symptomatic treatments.

Some clinical signs can specifically pinpoint the level of the nervous system involved. Internuclear ophthalmoplegia reflects highly localised pathology in the medial longitudinal fasciculus that links the pons to the midbrain. Tychicus Chen reports a patient with wall-eyed bilateral internuclear ophthalmoplegia (WEBINO) following etanercept for psoriasis (page 71). We asked Luke Bennetto and Christian Lueck to demystify the curious term 'wall-eyed' and to tell us what we need to know about WEBINO and how it differs from conventional internuclear ophthalmoplegia (page 4).

Charles Warlow, the founding editor of *Practical Neurology*, issued a challenge—and the prize of a copy of his book on stroke—to anyone who had diagnosed an intracranial vascular malformation using a stethoscope. Although Charles most likely had his tongue firmly in his cheek, Andrew

Larner works through the likelihood of making the diagnosis this way. He demonstrates convincingly that cranial auscultation is not a sensible use of clinical time (page 79)—its only value being to impress an audience—and that we should think as critically about performing and interpreting the clinical examination as we are with other investigations.

Hypnosis was one of the relatively few treatments available to Hughlings Jackson's patients, though was perhaps of more interest to Charcot. Does hypnosis have anything to offer patients in the 21st century? Wendy Phillips and colleagues summarise the history of hypnosis, what it is, how it is done, the current neuroscience underpinning it (page 42), and Mark Edwards provides a commentary on what it may offer to practising clinicians (page 2). We have other cases relating to different levels of the nervous system: HTLV1-related encephalitis (page 60); longitudinally extensive myelopathy from syphilis (page 75) and spinal glioma causing limb myorhythmia (page 77); and chronic inflammatory sensory neuropathy (page 82)—David Grundy, a Professor of Biomedical Science and ironically an expert on brainstem control, provides a dramatic personal experience of being temporarily locked-in following a basilar artery stroke.

There are also podcasts on the journal website (pn.bmj.com) for those wishing to explore the journal's best articles further. An editors' fire-side discussion (no actual fire is used) introduces each new issue (though we understand that this paragraph of commentary may be more than enough for most readers) and an editors' choice podcast, with its lead author answering those questions that we might all like to ask. Might be worth a listen.

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