What makes neurologists change what they do and how they practice? If asked, most clinicians will cite evidence, the outcome of clinical trials and new evidence for investigation and management brought together into clinical guidelines. They may acknowledge the cumulative experience of clinical practice and advice from experts—and perhaps experts’ well-written reviews in Practical Neurology (we hope).

New understanding of diseases can change practice. Autoinflammatory syndromes are becoming better understood and Neil Anderson and colleagues’ review on them is timely (page 145). Whereas autoimmune conditions arise through dysregulation of the adaptive immune system (mediated through B cells and T cells), autoinflammatory conditions reflect disruption to the innate immune system (mediated through monocytes, macrophages and neutrophils). Autoinflammatory conditions can be inherited (such as familial Mediterranean fever or cryopyrin-associated periodic syndrome) or acquired (such as adult-onset Still’s disease). Their clinical presentation is protean, with periodic episodes of inflammation of skin, muscle, joints, eyes with raised inflammatory markers—but also aseptic meningitis, meningoencephalitis, sensorineural deafness and peripheral neuropathy. Importantly they are amenable to treatment. We need to consider autoinflammatory syndromes in people with unexplained relapsing fever, raised inflammatory markers and varied neurological and systemic upset.

The weight of evidence guiding the management of women with epilepsy during pregnancy has been increasing, based on observational studies and patient registries. The strong evidence regarding the risks from sodium valproate has already significantly changed practice. John Craig and colleagues review the risks of epilepsy in pregnancy (page 98), notably highlighting recent evidence that lamotrigine dosage must be actively managed in pregnancy to limit the risk of seizures and sudden death.

The evidence relating to investigations usually focuses on how good the test is rather than how and when to use it. This is particularly true for genetic tests, which generally provide understanding of the disease rather than changing management, but have significance for other family members. Martin Turner and colleagues outline the background considerations and provide a framework for advocating genetic testing after every clinical diagnosis of motor neurone disease (page 107).

It seems ludicrous to suggest that post-traumatic amnesia is an emerging concept given that it was described in the 1930s, but it is benefiting from a critical re-evaluation. Peter Jenkins and colleagues (page 129) review the diagnosis and management, with an emphasis on collateral history and prospective evaluation using validated scales. Their illustrative clinical cases are rich in detail, including an interesting modern electronic collateral history (check it out).

Autism spectrum disorders have a prevalence of 1.5% but present more frequently than this to neurology clinics given their neurological comorbidities. Miriam Cooper and colleagues (page 120) provide practical advice to improve consultations for autistic patients. They give examples that illustrate how wide is the autistic spectrum, from those with intellectual disability to high functioning, and how we should adjust our clinic set-up and approach.

It is often worth considering why doctors might sometimes change practice contrary to the published evidence. Aidan Neligan and colleagues (page 94) challenge levetiracetam’s de facto position as generic first line antiseizure medication, particularly by non-neurologists, whose focus is often on the immediate management in the acute setting. Levetiracetam is easy to start and has few interactions but is not always the best long-term choice. There seems to be a case for wider dissemination of the evidence.

Ideas evolve. The pineal gland once (long ago) occupied the centre stage in neuroanatomy, literally and metaphorically, as the interface between the body and the soul. Costa Savva and Martin Turner discuss the progression of ideas about the gland (page 168), and its gradual decline in importance, ending up now as evolutionarily redundant and the site of common incidental findings.

Case reports rarely change practice, but rather they indirectly add to our clinical knowledge and provide a helpful reminder. We have cases of focal neurological complications associated with hyperglycaemia from Emanuele Bartolini (page 117), the patterns of visual loss associated with chiasmal lesions from Hajime Yoshimura (page 154), hemianopia as the presentation of posterior cortical atrophy from Ed Margolin (page 160), a challenging spondylotic myelopathy from Eoin Flanagan (page 162) and a myelopathy associated with intrathecal methotrexate from Pedro Rodrigues (page 141).

Sometimes, however, a single patient report triggers a change in practice. Neha Kumta and colleagues describe the experiences of a man who recovered from being locked-in with severe Guillain–Barré syndrome (page 126). He had been ventilated using low tidal volume ventilation with minimal sedation and treated with physiotherapy—strategies each based on evidence for better outcomes, survival and function. However, the low-volume ventilation caused significant distress and the passive physiotherapy gave significant pain. As Daniel Law and Matt Morgan discuss in an accompanying editorial (page 96), clinical management based solely on trial evidence for measured outcomes is not enough: we must also prioritise patient experience.

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