Talking through a challenging case with colleagues is often interesting and useful. Interesting, because thinking about difficult clinical problems is one of things that attracts doctors to neurology, and useful because the patient benefits from a virtual second opinion. Incidentally, requesting help will not reflect badly on you, somewhat counterintuitively, seeking advice from colleagues is more likely to improve their opinion of you and your competence—‘smart people ask for (my) advice’.1 The very act of describing the clinical problem to a colleague forces us to summarise the essential features of the patient’s presentation, and often simply articulating the problem out loud can help to clarify our thoughts about a patient’s problem. For example, we might make the mental leap that a patient’s ‘spells of weakness’ could be ‘periodic paralysis’, or reframe the symptom of ‘periods of unsteadiness with slurred speech’ as possible ‘episodic ataxia’. Sometimes just finding the right words is key, although this step in the diagnostic process, defining the clinical syndrome, can go unnoticed. For example, when a patient with a stroke has a scan showing intracranial haemorrhage, then ‘intracranial haemorrhage’ becomes the label. This is a useful though incomplete diagnosis, but a step that shapes how to take the diagnosis and management forward. Iain McGurgan et al discuss this further on page 128.

Sometimes naming the syndrome can take more thought. For example, a patient with status epilepticus of unknown cause on the intensive care who is not responding to treatment is a regular and worrying, if uncommon clinical problem. Recognising this as ‘new-onset refractory status epilepticus’ characterises the clinical syndrome and helps in the management, requiring not only close adherence to the standard status epileptics management protocol but also to focus on the underlying (often immune, sometimes genetic) cause. Although we generally discourage abbreviations in Practical Neurology, this is a good one to google, and doing so leads to an article by Laura Ritter and Lina Nashef discussing the spectrum of rare genetic and inflammatory disorders that can cause this very difficult presentation (page 119) and an approach to management.

Inflammation or infection of the brainstem and cerebellum is an unusual manifestation of encephalitis and has a different set of causes, and hence differing approaches to diagnosis and treatment. The term ‘rhombencephalitis’ encompasses this clinical and radiological syndrome, providing another useful summary term that clarifies this diagnostic step. Claire Rice et al discuss rhombencephalitis on page 108.

Axial muscle weakness is probably an under-recognised clinical syndrome that draws together the presentations of head drop and camptocormia. Waqar Wahed et al discuss how to evaluate clinically patients with this presentation, and how to approach the diagnosis (page 92). Three case vignettes in their report, each with a different cause, highlight the patient benefit that may follow a correct diagnosis.

Sometimes we need to have heard of a condition in order to recognise it. William Utley et al (page 171) describe an unusual clinico-radiological syndrome associated with antibodies to glial fibrillary acidic protein that responds to treatment, so worth diagnosing. Anyone who has not heard of vestibular drop attacks (previously and memorably named otolithic catastrophe of Tumarkin) might be perplexed by someone with Menière’s disease whose episodes throw them to the ground. Menière’s disease is uncommon and patients usually attend ENT colleagues but is certainly common enough for neurologists to know about it. Mansur Kutlubaev et al tell us what we need to know on page 137.

Neurologists also need some understanding of what neuroradiology can offer. Joga Chaganti et al (page 101) discuss how MRI can image the intracranial vessel walls—for example to distinguish vascular spasm from vasculitis—using black-blood imaging.

Sudden severe headaches prompt concern about subarachnoid haemorrhage; once this is excluded, look at the pituitary, another site where haemorrhage can occur—and often being symmetrical is easy to miss—as described by Jon Equiza et al on page 169.

In addition to the usual Test Yourself.articles—this time, ‘It’s all in the history’ (page 153)—we are introducing a shorter form of case-based question. In ‘Today’s Ward Round’ (page 161) readers are given a brief history, an image and a question. How will you fare? We recommend pausing, and perhaps discussing it with a colleague, before turning the page for the answer and discussion.

Several case reports take us through difficult clinical scenarios; someone with treatment-resistant CIDP and stable chronic lymphocytic leukaemia (CLL) who improved only after treatment of the CLL (page 143); a dramatic scleromyxoedema and neuropathy associated with Waldenström’s macroglobulinaemia (page 164); hyperreligiosity and frontotemporal dementia (page 173); some genetic detective work in a patient with young-onset frontotemporal dementia (page 149); and a classical illustration of the MR brain scan changes in CLIPPPERS (chronic lymphocytic inflammation with pontine perivascular enhancement responsive to steroids) (page 147).
Editors’ commentary

addition, we have the ever thought provoking Carphology and a Book Club report discussing a personal story of learning disability.

So, discuss the journal with your colleagues, ask them what they think; spark conversation—and go up in their opinion into the bargain.

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REFERENCE