Language provides us with names to categorise the world and becomes more sophisticated with use. Classification of everyday things comes naturally but we have a variable appreciation of our blind spots. If a neurologist were to try to distinguish between insects, most would probably apply a limited classification of flies (flies or bluebottles) not appreciating the diversity of species. However, when looking at butterflies, they would quickly realise they lacked the knowledge to name the many clearly distinct types. There is a parallel in how neurological diagnoses are applied, which also come with blind spots.

Dementia is a widely used term. The lay press often uses it as a synonym for the most common cause, Alzheimer’s disease, and typically presents the diagnosis as binary, someone has it or they don’t. This simplified approach has crept into medicine too, with a diagnosis of dementia being based on a score in a standardised test, such as the Folstein Mini-Mental State Examination or the Addenbrooke’s Cognitive Examination, thus missing much nuance. With disease-modifying treatments for dementia now in prospect, neurologists will increasingly need to make earlier and more specific diagnoses and to identify of the underlying pathology more accurately. It is still the clinical features that suggest the different diagnoses; Jeremy Johnson and colleagues provide a distillation of their extensive clinical experience in this area and explore the subtleties of cognitive disorders (page 300). They take us on a ‘walk around the brain’ using tables that repay careful attention, helping us to recognise and name useful clinical phenomena that might otherwise pass us by—the pigeon sign, ‘(Vicar of) Dibley’ sign, and bottom apraxia, to name a few.

Dissecting the nature of clinical phenomena makes it easier to recognise and understand them. Visual hallucinations that occur in Parkinson’s disease and dementia with Lewy bodies, in the Charles Bonnet syndrome and with thalamic lesions, have much in common despite their markedly different anatomical substrates; Rimona Weil and Andrew Lees explore what we can learn from these on page 327.

Corticobasal syndromes are rare. Like dementia they are heterogenous and their clinical features include some unusual phenomena (such as alien limb). These are beginning to be better understood and now include a broader range of phenotypes. Tim Anderson and colleagues provide a practical guide to their diagnosis and management (page 276).

Nodal and paranodal antibodies occur in some people with inflammatory neuropathies that have relatively distinct clinical and neurophysiological syndromes, notably an association with a nephropathy and a limited response to standard treatments for Guillain–Barré syndrome. Simon Rinaldi and his team provide an update and highlight how identifying such patients can allow for their more targeted treatment (page 284).

New investigations can change our understanding of diagnoses. Whole-genome sequencing is among the latest of the genetic investigations; it is an astonishingly powerful tool, but the very large datasets produced can cause significant problems in their interpretation. It is not infallible as some types of genetic disorders can be missed. Huw Morris and colleagues describe how to understand all this on page 322. Optical coherence tomography (OCT) has become a relatively standard test in ophthalmology to examine the retina and optic disc. It already provides opportunities to improve the management of patients seen by neurologists, such as those with idiopathic intracranial hypertension, while holding out the potential for more in the future. Neurologists clearly need to know about OCT and Clare Fraser and Christian Lueck provide a primer on page 313.

Some tests such as brain imaging commonly identify an incidental finding, such as pineal or colloid cysts. Having found them what is their best management? Michael Jenkinson draws from his wealth of experience to provide practical advice on page 292.

Neurophysiological investigations are essential in the diagnosis of neurological disorders, but who performs and reports on these investigations varies across the world. Matthew Kiernan puts the case that neurophysiological investigations are an extension of the clinical assessment and that the division between neurology and neurophysiology is artificial (page 274). An epileptologist to read EEGs and a neuromuscular neurologist to do peripheral neurophysiology might be more logical categories?

We have a range of clinical cases featuring mimics (for butterflies, mimicry is an evolutionary adaptation that provides a challenge for the lepidopterist). Syphilis, the great mimic, leads the way (page 340), but amyloidoma can mimic multiple sclerosis (page 344), and Cushing’s disease (page 351) or hypothyroidism (page 360) may each present neurologically. There are also Test Yourself articles and a Neurology Book Club report to round things off.

Incidentally, the UK has about 60 species of butterfly but about 7000 species of fly. Maybe the flies joining our summer picnics deserve a closer look?