From the earliest times, medical texts have been supplemented by illustrations. Whilst sometimes purely decorative, their main purpose is to provide an additional means of conveying meaning. The information content of a medical illustration depends on the skills of the illustrator and the technologies available for producing that image—manuscript, print from various original artefacts suitable for mass reproduction, photography and, now, electronic production. The principles of medical illustration are nowhere more explicit than in images of the nervous system. Little wonder, therefore, that some of the great examples are external and internal configurations of the brain. But since their purpose is to represent, rather than faithfully reproduce each and every feature of the subject matter, the artistic principles of composition, economy of line and contrast may make it necessary to dispense with detail and complexity, and the cartoon, line drawing, and black and white illustration still have their place in the age of sophisticated graphics and electronic image production. In his preface to the first edition of Neurological Differential Diagnosis (1975), John Patten points out that the text is ‘profusely illustrated, and although anatomical accuracy has been preserved, artistic licence has been taken whenever necessary to illustrate an important point. Each diagram is drawn from a special angle of view that enables the reader to visualize the area under discussion in situ in the patient’. The success of that book, and the admiration expressed by readers with respect to the illustrations, led Dr Patten to provide a much fuller account of his artistic training, and the origin of illustrations in his book, when the second edition appeared in 1996. In ‘An old Chinese proverb’, John Patten tells the story of medical illustration, with an emphasis on the nervous system, and reviews Netter’s Neurology by HH Royden Jones and Neurological disorders in famous artists edited by JULIEN BOGOUSSLAVSKY and Francois Boller (page 820). The astonishing contributions of Leonardo da Vinci—who depicted his own anatomical dissections—and Andreas Vesalius and Thomas Willis, each of whom employed professional artists to render the images of their anatomies, form a background to the stories of A Kilpatrick Maxwell, Max Broèdel and Frank Netter, working in the 19th and 20th centuries. Modestly, John Patten—formerly a consultant neurologist in the south of England—makes no mention of his own book but we learn something of the techniques he used from his informed comments on those who also have depicted neurological disease, thereby making ‘clear the wisdom of proverbial Chinese’.

Amongst three papers in the present issue dealing with epilepsy, Sarah Jamali and colleagues from institutions in Marseille and Rouen, France, and Amsterdam, The Netherlands, examine tissue from patients with mesial temporal lobe epilepsy using microarray technology and immunohistochemistry to show a reduction in neurotransmission (the serotonin receptor, HTR2A; neuropeptide Y, NPY1R), and provide evidence for complement activation leading to the formation of membrane attack complexes on entorhinal neurones (page 625). The mechanism of epileptogenesis is also explored by Mary Lou Solbrig and investigators from the University of California-Irvine, and the Scripps Clinic, California, USA, who draw together different observations relating to the aetiology of epilepsy to model the hypothesis that adolescent viral disease promotes seizures by reducing dynorphin expression in the dentate gyrus of the hippocampus in the rat (page 642): they show that Borna disease virus causes encephalitis, epilepsy, altered dynorphin and depletion with poor replacement and survival of dentate granule cells; conversely, dynorphin agonists protect against the various effects of pro-convulsants. From this analysis emerges a neurochemical framework for the mechanism of virus induced epilepsy that emphasizes the role of κ opioids. There is much uncertainty, and no little anxiety, concerning the incubation period of prion-related disease. Pietro Cortelli and a team from the Universities of Bologna, Milan and Genova, Italy, and Case Western Reserve, OH, USA provide some insight by mapping the evolution of clinical, polysomnographic and 2-[18F]-fluoro-2-deoxy-D-glucose positron emission tomographic abnormalities in carriers of the D178N mutation for fatal familial insomnia, and other family members and controls (page 668): they find that thalamic abnormalities may show up around 1–2 years before clinical onset. Members of the MRC Prion Unit at the Institute of Neurology, London, UK, under the authorship of Andrew Hill, describe molecular differences in the glycoform ratios of protease resistant disease-related prion proteins (PrPSc) in inherited versus sporadic, iatrogenic and variant forms of Creutzfeldt–Jacob disease (page 676): the demonstration that individuals with identical PRNP mutations make distinct PrPSc conformations further informs our understanding of genotype–phenotype correlations in this group of neurodegenerative diseases. Atsunobu Suzuki and colleagues from the University of Tokyo and Showa University School of Medicine in Toyko, Japan overcome the confound of poor motor responses and impaired face recognition in patients with Parkinson’s disease registering disgust to show that—compared with controls—these individuals do indeed underscore the recognition of unpleasant emotional experiences (page 707). Emotional responsiveness is also the theme of work reported by Alessia Nicotra and collaborators from Imperial College and the Institute of Neurology, London, UK, who confirm their expectation that autonomic denervation may disrupt brain activations and the perception of emotional
responses to angry faces when presented with an unpleasant electrical stimulation in the context of spinal cord injury: they suggest that autonomic denervation in spinal cord injury introduces affective vulnerability by attenuating properties of the anatomical substrate for pain recognition; the behavioural and motivational consequences for individuals with spinal cord injury are clear. fMRI is also used by Natalie Voets and a group from the University of Oxford, UK to study involvement of the right cerebral hemisphere in language processing by right-handed individuals with and without left hemisphere injury (page 754): using patients with temporal lobe epilepsy of left hemisphere origin, they associate a posterior shift in activation of the right, but not the left, inferior frontal gyrus both for phonemic and semantic tasks, and observe this alteration directly in one individual studied before and after left hemispherectomy for Rasmussen’s encephalitis. More specifically, while language tasks primarily involve the left pars opercularis, these activate different regions within the anterior insula and frontal operculum of the right hemisphere. [On the subject of naming errors, carelessly our January editorial confused William Wilberforce (1759–1833) with his third son Samuel Wilberforce (1805–1873), Bishop of Oxford from 1845 to 1870.] Peter Urban and a group from the Johannes Gutenberg-University in Mainz, Germany, analysed the sound of dysarthria in 62 acute cases resulting from stroke (page 767): difficulty with articulation is more obvious than the effects on phonation and prosody, although both occur; the frequency and severity of dysarthria correlate especially with left hemisphere lesions reminding us of conclusions reached by H Charlton Bastian, who first drew attention to aphemia (Bastian HC. On different kinds of aphasia. British Medical Journal 1887; 2: 931–6 and 985–90).

The lesions in all these examples of dysarthria involve the corticospinal pathways, and 40% recover absolutely, with nothing more than mild residual defects in the remainder. Christian Gerloff and colleagues (including our new Associate Editor, Mark Hallett) from the National Institute of Neurological Diseases and Stroke, Bethesda, USA and the University of Tübingen, Germany use a multimodal approach—EEG spectral analysis, EMG monitoring, transcranial magnetic stimulation and H15O-PET imaging—to characterize plasticity occurring in the context of left capsular stroke (page 791, and see cover): enhanced recruitment occurs in the premotor region of the ipsilateral hemisphere, the ipsilateral cerebellar hemisphere and extensive regions of the contralateral cortex where, compared with the ipsilateral hemisphere, cortico-cortical connections are expanded; and yet, effective conduction of the nerve impulse is still from the lesioned hemisphere through tracts projecting into the contralateral spinal cord. Nick Ward and investigators from the Institute of Neurology, London, UK, also examine changes in cortical activations consequent upon vascular damage to the corticospinal tract (page 809). Severity, graded by reduced response to transcranial magnetic stimulation, correlates with the extent of enhanced activation seen in the ipsilaterial posterior primary motor cortex; the contralateral primary motor cortex and superior cingulate gyrus; and bilateral premotor cortex, supplementary motor area, intraparietal sulcus and dorsolateral prefrontal cortex. Both studies illustrate ways in which the cortex adapts, partially and not altogether successfully, to subcortical lesions whilst retaining use of the crossed corticospinal tract; hence, plasticity operates by inter-hemispheric communications rather than through altered motor projections. In From the Archives we discuss two papers that established ground rules for the physiology of adaptation occurring after experimental lesions of the corticospinal tract. (The functional organization of the motor system in the monkey. I. The effects of bilateral pyramidal lesions. II. The effects of lesions of the descending brain-stem pathways. By Donald G. Lawrence and Henricus G.J.M. Kuypers. Brain 1968; 91:1–14 and Brain 1968; 91: 15–36).

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