The purpose of brains is to make movement. A brain has to identify what is going on in the environment and in its attached body and generate movements to stay alive and procreate to keep the species going. Movements of a person, which includes speech, are all that other people know of another person. The repertoire of movements that people can make is very broad, and even most ordinary movements are complex. Eating and dressing, for example, involve multiple body parts making a series of coordinated multi-joint movements. The movements of highly skilled athletes and musicians are so well tuned that most persons cannot match them. These complex movements that form the behaviour of everyday life are praxis movements. Their failure is apraxia. To call such a failure apraxia, of course, requires that the deficit cannot be explained by a more basic abnormality such as weakness, sensory loss, ataxia or aphasia. The range of capability of the brain to make movement is so large that there can be many types of failures. This has generated different classification schemes with a variety of different terms, sometimes with the same term meaning something else in a different classification.

The book by Goldenberg on apraxia contains a comprehensive description of different manifestations of apraxia and addresses the different classifications. Chapters 6 through 14 deal with topics such as imitation, body part specificity, and use of single tools. Chapters 1 to 5 give the historical background and set the stage for the rest of the book. I found the historical part particularly interesting, both as relates to apraxia but also ideas as to how the brain works.

While there were some historical precedents, the first well described cases of apraxia and pathological explanation were from Hugo Liepmann from 1900 to 1908 (Fig. 1). On the basis of detailed clinical assessment, Liepmann predicted brain lesions that were subsequently verified. After a series of cases, he came up with a theory of praxis and a classification of apraxia that arose from that theory. The movement formula was generated in the region of the occipital and parietal cortices and kinetic memories were in the region of the motor cortex, and movement was generated by a signal from posterior to anterior. Damage in the posterior region produced ideational apraxia, in the anterior region, limb-kinetic apraxia, and damage to the connection between them, ideo-kinetic apraxia. Similar ideas were coming from the early studies of aphasia such as those of Wernicke. Manuscripts of the day included diagrams with relevant brain regions and their connections. This all seemed rather successful until the attack from a distinguished series of British neurologists that reached its peak with Henry Head.

Head’s frontal attack on the ‘diagram makers’ came in the Hughlings Jackson Lecture for 1920, mainly dealing with aphasia, published in Brain in 1921 (Head, 1921). I looked back at that paper, and some quotations from that lecture make clear the strong feelings that he had.

‘Bastian published his well-known paper in 1869 which had such an evil influence on the subsequent course of the discussion. He started from the a priori assumption that we think in words, and that words are revived in the cerebral hemispheres as remembered sounds. He talked of lesions of special fibres and centres, and set the points on the catastrophic road to schemas and diagrams.
No one could write on aphasia without producing a new diagram of centres and the paths between them. Each author twisted the clinical facts to suit the lesions he had deduced from his pet schema.

How far the writers of this period were compelled to lop and twist the clinical facts to fit the procrustian bed of their hypothetical conceptions is shown by the famous case published by Wernicke in 1903 as “A Case of Isolated Agraphia.”

Head strongly supported the ideas of Jackson in this lecture with the notion of higher and lower levels of brain function, but that the brain was functioning holistically. It was not possible to identify brain centres for specific functions.

‘We know that speech can be affected by destruction of the substance of the brain, but this does not show that “the faculty of speech” is localized in any area of the cortex. We should as soon expect a special centre for eating as for speech; both are complex acts which do not correspond to any specific group of functions. No lesion, however local, can affect speech and speech only.’

Despite Head recognizing that such a view wouldn’t make it easy for neurologists, the view prevailed to a large extent. However, basic science moved on gathering evidence for localization of function in the brain, and the tide was turned in neurology, also on the pages of Brain by Norman Geschwind in his important two-part paper on ‘Disconnexion syndromes in animals and man’ in 1965.

As a quick personal aside, Geschwind and these papers were important in my own career. I was a freshman medical student in 1965, and Geschwind was a popular and influential lecturer at the time. I worked with Hubel and Wiesel in the summer of 1966 and introduced them to these articles, which they were impressed with. I then spent the summer of 1967 with Geschwind. Part I begins (Geschwind, 1965a):

‘THE early successes of the views of Broca and Wernicke led the classical neurologists to a mode of analysis of the disturbances of the higher neurological functions subsequently to be labelled with the derisive term “diagram-making”. Starting from the picture of the brain as a collection of sets of more or less specialized groups of cells connected by relatively discrete fibre pathways, these classical neurologists deduced a series of symptom complexes.

On the whole the period between the wars seems to have led to a loss of interest in analyses in terms of disconnexion. The criticisms of the holistically oriented neurologists, Head, Marie, von Monakow, and Goldstein probably contributed heavily to this decline of interest. The growth of holistic psychology under the Gestalt school and Karl Lashley and the rapid development of holistic schools of psychiatry probably all played a role, perhaps more by their effects on the general atmosphere of thought than by their specific critiques of the classical school.’

Geschwind then summarizes a huge body of animal and human data in support of localization of function including the importance of the connections between them in order to move information from node to node and carry out complex brain functions. He concludes with (Geschwind, 1965b):

‘For the past forty years there have been schools of thought which have stressed the importance of thinking of the patient as a whole, of seeing his responses as those of an integrated
unitary structure, even in the face of damage. The ramifications of this thinking in neurology, psychiatry, psychology and other fields must be well known to most readers. It should be clear from much of our discussion that this principle, while it may be useful in some cases as a stimulus, may be actively misleading when it is regarded as a philosophical law.’

Geschwind, as a neurologist in training, went to Queen Square to study higher cortical function with McDonald Critchley. He was so turned off by Critchley’s approach that he spent his time with Ian Simpson studying muscle disease. Perhaps it was these issues in part that led to his feelings.

This important battle in the history of neuroscience and neurology was played out on the pages of Brain, and has been won by Geschwind. These days, the localization of function and brain connectivity continue to be hot topics. MRI and EEG/MEG, as well as basic animal studies, are diving more deeply into anatomical and physiological connections with the development of progressively better techniques and mathematical tools such as graph theory.

Back to the book, in Chapters 6 to 14, the different clinical features are related to associated brain lesions, either by pathology or MRI. This core of the book is a valuable summary of the data. The story, however, remains complex, and, for example, post-publication is another large series with clinical–MRI correlation recently published in Brain (Buxbaum et al., 2014), with a commentary by Goldenberg (2014), giving rise to a slightly different model of different types of apraxia.

Chapter 16 reviews the sparse data on therapy of apraxia, and is a reminder that this area needs more attention. Chapter 15 is Goldenberg’s own conclusion about the physiology of apraxia. While recognizing specifically that all brain function must have an anatomical correlate, he appears to support the general Jackson-Head concept of higher and lower aspects of motor control. Higher is cognitive and lower is motor, and thus the title of the book, as the ‘cognitive side of motor control’. Is indeed the brain hierarchical in function or does it work as a synergistic whole with each piece playing its part? I think Goldenberg would like to find the mind in the brain (the cognitive side), and perhaps many others would like that too.

Mark Hallett
Human Motor Control Section, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, USA
E-mail: hallettm@ninds.nih.gov
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