One thing is clear; although ‘we do not find the term… apraxia in common use until we come within measurable distance of the present epoch of neurological activity’ the condition is by no means a new discovery. The concept is embedded in that of agnosia: ‘if I cannot recognise an object…I shall be unable to indicate its use’. This is apraxia of sensory origin but if the patient knows what is the object and ‘fumbles with it in endlessly futile ways, he is exhibiting motor apraxia’. The study of apraxia, distinct from difficulties with action through not sensing or having intact the motor apparatus for movement dates from (Hugo) Liepmann’s (1863–1925) classic case ‘which will remain a monument of clinical insight and examination ingenuity’. Dr (Samuel Alexander Kinnier) Wilson (1878–1937) rehearses the history of the subject.

From 1866, (John) Hughlings Jackson (1835–1911) recognized ‘power in his muscles and in the centres for coordination of muscular groups, but he—the whole man—cannot set them agoing… a speechless patient who cannot put out his tongue when told will sometimes… put his fingers in his mouth as if to help to get it out; and yet not infrequently, when we are tired of urging him, he will lick his lips with it’.

Seemingly, only (Arnold) Pick (1851–1924) writing in 1905, acknowledged this contribution. Comparable cases in the German literature are described as having ‘asymboly’, or loss of the memory pictures for an object, with loss of the ability to ‘turn new sense impressions to account, which is the main-spring of the memory pictures for an object, with loss of the ability to ‘turn new sense impressions to account, which is the main-spring of

MOTILITY, OR SENSATION, AND OF COORDINATION’.

Touring the cortex for disorders of cerebral function that impair movement he lists cortical blindness, deafness and sensory paralysis; cerebral ataxia; mind-palsy; agnosia, mind-blindness and mind- deafness; ideational agnosia; ideational apraxia; motor apraxia; and motor paresis or paralysis. On further consideration, the disaggregation of cerebral functions into these neat categories does not work so well. Agnosia produces apraxia (as the earlier commentators well understood), recognition involving assembly of the component sensory parts into a qualitative whole idea-complex. One of Dr Wilson’s patients, seen at the Bethlem Hospital, describes a match as a pen, declares that it is used to clean the teeth
and then proceeds to scour his finger nails. Absence of initiative (‘will-lessness’) and motor perseveration contribute to apraxia resulting in inertia in switching or hindrance in presenting the next idea and repetition of an already performed movement when a new one is intended which Liepmann designates as ‘tonic or clonic perseveration’. There is a contribution from inattention and an incapacity for retaining impressions although ‘we must beware of contentedly attributing everything to their action’. Apraxia may be least manifest for those movements that are most automatic and have the most associations with the victim’s previous occupations: males do badly using scissors and domestic knives but are less likely to be apractic for handling a pipe whereas females are often unable to mimic actions involved in smoking. Patients with apraxia seem content with their erroneous actions, indifferent to their errors and fail to profit by mistakes.

Diagnosis needs to account for the interplay of agnosia and sensory loss, especially cortical ataxia. The man who when asked to put his finger on his chin places it in his ear is hardly ataxic ‘even though some of the more amorphous movements of apraxics are less easy to distinguish’. The ataxic man is unstable in all his ways, whereas the apraxic may perform certain movements without the semblance of ataxia. It can be difficult to distinguish the movements of apraxia from some of those seen in hysteric. And Dr Wilson has been much exercised by the movements seen in some of his patients with (Thomas) Sydenham’s (1624–1689) chorea. The relationship to aphasia is complex. Either may be cortical, subcortical or transcortical, a classification first proposed by (Karl) Heilbronner (1869–1914): and ‘we may have aphasia with apraxia or apraxia without aphasia’. Wilson equates motor aphasia with apraxia of muscles moving the tongue, throat and larynx; and distinct from limb apraxia, which may or not co-exist, but can be regarded as ‘aphasia of the extremities’. The patient who cannot revive auditory word images cannot speak; if he cannot revive kinaesthetic images, he cannot move his limb spontaneously, such as in agraphia or playing a musical instrument, even though in neither situation is there paralysis of the muscles concerned.

The lesions responsible for ideational apraxia are often diffuse and distributed; and that may also be the situation in motor apraxia. Liepmann’s case showed a large lesion beneath the left first and second frontal convolutions but with many other areas of vascular damage in both hemispheres that may have been relevant. In 1905, Liepmann reports on 89 patients: none with left hemiplegia have evidence for apraxia in the non-paralysed arm whereas 20 of 41 with right hemiplegia, many of whom are aphantic, are unable to perform movements with the left arm. ‘The facts indicate that what the left hand can do is not in great part its own (i.e. the right hemisphere’s) possession, but is loaned to it by the left hemisphere’. Dr Wilson has experience of ipsilateral left-sided apraxia in the non-paralysed arm in cases of right hemiplegia with and without motor aphasia and in the absence of agnosia; and he is much impressed by cases in which the left-sided apraxia can be attributed solely to destruction of anterior callosal fibres connecting the left to the right hemisphere. Wilson resorts to diagrams showing disconnection of activity destined to innervate the left arm and originating in the left hemisphere (Fig. 1). He asks whether there is a ‘centre’ for eupraxia of both sides of the body in the frontal cortex on the left side analogous to Broca’s area concerned with innervation of the tongue and bulbar musculature, lesions of which result in motor apraxia? That is probably correct even though other cases, designated agnostic apraxia, arise from ‘disconnecting’ the left sensomotorium from the rest of the cortex (Fig. 2).

Six years later Kinnier Wilson and Dr F.M.R. (Francis) Walshe (1885–1973) report three cases with ‘tonic perseveration’ in the context of inability to relax a given movement through cerebral disease, as described by Liepmann, but which they prefer to describe as ‘tonic innervation’. It is not unlike the phenomenon of myotonia seen in (Julius) Thomsen’s (1826–1909) disease. AJ, a patient of Sir William Gowers (1845–1915) develops sudden left hemiparesis that progresses, with inability to relax his grasp for
20–30 s, or longer, after gripping an object; and he cannot mimic simple actions with the affected limb. There is bilateral optic neuritis (papilloedema) and Sir Victor Horsley (1857–1916) removes an endothelioma (meningioma) the size of a tangerine compressing the posterior end of the right frontal gyrus. The tonic innervation improves but AJ is left with hemiplegia that is still present at follow-up 5 years later. A patient of Dr (Howard) Tooth (1856–1925) experiences focal motor seizures on the left followed by progressive hemiplegia; he has bilateral optic neuritis and, after an attack with stupor in hospital, shows tonic innervation often taking several minutes to relax his grip or release a postural movement. Mr (Percy) Sargent (1873–1933) operates and removes a falcine endothelioma compressing the first and second frontal convolutions on the right. Apart from persistent hemiparesis, recovery is good and the tonic innervation disappears. Fanny Flynn, another patient of Dr Tooth, rapidly loses the use of her left arm and leg; she cannot relax her grip, and becomes forgetful with emotional instability. She has complete freedom of involuntary movements of the left arm—plucking the bedclothes, scratching herself or rotating her wedding ring (worn on the right)—but can perform not even the simplest activity of that limb to command nor let go when an object has been grasped (Fig. 3). Over time the severe motor apraxia with inability to imitate movements (Fig. 4), tonic innervation and agraphia persist and eventually she is discharged in statu quo.

Several observations can be made: tonic innervation is usually unilateral, provoked by voluntary movement, associated with corticospinal tract involvement partially sparing the arm, and may occur without apraxia. Although others have not commented on tonic innervation, Wilson and Walshe search the literature for suggestive cases starting with Liepmann’s patient who had difficulty letting go of an object at one stage in his illness before dense hemiplegia supervened. Amongst others, Kurt Goldstein (1878–1965) has reported a woman aged 57 who had a stroke causing the left leg to be ‘badly paralysed’ associated with severe motor apraxia of the arm; frequent pseudo-spontaneous movements with poverty of spontaneous actions and inability to initiate movements; tonic innervation of her left hand so that, after catching hold of her neck, she ‘nearly strangled herself’; agraphia; and ‘apraxic…incontinence of urine and faeces’. Being so similar to the authors’s own Case 3, the pathology described by Goldstein is of interest—softening in the distribution of the posterior branch of the right anterior cerebral artery resulting in damage to the gyrus fonicatus, posterior end of the superior frontal gyrus, the para-circular lobule and most of the right side of the corpus callosum. As with many of the other cases that Wilson and Walshe rehearse, they have their doubts on whether these are examples of tonic innervation and apraxia rather than akinesia, (‘Willenlosigkeit’; no ‘will-feeling’) and the modern reader would not disagree. Perhaps, only nine cases should be looked at in more detail in order to consider the nature and pathological anatomy of tonic innervation.

Wilson, in his earlier paper, and Liepmann agree that the phenomenon of perseveration is one of repeated performance of a given movement complex when, and only when, another is intended and despite recognizing that change is appropriate or being told to stop. There is a distinction between Liepmann’s phenomena of ‘intentional perseveration’ and ‘clonic perseveration’. These arise from fixation of a psychical idea, or ‘opisthomimesis’ due to passive preponderance and reinforcement of an old idea with fatigue in the intensity of new ones. Tonic innervation is...
more physiological in its mechanisms and occurs despite the patient’s efforts to inhibit the movement – indeed it is accentuated by those attempts: ‘It corresponds . . . to an impairment of function at the distal end of the psychomotor (afferent) limb of Wernicke’s reflex arc’. Tonic innervation is not apraxia: it occurs when functions of the corticospinal tract are slightly but not substantially impaired; is not related to hypertonicity; arises from cortical and transcortical disease; and occurs only with voluntary not reflex movements. As described by one patient:

‘I understood perfectly what was [required] and my intelligence appeared to me intact. Yet, having in my hand a common household article, the idea of how to use it completely escaped me, but I had sufficient presence of mind to remember that the best way to get the idea back would be to use the article mechanically without paying the least attention to it: the result was quite successful’.

Wilson and Walshe acknowledge that all cases of hemiplegia may show a degree of tonic innervation in so far as there is delay in relaxing when asked to do so. Inability to relax a movement suggests, in Sherringtonian terms, loss of reciprocal inhibition of willed movements:

‘as the patient voluntarily contracts his flexors . . . active inhibition of the . . . extensors takes place; when next he would reverse this action, he is able voluntarily to innervate the extensors, but there is no corresponding inhibition of the flexors’.

Conventionally, this phenomenon is occurring at the level of the spinal cord, but – given the evidence from pathology in Case 3 (Fanny Flynn) and that of Goldstein—might it be cortical or transcortical? Probably not: the authors conclude that ‘a spinal mechanism is at fault but it is thrown out of gear by defective impulses from the cortex . . . but in our ignorance of what is meant by voluntary as opposed to involuntary innervations, our view must remain merely a hypothesis’.

Peter Nathan (1914–2002) reports six cases of facial apraxia, in two of whom the disorder is more generalized, in order to consider the relationship to anarthria and aphasia. Following injury to the left anterior parietal region from a hand grenade on 8 August 1944, Case 1 is unable to utter a sound and has a right hemiparesis. Later he sings and imitates sounds around him but generally remains unable to utter other than producing some disjoined vowels and consonants. He is unable to perform facial movements to command but is fully accurate with his limbs in so far as the right-sided weakness allows. The facial apraxia persists. Case 2 is injured in the left frontoparietal region and is dysarthric with a right hemiparesis but his speech can just be understood. He has facial apraxia and is dyslexic with inability to assemble written instructions into a coherent whole. Within the limits of his right-sided weakness, the use of his limbs is normal. Case 3 suffers a road accident in Greece with a depressed skull fracture on the left, and when seen by Dr Nathan cannot read or write and has reduced speech fluency and facial apraxia; there is some residual right-sided weakness but all actions are performed normally with the limbs. Case 4 suffers a left temporal wound in which metal penetrates the left frontal region close to the falx. He is aphasic and dysarthric with difficulty reading, writing and calculating in the presence of a dense right homonymous hemianopia; he has facial apraxia but normal use of the limbs. Case 4 has shrapnel injury to the left parietal region. Dr Nathan finds him to have severe dysarthria with perseveration of sounds; alexia and agraphia; and inability to calculate. He has facial apraxia associated with severe paralysis of the right arm, inability to enact movements with the left due to ideational apraxia, and right-left disorientation. Case 6 develops difficulty using his arms on both sides and Dr Nathan finds him to have limited speech due to dysarthria with aphasia, and facial apraxia; he is also unable to perform actions with the right arm, the left being too densely hemiplegic to test. Sensation is normal. He is soon dead and autopsy shows carcinoma of the jejunum with metastatic tumours in both cerebral hemispheres. What can be learned?

‘It is obvious that if there is apraxia for facial movements, dysarthria will result . . . and facial apraxia is a common accompaniment of aphasia . . . a lesion of one hemisphere—and it is immaterial which—may suffice to cause bilateral apraxia of the face and . . . movements of speaking’.

Lesions of the left hemisphere resulting in apraxia are usually associated initially with complete lack of speech. Recovery, if it occurs, is faster for vowels than consonants because the apraxia is more marked for the tongue and lips than the larynx. Others have noticed this differential effect on speech sounds. Facial and speech apraxia are disorders of voluntary movement with retention of more automatic activities especially under emotive stimuli; with familiarity of the required movement; and if a rhythmic sequence is established as in singing. On localization of the underlying lesion, Peter Nathan produces a composite figure of the skull defects in his five cases and the parts of the underlying brain most likely to have been affected (Fig. 5). From this he concludes that the site of most vulnerability is large, spares the angular gyrus and temporal lobe, and most likely focuses on the lower part of the precentral gyrus concerned with movements of the face, jaws and larynx, or tracts connecting this gyrus to other parts of the cerebrum.

Now Dr Nathan reconsiders the views of Liepmann who eventually discarded the concept of a ‘praxis centre’ and preferred to define a territory most likely to be vulnerable from the point of view of apraxia. Liepmann classified ideokinetic apraxia in which automatic but not voluntary movements are retained; limb kinetic apraxia with destroyed or isolated kinetic engrams; and ideational apraxia in which the engrams are intact but the ‘ideational project is wrong’. Peter Nathan considers that whereas ideational apraxia is generalized, ideomotor apraxia is restricted in its distribution, the face being most commonly affected; but when ideomotor apraxia progresses and becomes generalized, there is also some ideational disorder. Liepmann argued that repetition of movement requires memory of the whole motor series; and apraxia occurs when the unconscious memory of innervations is disturbed. His concept of memory for function of the nervous system leaned on the work of (Carl) Wernicke (1848–1905). But too sophisticated a formulation based on distinction between memory for voluntary and involuntary movements in terms of motor engrams is unhelpful.

‘Liepmann’s explanations of apraxia . . . are based on a naive conception of neurology in which parts of the cortex are seen as containing memory
pictures; disturbances of function…[arising from] falling out of these areas, or blocking of interconnecting pathways'.

For Liepmann to claim that remembered patterns for movement are psychological manifestations of physiological activity of cortical cells in the ‘sensomotorium’ and that the brain tends to perform a series of movements in the same way as it has performed that sequence before, is merely to say that ‘practice makes perfect…rather we can go no further than to state that a psychological being makes decisions to move and a physiological being carries it out’. It does not follow that stimulating cells reproduces a stereotyped complex movement: the error of the mnemic idea ‘of cortical kinetics is due to its being based on the fallacious mosaic pattern conception of cortical physiology’. Peter Nathan quotes Hughlings Jackson:

‘the confusion of the anatomy and physiology is the bane both of neurology and psychology. It leads to superficial simplifications, and to crude popular “explanations” most of which are purely verbal’.

Hughlings Jackson recognized apraxia in the 1860s and formulated the condition in terms of the general principles of evolution and dissolution in the nervous system. It is a disorder of the motor cortex in which there is loss of movement but not paralysis. Initially, dissolution at the highest level prohibits ‘acting’ in a theatrical manner for movements most recently acquired—smiling, beckoning, nodding—when the patient ‘thinks’ but not when he ‘feels’; and he carries out many normal activities to which he is accustomed by habit. Later more primitive movements—speaking, chewing, swallowing or standing—stimulated by ‘feeling’ are also compromised and the patient may declare himself unable to carry out a simple command, explaining simply ‘I can’t’.

Much interest was shown in understanding apraxia and related disorders during the glorious days of descriptive neurology and its debate exemplified the wisdom of philosophers of the nervous system such as Hughlings Jackson. But these considerations are no less interesting to contemporary neurologists as the six papers on aphasia, alexia and apraxia in the current issue make clear.

Alastair Compston
Cambridge

Figure 5 Outline of lateral view of skull to show the region damaged in the first five cases. From Nathan (1947).